Proceedings of the conference of the medical specialists, Central Command and North-Western army : and of the joint meeting with the civil medical officers, Punjab.

Contributors

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S. B. Dennison

PROCEEDINGS

OF THE

CONFERENCE OF THE MEDICAL SPECIALISTS

CENTRAL COMMAND AND NORTH-WESTERN ARMY

AND OF THE

JOINT MEETING WITH THE CIVIL MEDICAL OFFICERS PUNJAB

Held at Lahore in February, 1944

ALLAHABAD:
SUPERINTENDENT, PRINTING AND STATIONERY, UNITED PROVINCES, INDIA
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1944

A CONFERENCE of the Medical Consultants, India Command, and the Medical Specialists, Central Command and North-Western Army, was held at King Edward Medical College, Lahore, from the 7th to the 10th February, 1944. A joint meeting with the Staff of the K. E. Medical College and Mayo Hospital, Lahore, took place on the last day.

All pre-arranged papers and discussions, (which are really short papers), are indicated in the text as (P) and (D) respectively. The speakers who took part in the discussions during the Conference, are indicated by separate paragraphs in the text. An index of all speakers will be found at the end. Graphs and diagrams could not be reproduced, owing to the shortage of paper. While every attempt has been made to reproduce these Proceedings as accurately as possible, all errors and omissions are deeply regretted.

Our thanks are due to Col. R. Hay, C.I.E., Inspector General of Civil Hospitals, Punjab and Lieut.-Col. S. N. Hayes, O.B.E., Principal of the K. E. Medical College, whose whole-hearted co-operation and help enabled the Conference to be held at Lahore under the best circumstances. We are grateful to Mrs. Knight, W.V.S., for typing the entire proceedings, prior to their submission to the press.

(Ed.)

1-4-1944.

ALPHABETICAL LIST OF MILITARY OFFICERS WHO ATTENDED THE CONFERENCE

ALDERDICE, A. A., Major, R.A.M.C.
ANDERSON, W. F., Captain.
ASHTON, B., Colonel, C.B.E., K.H.S., I.A.M.C.
BATHGATE, G. H. W., Major, I.A.M.C.
BHALLA, B. I. S., Lt.-Col., I.A.M.C.
BHATTACHARYA, B. P., Lt.-Col., I.A.M.C.
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BOSE, A. N., Lt.-Col., O.B.E., I.A.M.C.
BROOKS, W. St. J., Major, I.A.M.C.
CAMERON, J. D. S., Colonel, A.M.S.
CARRIER, P. L., Lt.-Col., A.B.R.O.
CHANDRA, M., Major, R.A.M.C.
CHATTERJEE, B. C., Major, I.A.M.C.
COBBAN, K. M., Lt.-Col., R.A.M.C.
CRADDOCK, A. L., Major, A.B.R.O.
CURSETJEE, H. J. M., Maj.-Gen., C.I.E., D.S.O., K.H.S.
DAUKES, H. W., Lt.-Col., R.A.M.C.
DAVAR, R. A., Major, I.A.M.C.
ELLIS, H. L., Major, R.A.M.C.
FREEDMAN, B., Captain, R.A.M.C.
FRIEDHEIM, I., Major (Miss), R.A.M.C.
FRIEDLANDER, H. A., Lieut., R.A.M.C.
FRIEDLANDER, R., Captain, R.A.M.C.
GORDON WILSON, Lieut.-Gen. C.B.E., M.C., K.H.S.
GREENING, G. Lyn., Major, I.A.M.C.
GRIEG, J. C. R., Major, R.A.M.C.
GUEST, A. I., Major, R.A.M.C.
HAMILTON, W. H., Major-Gen., C.B., C.I.E., C.B.E., D.S.O., I.M.S. (Retd).
HARDY, J. D., Major, I.A.M.C.
HARVEY, T. B., Major, R.A.M.C.
HAVILAND-MINCHIN, R. L., Lt.-Col., I.A.M.C.
HAY, J. D., Major, R.A.M.C.
HAY, R., Colonel, C.I.E., I.M.S.
HAYES, G. T., Lt.-Col., M.C., I.A.M.C.
HAYES, S. N., Lt.-Col., O.B.E., I.M.S.
HENDERSON, W., Major, R.A.M.C.
HINDS-HOWELL, C. A., Lt.-Col., R.A.M.C.
HYNES, M., Major, I.A.M.C.
KARAMCHANDANI, P. V., Lt.-Col., I.A.M.C.
KAUL, P. M., Major, I.A.M.C.
KELSALL, A. R., Lt.-Col., R.A.M.C.
KHURANA, B. S., Major, I.A.M.C.
KILOH, G. A., Major, R.A.M.C.
LAMPRELL, B. A., Lt.-Col., I.A.M.C.
LEHMANN, G. D., Major, I.A.M.C.
LIEBERMAN, D. M., Captain, R.A.M.C.

LINDSAY, D. K., Lt.-Col., O.B.E., I.A.M.C
LUTTER, F. T. F., Captain, R.A.M.C.
MacCARTNEY, D. W., Major, R.A.M.C.
MARTIN, J. S. S. Major-Gen., K.H.S.
McALPINE, D., Brigadier, A.M.S.
McCRACKEN, G., Captain, I.A.M.C.
MENON, K. P. G., Captain, I.A.M.C.
MILLER, J. A. W., Major, R.A.M.C.
MURTHI, G. V. S., Lt.-Col., I.A.M.C.
PASRICHA, C. L., Lt.-Col., I.A.M.C.
PRESTON, T. W., Lt.-Col., R.A.M.C.
PRICE, W. L., Ft.-Lieut., R.A.F.
PURI, J. C., Lt.-Col., I.A.M.C.
REYNOLDS, G. L. L., Captain, I.A.M.C.
ROGAN, J. M. G., Lt.-Col., R.A.M.C.
ROSS, D. C., Captain, R.A.M.C.
SCHLESINGER, B. E., Colonel, A.M.S.
SHARMA, G. L., Captain, I.A.M.C.
SINGH, Mohan, Lt.-Col., I.A.M.C.
SINHA, J, M., Captain, I.A.M.C.
SNELL, N. W., Lt.-Col., R.A.M.C.
TAYLOR, G., Colonel, I.A.M.C.
THOMAS, D. R., Lt.-Col., O.B.E., I.M.S.
THOMSON, A. M., Lt.-Col., R.A.M.C.
THOMSON, W., Major, R.A.M.C.
TREDGOLD, R. F., Major, R.A.M.C.
VARMA, O. P., Captain, I.A.M.C.
VAUGHAN, V. St. G., Major, R.A.M.C.
WALKER, W. I., Captain, R.A.M.C.
WALLACE, W. D., Captain, R.A.M.C.
WHITE, T. K., Colonel, I.A.M.C.
WINGATE, P. C. F., Major, R.A.M.C.

ALPHABETICAL LIST OF CIVIL OFFICERS WHO TOOK PART IN THE CONFERENCE

AGGARWAL, M. L., Dr.
GOYLE, A. N., Dr., Ph.D.
GREWAL, Khem Singh, Dr., Ph.D.
HASSAN, Dr.
KHAN, Mohd. Yaqub, Dr., M.B., B.S., M.R.C.P., D.M.R.E.
KHAN, Yar Mohd., Dr., K.B.
LUTHRA, J. N., Captain.
MEHTA, P. C., Dr.
PIRZADA, Dr.
SHAH, Riaz Ali, Dr.
SIDDIQUE, Dr.
SINGH, Man Mohan, Dr.
WIG, K. L., Dr., M.B., B.S., M.R.C.P.
YUSUF, Mohd., Dr., K.B.

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Proceedings of the Conference of Medical Specialists, Central Command and North-Western Army, Lahore, from 7th to 10th February, 1944.

7TH FEBRUARY, 1944—MORNING SESSION

1. The delegates assembled at 09·00 hours in the Patiala block of the **KING EDWARD MEDICAL COLLEGE, LAHORE,** and the meeting commenced at 09·15 hours, with **LIEUT.-GENERAL GORDON WILSON,** C.B.E., M.C., K.H.S., D.M.S. in India, in the Chair.

2. **LIEUT.-GENERAL GORDON WILSON**—" *Opening Address* "

General Cursetjee, General Martin, gentlemen, I deeply appreciate the privilege of saying a few opening words, on this important occasion. It is more than a pleasure to see this gathering of Specialists—it is a stimulus to imagination—and it is, to me, an incentive to do all in my power to help you and encourage you in your drive to secure better clinical standards in our hospitals and to maintain the high traditions of our profession.

It is one of the brighter features of war, that it almost invariably brings with it a great revival in the broader realm of medicine, in the fields of research, experiment, observation and treatment. New methods are brought to light : new drugs and chemicals, new diseases or old diseases in new guises attract our attention. New conditions of human activity bring their medical repercussions—new weapons produce new types of wounds. The remarkable thing is, that, despite the setbacks which occasionally and unavoidably arise in war time, due to the shortages of equipment, personnel and accommodation, keenness wins through. This is a great tribute to the spirit of our profession. It is my desire and that of all my consultants and advisers, to foster that spirit, and it is our duty to help you to give full expression to that urge for improvement of knowledge and technique, without which there would be no progress.

While I am on the subject of specialism, I want you to bear in mind your less fortunate brethren—the general duty officers and Regimental medical officers. With the increase in specialism, we now have one in every four officers a specialist and it may even be that we have to designate G. D. O.'s and R. M. O.'s as specialists, if we are to retain their keenness and interest in their important functions. I find that my consultants are always ready to shepherd my promising G. D. O.'s into the specialist fold. I hope that you will not work in too watertight compartments and that you will share your knowledge with your G. D. O. confrères and, if need be, share their work when occasion demands.

To amplify my remarks on the subject of equipment, personnel and accommodation, I want you to appreciate that the expansion of the Medical Services in India has been on a huge scale and it has been no easy task to keep pace with the medical requirements of so vast an army as has been raised. I know that, at times, you may have had a feeling of frustration at being unable to get all the "tools", etc., that you wanted, but in a world war the demands are so great and from so many different sources, that the supply and distribution of stores and equipment is fraught with the greatest difficulties. The more urgent operational theatre of war naturally receive priority in the first instance. Wars result in great changes in industry and it is no easy matter to adjust the adequate production of medical supplies in the initial stages. In this war, the colossal demand for anti-malarial drugs has been a problem in itself. New drugs come into use for specific purposes and adequate production has to be organized to meet the demand. I am glad to say that the supply of drugs and medical stores is now showing rapid improvement.

I shall be particularly interested to hear the results of you deliberations on the subject of scrub typhus, which has manifested itself in epidemic form, amongst troops in Assam and Bengal and recently, on a somewhat larger scale, in Ceylon. We must do everything we can to elucidate this condition of affairs and find out the best method of prevention.

I hope that you make a point of disseminating your information and knowledge and that you will aim at a uniformity of thought and outlook in your general medical problems in the Army—it is necessary for us to present a united from to the non-medical officers who too frequently fail to carry out medical advice, on the grounds of diversity of opinion among the doctors.

3. COL. SCHLESINGER—"*Medical Problems of Central Command and North-Western Army*"

I would like to take this opportunity, on behalf of the meeting, of thanking the three Heads of our branch of the service, in Central Command and North-Western Army—The D. M. S., General Gordon Wilson, and the two D. Ds. M. S., General Cursetjee and General Martin, for having honoured the Conference with their presence. We, in our hospitals, are faced with many practical problems and are the doctors who have to treat the actual medical casualties of the war. Naturally, in the course of our work, we are apt to develop a parochial view of our difficulties. Our three Generals have necessarily a bird's-eye picture of the medical situation. They have to take the broad view, involving priorities, emergencies and availability of men and supplies. Hence the value of the pooling of our ideas at a conference and, I repeat, we are delighted that they have found time to join us. We are all sad that Brigadier Marriott, the Consulting Physician to the Army in India, is ill and cannot be with us, and I am sure the Meeting would like me to send him a telegram on their behalf, expressing our sympathy and wishing him a speedy recovery.

The medical problems of Central Command and North-Western Army are many, and they have been largely covered by the subjects before us for discussion. Our part of India has now developed into an immense Training Centre, largely of recruits in all branches of the service. More recently, Training Divisions and specialized forms of training have also been inauguarated and they set particular problems of their own, in regard to new epidemics, such as typhus, the protection of troops agaipst malaria and the maintenance of health on periods of "light-scale" rations.

We are all aware of the poor type of recruit that is now often enlisted into the Army, only to block our hospital beds early in his career, or fall sick soon after reaching operational areas. Recently G. H. Q. endeavoured to stop this, by asking for quality rather than quantity, but in many centres their request has gone unheeded and we still see very poor material on our medical inspections and in hospital. There is more than a suspicion that, at the present time, quite a number from the Bengal famine area are solving their problem by making their way into the Army. This is closely related to one of the most important subjects we are about to discuss—malnutrition and anaemia. The problem in the Indian Army of today is, that quite a percentage start their Army life with a heavy debit balance against them. G. H. Q. has taken steps to cope with this, by instituting regular inspections and such measures as deworming and an extra milk ration when necessary. But our medical task is not rendered any easier by the average quality of recruit. Certainly some degree of anaemia exists in quite a number and, depending on the part of India from which they come, hookworm, chronic malaria, a poor diet since earliest childhood and, to a far less extent, Kala Azar, are the chief factors.

In the Base Hospitals of Central Command, hundreds of cases of anaemia, vitamin deficiency and malnutrition are being evacuated from the 14th Army. Here again, dysentery, repeated malaria and ankylostomiasis are the main aetiological factors in men whose diet maintains a bare reserve of haemopoietic elements and essential vitamins. During active operations, with transport difficulties, this small balance in the diet of the Indian soldier is often apt to become upset, with disastrous consequences. It is of some significance that these grave anaemias are rarely encountered in British troops, who exist on a much bigger margin.

In order better to compete with cases of anaemia, C. I. M. H. in Peshawar, in North-Western Army, started an Anaemia Centre, to which all serious cases in the District were sent. The same policy has now been adopted in Central Command, where we have established sixteen such centres and hope, by so doing, to be able to investigate and treat such cases better and concentrate the available supplies of drugs and vitamins there and put them to better use. We are endeavouring to improve blood transfusion technique in hospitals and to torpedo the prevalent idea, which is encountered in some quarters, that "the patient is too weak and anaemic to stand transfusion". The policy now, is to send senior medical officers, medical specialists

and officers in charge Medical Divisions, on blood transfusion courses and not, as often in the past, the most junior or most useless medical officer in the hospital.

I turn to malaria next and I know that the two A. Ds. M. will agree when I say that the chief problem here is still lack of discipline of units and individuals in anti-malarial measures. Personal protection is still the most important slogan to drive home. In Central Command, we are edeavouring to implement this by an instruction that every unit should select its own combatant malaria officer. who should receive some training. Antigas precautions have been well established in every soldier training at home in England, by a unit gas officer—why not in the case of malaria out here? The medical services can only act in an advisory capacity; it is for all units and all men to realise that it is their personal responsibility to carry out instructions. In connection with this, something will have to be done to afford W. A. C. (I)'s protection, now that more of them are coming into the service. The malaria rate among them is high.

In certain areas we have had good opportunity of witnessing the effect of suppressive and blanket treatment. Certainly the combatant and often the medical officer expects too much from these measures. We must all realize that the results achieved are neither 100 per cent.. nor afford a permanent guarantee against clinical malaria, and Col.Lamprell has no doubt something interesting to tell us about this. All such trials should be carefully controlled and observed, for all the information possible is required on this somewhat controversial subject. Malaria relapses are another great problem and I am speaking now of instances where there is no doubt that the therapeutic course has been properly administered and taken. Undoubtedly, a fair number of people exist who themselves develop no immunity and in whom the standard course merely controls the acute attack, but does not eradicate the disease. Two centres have been set up, for special investigation of such cases, in Delhi and in Peshawar and trial is being made of N. A. B. injections, as an adjunct to the standard treatment. This form of therapy is not new, but a decision for or against its value is required.

Another problem of malaria is the disposal of some cases with splenic enlargement. As already mentioned, it is these that form a considerable group of the patients in whom we encountered anaemia. Which cases should be retained and which discharged from the Army, and should any form of treatment be given?

Dysentery presents many points of interest. Certainly its diagnosis could be improved and how this is to be achieved, we shall hear from our colleagues at Delhi. The question is also frequently raised whether the prolonged starvation and time-honoured saline treatment is really necessary. In Indian troops, especially, I feel that poor-beverage, chicken soup, barley water and milk for days on end, is often overdone and may be the forerunner of some of our malnutrition and dysvitaminosis problems later. Amoebic dysentery has quite often defeated us all and is the source of many relapses, despite all forms of treatment, which has not, so far, been able to be standardized. Here, as in malaria, the British and American soldier suffers equally with the Indian. We should never omit sprue from our differential diagnosis, whenever we are confronted with a case of chronic diarrhoea, remembering that there can be all grades of this disease and that the close relationship of malnutrition, anaemia, pellagra, dysvitaminosis in general, and sprue, is gradually becoming established.

Tuberculosis has designedly been chosen as the main subject for discussion at our joint meeting with our civilian colleagues, for it has long been an all-India civil problem, which has not been as successfully tackled as others. In England, and probably most European countries, a man contracting tuberculosis in the Army, is discharged and treated in a civilian sanatorium, of which there are an adequate number. Here, none exist, to all intents and purposes. Short of filling our already overworked military hospitals with hundreds of cases of tuberculosis, arising in soldiers, but originating in civil life, and not attributable to military service, they have to be discharged to their villages, to infect their households and eventually to die. Certainly it is a parlous and pathetic state of affairs, but not one which the medical services of the Army can attempt to tackle in the midst of war.

On the neurology side, Brigadier McAlpine, the Consultant to G. H. Q. in this branch of medicine, is dealing with the common conditions seen in the Army in India. Both in Central Command and in North-Western Army, we have been rather concerned with the high mortality in cerebro-spinal fever, and a recent directive from G. H. Q., on this subject, should do a

great deal in expediting arrival at hospital, promoting early diagnosis and establishing bolder and more carefully supervised treatment.

In psychiatry, the problem, I think, which troubles us most, is the speedy disposal of the men with hysteria and its differentiation from malingering. Cure, so as to become an effective soldier, seems nearly always a forlorn hope, but much can be done in prevention. I would recommend a visit to the Surgical Division of the I. M. H. Rawalpindi, where the introduction of a system of rehabilitation and mass psychology P. T., by Lt.-Col. Andreasen, goes a long way in preventing the development of hysterical contractions and other such disorders, after injuries and operations on the limbs. Patients are there made to help themselves and re-educate their own muscles; the masseur and physiotherapy are kept in the background. Major Tredgold, will I am sure, have much to tell us on a subject that is largely foreign to the average physician.

Among the common epidemic, smallpox is largely a question of efficient vaccination, and the new A. B. 439 for officers, should tighten up the procedure for them. The clinical course of typhoid fever may seem strange to many R. A. M. C. officers, fresh from civilian practice, where previous T. A. B. inoculations do not usually complicate the signs and symptoms. I think that we should all strive for an earlier diagnosis, and impress on those under us, the frequent call for dual diagnosis, in this country, where malaria so often obscures the clinical picture. Typhus is now prevalent in certain parts of Central Command, following the special training, now under way. Here, there is good opportunity for further advance in our knowledge of the aetiology and its relationship to the somewhat complicated agglutination reactions.

Medical training is an important subject for discussion, on our programme, and, as you see, we are trying to approach it from all angles. Our difficulty is, that we have, largely, to rely on the sources, both British and Indian, now available in this country. Out of these, we are expected to create an efficient enlarged medical service, and provide specialists—an almost impossible task in some respects.

Finaly, I cannot end without reference to the Review Board. In North-Western Army and in Central Command, I have acted as its whipping boy! So much has this been the case, that we have invited the sponsors of that much-reviled Board, to come and support it, and I have advised them to appear in their thickest armour.

ANAEMIA AND MALNUTRITION

4. MAJ.-GEN. MARTIN, in his introductory remarks on this subject, spoke from the administrative point of view, on the saving of man-power. He laid emphasis not only on the question of malnutrition, but also on the importance of early diagnosis of avitaminosis, in view of the fact that Riboflavin deficiency has been observed in several cases.

5. MAJOR HYNES—"*Anaemia in the Indian Army*" **(P)**

The importance of anaemia in the army is twofold. Firstly, it reduces the efficiency of the soldier—he is listless, tires easily, and has poor resistance to infection. Secondly, it is an index of malnutrition, for although the precipitating cause of anaemia in the sepoy is usually malaria or ankylostomiasis, these diseases only very rarely produce severe anaemia when the diet is adequate.

The Classification of Anaemias

Three measurements are important in the classification of anaemias—the haemoglobin, the mean corpuscular volume (M. C. V.), and the mean corpuscular haemoglobin concentration (M. C. H. C.). The haemoglobin measures the degree of the anaemia. According to whether the M. C. V. is large, normal, or small, the anaemia is called macrocytic, normocytic, or microcytic. If the M. C. H. C. is normal the anaemia is orthochromic, if it is low the anaemia is hypochromic.

In the army we are concerned almost entirely with two deficiency anaemias. Deficiency of a substance present in marmite and liver—Will's factor—causes a macrocytic anaemia. Deficiency of iron causes a microcytic hypochromic anaemia. In practice the two are very commonly combined, and, according to the relative preponderance of the two deficiencies, we have a hypochromic anaemia, which may be macrocytic, normocytic, or even (very rarely) microcytic. In all three, the colour index will be low, yet liver will be necessary for cure.

The Diagnosis of Anaemias

A complete blood investigation is a rather complicated laboratory procedure, which is only possible in a few special centres. But an estimation of the haemoglobin and an examination of the stained blood smear will give all the information necessary for the diagnosis and treatment of nearly every case of anaemia.

Haemoglobin Estimation

The haemoglobin should always be expressed as grammes per 100 ml. of blood (gram. per cent in brief). In England "100 per cent. haemoglobin" usually means 13·8 gram per cent., but in other countries it means anything from 14 to 18 gram. per cent. The confusion is so great that it can only be cured by abandoning the percentage expression.

The Sahli or acid haematin method, is the most convenient for field use. Its great disadvantage is that the intensity of the colour increases progressively, rapidly for 5 or 6 minutes, and then more and more slowly for about two hours. The higher the temperature, the more rapid the colour development. Each haemoglobinometer standard is calibrated for some definite time and the matching must be made at this time, after mixing the blood and acid. The standard time is often 5 minutes, when the colour is still increasing rapidly, so that an error of a minute is important. Moreover, the reading at 40°C, a moderate summer temperature in the tropics, is 10 per cent. higher than at 20°C. A standard time of 20 minutes is better, then, if the matching is done a few minutes late, the haemoglobin will be read only a little too high, and the extreme temperature variation will be only about five per cent.

The inaccuracy of the Talqvist method is now universally recognized. The best that can be said for it is that it gives a very rough measure of the haemoglobin between 40 and 80 per cent. (100 per cent. Talqvist: 13·8 gram per cent.). Figures above or below these limits are practically meaningless.

The Examination of the Stained Smear

If cells larger than normal are present on the smear, then Will's factor deficiency may be diagnosed. If there is doubt about the size of a cell, it may be measured with an eyepiece micrometer; or, if this is not available, the cell may be compared with the small lymphocyte, which is 7M in diameter. Nearly all the macrocytes may be swept to the edges and tail of a badly spread film, so these areas should always be examined, unless the cells there are obviously distorted.

Hypochromia is shown by incomplete "filling" of the red cells—an appearance very easily recognized after a little experience.

The Red Cell Count

The ordinary red cell count is grossly inaccurate—the report "R. b. c.: 5·12 millions per cu. mm." really means "The red cell count probably lies between 4·5 and 5·5 millions per cu. mm." Its only practical use in ordinary laboratories is to permit the calculation of the colour index, which, in India, gives no indication of the type of anaemia present. Finally, it wastes five or ten minutes of a technician's time. In fact, there is so justification for asking for a red cell count, when a complete blood investigation (with haematocrit, etc.) is not possible.

Normal Blood Standards

The blood standards of healthy upper-class Indians are fully equal to those of Europeans, and the same standards should be reached by fit sepoys.

Surveys of coolies have shown the haemoglobin to range from 3 or 4 to 12 or 13 gram per cent. Those at the lower end of the scale are obviously suffering from anaemia, but what of those at the upper end, who are better than their fellows, but still anaemic by European standards? Deworming, anti-malaria treatment and full doses of all the haematinics, will not raise the coolies' haemoglobin above 12 or 13 gram per cent. His cells become fully saturated with haemoglobin, but remain smaller than normal—that is, he has an orthochromic microcytic anaemia. This persistently low haemoglobin has usually been ascribed to the coolies' inadequate diet, and especially to deficiency of foods of animal origin. (We hope to test this hypothesis by observing the response of recruits to a good diet.)

The Deficiency Anaemias

Iron Deficiency Anaemia

Iron deficiency anaemia may be due either to an inadequate iron intake, or to excessive iron (i.e. blood) loss. Usually both factors must be considered—the greater the iron intake, the greater the blood loss which can be borne without anaemia developing.

The sepoy's iron intake is certainly not inadequate, for his diet contains at least three times the minimal requirement. He may lose blood by hookworm infestation, but it is only rarely that the Indian adult, who has developed considerable immunity, has an infestation heavy enough to produce severe anameia simply by haemorrhage. Heavy infestation is, however, commoner in severe anaemia than in the general population, and may be regarded as the precipitating cause of anameia in those made especially susceptible by some other unknown factor—possibly dietary.

Nutritional Macrocytic Anaemia

This disease was discovered in India and called Tropical Macrocytic Anaemia, but, since it is also common in temperate climates, the present name Nutritional Macrocytic Anaemia, (N. M. A.), is preferable. It is defined as a macrocytic anaemia cured by marmite or liver, and it is thought to be due to deficiency of a substance—Will's factor—present in these foods. Will's factor occurs naturally in association with Castle's extrinsic factor and the vitamin B complex, but it is not identical either with extrinsic factor or any known vitamin. It is used up quantitatively in haematopoiesis, so that any increase in blood formation increases the demand for it.

N. M. A. is usually due to a combination of a low intake and increased utilization of Will's factor. The diet of the poor Indian and, to a lesser extent, of the sepoy, appears to be deficient in Will's factor, probably because it contains so little food of animal origin. The most important cause of increased Will's factor utilization by the sepoy is chronic malaria. Not only parasitized red cells are destroyed, for the hypertrophic reticulo-endothelial system phagocytoses normal red cells. Haematopoiesis increases to compensate for this loss, so that more Will's factor is used. If the utilization exceeds the intake, macrocytic anaemia develops and the abnormal red cells are phagocytosed even more readily than normal ones. Haematopoiesis and Will's factor utilization increase still further, and the patient slides rapidly down a vicious spiral.

The increased blood formation necessitated by blood loss in ankylostomiasis may similarly precipitate N. M. A. and the blood will then show evidence of iron as well as of Will's factor deficiency. Such a dimorphic anaemia, but with malaria as the predominant cause, is very often seen in the Indian Army, and can only be cured by giving both liver and iron.

Macrocytic anaemia may also be caused by malabsorption of haematopoietic principles. This is most clearly seen in sprue, but other forms of chronic diarrhoea may well have the same effect.

The Treatment of Anaemia

Iron deficiency anaemia is very readily cured by the administration of iron and the larger the dose given, the quicker will be the recovery. 27 grains of ferrous sulphate or 120 grains of ferri et ammon, cit. daily, are optimal.

N. M. A. can be cured by marmite or liver by mouth, or by liver extract parenterally 1 oz. of marmite a day is usually enough, but refractory cases may need 2 oz. The Indian takes it most readily mixed up in a cupful of water. ½ lb. of raw liver a day is an effective and simple treatment, when the patient's prejudices permit.

The dose of liver extract necessary, is much larger than in P. A. Crude extracts, of the campolon type, are preferable; the ordinary case needs 15—20 cc. weekly, the refractory one needs double this. Highly concentrated extracts of the anahaemin type are not to be recommended. Most cases will respond to very large doses (e.g. 2 cc. anahaemin daily), but there is no doubt that these extracts are relatively poor in the curative factor.

Blood transfusions are urgently necessary if the haemoglobin is below 2·8 gram. per cent., and may have to be repeated for weeks, to tide the patient over a refractory period. When there is severe anaemia and heart failure, it is easy to kill the patient by overloading the circulation, and small daily transfusions may be given by a slow drip (e.g. 300 cc. at 10 drops per minute). Such slow drops require constant attention (unless Marriott and Kekwick's regulator is used), but an intelligent nursing sepoy can be taught to manage them admirably.

If there is evidence of ankylostomiasis or malaria, these will, of course, be treated also. Indeed, if malaria is at all severe, the anaemia will not respond until the fever is suppressed by quinine or atebrin.

To summarize: the Indian's blood should be as good as the European's, but the poor Indian, on a defective diet, has at best a microcytic orthochromic anaemia. Any extra strain on the

blood-forming tissues, readily turns this into a severe usually dimorphic anaemia. The sepoy, nowadays, comes from this class; the problem which we are to discuss, is how to raise him above it.

6. LT.-COL. A. M. THOMSON—"*Anaemia from the Nutrition Point of View*" **(P)**

Anaemia has been called the outstanding sign of malnutrition in the Indian Army. There can certainly be no doubt as to its great importance, whatever its aetiology. I propose to discuss, shortly, the extent of our actual knowledge of dietary intake, as a factor in the production and prevention of anaemia. First of all, let us consider what is known about anaemia in India, as related to Indian dietary habits in civil life.

Diet in general

The first fact we have, is that the population of India, among whom anaemia is common, eat a very poor diet. The composition of this diet is singularly uniform, even in areas separated by thousands of miles. Rice is the staple food, and is supplemented by a small amount of dhal and vegetables. Only a fraction of the vegetables are of the green leafy variety. The cooking medium is a small quantity of vegetable oil. Foods of animal origin—meat, fish, eggs and milk—are eaten either in very small quantity or not at all. On analysis, this diet is shown to be more or less grossly deficient in almost all constituents necessary for healthy life, and its ill-effects on physique are reflected in the poor quality of recruits, now being accepted in the army. The labouring population, which consumes this diet, is almost universally infected with parasites—malaria and hookworm being perhaps the most important. This diet is obviously inadequate to prevent anaemia in such a population. Is it also inadequate for haemopoiesis when parasites are absent? The evidence on this point is inconclusive, but it has been reported that, in certain estates in the Nilgiris, where such a diet is consumed, malaria and hookworm are not prevalent. Local research workers state that *severe* anaemia is uncommon among the coolies—a very different picture from that prevailing on, say, Assam tea estates. I do not know if Nilgiri coolies exhibit a "normal" microcytosis, like Assam coolies. There are, accordingly, certain grounds for the belief that even this very poor rice diet contains just enough of the various haemopoietic factors necessary to control anaemia in a population free from parasites. The margin of safety is, however, negligible, and, since the majority of labouring Indians are infested, anaemia remains common. We have few precise haematological data on populations from the wheat-eating provinces of India. Indian diets, based on what usually contain more dairy produce than rice-diets and small amounts of meat, are commonly consumed. My first impression of recruits from the Punjab, is that the standard of nutrition now prevalent, is not so markedly superior to that of, say, Madrassis and Gurkhas, as one might have expected, and there is little doubt that anaemia is relatvely common among them. These wheat-eaters are also the victms of endemic hookworm infestation and malaria. I think it is fair to conclude, that in India, anaemia as a simple dietary deficiency state, is extremely rare. It is the product of parasitic infestation and a bare subsistence level of nutrition. In war, nutritional planning must take into account the effects of hookworm, malaria, and other parasites. Indian Army rations should provide a sufficiency of all necessary haemopoietic factors, to permit of maintaining maximum bodily reserves. This may not stamp out anaemia, but will reduce it. Let us, therefore, consider the nature of these haemopoietic factors. Of identified dietary constituents, iron and protein are directly concerned. Certain specific haemopoietic factors which have not yet beent isolated are also required. I must not be dogmatic about other dietary constituents, because time is short. There is no sound evidence that fat, carbohydrate, calcium, phosphorus, or any of the identified vitamins have a specific function in human haemopoiesis. The same holds good for trace elements, such as copper and cobalt. I shall, therefore, ignore these doubtful factors and concentrate on iron, protein and the unknown haemopoietic principles.

Iron

The iron content of a diet is estimated by chemical analysis, and may be determined as total iron and ionisable iron. The ionisible iron is sometimes known as ' available iron,' because it was considered for a long time that only inorganic iron can be used by the body, More recent research has shown, however, that some organic iron compounds are able to stimulate haemopoiesis and that under certain conditions, inorganic iron is made unavailable. We therefore cannot say what fraction of the iron in a diet is able to be absorbed and utilized. We do not know exactly what happens after iron is consumed. It is not excreted by the body, so there must be a mechanism which regulates absorption according to requirements. The nature of this mechanism

is unknown, but absorption seems to depend upon the state of the body reserves of iron, not upon the presence of anaemia. The amount of iron present in a diet, therefore, has little direct physiological significance. In the circumstances, all we can do is adopt a provisional figure as likely to cover requirements under normal conditions. In Western countries, it is generally accepted that, if the daily diet contains 12 mgm. of iron, this amount will be adequate for all the needs of a healthy man. It is impossible to state what level should be aimed at, for a population of unhealthy men, such as those with ankylostomiasis. As far as the Indian Army rations are concerned, 30—70 mgm. of iron are provided, that is, 2½—6 times the "optimum" for health. It is idle to assert that this may be too little for war conditions, because there is no ready method of increasing the amount to any significant extent, by means of normal foodstuffs. And, in any event, we do not know what physiological significance the figures have.

In ankylostomiasis, iron is lost through bleeding and clinical experience indicates that hookworm anaemia can be controlled satisfactorily with iron, even without deworming. The degree of anaemia has been thought to be approximately a function of the amount of iron lost through bleeding and the amount absorbed from diet or medicine. I have, however, no knowledge of any quantitative study of iron losses in ankylostomiasis, nor is it apparent why so few observers have found a correlation between degree of anaemia and hookworm load. It may be an over-simplification to say that hookworm anaemia is a simple matter of loss and gain of iron.

In malaria, there is no loss of iron, since that liberated from destroyed red cells, can be used again in new haemoglobin formation. It follows, therefore, that iron deficiency should not be a feature of post-malarial anaemia. Successful iron therapy in such anaemia, if not fortuitous, may be due to a specific stimulation of haemopoiesis by large doses of iron.

Protein

Protein is not specifically concerned with haemopoiesis as is iron, and anaemia due to protein deficiency has not, so far, been described. The work of Whipple and his colleagues has shown that blood proteins exist in a state of dynamic equilibrium with the protein reservoirs of the body, which are stocked from the diet. Thus, protein intake certainly can affect blood regeneration in anaemia. We have, however, few proved facts about quantitative and qualitative requirements of dietary protein in man. On an ordinary western mixed diet about 70 g. protein daily, is considered optimal. The Indian Army ration contains over 100 g. of protein, of which about 5 to 15 are from animal sources. Animal protein, generally, has a higher biological value than vegetable protein, and there is a widespread belief that it has special significance in the prevention of anaemia. This belief seems to arise mainly from two observations. One is that the ordinary diet of the Indan labourer is outstandingly poor in foods of animal origin and concurrently the population suffers from anaemia. The other is that British troops are relatively free from severe anaemia, as compared to Indian troops. If this fact is due to a nutritional difference, the outstanding item consumed largely by one and not the other, is meat. Now, animal protein is the outstanding constituent of meat and other foods of animal origin, but the evidence linking it with anaemia is rather slender. In health, a purely vegetarian diet can provide protein adequate for all needs, provided amino acid supplementation is effective. We do not know how protein requirements are affected in a body infested with parasites, but certainly protein losses are not severe. No proof has yet been offered that the total protein of the existing Indian rations, is not fully adequate for haemopoiesis under service conditions, in spite of the low animal protein intake. The observation that certain selected cases of severe anaemia have a lowered serum albumin, has, in itself, no direct significance in relation to habitual protein intake. We must, therefore, regard the case for more animal protein as lacking proof. The observations linking anaemia with small consumption of foods of animal origin, may be explained by other factors than animal protein.

Will's Factor

Will first postulated an unknown dietary factor, present in liver and marmite, which cured a macrocytic anaemia, common in the tropics. It is doubtful if this nutritional macrocytic anaemia has ever been described in cases without some unusual strain on haemopoiesis, such as that of pregnancy or malaria. We may, however, consider it as a conditional deficiency disease. Since Will's factor is found in liver and marmite, it may be an unidentified component of the vitamin B2 complex. Meat is known to be a good source of many of the vitamins of the B complex group, and is known to contain the extrinsic factor of Castle. This may explain, in part, the high value placed on meat, in the control of anaemia. Other foods of animal origin, such as milk and fish, are also good sources of various members of the vitamin B2

complex, though there is no particular evidence that they have any special value in haemopoiesis. Indeed, Whipple states that dairy produce and fish are relatively inert for stimulating new haemoglobin production in dogs with secondary anaemia.

The Indian Army Ration

We may conclude that the known facts about identified dietary constituents are of singularly small help in designing a ration for an army which requires protection against anaemia. It is, however, reasonable on many grounds, that if the ration is well-balanced in terms of known constituents, it is likely to provide adequate reserves of the known and unknown haemopoietic factors. There is, in addition, some circumstantial evidence that foods of animal origin and. in particular, meat should be liberally represented.

I have not time to deal with the Indian Army as a whole, but want to explain why meat and milk are not more adequately represented. The fact is, that no more meat, or other food of animal origin, is available. The Q. M. G. has been unable to implement our expressed recommendations from Indian sources and it is now clear that we cannot expect much more help from overseas. That is the reason why so little change has taken place, in spite of medical advice. I must impress on you that, if every Indian soldier actually receives the maximum amount of animal foods now authorized in Field Service Rations, it will be a great achievement. And until that is accomplished, increases in the Scales are out of the question. We in the Medical Directorate are fully aware of the importance of the anaemia problem and are ensuring that all the facts are known in the proper quarters. We are also going to considerable lengths in obtaining a more complete and accurate picture of the situation, through the work of Major Hynes, and many others. When preliminary work is completed, we may be able to examine further, the relation of anaemia to nutrition, under proper experimental conditions. Such an investigation will also have to take account of such factors as parasites and standards of recruitment. From the point of view of the medical specialist, it is clear that measures in the field of preventive nutrition cannot be expected to have much effect on the incidence of anaemia for some time. It therefore falls on you to ensure that men do not leave hospitals ill-nourished and with anaemia still extant. I hope that you will also experiment with treatment under controlled conditions and determine the parasitology of anaemia in conjunction with pathologists.

Another type of investigation which is of great value in planning a campaign of prevention, has been virtually ignored, as far as I am aware. I mean an investigation of the epidemiological statistics of anaemia. How many cases occur among a given body of troops under given conditions? What classes of troops are most prone to anaemia? Does length of service in the army lessen liability, and so on? In these matters we have almost no exact information. I should like very much to have a precise statistical analysis of the wastage caused by anaemia, since this is the kind of information which is understood at once by the General Staff. And precise figures are always more powerful incentives to reform, than any number of general impressions, no matter how unanimous the latter may be.

7. At this stage of the Conference, **GEN. GORDON WILSON,** said, "I deeply regret my inability to attend the complete session of the Conference, but I feel certain it will be an unqualified success and of benefit to the Army. We owe a debt of gratitude to Colonel Hayes for the excellent facilities he has placed at the disposal of the Conference, and which I am sure you all deeply appreciate. I must refer with pleasure to the presence of a very distinguished officer, late of the I. M. S.—General Hamilton—and I am sure we are all glad to see him here. I congratulate Colonel Schlesinger for his great initiative and keenness in organizing this Conference.

Gentlemen, I wish all success to your deliberations."

After Gen. Gordon Wilson had left, the Meeting was resumed, with **MAJOR-GENERAL MARTIN** in the Chair.

8. LT.-COL. KARAMCHANDANI—"*Nutritional Diarrhoea*" **(D)**

The association of chronic diarrhoea with infection through pathogenic organisms, has long been recognized. This infection may be derived from either a septic focus in mouth, nose or pharynx; or appendix or colon chronically inflamed by ingestion of pathogenic organisms. Our minds have been so much focussed on this aspect of diarrhoea, that search for pathogenic organisms has, heretofore, been the *sine qua non* for diagnosis, and dissociation from this ingrained notion becomes difficult. So that, if chronic diarrhoea happens to be accompanied by some flakes of mucus and blood, it is apt to escape correct diagnosis. This happened when some cases of intractable

diarrhoea with some mucus were reported. The medical officers, when satisfied that there were no offending pathogenic organisms, treated them symptomatically, with the result that cure was never obtained.

Intestinal carbohydrate dyspepsia, giving rise to diarrhoea as a result of excessive growth of enterococci; deficient digestion of proteins, resulting from gastric or pancreatic deficiency, leading to non-infective putrifactive diarrhoea; pancreatic deficiency, causing fatty diarrhoea; obstruction of lacteals, due to disease of mesentric glands or deficient bile, ending in chylous diarrhoea (i.e. excess of fatty acids and soaps); and gastrogenous diarrhoea, when gastric juice is deficient and an abnormal number of organisms reach the intestines, have long been understood and recognized as distinct entities. Various names, e.g. steatorrhoea, creatorrhoea, chylorrhoea, etc., have been coined for these types. Although such has been the panorama, still the infective portion of the disease has been put in such a bold relief, that its non-infective side has been almost ignored.

It is, therefore, this side of the picture that I wish to emphasize. A case of diarrhoea, whose cultural investigations of stools were consistently negative, came under my care. It was only when the patient died and a post-mortem examination performed, that the real nature of the disease became apparent, its gravity understood and experience taken advantage of, for future guidance. The histo-pathological report on this case is given below:

> "The mucosa shows desquamation of the epithelial cells, together with necrosis, the small ulceration being limited as far as the submucosa. The submucosa is markedly oedematious; the blood vessels are dilated and extravasation is noticed in the core of of villus-like processes; there is noticeable perivascular round cell infiltration. The muscularis is oedematous, more marked in inter-muscular plane; very little inflammatory reaction is noticed. The serosa is unaffected. Histo-diagnosis: ulcerative colitis of unknown aetiology."

I shall not discuss how the above pathological changes are consistent with nutritional insufficency. Suffice it to say that the vitamin supply, particularly of vitamin B, is as important as energy-providing substances, its deficiency tending to cause the disease under discussion. Just as sore mouth and tongue, erosing at angles of mouth, unevenly coloured tongue with enlarged papillae or denuded epithelium, are caused by B2 deficiency, so also are the desquamation of epithelial cells of the intestinal mucosa with necrosis and ulceration of the same, oedema of submucosa with dilated blood vessels and extravasation of blood cells and the swelling of muscularis with little inflammatory reaction, the result of consistent B2 deficiency. That the primary cause operating in these cases was nutritional insufficiency, was proved the rapeutically. I opened a separate ward, where 18 such cases were successfully treated with the undermentioned administration:

> Milk ounces eight, two-hourly, from 8 a.m. to 10 p.m., i.e. pounds four in 24 hours. Essence of chicken 25 cc. with vegemite one teaspoonful, three times a day. Orange juice with min. ten of dilute hydrochloric acid in it, twice a day. Calcium lactae grains ten, three times a day. Liver 2 cc. hypodermically, daily for six injections. Camphorodyne min. fifteen, three times a day. Diet was gradually built up, first by increasing milk to ten ounces two-hourly; then adding four ounces of toasted bread, two half-boiled eggs during the second week. Later on, the toasted bread was increased to eight ounces; minced meat three ounces and two bananas were added. After this, the patient was put on convalescent diet, which was usually by the end of the third or beginning of the fourth week. The patient was discharged after six weeks.

Out of 18 cases, 16 responded favourably to treatment; one case was diagnosed Tabes mesenterica and the other, an advanced case of anaemia.

9. LT.-COL. BHATTACHARYA—"*Avitaminosis in Persia*" **(D)**

This paper is based on observation of 4,500 medical cases of Polish nationality, treated in an I. G. H. in North Persia. The period covered is from June to December, 1942. Of these cases, 25·8 per cent. (1,167) were suffering from effects of malnutrition. The patients gave a history of poor diet in Russian prisons, concentration camps and occupied territories, over a period of nearly two years. Black bread and soup formed the staple articles of diet. Here are a few sample diets:

> (1) W. J. age 29. In prison—Vegetable soup, porridge and 300 gm. of bread daily; meat once a week (50 gm.),

(2) L. S. age 27. In occupied territory—Soup, porridge, 600—1,000 gm. of bread and potatoes cooked in mustard or cotton-seed oil, daily; meat 50 gm. twice a week.

(3) S. J. age 24. In concentration camp—700 gm. of bread and soup daily; 50 gm. of meat on alternate days; 20 gm. of sugar once a week; cooking in margarine.

The main groups of cases were:

Gr. 1 Pure inanition cases without symptoms of specific disease. 226 cases with 4 deaths (22 per cent. of the total malnutrition cases).

Gr. 2 Scurvy, 22 cases (1·8 per cent.).

Gr. 3 Nutritional oedema, 108 cases with 7 deaths (9·2 per cent.).

Gr. 4 Pellagra formed 64·2 per cent. of the total malnutrition cases, 135 were fully-fledged cases and 614 had mild pellagrous symptoms.

This short paper will be confined mainly to the consideration of some aspects of pellagra.

Seasonal incidence—Number of admissions for pellagra was 2½ times more in October—December than in the previous quarter.

Sex and age incidence—mostly males between the ages 20—50; this freedom of children, old men and females, may be explained, apart from other reasons, by the fact that adult males had to do the hardest work, in mines, in fields and in construction works in Russia. Wilson in Egypt (1921) did lay stress on the importance of hard labour as a predisposing factor for pellagra, as it creates a relative protein deficiency.

The constant features—present in all cases, in varying degrees, were: (1) Emaciation, (2) Diarrhoea, (3) Erythema and/or hyperkeratosis of skin, (4) Tongue changes.

The followng interesting symptoms were noted:

General—A placid resigned mood, rather than irritability or depression.

Alimentary—Hunger was far commoner than anorexia. We often discovered bottles of strong Persian vodka and loaves of bread, under the pillows of even moribund cases.

Tongue—In mild cases, redness at the tips was a constant feature. A large flabby tongue with superficial fissures was common, a raw smooth tongue in fully-fledged cases.

Diarrhoea—was invariably present, often with blood and mucus in stools.

Skin-Patches—of erythema on both cheeks, resembling malar flush of hectic fevers, was constantly seen in mild cases. In slightly more advanced cases, a butterfly-shaped erythematous patch on the bridge of nose and cheeks was a common feature. Along with this, scaliness and harshness of the skin was noticed on the back of hands, forearms and legs. Advanced cases showed hyperkeratosis and desquamation of skin, in parts exposed to sun. There was no special incidence of dermatitis on pressure points.

Nervous System—Symptoms due to involvement of the N. S. were rare. Only 7 cases of polyneuritis and one case of ataxia were seen.

Ocular Signs—Those commonly noticed were intense photophobia, visual fatigue, contraction of palpebral fissures and conjunctival congestion.

Mental Symptoms—While mild disturbances of mood were common, grosser mental complication were infrequent. Acute Encephalopathic Syndrome with acute delirium followed by stupor, was seen in 2 patients only; 14 cases of post-pellagral dementia were admitted; 12 cases with affective syndromes and 7 with schizophrenic type of reaction were also seen; the role of avitaminosis in the causation of the latter two types, might have been important.

Treatment—A liberal mixed diet, special items being marmite and tinned salmon: Nicotinic acid tablets given orally in doses 100—500 mgm. Average dose was 300 mgm. over a period of 3 to 4 weeks. Vit. A & D, conc. ascorbic acid and iron, were other drugs used in some cases. The early signs of improvement were disappearance of the malar flush, return of appetite from voracity to normality, improvement of diarrhoea; the mental condition of the cases improved and they beame more social and brighter.

Conclusion

A. Mild symptoms of deficiency of fat-soluble vitamins were frequent; malignant symptoms, producing definite disease, very rare.

B. Pellagra was the commonest deficiency disease encountered. Lack of protein and hard labour were important factors in the aetiology, in addition to the main cause—lack of vitamin B complex in the diet. The fact that carbohydrates formed the staple article of diet, additionally contributed to this vitamin B deficiency in the system. The symptoms exhibited by the cases, are in keeping with the modern conception of pellagra, that it is a true polyavitaminosis; thiamin, riboflavin and often ascorbic acid are concerned, as well as nicotinic acid.

C. Polyneuritis, supposed to be related to vitamin B deficiency, was very rare.

10. MAJOR WINGATE—"*Anaemia Centre in Peshawar District*" **(D)**

Summary

Some impressions gained as a result of 9 months' work at the above centre, are as under:

Multiple aetiology was present in most severe cases seen, recurrent malaria and chronic ankylostomiasis being factors in a high proportion. A mixed group, in which chronic diarrhoea was present was described. An apparently non-specific colitis was found at autopsy in a few fatal cases, with a marked thinning of the small gut wall. Inference was drawn from these and similar cases that, whatever the primary cause, the secondary effects of malabsorption were important in hindering progress. The need was stressed for parenteral therapy to supplement oral therapy in these case, sometimes for long periods.

11. CAPT. SHARMA—"*Anaemia and Malnutrition*" **(D)**

Nutritional deficiencies, like avitaminosis, are the result of variable participation of a number of factors, affecting intake, absorption and utilization of essential food factors.

Intake—Deficient intake, to cause even a moderate anaemia, has got to be pronounced and prolonged. Severe anaemias, due to this factor alone, are rare in military practice and respond quickly to treatment consisting of high caloric and protenous diet, with yeast or marmite, and iron. Out of 87 cases of anaemia under my observation recently, only 4 cases were of this type, with service varying from 1—3 months; three out of four being strict vegetarians, who refused to take the standard malnutrition diet, were unwilling to continue in the service and did not co-operate in treatment at all. Their progress was very slow and they had to be discharged from service. The fourth case was that of a Kashmiri Mohd., admitted in a very precarious condition, with R. B. count below one million and 20 per cent. Hb. He refused treatment and proved fatal. It is not yet ascertained up to what extent and how, deficiency in animal proteins accounts for causing anaemia, but additional animal proteins in diet of malnutrition and deficiency anaemias is of definite theapeutic value.

Deficient Absorption—Is responsible for causing a great majority of severe anaemias, most intractable cases being the result of dysenteries, sprue, chronic diarrhoea and other intestinal diseases. In terms of Castle's Hypothesis regarding causation of macrocytic hyperchromatic anaemias, i.e.

$$\frac{F+G}{I}=L.E.$$

it is increase in denominator or intestinal impermeability which accounts for the majority of the macrocytic anaemia of malnutrition. The blood picture in these cases is identical with that of Addisonian Pernicious Anaemia, the only points of distinction being (1) Hcl. in gastric juice is normal, (2) no increase of bilirubin in blood plasma, (3) C. N. S. lesions are absent, (4) typical atrophic changes in tongue and buccal mucous membrane are frequently absent. Vandenberg's test and fractional test meal are of great importance, to differentiate these conditions. Treatment:—High caloric diet, rice in proteins of animal origin, e.g. meat, liver, butter, cream, milk, eggs, yeast or marmite and iron, in addition to treatment of intestinal condition and blood transfusion when indicated, all improve the blood picture and general condition of the patient; quite a number of anaemia cases from the Assam front were of this category.

Defects in Utilization of Food Essentials—may be the result of innumerable human maladies—malaria and ankylostomiasis being two important causes—often causing severe microcytic hypochromatic type of anaemia. The incidence of malaria and ankylostomiasis in victims of dietary deficiencies exaggerates the anaemic condition; by haemolytic process in the case of malaria, and, in the latter case, by action of liberated toxins, the nature of which is yet unknown, is superadded to blood loss caused by the presence of these worms. The

quantity of blood drawn by one worm daily, is estimated as being 0·38—0·84 ccm. (Strong, 1942). The amount of blood lost, as such, is not the only factor causing anaemia of a severe type, if the blood regeneration capacity is normal. Malnutrition and chronic infection combined (e.g. in malaria and ankylostomiasis) depresses the haemopoietic capacity of bone marrow to such an extent that even a smaller amount of blood lost or destroyed, cannot be replaced. Appropriate treatment of these infections is of great importance, in addition to treatment of anaemia and malnutrition.

12. MAJOR DAVAR—"*Anaemia and Malnutrition*" **(D)**

Malnutrition is fairly common among Indian troops in the Eastern Army and a large number of cases are seen in the field areas. It is significant that this condition is not generally seen amongst British troops.

The constant aetiological factors are malaria and dysentery, accompanied by vitamin deficiency and bowel infections, with ankylostoma, tapewormn, etc. In my opinion, the more common of the two conditions is dysentery, which was present in as many as 80 per cent. of the cases. The common sequence of events is as follows:

The patient gets one or two attacks of malaria, or, more commonly, of dysentery, and improves with ordinary treatment, but in other cases, these attacks recur from time to time. In such cases, after the initial two or three attacks, blood, or blood and mucus, disappear from the stools, to be replaced by frequent attacks of diarrhoea with offensive liquid stools, accompanied by griping and tenesmus. As this condition persists, progressive malnutrition, multiple avitaminosis especially of B group, and emaciation supervene and the anaemia, especially of secondary type, becomes a marked feature. The patient further develops a more severe type of anaemia and glossitis, especially of atrophic variety. Now, two factors are present, namely (i) diarrhoea with toxoemia, and (ii) starvation due to anorexia and to difficulty in assimilating food. It is my opinion that, in the large proportion of cases, there is a primary bacillary dysenteric infection, and malnutrition, avitaminosis with anaemia supervene. It does not supervene in severe cases of dysentery, which are thoroughly treated in the early stages, but is more common in mild cases, which do not receive adequate attention.

The stools, though they show all the characteristics of bacillary dysentery, do not always give positive culture results; negative culture reports do not indicate absence of dysenteric infection. I recently saw ten B. O. R's, from one Unit, with typical signs and symptoms of bacillary infection, but, of the stool cultures made from fresh stools, only one showed B. Flexner growth.

In the late, severe stage of anaemia, about 25 per cent. showed the characteristics of macrocytic hypochromic type, with colour index over one.

(i) *R. B. C. Count*

Below 1·5 million—2 per cent. of cases.
Between 1·5 and 3 million—50 per cent. of cases.
Between 3 and 4 million—48 per cent. of cases.

(ii) *Hg. per cent.*

Below 30 per cent.—5 per cent.
Between 30 and 50 per cent.—40 per cent.
Between 50 and 70 per cent.—50 per cent.
Above 70 per cent.—5 per cent.

(iii) *Colour Index*

Below 1—75 per cent.
Above 1—25 per cent.

These blood counts may seem comparatively high, as they were not done immediately on admission, but after the patients had been treated for some time.

One of the characteristics of anaemia of malnutrition and avitaminosis, is its very slow response to treatment. It takes a long time for the blood picture to reach its normal level. After blood transfusion there is rise in R. B. C. and in Hg. per cent. which is not always maintained unless transfusions are repeated; and even in such cases, the progress is very

slow. Severe cases of anaemia require eight to nine months or more of treatment, for the blood to reach its normal level.

DISCUSSION

13. LT.-COL. COBBAN said that, in the Anaemia Centre at Delhi, ankylostomiasis had been found to be rare as a cause of anaemia, most of the cases being due to malaria or chronic diarrhoea following dysentery. The anaemias due to malaria were of two types—the acute and the chronic. It was further found, in some cases of anaemia without apparent cause, that progress was not made, in spite of full treatment and diet. In all these cases, eventually, malaria parasites were found in the blood and, after a course of anti-malarial treatment, recovery was rapid.

14. LT.-COL. MURTHI gave the details of an investigation that was carried out on a small series of cases, in the Anaemia Centre at Moradabad:

"*Actiology*—An analysis as to the causation of the anaemia made by Capt. R. N. Chatterjee, M. O. in charge of the ward, is as follows:

Malaria 54 per cent. ankylostomiasis 9 per cent. malaria and ankylostomiasis 9 per cent., dysentery or diarrhoea 12 per cent., malaria and dysentery 3 per cent. post haemorrhaegic 3 per cent., unknown causes awaiting investigation 9 per cent.

Nature of the Anaemias

Hypochromic 40 per cent., Normochromic 30 per cent. Hyperchromic 30 per cent. In the absence of a Haemocrit, attempts were made with a graduated centrifuge tube, using the standard oxalate solution, but the results were not in conformity with anticipations and hence I am unable to present the nature of the anaemias with reference to the Mean Corpuscular Volume and hence the diameter.

Anaemia in Malaria Cases

50 per cent. of malaria cases showed a Normochromic anaemia and in these the malaria had been of a relatively recent date. 39 per cent. of the cases showed a hypochromic anaemia and, in these subjects, the attacks of malaria were recurrent and the spleen was very enlarged and hard. From the above, it would appear that anaemia in recent and fresh malaria is due to haemolysis occurring in the paroxysms, while in the anaemia of chronic and relapsing malaria, it is due to the additional factor of iron deficiency in the system or its defective utilization. 10 per cent. of the cases showed a hyperchromic anaemia, but further investigation may show additional factors. For the present, I should consider that hyperchromic anaemia in uncomplicated malaria is not common.

Malnutrition and Anaemia

6 per cent. of the cases, only, showed any signs of malnutrition and, even in these, the intestinal disturbance answered for the malnutrition and hence I consider that anaemia of nutritional origin has not occurred in my series.

Therapy

The indiscriminate use of liver extract in anaemia is not indicated."

15. COL. TAYLOR said, "The two charts on the epidiascope show the sickness rates of B. T. and I. T. in Assam and in Eastern Army, during 1943. In the one from Assam, the sickness rate from July onwards, for Indian troops, was considerably higher than for British troops, unlike the rates elsewhere in Eastern Army and, I understand, the rates for the rest of India, where the B. T. rate is higher than the I. T. rate.

The reasons for this are associated with the shortage of supplies of animal protein, fruit and vegetables, from May onwards, for Indian troops in forward areas in Assam, and Indian troops in these areas were fed mainly on *atta*, rice, *dal*, sugar, tea, condiments and a small quantity of milk, for months, a diet similar to that given by Goldblatt to his convicts in his pellagra experiment, reported in 1920. These men, after a few months on this diet, in the highly malarious areas existing in Assam, develop symptoms of anorexia, flatulence, dyspepsia and soreness of the tongue, in the early stages. The tongue condition is a glossitis, with red enlarged papillae, in the early stages. These cases later develop diarrhoea and progressive wasting and anaemia. On 30th September, 1943, over 1,000 cases of this type were in hospitals in these areas and many were evacuated to Central Command. With

poor feeding in hospitals, the condition is often made worse. These conditions did not arise in British troops or in Indian troops elsewhere in Eastern Command, and, did not arise in the Arakan, where troops received the F. S. Scale of animal protein, though subject to the same conditions of malaria.

We are not in a position to criticize policy. But, with reports of the sort we have heard this morning, of inadequate food supplies for the Army, we naturally all wonder whether it is sound to recruit large numbers of poor-type Indian recruits, whom the Army is unable to feed adequately, in forward and other areas. We, as Medical Specialists, are at least called upon continually to deal with the results of this policy."

16. LT.-COL. PASRICHA—"*General Remarks on Treatment*" (P)

In the few minutes allotted to this talk, it is not possible to do more than draw attention to some of the more important points. If I am dogmatic and, at time, provocative, it is for the purpose of eliciting information and inviting discussion on a subject which is one of the most important to the Military Medical Services in India, as it is to the civil practitioners of India.

The Anemia and Malnutrition Problem

We have anaemia in the Army, more so in the I. O. R. than in the B. O. R., more in some communities, more in certain types of workers, more in certain areas than in others. Far too many of the recruits to the Army are anaemic, some so pale that one would advise them to seek admission to a hospital, rather than join the Army. Here are some findings in a training centre, which I recently visited. Of the new recruit, some 20 per cent. were obviously pale, of those who had six weeks' training, some 15 per cent. were anaemic, a difference which is of no significance. Next, I examined a batch of trained men, ready to be sent to forward areas. They looked fit when viewed from afar; they had apparently good muscle, their march and their bearing was that of apparently healthy men, but, when examined from near, by the simple method of a "tongue parade", some 20 per cent. were grossly anaemic. This was confirmed by the estimation of haemoglobin, using for this purpose the crude Tallqvist' method. All had haemoglobin below 60 per cent. and of these, 70 per cent. had haemoglobin below 50 per cent. a somewhat disconcerting picture; More of this later.

Let us first study the problem of anaemia in India on its broader base. Until about two decades ago, haematological studies in India were practically non-existent; whatever data was on record was poor. In 1926, Balfour and Wills laid the foundation of haematological survey in India, and their studies have been continued by a series of workers, culminating in the work, over a period of eight years, at the Calcutta School of Tropical Medicine. From this work, one can interpret the picture in considerable detail. The facts that stand out in bold relief are:

(1) City-dwelling Indians, living on a good mixed diet—the blood picture conforms closely to European standards. In rural populations and in Indians of the inferior economic strata, the haemoglobin level is lower; in some areas, e.g. Assam, much lower than in others.

(2) This high incidence of anaemia in the general population is not one specific condition with one common cause, but is due to several aetiological factors, some known and some still to be discovered; of the known, the most important are hookworm, intestinal infections, and a low available iron intake, as causes of microcytic subchromic anaemia; and specific and general dietary deficiency, alone or in combination with malaria, as the cause of macrocytic superchromic anaemia. These aetiological factors are often combined, resulting in anaemia which is both micro- and macracytic in type the so-called "dimorphic" anaemia.

(3) The high incidence of anaemia in the rural population of India results, in the case of women, in a very high incidence of anaemia of pregnancy, with alarmingly high mortality. Just as the women develop severe anaemia in pregnancy, so the men of this population, of whom a very large proportion have some form of dys-haemopoiesis, are liable to develop severe anaemia under the stress and rigours of army life. This breakdown is particularly common in ancillary troops.

Treatment of Anaemia.

Once we appreciate the background against which a disease is being studied, the treatment can be rationalised. The treatment should begin, not in hospitals, but in training centres. It is the failure of adequate treatment in these centres, that eventually fills the Base Hospitals. A few words about the treatment at the Centres: deworming and extra milk for all recruits from areas of heavy hookworm infection should be insisted upon. In addition, the M. O. i/c and the O. C. Training Centre, should carry out frequent "tongue parades" and augment the treatment of those who are obviously anaemic with a course of iron. True, the iron often does not raise the haemoglobin as it does, for example, in chlorosis, but it is worth trying as an experimental measure, until such time as something better can be found. The Medical Services insist on men—fit men—even though they be labourers, sweepers, nursing orderlies, or members of the R. I. A. S. C.

Treatment of and Established Case

I shall confine my remarks to the patients as seen in an I. B. G. H., of which I have first-hand information. The first essential is the complete investigation of a case. In the study of anaemia, I would stress those investigations which are the barest minimum, e.g. the haemoglobin estimation (using a reliable method), R. B. C. and reticulocyte counts, cell volume and W. B. C. count, in order of importance. Others, such as the examination of urine and stool are often necessary. There are many pitfalls and difficulties in the interpretation of laboratory findings, e.g. the haemoconcentration in an individual on admission, which, unless one is aware of the condition, may give a misleading picture. Once the type of anaemia is known, treatment is easy. Iron for the microcytic subchromic iron deficiency anaemia; and liver for the macrocytic superchromic crude-liver deficiency anaemia, remembering always that both types often co-exist; when the dominant type is treated first, and, later, the other type becomes apparent. For the very severe—and we attach more importance to the clinical condition of the patient, rather than the R. B. C. count—blood transfusion has been our main sheet-anchor. We prefer large whole blood transfusions, given slowly, and repeated, to maintain a satisfactory haemoglobin concentration. These transfusions are particularly useful in chronic cases, when, in spite of treatment, the general condition continues to deteriorate. When facilities for blood examinations are not available, and this is by no means infrequent, owing to lack of trained personnel, lack of apparatus or a deluge of patients, it is a good working rule to give a course of iron and observe the result. If there is no improvement, give liver in addition. It is my impression that some of the available liver preparations are not effective. The reticulocyte response after their exhibition is poor, but I have not any controlled data on this point to present to you. It is an impression which is worth further investigation. In this empirical form of treatment, it is worth remembering our background, that in India, the dominating defect, as far as the haemopoietic system is concerned, is a hypochromic, microcytic, iron-deficiency anaemia.

And now I come to a very important part of treatment—the diet. Many of the patients that I saw last autumn, were living skeletons—a mass of bones, covered with a minimum of tissues—some with diarrhoea, some still having bouts of malaria. Here is another impression, without any controlled data, these thin miserable specimens of humanity appeared to suffer from more frequent relapses of malaria than the ordinary individual. These patients had been kept for a considerable time on "light diet", consisting mainly of fluids, soups, whey, lime-juice, albumen water, supplemented in some cases with cocoa, Horlicks, porridge, egg-flip or bread and butter. They were being starved and undoubtedly suffered more from the medical attention (or, more correctly, lack of medical appreciation) than from their natural enemies. These patients were put on a high protein, high vitamin, low residue diet, containing normal amounts of carbohydrates and fats. Many of these patients had lost their appetite—and who would not, if fed for any length of time on slops? The Indian appetite, dependent as it is, by his nature, on spices and condiments, revolts against the continuous exhibition of egg-flips, whey, cocoa, Horlicks and other similar insipid preparations. The desire for food sickens until it finally dies. Such men do not need medicines to stimulate their appetite; they need the aroma of spices, the visual stimulus of foods which they know and which they relish.

An invalid kitchen was started, run entirely by voluntary workers, all Indian ladies. They were encouraged to use their ingenuity to produce suitable Indian diets. They produced a series of excellent dishes, e.g.

(1) *Khicheri* (rice, gram and butter).

(2) *Egg Halwa* (eggs, butter and sugar)—something like buttered eggs, but sweetened; salted for a change.

(3) *Curries*—Chicken, liver, fish, vegetable, etc. These are not curries of the kind prepared in hotels or messes, which would upset all the most hardy stomachs, but more like English stews, with light flavouring of fresh spices, of the light *korma* type.

(4) *Pish-Pash* (rice, chicken and butter)—often enriched with liver soup and liver minced, fried or roasted.

(5) *Milk products and puddings*—

(*a*) Khir-rice, milk and sugar, flavoured with Indian flavourings, e.g. rose-water, *kiara*, cardamoms.

(*b*) *Dhai* (curdled milk—the lactobacillus acidophilus milk) either pure or various preparations of it, containing vegetables; sweetened for those who prefer it sweet.

(*c*) Bread and butter pudding—the ordinary bread and butter pudding, but with Indian flavours, as in (*a*) above.

(*d*) Custard puddings—and jellies with milk, flavoured as above.

(6) *Soups*—chicken, liver, vegetable—flavoured lightly with spices.

(7) *Predigested foods*—Papain digest of meat, chicken, liver, or "moong" dal.

(8) Adjuvants—

(*a*) Lime pickle.

(*b*) Raw papaya chutney.

(*c*) Raw tomato chutney, etc.

(9) Liver Preparations—

(*a*) Soup.

(*b*) Curries of "*korma*" type.

(*c*) Liver-juice raw [see 10(*b*)].

(*d*) Liver pieces grilled—particularly palatable.

(10) Yeast and Yeast preparations—

(*a*) Bakers' yeast, freshly prepared—4 oz. per patient.

(*b*) "Jhosh" cocktail, containing—

(i) yeast, 4 ounces.

(ii) lime-juice, 1 drachm.

(iii) liver juice raw, 1 drachm.

(iv) sugar to taste, or, when sugarcane is available, fresh sugarcane juice.

(11) Gland Dish—a curry of kidneys, liver, sweetbreads, etc.

(12) Sprouting Gram—conveniently mixed in the chutney, relished greatly by patients.

These are some of the dishes prepared in the invalid kitchen, by women voluntary workers. Many other articles are prepared, and the ladies make every effort to cook special dishes at the request of the medical officers. The ladies not only cook tempting dishes, to stimulate jaded appetites but follow it up by contacting the more seriously-ill patients and enquiring whether the food was to their liking, whether it was seasoned properly; in this and in many other ways, these ladies have converted a hospital diet into a "home" diet. The results of the establishment of such a kitchen have been astounding; in many cases; the thin miserable individuals have filled out, often in a remarkably short period. These patients need feeding, and, even patients with mild degrees of intestinal fluxes, improve remarkably when fed liberally on a diet which appeals to them. There is a real need for the establishment of special invalid kitchens, staffed or supervised, wherever possible, by voluntary lady workers. In the kitchen established at Lucknow, we have Hindu, Mohammadan and Christian ladies, working together and there has never been any objection from any of the patients.

And, lastly, vitamins and their role in the treatment of anaemia and allied conditions. Although used, often without any real understanding of their value, their limitations, their role in the treatment of the cases, is limited. Their real value is the treatment of patients suffering from dysvitaminosis, in syndromes where lack of a particular

vitamin is established. In such patients, the response to adequate dosage of the vitamin concerned, is striking. The vitamins are often used as garnishes in other treatments, a little sprinkling of one or several preparations is prescribed, more or less as a routine; choice of these are often governed by fancy, and results by fortune. This is a confession of failure and a sheer waste of these precious and powerful therapeutic agents.

17. MAJOR LEHMANN—"*Concentrated Blood Transfusion in Anaemias*" **(P)**

Before reading this paper on Concentrated Blood Transfusion, I must explain that this work was not carried out as pure research, but as an endeavour to save life at a time when, apart from the increased war-time work at the District Laboratory, we were running a Blood Bank and processing serum, and I was not able to get any details from case sheets, as the I. G. H. concerned has now left Meerut District. I have, however, all the blood counts, etc., collected at the time, and the types of treatment given, with the results.

The Cases

In the latter part of 1942, many cases of anaemias, following malarias and dysentery, were coming from Burma and being passed back from clearing stations to Base Hospitals. On the way, very little treatment and few examinations were done and cases were arriving in Meerut with red cell counts as low as 700,000 per cmm. and Hb. 1·7 grm per cent. Treatment with iron and liver, combined with special diets with low roughage and high calorific and vitamin content, were tried, but it was soon seen that, even in the cases which were afebrile and showed no signs of ankylostoma infection, the haemopoietic tissues were not stimulated sufficiently to improve the blood pictures, but, on the contrary, these continued to deteriorate. Whole blood transfusions were given to two of the patients, but as these cases died soon after, the O. C. Hospital forbade the medical officers in charge of the cases to continue this treatment. The bloods given were carefully matched and there were no signs of incompatibility. It was considered at the time that the extra volume added to the already increased blood volume of an anaemia, was too much for a heart, whose muscle walls were greatly weakened by lack of nutrition over many weeks. During September and November, 1942, out of 30 cases treated in these ways, 13 died and, although complete post-mortems were carried out, nothing abnormal was found, which could not be accounted for by the anaemia *per se*, following chronic malaria and bacillary dysentery. With the exception of one case, in which a large haemorrhage was found to be present in one supra-renal, no other glandular change were discovered.

Concentrated Blood Fluids.

(i) At that time, we had a Blood Bank and were separating and processing serum at the District Laboratory, Meerut. We were, therefore, prepared to supply citrated whole blood in sufficient quantities (about 100 bottles) as required. The difficulty here, however, was that the blood would have to be given very slowly at the rate of 20 drops a minute (1 pint in 8 hours) and as, on some days, five or more transfusions were required, sufficient apparatus was not available. Also, constant watching is necessary to prevent blockage in very slow transfusions and sufficient trained staff were not at that time available. Oxygen could not be obtained to agitate the fluid and thus prevent the settling of the cells. It was therefore decided to prepare sterile concentrated blood cell fluids from the blood clot left over after separating the serum. It was considered that this would have one great advantage over whole blood transfusion, i.e. the same number of cells carried in smaller volume with the concomitant advantages of (*a*) quickness and ease of administration and (*b*) less strain on the heart, while, at the same time, putting new oxygen-carrying cells into the circulation to feed and stimulate the haemopoitic tissues; that is, to make ½ pint of concentrated blood do the work of 1 pint of ordinary blood. The first concentrated blood cell fluid transfusion given, was a small one—100 cc and 5 million R. B. C. per cmm, as we had no knowledge at that time of any work done with concentrated cell fluids and did not know whether there might be severe reactions. The result from this seemed good, an increase from 700,000 to 900,000 R. B. C. per cmm, so these small tranfusions were continued—75—125 cc 5—7·5 million R. B. C. per cmm.

(ii) *Types of fluids*—Two types of cell concentrations were supplied, (*a*) Cells in serum, (*b*) Cells in plasma.

(iii) *Method of preparation*—(*a*) After allowing human blood to clot for 24 hours, it is seen that, apart from the clot and the serum, there is always a layer of loose blood cells. For the purposes of producing serum for processing, bottles of 500 cc. of blood having been collected, they were allowed to clot for 24 hours. The serum was then removed and about 100 cc. of loose cells and serum were left with the large clot. By shaking this, more cells were detached from the clot. When the fluid was considered to be sufficiently concentrated, it was blown over through a fine mesh filter into sterile bottles. The air used for this purpose was first cleaned and sterilised by passage through filters containing oil and Dettol. Samples of blood were then examined microscopically for the presence of clot, and counts done for concentration. (*b*) When cells in plasma were used, donors were bled specially for these, into Horlicks bottles, fitted with rubber diaphragms, containing the requisite amount of citrate. These were allowed to stand for 24—48 hours in the refrigerator. The supernatant plasma was then blown off until the required concentration was produced. The bottles were then well shaken and a small amount of the concentrated blood in plasma blown off for an examination of the condition of the cells and a count. (*Note*—Another and simpler method is now being used in some hospitals. For this, the blood is taken from the donor, the bottle is then fitted for blood-giving, turned upside-down and left overnight in the refrigerator. Next morning, the blood transfusion is given in the ordinary way without shaking the bottle. The layer of concentrated cells being given and a large proportion of the plasma left in the bottle.) Cells in serum were used at the beginning, until we were informed that there was a danger in using serum had been in contact with clot for more than 72 hours, as a vaso-depressive substance had been shown to develop. For this reason, cells in plasma were used for the later transfusions. The only difference that we could discover was that we had some reactions with these latter, (although pyrogen-free water was used), which were not seen with the former.

(iv) *Concentration*—A normal blood transfusion, given without concentrating the cells by any method, is about 4,000,000 per cmm, e.g. 400 cc. of 5,000,000 R. B. C. per cmm. blood+100 cc. of citrate, gives 500 cc. of 4,000,000 R. B. C. per cmm blood. The bloods used for these cases were 5—7·5 million per cmm. i.e. an increase in concentration of 25 to 90 per cent.

Method of Transfusing

These small infusions were quickly given, without any special apparatus, in about 20—30 minutes. The fluid was poured from the bottle into a small Roger's-type saline infusion apparatus and thence into the vein, through a wide bore needle. Generally, there was no need to cut a vein.

Results

All the cases with counts of less than 2,000,000 on admission have been collected for the purpose of survey. All those treated by concentrated transfusions being placed in one group (Gr. ii) and all by other methods in (Gr. i). (Group i) consists of 30 cases of which 13 died, giving a mortality just over 43 per cent. (Group ii) of 20 cases of which 2 died, giving a mortality of 10 per cent.

Discussion

(i) *Cause*—As all these cases gave a history of dysentery and malaria, it would appear that the anaemia was caused by a combination of blood destruction by the plasmodia and lack of absorption, due to the state of the intestine, of some factor required to stimulate the marrow to activity. Similar cases arriving in Benares were shown to be pellagra. In the recent epidemic of pellagra in the Sudan, the precipitating factor appeared to be a long continued bacillary dysentery in people living on a border-line or deficient diet. The cases referred to in this paper had been on deficient diet and all had or had chronic dysentery. Some of them were treated with vitamin B, but without success, but it is generally accepted that, pellagra cases in which the diarrhoea is not cured, will not respond to vitamin B therapy. I have not, however, been able to obtain the details in these cases. They showed, however, no skin lesions or any of the typical cord lesions or burning pains in the epigastrium; one or two, however, complained of sore tongues. The blood pressures are not available. None of the cases that died, showed the typical atrophy of the ileum. These cases were not, therefore, diagnosed as pellagra.

(ii) *Effect*—The original intention in giving concentrated cells was to give in 2—300 cc. the equivalent of 1 pint of blood, cells, and thus increase the blood count by 500,000 per cmm, per transfusion, taking the patient out of the danger zone and, at the same time, gradually building up the blood until his own haemopoietic tissue would take over. When, however, it was found that the small transfusions stimulated the haemopoietic tissue to rapid blood production (Reticulocyte counts is some cases of 20 per cent.) these were continued. The changes in the blood were much more (1,000,000 per cmm, increases in three days) than could have been accounted for by the actual number of cells added (100 cc. of 8 million per cmm, blood would only show an increase of about 100,000), and so one must assume that they stimulated production.

(iii) *A Partial Analysis*—For this, the cases have been divided into three sections: (*N.B.*—Graphs have been omitted. *Ed.*)

(*a*) R. B. C. count below 1,000,000 per cmm.

(*b*) R. B. C. count between 1,000,000 and 1,500,000 per cmm.

(*c*) R. B. C. count between 1,520,000 and 2,000,000 per cmm.

(*a*) Seven cases—

(i) Good response—relapse during fever (one case).

(ii) Slight relapse after first injection—two required before a good response was obtained (one case).

(iii) Good response—rapid increase after one injection (one case).

(iv) Good uninterrupted response (two cases).

(v) No response—gradual decrease—aplastic (two cases).

Mortality—29 per cent.

Relapse rate—60 per cent.

(*b*) Three cases—

(i) Rapid increase after only one injection (one case).

(ii) Very rapid increase after one injection 1 million in 3 days, 2 million in 12 days. Relapsed but recovered and blood picture went to normal with no further treatment (one case).

(iii) Rapid increase—later relapse—rapid response after further injection (one case).

Mortality—Nil

Relapse rate—66 per cent.

(*c*) Eleven Cases—

These cases all did very well, only four out of eleven showed relapses, one of which was within the limits of error of blood counting and can be disregarded.

Mortality—Nil

Relapse rate—27 per cent.

It is difficult, as mentioned at the beginning, to give a very complete analysis from these figures, as insufficient counts were taken. It seems, however, that if these concentrated small transfusions are given to cases before they fall below 1,000,000, all respond and recover and that the earlier that they are given, the less the relapse rate. This, however, was difficult to analyse, as no details of the attacks of malaria from which some of the cases suffered, are obtainable.

(iv) *Statistical Evaluation*—Assessment of significance. As only a few cases in all were treated by this method, it was necessary to assess whether the change of mortality could be due to chance, or whether it was significant. Using the formulae for standard deviation and error, it can be shown that the odds against this change of mortality being the result of chance are 1: 80. These are vey long odds and the results are therefore significant.

N.B.—This is not put forward to take the place of whole blood transfusion, but to show what can be done in an emergency, when whole blood transfusion cannot be given, for lack of sufficient apparatus or personnel, or when it is forbidden.

(*The response to treatment by concentrated blood, recorded on charts, was demonstrated by epidiascope*).

18. MAJOR ELLIS—"*Blood Transfusion*" (D)

My remarks on Blood Transfusion, during the next five minutes, will be confined entirely to its application in those cases of anaemia in whom malnutrition is a predominant factor. I suggest to you that blood transfusion, in such patients, is performed for one of two main reasons: (1) to save life, either because the degree of anaemia is too severe to wait for haemopoietic substances to act or because such remedies have been found relatively ineffective; and (2) to reduce, if possible, the time spent by these patients in hospital.

I consider that you can attain neither of these objectives by the administration of small quantities of blood, whether it be in the form of 250 cc. of a citrate mixture or 150 cc. of properly concentrated red cells. I advise instead, the transfusion of one to two pints of fresh blood, given at a slow drip rate and repeated after a few days, when necessary. Some medical officers are a little nervous of blood transfusion, particularly of large quantities, because a small number of fatalities have been reported from one or two hospitals. It may interest you to know that in my own Unit, we have successfully transfused a small number of patients with haemoglobins below 2·5 grams. The quantities administered in any one transfusion have not been less than two pints and, in one or two instances, have exceeded three. I would also like to draw your attention to the fact that, in practically all those fatal cases whose record sheets I have been allowed to see, death is stated to have occurred, not after large transfusions, but following small ones—in some cases of no more than 50 cc. and I am satisfied that these results have been largely due to the administration of contaminated material, or of the transfusion of fluids through dirty apparatus.

The previous speaker has told us that a somewhat dilute suspension of R. B. Cs. in serum was used at his hospital, as two deaths had occurred following a pint transfusion of ordinary blood. This would suggest that perhaps the plasma provided the responsible lethal factor. There is no evidence that I know of, experimental or otherwise, to substantiate this reasoning. Providing that the necessary care and precautions are taken, neither cells nor plasma should, in my opinion, cause ill-effects. I myself have found, that reactions with plasma are, in fact, far less frequent than they are with ordinary citrated blood.

In the preceding paper it has been stated that Transfusion Service did not approve of the administration of serum and cells, collected from around the blood clot, after the clear serum had been siphoned off. The reasons why this was deprecated are, namely:

(*a*) Because such a mixture was not a proper red cell concentration, the percentage of R. B. Cs. being not appreciably higher than that of ordinary citrated blood, whilst the increased risk of contaminating the material was much greater.

(*b*) Because it has been shown by various workers, e.g. Zipf, Fiske and, more recently by Reid and Bick of Melbourne, that when blood clots, adenyl compounds are produced. These substances are vasodilator in action and have been shown to be partly responsible for the toxicity to laboratory animals—rabbits, etc.—of fresh defibrinated blood or serum. These substances are not present in human serum after it has been stored for a little time. Another substance, which I will not go into now, is also reported by some workers to be liberated by platelets during the process of clotting.

It has been stated that such mixtures can be given far more quickly than the ordinary transfusion. I think that most physicians who have used the red cell concentration are agreed that it is most important that they should be given very slowly indeed.

I do not wish to imply that concentrated red cells do not fulfill a very useful therapeutic role in some conditions, but I would draw your attention to the fact that, in the particular series of cases at present under discussion, the total proteins are very often on the low side. Is there any harm in transfusing such patients, not only with red cells, but also with protein? Secondly, it is most important for us to remember that when fresh blood was collected under comparatively ideal conditions in the United Kingdom, the contamination rate was often as high as 5 per cent. This figure is inevitably increased when red cell concentrates are prepared, even when this is done by experts in the most ideal surroundings. In India, contamination, as you are aware, is a very much more serious menace to work of this sort, than in England. Surely then, we are not justified in taking unnecessary risks and I would urge that, if we can treat our patients every bit as satisfactorily by employing the more simple measures of blood transfusion, then we should do so.

19. MAJOR CRADDOCK—*"Iron Therapy"* (D)

I well remember a physician in London introducing a lecture with the story of a woman, suffering from iron deficiency anaemia, whose haemoglobin was 60 per cent. She was not very co-operative and he sent her away with strict instructions to take 90 grains of Blaud's pills daily, without fail. She consulted him again after ten days and her haemoglobin was found to be still 60 per cent. The physician accordingly asked her, "Are you quite sure you've taken your medicine regularly?" "Quite sure," she replied. He then faced her and said very deliberately, "Woman, you lie!" Whereupon she broke down and confessed. It was a reasonable certainty, in this case, that the haemoglobin would have risen to 70 per cent. and possibly 75 per cent. In a series of cases, who received 9 grains of ferrous sulphate daily, the mean average daily increase in haemoglobin was 1·2 per cent. Such a mathematically predictable response to iron is the basis of the confident attitude towards iron deficiency anaemia in non-tropical countries, but I have yet to see similar satisfactory results recorded consistently in India.

This means that either the anaemia so treated is not a simple iron deficiency anaemia, or that the treatment is inadequate because of faulty administration of iron or poor absorption.

It is my belief that a simple iron deficiency, anaemia is, in fact, not common among Indian troops, partly because it is less common in men anyway, but chiefly because of the frequency of nutritional, especially vitamin deficiencies, and of helminthic infections among them. It is well known that anaemias, partly or wholly attributable to vitamin deficiency, will not respond to iron alone, however adequate the dosage. In many cases, however, even if these factors, together with achlorhydria, if present, are taken into account and adequately treated, the anaemia does not respond until parenteral liver therapy is instituted. Liver appears to be far more necessary for haemopoiesis, even in apparently simple anaemias, than in temperate countries. I am prepared to modify this view, however, pending the results of certain controlled experiments, which we are now carrying out in the anaemia centre attached to our hospital.

Faulty administration of iron has probably played a part in the past, but this should not occur with ferrous sulphate, which, all authorities now seem to agree, is the most suitable preparation. As iron is absorbed from the duodenum and upper jejunum, in the form of its ferrous salts, this attitude would seem to be well founded. Ferrous sulphate, also, has a high utilization percentage; 9 grains contain 180 mgm. of iron, of which 14 per cent. (25 mgm.) is utilizable. This is well in excess of the 7—10 mgm. estimated to be the normal average daily requirement for men. Half of our iron deficiency anaemia patients now receive 5 grains of ferrous sulphate t.d.s., prepared by trituration with glucose, as recommended recently. The other half receive 9 grains of ferrous sulphate in one dose daily put up in gelatin capsules to ensure that oxidation cannot occur before the salt reaches the gut. Given in this way it is simple, convenient and inexpensive. In addition, all patients receive fresh brewers' yeast, 2 ounces daily. Acid hydrochlor. dil. is given only to patients with achlorhydria or hypochlorhydria, and ascorbic acid only to those who are thought to be deficient in it.

All patients, except those undergoing controlled experiments, receive the following diet, while in hospital. This diet is chosen, not only because it is rich in iron—it might be said that this is unnecessary if medicinal iron is being given in adequate dosage—but because, in general, the foods rich in iron are rich in vitamins also. On this regime, the best results we have had so far, was a gain of 25 per cent. of haemoglobin in 24 days. This patient received only ferrous sulphate and yeast, in addition to the diet. Nevertheless, our experience to date suggests that, until the results of controlled experiments in the anaemia centres now established, give clear indications regarding the value of simple iron therapy, it is more economical, from the point of view of returning men to duty in the shortest possible time, to combine iron with other haemopoietic agents.

(The following diet chart, showing total and ionisible iron contents of various items, with their caloric values, was demonstrated on the epidascope).

Diet in Anaemia Ward

Basis : Milk diet, plus extras

Time				Total Fe. mgms.	Ionisable Fe. mgms	Calories
07.00 hrs. ..	Tea	Milk ..	10 oz.	0.70	10.70	200
		Sugar ..	1 oz.	..	..	100
09.00 hrs. ..	Egg flip ..	Egg ..	1	1.80	1.80	60
		Milk ..	10 oz.	0.70	0.70	200
11.00 hrs ..	Porridge	Milk ..	10 oz.	0.70	0.70	200
		G. syrup ..	1 oz.	0.50	0.45	90
12.00 hrs ..	Midday meal ..	Rice ..	5 oz.	0.95	0.80	500
		Dal ..	1½ oz.	3.20	2.10	135
15.00 hrs. ..	Afternoon meal ..	Liver ..	8 oz.	15.50	14.00	400
		Vegetable* ..	8 oz.	1.50	1.20	100
		Bread ..	8 oz.	2.50	1.90	400
		Butter ..	2 oz.	..	..	400
18.00 hrs. ..	Evening meal ..	Rice ..	5 oz.	0.95	0.80	500
		Dal ..	1½ oz.	3.20	2.10	135
		Egg ..	1	1.80	1.80	60
		Milk curd ..	10 oz.	0.70	0.70	200
21.00 hrs. ..	Cocoa	Cocoa ..	1 oz.	0.45	0.40	..
		Sugar ..	1 oz.	..	..	100
		Milk ..	10 oz.	0.70	0.70	200
		Total ..		38.35	32.10	4,220

(1) * includes potatoes and spinach when available.
(2) All the milk may profitably be given as *Dhai*.
(3) Fruit should be added to the above diet.

20. COL. CAMERON spoke as under:

"I had the pleasure of discussing the anaemia of ankylostomiasis with Castle and Rhoads, before they published their paper on the Puerto Rico experiments. They went to Puerto Rico, expecting to find an anaemia due to deficiency of absoption of specific factor; they returned convinced that it was due to blood loss, and could be cured by a diet adequate in protein and iron. This leads me on to the consultant physician's hobby-horse—a sufficiency of protein, animal protein, in the diet. Chittenden's original experiments pointed one fact—that the minimum was not the optimum. We must strive for an optimum supply and not be satisfied wih the minimum we are offered. "A man's grasp must exceed his reach, or what's a heaven for?" asks Browning. We of the hospitals must accept part responsibility for many cases. Often hospitals offer too low diets and too often believe that, in giving chicken essence ampoules, they are doing all that is necessary for the patient. We must get rid of this idea—chicken essence ampoules do not provide food. We are taking steps to get rid of this, by obtaining no further supplies of these ampoules.

Deaths have occurred from transfusion in anaemia cases. Care must be taken in the rate of administration; these patients must be treated like cardiac cases and given blood at half the rate used in normal circumstances. I am against the small, non-repeated, cocktail transfusion.

Major Ellis emphasised an important point. We must always keep in view, in our treatment, the need for the earliest possible return of the patient to the Unit, and adopt any measure to expedite this.

I agree with Major Craddock in his views on iron therapy. But where, I wonder, are we to get all the potent liver extract?"

21. COL. SCHLESINGER asked whether he had heard Lt.-Col. Thomson correctly. He had implied that iron therapy would rectify the anaemia caused by ankylostomiasis, even without deworming. Surely that was not the case. The aim should be to improve the anaemia first, if it were severe, then deworm, and then continue with iron therapy until the blood count became normal.

7th FEBRUARY, 1944—AFTERNOON SESSION

The meeting closed at 13.15 hours and was resumed at 14.30 hours, with **COL. CAMERON** in the Chair.

NEUROLOGY

22. BRIG. McALPINE—"*Common Neurological Conditions in India Command*" **(P)**

In some medical minds, the word "neurology" conjures up a picture of student days and of anxious moments before certain examinations. Consequently, there may remain an impression that all matters neurological are difficult. It is true that certain aspects of this subject can only be tackled by someone who has had specialized training. However, in India Command, the average neurological case is straight forward and requires common sense rather than theoretical knowledge of anatomy for its elucidation. Therefore we rely on you, as medical specialists, to tackle these cases. There is now a Neurological Centre at 126 I. B. G. H. Poona, to which difficult and obscure cases should be referred.

There is a tendency today, in medicine as a whole, to place too much reliance on the results of laboratory tests and X-ray examinations. A carefully taken history and a systematic examination, especially in a neurological case, should always form the real basis of your diagnosis. I wish to emphasise the value of history-taking. This should cover, not only the events of the illness in chronological order, but also provide information as to the type of individual. It is not difficult to sum up an individual by means of certain questions—What was his school record? What jobs did he hold as a civilian? What interests did he have outside his work, especially in the social sphere? How has he adapted himself to army life? Has he been in action? if so, with what effect? Is he happy in his present unit? The answers to these questions and the manner in which the patient gives them, will usually help you to decide whether the patient belongs to the category of the "neurotic sick". A common cause of prolonged hospitalization is failure on the part of an M. O. to make up his mind about a case, in which the symptoms suggest organic disease, but the results of examination are negative or inconclusive. If, in such a case, the M. O. spent an extra quarter of an hour assessing the type of individual, the information so gained, would often enable him to come to a conclusion about the case at a much earlier date and so would reduce considerably, the time spent by the patient in hospital. Brigadier Bennet has informed me that he has instructed psychiatrists to impress on M. O's, the importance of this part of the history and, from my own experience during this war, I can assure you of its value. 90 per cent. or more of neurological cases in Indian Command fall into one of six categories: meningitis, acute poliomyelitis, peripheral neuritis, head injuries and their after-effects, epilepsy, and, lastly, vascular accidents.

Meningococcal Meningitis—During the first eleven months of last year, 1,750 cases occurred in India Command. This is a small number, considering the size of the Army in India, and it speaks well for its hygiene. Colonel Schlesinger informs me that 800 cases occurred in Central Command during the same period, nearly 50 per cent. of the total number. This is understandable, in view of the large number of recruits in the Command. The mortality in Central Command was 18 per cent. I have no figures for the other Commands or Armies. This figure is double what it should be and must be reduced. Colonel Schlesinger has made certain recommendations and these have been included in a recent Technical Instruction on this subject. He believes that the main reason for this relatively high mortality is a failure, on the part of the I. O. R. to report sick until the disease is well established. Unit M. O's. and, in the case of units without M. O's. the officers of the unit and personnel of M. I. rooms, must be on the watch for the man who becomes ill with headache, fever and vomiting. Such a case must be brought to hospital at once. Lastly, we now have in India, small supplies of sulphadiazine, the most potent of the sulphonamides in the treatment of meningitis. The supply for this Command will be controlled by Col. Schlesinger. It will only be used in cases of pneumococcal meningitis and in other types of meningitis that have resisted treatment with sulphapyridine.

Acute Poliomyelitis—Fortunately there was no epidemic during 1943, in India Command. No case has been reported in an I. O. R., presumably because Indian populations are well salted in childhood. The mortality in British personnel was between 15 and 20 per cent. 50 per cent. of those that survived shewed more or less serious residual paralyses. Of late,

attention has been drawn to the intestinal tract, as a portal of entry for the virus. This raises the question as to whether, in India, such conditions as dysentery, typhoid, or infective hepatitis, may occasionally pre-dispose to the disease. I have notes of several such cases and would be glad to hear of further ones. As you know, convalescent serum, given in the pre-paralytic stage of the disease, has had a good trial in America and Australia; the results are not encouraging and therefore this form of treatment is not advocated. Rest in bed and correct posture for the paralysed limb are the essential points in treatment during the first few weeks. Lastly, the question of the attributability of this disease to military service, is now under consideration.

Peripheral Neuritis—85 per cent. of cases of polyneuritis in B. O. R's. in India Command, are due to diphtheria. I have not heard of a case of diphtheritic polyneuritis in an Indian. 40 per cent. (approximately) of diphtheritic cases follow cutaneous infection. You should make yourself familiar with the appearances of cutaneous diphtheria and its varied forms. In about half the cases of polyneuritis following cutaneous infection, a history of local neuritis can be obtained. This is usually sensory in type and is characterized by numbness and sensory loss around the lesion. Never fail to ask a patient with polyneuritis of uncertain origin, this question. "Did you notice anything wrong with your eyes, before your legs were affected?" In over 50 per cent. of diphtheritic cases, the answer will be, "Yes, my eyes seemed blurred when reading", or words to that effect. Remember that this symptom may only last a few days and, on examination, no actual paresis of accommodation may be detected. Lastly, I would stress the great importance of early diagnosis and treatment of diphtheria, since, the longer that serum treatment is delayed, the higher the incidence of paralytic sequelae.

Accidental Head Injuries—The incidence of this type of injury is fortunately lower than in certain other theatres of war. I hope that you have all established a liaison with your surgical colleagues in the treatment of these cases, which, as a rule, present a medical problem, rather than a surgical one. As you know, the whole outlook on head injuries has been materially altered during the present war. It has been found that, in the Army, reassurance about such symptoms as headache and giddiness, coupled with early rehabilitation, results in a return to duty of a relatively high percentage of cases. Headache is a common symptom in India—the patient often gives a history of a head injury and attributes the headache to it. It is up to you to dispel this bogey, by correctly handling head cases in the first week or two after the injury.

Vascular Accidents—I have in mind, two types of lesion—the one arterial, and the other venous.

(*a*) *Arterial Cerebral Thrombosis* is a well-recognized condition, and, in men of military age, is not uncommonly due to a syphilis. We have seen hemiplegia of sudden onset in I. O. R's. in whom syphilis could be excluded. A few of these cases came on after malaria, not necessarily of the cerebral type; in some, there was an interval of one or two weeks, before signs of hemiplegia appeared. Perhaps Major Craddock will be able to tell us about these cases They require further study.

(*b*) *Thrombophlebitis of Cerebral Veins;* recently attention has been drawn to this condition, more particularly in women during the puerperium. Other conditions that may give rise to it are severe infections, such as typhoid, septicaemia, etc. Thrombosis of a cerebral vein interferes with the function of that part of the cortex which it drains, and clinically results in a fit or repeated fits and loss of function, such as a hemiplegia. In some cases, thrombosis may involve the superior longitudinal sinus and, by preventing the absorption of cerebrospinal fluid, may lead to a rise of intracranial pressure. The clinical picture of cerebral thrombophlebitis is now easy to understand, e.g. an abrupt onset of headache, one or more fits, mental confusion or coma and, in some cases, a hemiplegia. Papilloedema may follow, if the superior logitudinal sinus is thrombosed. The cases may resemble an abscess or a tumour. Major McGregor (No. 2 Neurosurgical Unit) has studied a few cases which passed through his unit during 1943. One of the early cases was explored. Nothing was found except a tense brain and a thrombosed cerebral vein. The prognosis is quite good in most cases. Raised intracranial pressure should be treated by repeated lumbar puncture, but here I must issue a note of warning. Unless you are certain of the diagnosis, it is dangerous to carry out repeated lumber puncture when intracranial pressure is high. I would

suggest that you transfer a doubtful case to 126 I. B. G. H. Poona. In contrast to the hemiplegia following arterial thrombosis, that which may accompany venous obstruction clears up quickly. Bear this condition in mind, as an explanation of certain cerebral complications met with in febrile state and which, up till now, we have thought of in terms of arterial thrombosis.

We are hoping to investigate clinically and pathologically the cerebral sequelae of heat stroke. As you know, sequelae sometimes follow this condition, the two commonest being a cerebellar picture and dementia of varying degrees. Notes of such cases are requested.

Lastly, Col. Schlesinger has asked you to let him have notes on any unusual neurological case. By piecing together evidence, from notes on single cases, we may be able to learn something about some of the neurological problems that exist in this country.

23. MAJOR CRADDOCK—"*Plasmodial Encephalitis*" (P)

The object of this paper is to describe certain cases of malaria, complicated by lesions in the central nervous system, to discuss the possible pathological causes of the symptoms and signs encountered, and to make certain suggestions regarding their prevention and treatment.

Clinical Manifestations.

(*a*) *Involvement of the pyramidal tracts*—Of 13 cases admitted to No. 1 Burma General Hospital, 8 showed pyramidal signs, and of these, 5 died, 2 recovered completely, and one recovered but was left with persistent hemiplegia. The earliest signs are, spasm of the jaw, diminished voluntary power with increased muscle tone and tendon reflexes, loss of abdominal reflexes, sphincter disturbances, and, less commonly, ankle clonus and extensor plantar response. Other signs, which may have the same origin, are dysphagia and dysarthria, and the fully-developed picture may be that of quadriplegia, hemiplegia, paraplegia, or monoplegia. All these types have occurred in our series, but only three cases will be described at this point.

Case 1. *Cook, aged* 21—

C/o fever and rigors. After admission vomited and became unconscious. O. E. Cheyne Stokes respiration. Pupils moderately dilated, unreacting. Fundi normal.

Spastic contraction of masseters. Increased muscle tone in upper limbs, diminished in lower limbs.

Abdominal reflexes and knee and ankle jerks absent.

Marked and sustained ankle clonus. Plantar response not obtained.

Retention of urine present. Became very jaundiced.

Spleen large and soft. B. P. 90/65. Blood smear M. T. rings. Lumbar puncture: Fluid clear and not under increased pressure (105 mm).

Given intravenous quinine and glucose and rectal hypertonic mag. sulph. after which he recovered consciousness but quickly relapsed and died.

Autopsy. Petechial haemorrhages scattered throughout white matter but concentrated into a dense pattern in the internal capsules on both sides. Microscopy showed the capillaries closely packed with M. T. parasites.

Case 2. *Rifleman, aged* 25—

C/o fever and rigors on alternate days at intervals throughout the preceding 2 months.

O. E. Extremely restless. Spleen palpable and soft.

During examination became semi-comatose and incontinent of faeces.

Clasp-knife spasticity of both lower limbs.

Abdominal reflexes absent on the right side.

Sensation to pinprick absent over the whole body.

The next day both upper limbs were weak and spastic and the day after, the spasticity of the lower limbs had passed off but voluntary power was still lacking. Full power returned 2 days later and on discharge 2 weeks afterwards the C. N. S. was normal.

Treatment was intravenous quinine.

(*N. B.*—This was the only case in which parasites were not demonstrated, and the diagnosis was malaria, clinical).

Case 3. *Signalman, aged* 20—

Suddenly fell unconscious on parade. On recovery, found his legs stiff and useless and could not open his mouth or speak.

O. E. Conscious. Temperature normal.

Very marked spasm of jaws. Unable to speak or swallow and unable to write. Neck stiff.

Lower limbs: no voluntary power and very stiff. Excruciating pain if attempts are made to bend them.

M. T. rings in blood. Given anti-malarial treatment and slowly recovered.

One month after admission onset of frequent attacks of vertigo and tinnitus and transient unconsciousness. No fever.

M. T. rings again found in blood. Second full course of treatment given.

A month later examination showed spastic paresis of left lower limb with impairment of all forms of sensation over the left half of the body. Partial deafness of the nerve type was also present on the left side.

This patient orginally had a bilateral pyramidal lesion and was finally left with a monomplegia and a hemi-anaesthesia, possibly due to a lesion of the posterior limb of the right internal capsule, with involvement perhaps of the thalamus.

At this stage it will be interesting to consider the mode of production of the dysarthria and trismus, which were a marked feature in this and many other cases. From the cortex, the motor fibres concerned in articulation, pass downwards in the pyramidal tracts and, after decussation, end in the trigeminal and facial nuclei, the nuclei ambigui and the hypoglossal nuclei in the pons and medulla, whence the lower motor neurones run in the corresponding cranial nerves to the lips, soft palate, tongue and larynx. The articulatory muscles on each side, appear to be innervated by both cerebral hemispheres. Hence, a unilateral pyramidal lesion does not produce permanent dysarthria. Dysarthria is, however, produced by bilateral pyramidal lesions. Following such lesions, the articulatory muscles are weak and spastic and the tongue appears smaller and firmer than normal and the jaw jerk is exaggerated. (Brain, 1940).

(*b*) *Coma* is probably the most frequent of all manifestations, but, as it is probably an expression of a generalized cerebral involvement and common to encephalitis of all types, I will not discuss it at length. The case histories showed that coma may terminate in four ways—

(i) Death without recovery of consciousness (Case 9).

(ii) Death after a lucid interval (Case 1).

(iii) Recovery but with persistent residual paralysis (Case 3).

(iv) Complete recovery (Case 2).

(*c*) *Mental Disorder*—Delirium and great mental excitement are not infrequent. One of our patients showed a more unusual picture, namely an alteration in behaviour and character.

Case 4. *Ambulance sepoy, aged* 30—

During the night, the patient approached one of the Sisters as she was returning to her tent. He looked queer and acted very strangely, his eyes had a fixed and wild appearance and his speech was muttering and unintelligible. He approached the Sister in a menacing way and she believed that he was about to assault her. She called for help and he was placed under arrest. The next morning his behaviour was still very strange and he was kept in bed with difficulty, though his temperature was 102 and rose rapidly to 104. At this point, generalised epileptiform convulsions supervened and several fits occurred in rapid succession, followed by deep coma. Very rapid shallow respirations were present and the pulse was 160. There was conjugate deviation of the eyes to the left and the pupils were pin-point and unreacting. The patient rapidly succumbed after an intravenous injection of quinine, which was succeeded by a further fit.

At autopsy there was gross oedema of the brain, which was bulging on section, and the histological report on the brain tissue was "Section of brain shows peri-capillary transudation of serum. The neurocytes stain badly. Schizonts and gametocytes of M. T. parasites found in the capillaries."

(*d*) *Convulsions*, both generalized and of Jacksonian type are common. I mention them merely to draw attention to the suspicion which should attach to fits occurring in a patient without an epileptic history, especially if status epilepticus should supervene, as in the case just quoted.

(*e*) *Focal Lesions in the C. N. S.*—Hyperpnoea is one of the earliest signs of central respiratory disturbance. It precedes the more familiar signs, namely, sighing respiration, Cheyne Stokes rhythm, etc., and should always arouse suspicion of cerebral involvement in a just quoted.

Case 5. *Sepoy, aged* 21—

C/o fever for 4 days. B. T. parasites found in the blood. The patient was very ill, with extremely dry furred tongue, but no cerebral symptoms occurred until 6 days after antimalarial treatment was begun, when the patient passed urine into the bed.

O. E. The patient lies vacuously in bed with the mouth drooping open and the face devoid of expression, resembling encephalitis lethargica. He is quite unable to speak, but the jaw is flaccid.

The pupils are unequal, but react.

Loss of voluntary power in the left lower limb, which is slightly spastic with exaggerated tendon jerks. The plantar response could not be obtained on the left side. The left cremaster reflex was diminished bu the abdominal reflexes were normal. No sensory changes.

Four days later, speech began to return and facial expression was gradually recovered. A month later he was convalescent, but there was still evidence of bladder and bowel dysfunction. The bladder could become distended to the umbilicus without arousing a desire to micturate, and the bowel was choked with scybalae. When finally discharged, he was completely normal.

This patient had a true aphasia, probably due to a lesion in Broca's area. In addition, he had an extra-pyramidal syndrome, due to a lesion of the corpus striatum, which has also been described in meningococcal encephalitis (Banks & McCartney, 1942). I have not seen the established Parkinsonian syndrome following malaria, but there does not seem any reason why it should not occur. Vertigo and tinnitus, which occurred in Case 3, are probably to be attributed to a lesion of the tegmentum of the pons, rather than a peripheral neuritis. This patient was left with persistent partial deafness. Other focal lesions are indicated by signs described in the case histories.

(*f*) *Meningism* was present in 5 out of the 13 cases already mentioned. In two, the C. S. F. was normal, in the third r. b. cs were present and in the last two, xanthochromia. In one of these, a fibrin clot appeared on standing and tuberculous meningitis was suspected, but jaundice and splenomegaly were present and M. T. rings were demonstrated in the blood (Case 10). The resemblance to tuberculous menigitis was further enhanced by the presence of gelatinous exudate over the base of the brain at autopsy. Malarial meningo-encephalitis is, therefore, one of the conditions in which Froin's syndrome may occur. The haemoglobin and the increased protein are, presumably, due to haemorrhagic aseptic meningitis, or to capillary rupture into the subarachnoid space.

Diagnosis—It is a safe rule that acute central nervous disorders, which cannot be attributed with certainty to any other cause, should be regarded as protozoal and treated accordingly. The absence of fever and of malarial parasites from the blood, are not safe criteria in excluding malaria and should not be relied upon nor allowed to delay treatment. Neither should the presence of B. T. parasites in the blood be allowed to induce a false sense of security; malignant tertian parasites may be lurking in the recesses of the brain. Reliance should rather be placed on the predominance of signs above-mentioned, if associated with a suggestive history, and splenomegaly.

Differential Diagnosis—The following are conditions which we have actually had to consider and exclude, in coming to a decision regarding cases finally diagnosed as cerebral malaria, (or as one of the conditions specified*).

(i) Meningitis from all causes, including tuberculous meningitis.

(ii) Coma from all causes, including alcoholic poisoning, encephalitis lethargica*, and diabetes and heatstroke.

(iii) Acute disseminated encepho-myelitis, including post-vaccinal encepho-myelitis*.

(iv) Tetanus.

(v) Subarachnoid haemorrhage.

(vi) Apical pneumonia with mental excitement and confusion*.

Lumbar puncture is a *sine qua non* in differentiating many of these conditions; when a protozoal cause cannot even then be excluded, the patient should be given the benefit of the doubt by the institution of anti-malarial treatment.

The Prognosis is always grave and is adversely affected by pre-existing anaemia, by debilitating conditions like dysentery, and by co-existing infections like pneumonia, which is a very frequent complication. Delay in hospitalization or in any treatment, has an unfavourable influence on the outlook. A persistently low blood pressure is of grave import, as also is widespread involvement of the nervous system, though remarkable powers of recovery are sometimes shown.

Discussion of Pathology—Plasmodium falciparum sporulates in the internal organs and it is not surprising, therefore, that organs with a dense vascular bed, such as the brain, should sometimes suffer heavily. The main pathological features are familiar and are depicted in the diagram below, which attempts to show why the cerebral infection carries such grave menace to life. This, in my opinion, is because suitable conditions exist for a progressive diminution in the supply of oxygen to the brain, the cells of which are notably susceptible to the effects of anoxia. In the brain itself, a vicious circle is established, leading to what may be termed a local or dissociated anoxaemia. In the rest of the organism, the effects of the protozoal invasion result in a generalised anoxaemia, which still further impovershes the devitalised nerve cells. If the anoxia be sufficiently severe and prolonged, irreversible changes will take place, death or paralysis resulting, according to the site and extent of the lesions.

Treatment—It is obvious that once irreversible changes have taken place in vital areas of the brain, the patient is beyond human aid. It cannot be too strongly emphasised that it is impossible to predict whether a given malaria patient is or is not likely to develop encephalitis, and the only safe rule, therefore, is to regard every patient diagnosed or suspected to be suffering from malaria, as a potential case of protozoal encephalitis, and to concentrate on preventing the onset of this complication. A study of cases reveals that encephalitis is mostly to be feared in patients whose fever is imperfectly controlled. This may be due to (*a*) delay in treatment, (*b*) failure to ensure that quinine is administered and absorbed, or failure to give by appropriate routes or for a long enough period. The former may be due to attempts to establish a diagnosis, owing to the official dislike of "Malaria clinical". Insistence on an exact scientific label always involves the danger of delay, a danger which is further enhanced by the tendency of pernicious infections to be "M. P. negative". To counteract this, I often repeat the slogan "A live clinical is better than a dead M. T." This danger is rightly emphasized in the introduction to the Blue Book on this subject. Another prevalent cause of delay is the practice of transferring cases to special malaria wards, which, though good in itself, demands that special precautions be taken to ensure that treatment is not withheld because of the transfer. As regards (*b*), when fever remains after two days of quinine, it is wise to continue quinine intravenously, combined with intramuscular mepacrine. Another practice which we have adopted, is to give intravenous quinine to all malaria patients whose temperature on admission exceeds 104. I have the impression that this has been of value in preventing the onset of pernicious manifestations.

For the treatment of encephalitis, when it occurs, I think that dehydration is of value, when the onset is sudden and overwhelming. There is some reason to believe that, in these cases, cerebral oedema may be responsible for the coma and, if this can be overcome, anti-malarial treatment may be effective in combatting the underlying encephalitis. Accordingly, I give intravenous hypertonic saline (15 per cent.) and glucose (50 per cent.), plus rectal mag. sulph. 6 ounces of a 50 per cent. solution, until consciousness is restored. In cases, however, where encephalitis supervenes on a severe malarial illness, dehydration is already severe, owing to toxaemia and I am, therefore, more inclined to give isotonic infusions and to give the first pint very quickly, to restore the blood pressure and thereafter a continuous drip for so long as considered necessary. To this drip should be added glucose and quinine, though the first intravenous quinine should have been given before this. The sheet-anchor of treatment is still intravenous quinine and, in a condition which has such a high mortality, you are very unlikely to kill the patient with quinine, but much more likely to kill him with insufficient quinine. Three doses of 6 grains can safely be given and even more if necessary, though the chances of success, if the first three have failed, are small. The interval between the early doses should be short, if the indications are urgent. Other measures aimed at combating the anoxaemia, e.g. oxygen and blood transfusion, may be of value, though I have not been able to give the latter a sufficient trial. In the absence of hypotension, the patient may be nursed in the sitting position, to promote drainage from the brain.

Further Case Histories

Case 6. *Sepoy, aged* 45—

Admitted with right lower lobar pneumonia. Treated with sulphpyridine (total gm. 68) for 19 days by a zealous Medical Officer. Fever recurred on 25th day and cerebral signs appeared on 30th day, when patient died. The signs were—ptosis of left eyelid, aphasia, absent abdominal reflexes and knee and ankle jerks, and meningism.

Autopsy : Brain congested and oedematous, petechial haemorrhages in temporal and frontal lobes M. T. parasites in capillaries. Grey hepatisation of the lower lobe of the right lung.

Case 7. *Havildar, aged* 40—

M. T. and B. T. parasites in blood. Treatment started on 7th day of illness. On 10th day, spasm of jaw and difficulty in articulation. Diminished abdominal reflexes. Marked hyperpnoea. Neck muscles tender. Frequent twitchings of facial muscles. Died with hyperpyrexia. Post-mortem refused.

Case 8. *Sepoy, aged* 25—

Infected at same source as Case 7. M. T. rings in blood on 4th day of illness. Pneumonia with leucocytosis on 7th day. Given M & B gm. 21. On 10th day spasm of jaw, restless movements of arms, generalised rigidity, stiff neck. C. S. F. yellow. Rapid deterioration. Before death massive haemorrhages in skin of buttocks and petechiae in axillae. Post-mortem refused.

Case 9. *Sepoy, aged* 31—

Infected at same source as Case 8. B. T. rings. On 3rd day of treatment fever uncontrolled and became delirious and unconscious. Died suddenly.

Autopsy: Haemorrhages in visceral pericardium and pleurae. Brain smear showed capillaries packed with M. T. parasites.

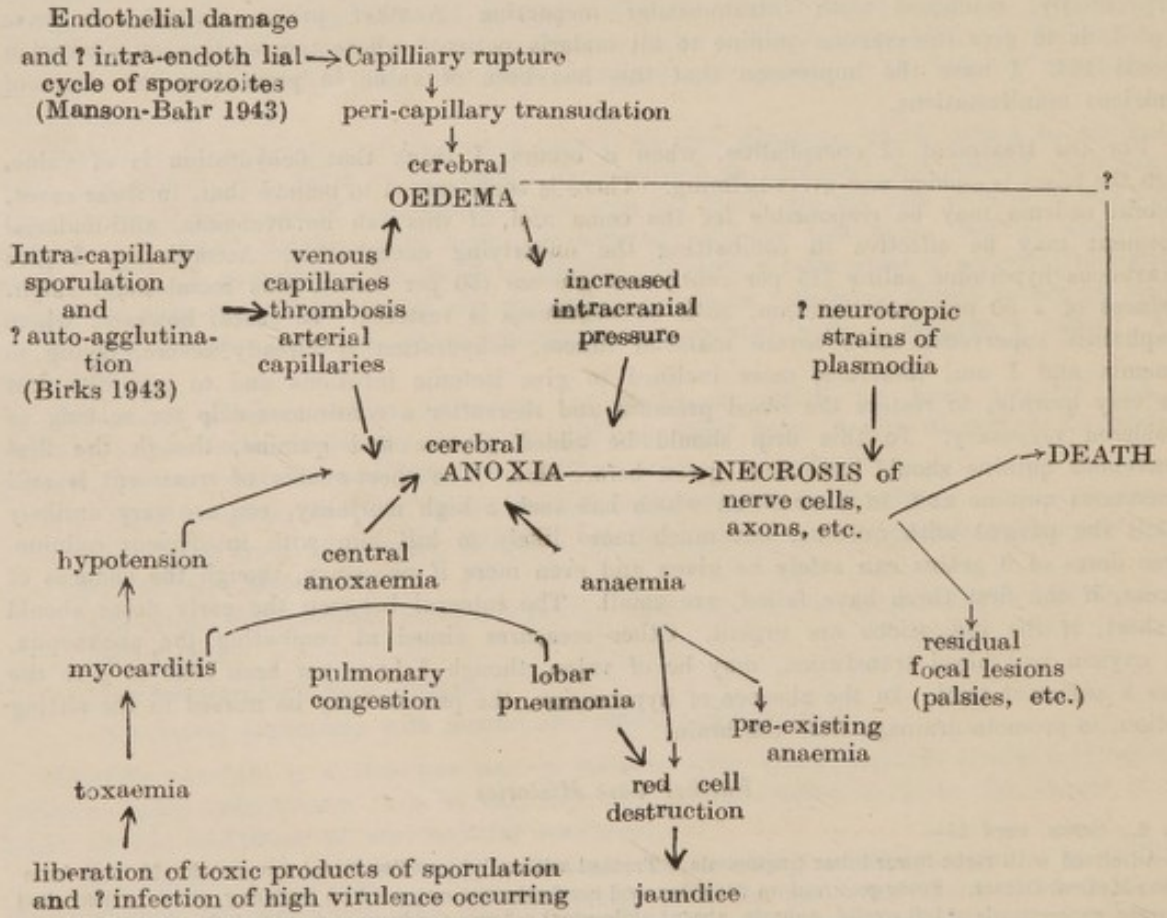
Case 10. *Sepoy, aged* 22—

Vomiting, headache. Spleen enlarged, jaundiced. M. T. rings. Developed retention of urine, stiff neck restless. 2 days later cerebral and meningeal irritation. Right plantar response extensor. C. S. F. pressure 170 mm, straw yellow, opalescent. Next day, C. S. F. pressure 220 mm, straw yellow. Fibrin clot formed. 3-4 lymphocytes/cmm, large nos. of rbcs. No M. P. in rbcs. Retention of urine persisted. Cold sweats and sighing respiration before death. (Autopsy: Meninges very congested, gelatinous exudate over base of brain. Report on brain sections not received.)

Case 11, *Naik, aged* 25—

Headache, high fever, pain in back, marked congestion of conjunctivae. Blood negative for M. P., C. S. F. clear, no pus cells. Died 8 hours after admission. Autopsy: Petechial haemorrhages in skin and in all internal organs, some confluent. Cloudy swelling of heart, liver and kidneys. Brain, oedematous, convolutions flattened; smear from surface showed gram-negative intracellular diplococci. Pneumococci were grown from blood taken from the heart. Report on sections:—brain—capillaries are dilated and contain thrombi, moderate degree of pericapillary transudation, schizonts and gametocytes of M. T. parasites present; suprarenals—extensive medullary haemorrhages.

The Diagram referred to under Discussion of Pathology



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DISCUSSIONS

24. LT.-COL. KELSALL said, "It is taught in West Africa, that in cerebral malaria, although capillary blood may show no parasites, a film from the venous blood is always positive. Is this the opinion of the members of this Conference?"

25. MAJOR GUEST enquired whether a patient, suspected of cerebral malaria, could have a normal or a slightly raised temperature at the time of examination; and whether paralysis

of lower motor neuron type, recently described as 'infective neuritis' could be ascribed to malaria. Several cases with this type of paralysis had been seen in patients who had received malaria treatment a few months previously.

26. COL. SCHLESINGER said that he had listened with the greatest interest to Major Craddock's paper and particularly to his description of mental symptoms associated with a severe M. T. infection. He asked whether there was any way of differentiating mental changes occasionally arising after mapacrine therapy and those which the speaker had described as being due to the malaria infection itself.

27. COL. TAYLOR said, "May I ask what are the views on the use of concentrated solutions of glucose, saline, to produce dehydration of the possibly oedematious brain, in cerebral malaria? The situation is difficult, because the general circulation may be dehydrated and depleted of salt, often the result of long journeys in ambulances, and yet, the brain in certain cases of cerebral malaria, may show some oedema. The pressure of the cerebro-spinal fluid is not necessarily a guide, as some post-mortems show oedema of the brain in cases in whom the manometer showed a low C. S. F. pressure; oedema is found in a minority of cases at autopsy. Dehydration of the general circulation is a very common and serious finding in the hot weather. To my mind, the general dehydration and salt depletion need treatment first. It is a subject needing clinical and pathological research."

28. MAJOR CRADDOCK, in his reply, said that (i) he would prefer to refer that question (para. 24) to a malariologist; (ii) he had not yet seen any case of palsy of a lower motor neuron type which could be conclusively attributed to malaria (para. 25); (iii) in the only two cases he had seen, the malaria had been controlled when the mental syptoms appeared during the mepacrine course, complete recovery occurred and it was assumed (but not, of course, proven) that they were due to mepacrine and not to malaria (para. 26); (iv) this question (para. 27) was covered by the treatment section of his paper, which there was not time to read at the Conference. (Now fully reproduced. *Ed.*)

29. BRIG. McALPINE said, "Major Craddock must be congratulated on the way in which he has tackled the neurological complications of malaria. There are just two points on which I should like to comment. The nature of the pathological changes in the brain, in cerebral malaria, are far from being settled. During a recent visit to No. 20 U. S. A. G. H., I was shown sections from 8 cases. In about half of these, there was remarkably little in the way of oedema or capillary changes. We want to learn more. D. D. H. & P. has made arrangements, by which neuropathological material will be examined at 126 IBGH, Poona. Ask your pathologist to send brain and spinal cords from this and other conditions, in formalin, in a suitable container. A report on the examination will be sent by the neuropathologist at that hospital. Secondly, I would suggest to Major Craddock that, in view of the lack of uniformity of the changes in the brain in cerebral malaria, it would perhaps be wise to forego the attempt at localisation of the varied signs, met with in this condition."

30. LT.-COL. ROGAN said, "The question has been raised whether coma can occur in afebrile cases of cerebral malaria. The answer is—undoubtedly. In areas where M. T. malaria is prevalent, cases of cerebral malaria may not infrequently be admitted to hospital, in a state resembling surgical shock, with a subnormal temperature. Later, as the condition improves, pyrexia usually develops. Regarding the possibility of cerebral malaria occurring with negative blood slides, it is the opinion of most physicians who have had extensive experience of malaria, that this can occur, but very rarely. In the great majority of cases, parasites can be found with careful and repeated search, especially if the thick drop technique be applied. With regard to mepacrine psychosis, it should be realised that this condition is very rare and that the majority of cases thus diagnosed in forward areas, have, on investigation been shown to be suffering from cerebral symptoms, arising from inadequately treated M. T. malaria. Mental aberration due to M. T. malaria is not at all uncommon in hyperendemic areas and is always an indication for energetic treatment.

31. LT.-COL. KARAMCHANDANI mentioned two cases:

Case 1. One I. O. R., showing signs of acute confusional insanity, was brought at 20·00 hrs. to I. M. H., Pishin (Baluchistan); he was violent; with temperature normal; did not sleep during the night; on the following morning, he was reported to have received corporal punishment to quieten him and the blood examination showed heavy infection

with M. T. parasites; highest temperature, 99°F; was successfully treated with quinine and plasmoquine.

Case 2. One B. O. R. reported at B. M. H. Ferozepore, with signs of polyneuritis of both lower limbs; normal temperature; after 2 days temperature rose to 102°F and the blood slide showed malaria parasites; the signs cleared up without anti-malaria treatment.

32. MAJOR DAVAR said that he had seen cases of cerebral malaria with a temperature of 99°—100°F, and described his first case of afebrile cerebral malaria. A sepoy was admitted in an unconscious state, with a note ti say that he had been sick the previous evening, had a convulsion, and vomited blood in the morning and become unconscious. On examination, his pulse was imperceptible, temperature 97°F, spleen three fingers below costal margin. He was given I. V. quinine—gr. 6 immediately; blood slide showed M. T. parasites; another I. V. quinine—gr. 6 with a pint of glucose, saline followed; the result of an enema showed large quantity of dark blood; catheterised urine was clear; developed extensor sign on one side on the same evening; a third I. V. quinine was repeated; during the night he vomited a large quantity of blood in bed and, on the following morning, showed extensor sign on the other side. C. S. fluid was clear; Total W. B. C. 21,000 per cmm, Poly. 70 per cent., Lympho 28 per cent. and large Mono 20 per cent. Subsequent blood slides were negative to M. P. Patient regained consciousness on the third day, but his mental condition was not quite clear. Mental symptoms and abnormal behaviour were frequent in malaria patients.

33. COL. CAMERON, in winding up the discussion, said :

" In the U. K., syphilis is regarded as the great imitator of diseases: in India, malaria takes its place. This is certainly so in neurology and, in many cases, cerebral malaria may be either the sole cause of neurological signs and symptoms, or an associate of some neurological condition. Where M. T. malaria abounds, it is a sage rule to treat malaria until its absence is proven in such cases. The remarks by Lt.-Col. Kelsall have been borne out in my discussion with R. A. M. C. officers from West Africa. They state that parasites can always be found in venous blood in cerebral cases—but they do not withhold treatment in suspected cases if the venous blood is negative. I. V. quinine is, of course, the treatment. It is frequently given in glucose, saline, by drip. In adopting this measure, one must beware of failing to build up a sufficient concentration early. An initial I. V. injection, given slowly, followed by drip administration, is probably the better procedure. We must beware of the combination of meningococcal meningitis and malaria; a lumbar puncture is advisable in the large number of cases of cerebral malaria."

TYPHUS

34. COL. TAYLOR—"*The Incidence of Diseases of the Typhus Group in India*" **(P)**

Definition of Typhus Group—

Until recently, the name 'typhus fever' has been used only for an epidemic continued fever of a clear-cut clinical epidemiological type. During this century, there have been descriptions from many parts of the world, of diseases, given many names, akin to classical typhus, but differing in being primarily diseases of lower animals, which are not conveyed from man to man, but from animals, mainly from rodents, by ticks, mites and fleas. In their clinical features, causal organisms and pathology, they are very closely related to epidemic typhus, but, being only incidentally conveyed to man, they are non-epidemic sporadic place diseases. They may be defined as "a group of febrile diseases of varying severity and clinical manifestations, caused by micro-organisms of the genus Rickettsia and transmitted to man, mainly, if not entirely by the agency of insects." (*Napier.*)

Rickettsia are small micro-organisms, incapable, as far as it is known, of existing in the absence of living cells, but visible to ordinary miscroscopic examination in stained films and held back by the ordinary bacterial filters. There are at least three morphological distinct types, a bacillary form, a diplococcal form and an indefinite form, corresponding to the three types of endemic typhus.

Brief History of Typhus—

Epidemic typhus is a disease of great antiquity, and some of the cases of epidemic fever, described by Hippocrates, were undoubted typhus. There were frequent outbreaks, associated with wars and famines, reported wherever records exist. The name 'typhus' came into use

about 1800. In 1837, Still and Gerhard independently showed that typhoid was a separate disease. Lind, an East India Company surgeon, had found in the 18th century, that the infective agent of ship typhus sticks to the garments of patients and those who come into contact with them. He therefore ordered doctors and nurses to change their dress before leaving a ship or hospital harbouring such patients, and to burn everything worn by them. In 1909 Nicolle showed that lice were a vector, by producing the disease in a chimpanzee, by subcutaneous inoculation of blood from a patient, and then transmitted the disease to monkeys, by the bites of lice which had fed on the infected chimpanzee. In 1910, Ricketts first described pleomorphic organisms, which now bear his name, on the bodies of infected lice. In 1916, Weil and Felix, using Bacillus proteus X 19, which had been isolated from typhus patients, worked out the test which is known by their names. Megaw, in 1917, drew attention to the existence of endemic typhus in India, by analogy with Rocky Mountain Fever, on which much work had been done in America. Fletcher, in 1927 and later Lewthwaite and Savoor in Malaya, described and separated two types of endemic typhus, scrub typhus, corresponding to Tsutsugamushi fever of Japan, spread by a mite, and endemic murine typhus, spread by fleas. Much work on similar lines had been done in Japan and Sumatra, in which Rickettsia were isolated and the relations of the vectors and animals reservoirs shown.

Recently during the war, epidemic typhus has flared up in Russia and Poland. Much work has been done in isolation of Rickettsia and in the preparation of vaccines. Rickettsia have been grown in the chorio-allantoic membrane of the developing chicken embryo (Zia 1934) and by Cox in 1939, who found that they multiply freely in the yolk sac of the fertile hen's egg. Castaneda grew them in the lungs of infected rats and mice in 1939. Agglutination reactions against Rickettsia and complement fixation tests have been developed, to identify the type of rickettsia, as well as animal reactions, such as the Neill-Mooser reaction in the guinea pig, and the reaction in the anterior chamber of the rabit's eye, following an injection of K strains of Rickettsia.

Classification—

There have been two main classifications. Megaw (1921—1930) after calling attention to endemic typhus in India, classified typhus according to the vectors, into epidemic louse-borne typhus, and non-epidemic typhus, sub-divided into fleaborne typhus, tick typhus and mite-borne typhus. The main objections to this classification, which has served a very useful purpose, are that often the vector is unknown and unproved, (in India, no vector has yet been proved to to carry any form of typhus) and, secondly, the same clinical type of typhus may be carried by various vectors. Felix has suggested a provisional classification, which is now in common use, based on the serological findings of the Weil-Felix reaction. There are three main serological types. Type OX 19 includes epidemic louse-borne typhus and the endemic murine flea-borne typhus. Type OXK includes Tsutsugamushi and the similar scrub typhus of Malaya, the Dutch East Indies and Australia. The 'Type Undetermined' shows a positive Weil-Felix reaction for OX2, OXK and OX 19. The Rocky Mountain Spotted Fever of America is the classical example, though similar fevers are found through all sub-tropical countries.

Thus, the classification in common use is X 19 type, sub-divided into epidemic and endemic, K type, and the undetermined or mixed type.

The Weil-Felix Reaction—

The common feature of the majority of the rickettsial diseases (Q fever is an exception) is that the patients show agglutination reactions, either with Proteus OX 19 or its variants OX2 or OXK. These strains of Proteus are neither the causative agents nor secondary invaders. The K strain of Proteus was a variant of X 19, taken to Malaya (Kingbury). Castaneda and Zia (1933) have shown that Proteus X 19 and Rickettsia prowazeki, possess a common antigenic factor, probably a carbohydrate.

In X 19 types of typhus, the reaction becomes positive after the first week in a dilution of 1/125 and, at the end of the febrile period, may be as high as 1/5000 or higher. In OXK strains, there are difficulties, which are often not appreciated, which can be summarised as:

(1) OXK titre often may not rise until about the third week of the disease and often not until much later. In one recent case, described by Luck and Parker in Calcutta, there was a significant rise from OXK 1/250 to OXK 1/500 on the 62nd day of the disease, and to OXK 1/1000 on the 66th day of the disease, the fever, having terminated on the 25th day.

Thus, cases dying in the first two weeks of this disease, may have a negative Weil-Felix, or the W. F. may be done only once, before there is a significant rise in the K titre, which may occur days or weeks after the temperature has returned to normal. In both circumstances, the diagnosis may be missed.

(2) A less common difficulty is a rise of X19 and/or X2 in the first two weeks of the disease, falling about the 30th day and rising in K at the 40th day. An instance of this is recorded by Lewthwaite and Savoor in rabbits and examples in patients have recently been seen in the Army in Bengal.

(3) The K response stays at a significant level for a short period, i.e. rarely more than 60 days, unlike most infections.

(4) False positive Weil-Felix reactions are reported in undulant fever, up to 1/240 (Kemp, Wright and Wayne 1933), and in chronic Brucellosis, in chronic Sinuses, in leprosy and in an Empyema sinus (Savoor) and in Rat-bite fever (Das Gupta), in a case of amoebic abscess (de Monte) and, possibly, in some cases of Trachoma.

(5) There may be a significant rise of the T. O. agglutins. In a case in the Ranchi 1943 outbreak (Birnbaum), the Weil-Felix, on the 15th day of disease, was OX2 34, OX19 2200, OXK 85, and the T. O. agglutination was recorded at 2560, having risen from 320 on the 9th day and falling to 640 on the 24th day. Thus, if the Weil-Felix reaction had not been done, the agglutination reaction would have suggested the enteric group. A title of 320 T. O. was recorded in 6 cases out of 20 cases of typhus of the mixed undetermined group, in this outbreak.

It is thus easily understood how cases may be missed or wrongly diagnosed.

The newly-introduced Rickettsial agglutination and complement fixation may help, as they are said to be more specific and to become positive earlier. As far as I know, they have not as yet been used in India. Van Rooyen and Bearcroft in 1943, published their experience with the agglutination rickettsial reaction in the Middle East. They recommend that it should be done in all suspected typhus cases, in addition to the Weil-Felix reaction.

Modern work on laboratory animals has helped to identify typhus fevers of varying names, in different localities. The main work is on the susceptibility and acquired immunity of guinea pigs, rabbts and mice, to rickettsal diseases. The Neill-Mooser reaction is the scrotal swelling in guinea pigs, resulting from intraperitoneal injections of rickettsia, containing blood of the endemic X19 or indeterminate OX2 type. The eye reaction of rabbits is the result if injecting K strains of Rickettsia into the anterior chamber of the eye, in which they multiply and produce an iridocyclitis.

Experience is showing many clinical variations with different strains of the same type of typhus, depending on race, colour and nutrition of patients. Apparently, the inhabitants of a district may be partially immune to the local type of typhus, which may be fatal to the newcomer. Modern work may well explain these anomalies. They are obviously important to the army.

Brief History of Work on Typhus in India—

There has been little original work in India, though all types of the disease appear to be widespread. Megaw's suggestion in 1917, that the disease from which he himself suffered was tick typhus of the Rocky Mountain Fever type, was based on the fact that he was bitten on the neck by a tick, 21 days before the fever began, and on clinical grounds. No serological or other work was done. The name "tick typhus" came into general use when Megaw later introduced his classification by vectors. But, it is not always appreciated, that in India the vector or vectors have not been isolated or shown conclusively to carry various types of typhus. Covell in 1936 and later, isolated strains of Rickettsia of X19 type in the Simla hills, from rat fleas, and isolated OXK strains from patients. He was not able to show conclusively how the disease was transmitted and urged that much further work was necessary. Savoor (private communication) has, I understand, recently isolated X19 murine and K strains of typhus from patients in Bombay. Heilig in Mysore, isolated a strain from cases resembling Rocky Mountain Spotted Fever, the sera of which were sent to Topping in America. The complement fixation tests showed a close relation to R. M. S. F. with an unexplained cross-reaction in low dilutions to endemic X19 typhus, which has only occasionally been recorded in R. M. S. F. Recently, in the Army outbreaks in Bengal, and Assam, Sayers and Parker have isolated in guinea pigs, rabbits and mice, four K strains in patients, on which Savoor in Bombay is now working. They show the characters of the rickettsia from Tsutsugamushi and Malaya scrub typhus, the K type.

The need for more work in India has continualy been stressed. Until this has been done, the differentiation of the various types of typhus in India will continue to be unsatisfactory, depending on clinical grounds and on the Weil-Felix reaction.

Brief History of the Occurrence of this Disease in India—

It is obvious from what has already been said, that reports of these diseases in India must be accepted critically. Undoubtedly, many cases have been missed and, when diagnosed, they have often been wrongly named.

Lind, the East Indian Company surgeon, mentioned above, was familiar with ship typhus in the 18th century. A possible epidemic is recorded in 1810, in Madras, which destroyed 106,000 people. There were said to be epidemics in the Nilgiris in 1831, and in jails in Agra, Meerut and elsewhere, during the Mutiny, also in the Yusufzai country and the jails of Peshawar and Rawalpindi in 1869. At the Indian Medical Congress of 1894, typhus was said to be endemic in the trans-Indus districts, from Baluchistan to the Yusufzai country, in Hazara and in the Himalyan hill tracts, especially Kulu. In 1905 and 1907 at Peshawar, Husband and MacWaters described over 120 cases in the 1st and 6th Mule Companies, on their return from Tibet. This outbreak led to the evacuation of their lines. They recorded that "the disease is well known in Kashmir, amongst natives, among whom it terminates in August, when there is an exodus from villages to higher pastures." A small outbreak was recorded in Peshawar jail in 1908. In 1913, McKecnie described cases of fever, resembling typhus, in the Kumaon hills and later, Megaw's interest, after suffering from the disease himself, led to reports of what was called 'tick typhus' from all over India. In 1924, an I.M.S. Officer, Maj. F. W. Cragg, was sent to Kashmir, to investigate typhus, and died here at Lahore from the disease, on his return. Boyd, in 1935, collected 110 cases from the Army records of 1934 and classified them serologically, according to Felix's classification, into type X19 (of which he considered there were two varieties, the Ahmednagar-Poona type and the Bangalore type), the OXK group, and the OX2 undetermined mixed group. It was then realised that all types of non-epidemic typhus were widespread throughout India. Covell, in 1936, isolated X19 murine strains of Rickettsia from rats in Kasauli and OXK strains from patients. Shortt, in Madras, collected a series of Madras cases. No further work of significance was recorded up till the war. It was thus thought that epidemics of X19 louse-borne typhus had occurred in the colder parts of India and that the endemic types were not uncommon in all parts of India. It was impossible to come to any conclusion as to the frequency of the disease in the civil population, because, owing to the lack of hospitals and laboratories, the Weil-Felix reaction was rarely done. In the teaching hospitals, a few cases diagnosed. In the army it was a sporadic disease and no outbreaks of epidemic proportions had occurred. In British troops, for the period 1933/42, there was an average of 29·6 cases each year and the Indian troop ratio per 1000 varied between 0·09 to 0·6 per 1000 per year (figures supplied by Lt.-Col. E. Hall).

Recent Outbreaks in the Army—

I will just describe the outbreaks in Eastern Command and Fourteenth Army for 1942/43, because they are the largest outbreaks of the endemic type yet described in troops in India and because I am familiar with them.

In Ranchi, between 27th December, 1942, and mid-February, 1943, there were 35 cases in British troops, who had recently been doing jungle exercises. There was the usual sudden onset, with severe headache and general muscular aches; rash, in all cases, appeared on 3rd and 4th day, spreading to all parts in the severe cases, including palms and soles, but not the scalp. Two Officers said that they had been bitten by ticks, but the other cases denied being bitten by anything except mosquitoes. The Weil-Felix reaction was of the mixed indeterminate type, with raised OX2, OX19 and OXK. The OXK reaction was low or absent. There appeared to be two types of OX2 response, a titre rising to 1,500—3,500 (in one case to 30,000) and, in the other, not above 300.

The second outbreak of 20 cases, with 4 deaths, in one British Unit in Calcutta (The Royal Warwicks) in the autumn of 1942, forms part of Capt. Walker's paper. This group of 20 cases had an OXK reaction (with the exception of one OX2 case, imported from Ranchi), in contrast to other cases which have occurred throughout the year in Calcutta, which have commonly an X19 reaction. (See note from District Lab. Calcutta, below).

The third outbreak of 116 cases, between 16th June and 29th December, 1943, was reported by Lt.-Col. J. W. Lusk at the I. M. H. Alipore. It occurred mainly in two Indian regiments,

the 2/8 Punjabis and the 6/11 Sikhs, in whom there were 38 and 11 cases respectively, stationed at Jhingergaucha, a village 80 miles from Calcutta. These cases were all of OXK type. The remaining 68 cases were from Calcutta, where there was a fairly even monthly incidence of the disease. The Weil-Felix reaction was mainly OXK, but some mixed types and a few OX19 were encountered from Calcutta (see Note from District Laboratory, Calcutta, below). There were all degrees of severity, ranging from mild chest symptoms and signs, to generalised rashes with profound toxaemia and death. There were, in all, 16 deaths, a mortality rate of 13·8 per cent. No primary eschar was noted and there was no glandular swelling. In 22 cases (19 per cent.) the total white cell count was above 10,000, but no marked lymphocytic or monocytic increases was noted. Lt.-Col. Lusk and Major Parker of the District Laboratory, Calcutta, have made an excellent study of these cases, clinically, serologically and histologically, which will be of great value.

The fourth and largest outbreaks, was in Fourteenth Army. I have not yet received a report of the total number of cases, but over 300 were admitted into medical units of 4th Corps, between the end of September, 1943, and the end of that year. The cases were all of the OXK type and some closely resembled Tsutsugamushi fever. Two sub-divisions where made locally, the ' River Fever ' type, with generalised enlarged and often tender lymphatic glands, deafness, sore throat and fever, duration an average of 18 days; and the "Indian" type, with average fever duration of 14 days, with no glands or deafness. The Weil-Felix reactions were alike, a strain of rickettsia of the K type has been isolated by Sayers, Parker and Savoor, and work is now being done on it. The death rate was low, 6·9 per cent. though this figure may need revision, as cases may have died in forward units, possibly undiagnosed. The cases differ from the Calcutta cases, in having a primary eschar in 15 per cent. of B. O. R.'s, in the frequency of the enlarged, tender glands and, in some cases, a rise of lymphocytes in the differential count. The disease appeared milder in Indians, in whom a rash was less frequently seen. The military interest in this 4th Corps outbreak, is that it occurred just after the monsoon, as operations were beginning. 130 cases occurred within a few weeks, in two Companies of one British battalion (The Devons), who were on the side of a hill, conspicuous because it alone, for many miles around, had palm trees on its slopes. As you are probably aware, in Malaya, the rodents with their parasitic mites, frequent areas carrying K strains of Rickettsia, where certain palms grow. We do not as yet know whether these palm trees of the Assam-Burma-border have anything to do with this outbreak. It is assumed to be mite-borne, although there is no final proof. There are no consolidated figures for Eastern Command or Fourteenth Army, so that the total number of cases diagnosed, is not known.

The records of Major Parker, at the District Laboratory, Calcutta, during 20 months in 1942 and 1943, which include the two outbreaks already recorded above, in and near Calcutta, show that there were outbreaks of K typhus in November and December 1942 of 17 cases, and in August, September, October and November 1943 of 57 cases at Jhingergutcha, near Calcutta. A few cases occurred in affected and other units of K typhus throughout the year. Cases of X19 typhus occurred throughout the year, with a slight rise in August and September. Thus, in and near Calcutta, K and X19 typhus are endemic, with outbreaks at the end of and after the monsoon. From the K patients, four strains of rickettsia have been isolated in rabbits, guinea pigs and mice.

Other outbreaks have been recorded recently in the Army in India. Lt.-Col. T. A. A. Hunter, R. A. M. C., recorded 11 cases occurring in November and December, 1941, in Colaba, among troops doing jungle training near Bombay. The cases differ from other types of typhus described, in that the rash appeared at the beginning of the disease, or soon after it, and was the most striking characteristic, as the rest of the symptoms were mild. There was no primary eschar. The Weil-Felix showed a rise in OX2 in 6 cases (maximum 1/1250) and a negative result in the remaining 5 cases, during their 3 or 4 weeks in hospital. They were given the name of 'V' fever, Vada being the locality of their origin. Similar cases are recorded in America.

A/S A. J. H. de Monte, I. A. M. C., has reported 11 cases from a P. O. W. camp at Bairagarh, with a high mixed OX2, OX19, rise in agglutins in 9 of the cases. Major Bardhan, I. M. S., has reported 41 cases of XK type from the Brigade Laboratory at Jhansi in 1943, and I have no doubt there are other reports.

Southern Army have informed me that 54 cases were reported during 1942, and 76 during 1943, of all three endemic types, and Central Command had 89 cases during 1943, again of all three endemic types.

Lt.-Col. Cotter, the Public Health Commissioner of the Government of India, has informed me that, on the Persia-Baluchistan border, between April and June 1943, a total of 775 cases were reported from Zahidan and Mirjawa, among military personnel and civilians. I have no information as to its type.

Cases have also occurred among men on leave in Kashmir, where the situation will be described later.

Thus, the position as regards the Army in India is—

(1) Moderate number of cases are being reported from all parts, of all endemic types, of which the vector has never been proved in India. From work elsewhere and from inference from Weil-Felix results, it is probable that the vectors are fleas, ticks and mites, but this again needs proof. It is also almost certain that many other cases pass undiagnosed. These cases occur throughout the year.

(2) Large outbreaks of the K type of typhus, closely resembling Tsutsgamushi and the Malayan scrub typhus, have occurred in troops operating in the jungle conditions of Bengal and Assam, occurring at the end of the monsoon. The vector is unknown, but, from inference, it is presumed to be a mite. From the Army's standpoint, these outbreaks, occurring as the monsoon clears, are obviously important, because this is the time when operations begin.

(3) Large outbreaks on the borders of India have occurred, in cold mountainous country, probably either louse or flea-borne X19 type, which may be of considerable importance in later campaigns.

(4) Smaller outbreaks of the undetermined mixed type have occurred in troops training for jungle warfare, presumably due to ticks.

(5) The possibility of flea-borne endemic typhus in China is discussed later.

(6) Great differences in severity are reported, from mild cases with a few days fever, chest signs and symptoms and no rash, to very severe toxic cases, dying within a few days. It is probable that many cases are never diagnosed.

(7) Crowding of hospitals with severe cases, as recently happened in Assam.

Recent Outbreaks Reported from Civilian Sources—

Lt.-Col. E. Cotter has also supplied the following information, of recent outbreaks :

Afghanistan : a typhus epidemic (no figures available) broke out in Kabul and neighbouring villages, in the beginning of 1943. A few cases also occurred in Kandahar and Jalalabad.

North-West Frontier Province : a few cases were reported in Peshawar City in 1943. The cases were said to be of the louse-borne variety and the disease was mild in character.

Kashmir : in 1943 there was a serious outbreak in verious parts of the Jammu and Kashmir provinces. There were, in all, 1,523 cases and 408 deaths, up to the end of 1943, the main bulk of the cases being in March, April and May. According to the Director of Medical Services, typhus is endemic in Kashmir. There are yearly outbreaks, associated with the yearly migration of the Gujars, a wandering shepherd tribe, from their houses in the hills, to the lower regions of Kashmir, during the winter.

From the information received from various provinces, there is confirmation that small numbers of all three types of typhus occur. Here in Lahore, in March, 1943, Dr. Goyle has given me records of 4 cases of X19 type, occurring in the Borstall Jail.

Records from Burma, China, with reference to future operations—

The Inspector General of Civil Hospitals, Burma, has supplied the following information :

" The incidence and mortality of the typhus group of fevers was notifiable in urban areas in Burma. There was no information about the incidence and mortality from typhus in the rural areas." A few cases were reported from towns, between 1933 and 1938. In 1939, the number of cases of (*a*) louse-borne typhus noted in Burma hospitals, was 56 with no deaths, (*b*) the tick-borne typhus numbered 4 with 1 death, and (*c*) unknown vector was 76 with 6 deaths. These reports are obviously scanty and do not concern rural districts, where the Army is likely to operate.

In 23 days of September 1941, 101 cases of endemic typhus of K type, were reported in one unit, camping in the jungle. The usual early difficulties of diagnosis arose, until the Medical Officers became 'wise' to the disease. It was assumed that the vector was a mite, but no proof was obtained. Major Coombes, I. A. M. C., who saw the cases, tells me that they were from 2 companies of Punjabi Mussalmans, although a neighbouring Sikh company escaped. He says that there is "any amount of the K typhus in Burma", with highest incidence in August and September, at the end of the monsoon. Possibly this seasonable incidence is not only due to the habits of the vector and the animal reservoir, but because man keeps out of the jungle during the rains and goes into it when the rain stops, for his own doings, or, in the case of the Army, for training or actual operations.

In Assam, the Inspector General of Civil Hospitals writes that a small number of cases, of all types, are yearly diagnosed, but, owing to the nature of the laboratory services, these cases do not represent the true picture of the disease.

Typhus is endemic and epidemic in China. In Yunan, bordering on Burma and China, Capt. Hsu, I. A. M. C., tells me the endemic flea and possibly louse-borne typhus is mild, with a low mortality. In the European, going to Yunan, the disease is much more severe, with a high mortality. In 1942, a Friends' Ambulance Unit went there. Of 20 Europeans in it, there were two deaths from typhus, within a few months. The remainder were inoculated with Cox's vaccine and the outbreak stopped. There is universal X19 typhus, throughout China. Capt. Hsu, Pekin, tells me that there is no K typhus. He also told me of Raynol's report from the Pasteur Institute, Shanghai, of epidemic louse-borne typhus, arising from endemic flea-borne typhus, an occurrence which has been reported elsewhere.

One word about the value of vaccines in typhus. Cox's and Weigl's vaccines are of definite value in X19 typhus. In the indeterminate group of R. M. S. F. there is proof that the appropriate vaccine is of value. But, in K typhus, there is no evidence that any vaccine is of value. Kawamura (1937 and 1939) found that inoculation of a weakly virulent strain of K strains, produced a mild illness in a man, followed by immunity to a strongly virulent strain. As yet, this cannot be recommended for general use.

Thus, in future operations, the army in Burma, China and Malaya is likely to encounter cases, if not outbreaks, of both epidemic and endemic X19 typhus and typhus of the K type. With the present knowledge of outbreaks of K typhus, these outbreaks may be serious.

Q Fever

Col. C. Pasricha described an outbreak of Q fever, in Dehra Dun, in 1941/42, of 400 cases of which, 'the diagnosis was clinical, confirmed later by highly suggestive agglutinations, done by Burnett in Australia.' (personal communication).

Clinical Incidence—

It is not the purpose of this paper to describe the clinical varieties of the disease, which Capt. Walker is doing. The wider the study of the disease, the greater the diversity of signs and symptoms. The following list is based on recent experience of difficulties of diagnoses.

(1) During the early fever stage—malaria, cerebral malaria, influenza, enteric group, plague, glandular fever, dengue, encephalitis.

(2) When rash appears—smallpox, measles, haemorrhagic diseases, scurvey, haemoptysis, and haematemesis of various causes, bowel haemorrhages, septicaemias, dengue.

(3) With chest signs—bronchitis, pneumonia, pharyngitis and pneumonitis.

(4) In post-fever stage, the disease may be diagnoseable by the Weil-Felix: gangrene of the extremities has been seen in three cases, phlebitis and thrombosis of many different blood vessels.

(5) Post-mortem examination of early cases, dying before the Weil-Felix is positive—septicaemia, particularly of enteritis (Gaertner) (*Major Parker*). Some cases, coming to post-mortem, have been missed, as typhus was not considered.

In all suspected cases, a Weil-Felix should be repeated at regular intervals.

Work needed in India—

It is obvious that work is needed in India, along two lines. Firstly, a research team of a first-rate Entamologist and Bacteriologist, with knowledge of rickettsial diseases, to investigate the vectors and animal reservoirs and the kind of country in which they are found. Practically no work has been done on Indian mites (Prof. D. M. Roy, S. T. M. Calcutta) and no

vector in India has ever been shown conclusively to carry the disease. The finding of the vectors would appear to be important for the army, because, with added knowledge, localised areas where the disease is common, may be avoided and adequate preventive measure taken against bites. Further knowledge of the seasonal incidence might well be of value, in preventing and controlling outbreaks. A second expert team to investigate the possibility of vaccines is needed. Recently in Assam, an American vaccine (which was not K) was used when there was an outbreak of the K type. There is little evidence of cross-immunity with strains. My own view is, that the work on finding the vector, and the prevention of bites, will yield more valuable and quicker results than vaccine preparation of the K strains, which bristles with difficulties. It has yet to be shown that a K vaccine is of value. If and when the army moves into China, the possibility of X19 epidemic and endemic typhus will arise, with all the paraphernalia of prevention of lice and fleas. Here the newer vaccines are of proved value. I believe that requests have been sent to England, for research of this nature.

Summary—

It has now been shown that :

(1) Endemic typhus, of all three types, is fairly common in India.

(2) Outbreaks of the K and undetermined type have occurred in troops, operating in jungle conditions in Eastern India and Burma, which are of considerable military importance.

(3) The possibility of outbreaks of epidemic or endemic X19 typhus is considered.

(4) Lines for research are discussed.

(5) The need for consideration of a diagnosis of typhus, in all doubtful cases, and some of the pitfalls in making the diagnosis, are described.

(The pictures of haemorrhagic lesions in the lungs, intestines, caecum and brain, were demonstrated by epidiascope.)

35. CAPT. WALKER—"*Clinical Observations in Typhus*" **(P)**

Typhus fever is a new experience for those of us who have come recently from the U. K. and, in some parts of India, it is common. In one year I have seen forty cases among British troops in Calcutta. The disease appears to vary in its clinical and serological features, within narrow geographical limits. Cases occurring in Calcutta differed in both respects, from those occurring in Ranchi, only 200 miles away.

The vector in Calcutta has not been identified. The agglutination reactions indicate a mite-borne type of typhus. No vermin were found on any of the patients and no primary tache was observed.

The onset is usually sudden and severe. There are no characteristic early features. The symptoms are those of any severe fever—generalized aching, headache and cough. The eyes are suffused and, commonly, photophobia is present and may be prominent—a useful diagnostic point, though too much weight should not be placed on it. The patient's mental condition is altered in all but the mildest cases. He is in a state of drowsy resistance. Frequently he lies on his side, curled up in bed with his back to the light, resenting examination.

On the first and second days, muscular twitchings are sometimes seen. They affect all the visible superficial muscles. A twitch affects a strand of a muscle, throughout its length. It may be insufficient to cause movement, or the head and trunk may be jerked violently.

Usually there is generalised enlargement of the lymph glands, which may be tender, and palpable enlargement of the spleen.

The temperature rises rapidly to 103-104 in the first hours, with a corresponding tachycardia. High continued fever was maintained throughout the illness. In the cases which occurred in 1942, the fever subsided by crisis on the 14th day. Later in the year and in 1943, the character of the termination changed. The fever was more prolonged and subsided by lysis over a week. I can offer no explanation as to why the fever should change its character so markedly, in so short a time, for, in every other respect, the disease remained unchanged.

Some degree of bronchitis is usually present and, in severe cases, this develops rapidly into pneumonia by the end of the first or the beginning of the second week. It develops much more rapidly than the hypostatic pneumonia, which so commonly terminates a severa illness. The rapidity with which this complication develops, must have an explanation, and the only one I can offer, is that the cardiovascular changes, which are so pronounced in typhus, affect

the pulmonary circulation in such a way that some degree of pulmonary oedema developes and forms a suitable nilus for the multiplication of pathogens.

The rash is variable. It may not appear throughout the illness. Characteristically, it develops between the 3rd and the 10th days. The initial erythema of the skin becomes broken by pale lines and, at the same time, it darkens to a dull red colour. Superimposed on this mottling, are darker red. slightly raised papules, verying in size up to a centimetre or more in diameter. The trunk is most densely affected, the rash becoming less intense towards the periphery. Commonly, the exanthem is merely a few dull red macules, about ½ a centimetre in diameter, located on the upper abdomen and lasting a few days only. When the rash is scanty, the underlying mottling may be absent. When severe and affecting a wide area, oedema of the skin is pronounced, particularly of the face, which is red and bloated. The duration of the rash varies. When well-developed, it persists until the third week, fading gradually and leaving a brown pigmentation on the affected area, sometimes with desquamation. The oedema of the skin, which I have just mentioned, is not oedema of the subcutaneous tissues, as in nephritis or congestive heart failure. It is an oedema of the epidermis, similar to that seen in smallpox. There is no pitting on pressure. It is due to dilation of the superficial capillary bed. If this is so, then it is possible that these patients lose fluid into the skin, a condition analagous to that seen in burns. I started to investigate the haemoconcentration in such cases, but, unfortunately, I was only able to examine two, before I was posted to an area where typhus does not occur. One of the two was a mild case, with a scanty rash and, as might be expected, there was no variation in the haemoglobin concentration throughout. The second case had a severe rash and might have been expected to show some variation, but none was observed. His daily venous haemoglobin did not vary by more than 5 per cent. This line of observation could be developed by someone with suitable opportunity.

The patient rapidly becomes extremely ill and, about the beginning of the 2nd week, he may be in the typhoid state. Retention of urine may be the cause of restlessness at this stage.

In no other acute illness are the changes in the action of the myocardium so strikingly observed. In the first few days, the heart is normal, apart from the tachycardia. In unfavourable cases, the quality of the sound changes as the pulse rate rises. The first sound alters distinctly and becomes of higher pitch and shorter duration. The rhythm alters, so that, in 48 hours, tic tac rhythm is obvious. This change may be seen before serious deterioration is noticed in the patient's general condition and it is of serious import. Later, extra systoles or auricular fibrillation appear.

In a favourable case, the clinical condition of the patient remains constant, after full development of the fever, until defervescence. In the cases which terminated by crisis, convalescence was rapid, whereas, in those which ended by lysis, convalescence was prolonged. Considerable emaciation is usual in a severe case.

Complications are few. Disseminated pneumonia is common and appears about the end of the first week. It may be associated with haemoptysis. Intestinal haemorrhages of some severity occurred in one case. Tympanites may develop and is a cause of considerable discomfort and embarrassment of respiration. No other complications were observed.

In Indian typhus, the serological findings vary in different areas. 29 cases showed high titres to OXK, 2 agglutinated simultaneously OX2, OX19 and OXK: 1 agglutinated OX2; and one OX19. In 7 cases, serological findings were negative, but the clinical picture in those cases included the rash, the enlargement of lymph glands and splenomegaly, and 2 were associated with other cases of typhus, so that the diagnosis could not reasonably be doubted. Four of these patients with negative serological findings, died. P. M. revealed the slight generalized enlargement of lymph glands and other causes of death were excluded.

Owing to the frequent movement of units, it was not possible to follow up cases. However, one patient was observed weekly, for over two months after discharge from hospital. The titre at the peak was positive to OXK, in a dilution of 2500. During convalescence it fell rapidly to 500 and remained at that figure until contact was lost with the patient. Another case was re-admitted for a mild acute bronchitis, about two months after his discharge and the titre was 250 to OXK.

The diagnosis of typhus is difficult in the early stages. In the first few hours, it is clinically indistinguishable from malaria and cerebro-spinal fever and, in the first few days, from typhoid fever and smallpox. Malaria is readily excluded by examination of the blood and the

failure of response to intravenous quinine, as these cases are frequently mistaken for cerebral malaria and treated as such, in the absence of parasites from the peripheral blood. The severity of the onset, with irritability and photophobia, suggests cerebro-spinal fever. In the first hours, lumbar puncture is the only method of differentiation. Cases of severe dengue simulate typhus closely and diagnosis may be impossible until the course of the illness reveals the true condition present. The onset of fevers of the enteric group is usually more gradual, but differentiation of the two diseases is impossible, in many cases, until a specific serological result is obtained. The onset, course of the fever and such complications as typanites and intestinal haemorrhage, are similar in both conditions. When the rash is typical of typhus, identification is easy, but many cases show only a few pink macules on the abdomen, similar to the specific rash of typhoid fever. Smallpox is the only other acute febrile illness with which typhus can be confused. The onset in both is severe, with similar symptoms and the typhus rash may appear on the third day and resemble that of smallpox. Cases of smallpox have been admitted into the typhus ward and, conversely, cases of typhus have been admitted into the smallpox ward, in both instances, after due consideration by experienced observers. Differentiation may be impossible until the development of the rash on the fourth and fifth days, indicates the true diagnosis.

Isolation is unnecessary, as case to case infection does not occur. For efficiency in nursing and general management, however, a special ward with experienced staff, is desirable. As the fever is prolonged and debilitating, a high caloric diet is essential from the beginning. A diet of about 3000 calories should be used and amplified with glucose and cream. It is important to ensure an adequate fluid intake. If the daily output of urine is over 40 ounces, it can be assumed that the intake is sufficient. When the patient is incontinent, as is often the case, at least one hundred ounces of fluid must be given in the twenty-four hours. If the diet is given as two-hourly feeds, between 7 a.m. and 9 p.m. (a solid meal being given at 1 p.m.) each feed, consisting of five ounces of milk made up to ten ounces with some suitable flavouring agent, seventy ounces of fluid is provided; 10 ounces of fruit juice can be added, three times in the twenty four hours. When consumption of sufficient fluid by mouth becomes impossible, in the later stages of the fever, intravenous injection of 10 per cent. glucose is necessary. Each pint contains 50 G. of glucose, representing 200 calories. Twelve pints, or more, can safely be given intravenously in 24 hours. In hot weather, when the patient is sweating freely, estimation of the urinary chlorides is necessary, to prevent chloride depletion. If the chlorides fall below 2 Grammes per 100cc of urine, normal saline or hypertonic (1·6 per cent.) should be exchanged for the glucose solution, until a larger output is obtained. When intravenous infusion of fluids is used in such cases, the saphenous vein, two inches above the infermal malleolus should be exposed in a transverse incision and a canula tied in. Constant attention is required, for the canular readily becomes blocked. If necessary, the vein in the other leg should be exposed, or one in the forearm, above the wrist.

Although the sulphonamides are useless in combating the specific infection, sulphapyridine is of definite value for the pneumonia, which so commonly complicates these cases.

The tympanites, which causes discomfort and embarrassment of respiration, is most satisfactorily dealt with by prostigmine ½—1 cc, repeated, if necessary after an hour.

Convalescent serum was used in some earlier cases, when a suitable donor was available, but the impression gained was that this measure was valueless.

36. LT.-COL. BINDRA—"*Observations on Scrub Typhus, in Addu Atoll (Maldive Islands)*" **(D)**

The period covered by the investigations was from May to December, 1943. These islands, with their limited fauna and flora, and with comparatively small area, make the understanding of certain aspects in the transmission and spread of this disease and the problem of its control, comparatively easy. The Atoll is formed by a group of small coral islands. The soil is sandy and the vegetation, though plentiful, is limited in its variety; cocoanut trees predominate and there are a few other trees, e.g. bread-fruit, papaya, etc. The uninhabited areas are covered with thick scrub, growing to a height of 5-6 feet. The animal kingdom is represented by rats, lizards, snakes and a few varieties of birds, including flying foxes. Large numbers of chickens are to be found in the villages, but there are no other domestic animals. Amongst the insects, there are flies and mosquitoes of various types.

Typhus fever was found to be endemic on all the islands, comprising the above-named group, and cases were admitted from amongst troops stationed on every one of these islands. In the dry season, contact with scrub could be traced in almost every case admitted into hospital with this disease. Units, e.g. Auxiliary Pioneers, and those engaged in anti-malarial work, whose walk brought them into close contact with scrub, were most heavily affected, with as many as 50 per cent. of these men being admitted. As a contrast to this, the hospital situated in a clear area, with a total population of over 600, had only six cases, over a period of eight months. Another significant fact emerged, when a Field Battery, stationed on one of the islands, embarked on clearing the area surrounding the camp. Within a few days, the typhus rate in the unit went up from 1-2 cases per month to about 40 per cent. of the total strength finding their way into hospital inside six weeks. The work had to be stopped. There was a marked rise in the incidence of the disease when the rains started. The admission rate went up by 300—400 per cent, as compared to the previous month. At this stage, even units which were previously comparatively free of the disease, started getting cases, and contact with scrub was no longer a constant feature. The obvious conclusion from the above, would appear to be that the rats were flooded out of their holes and were forced to seek shelter in the huts and tents, thereby coming into close contact with the men.

Reservoir of the infection and vector—

Even though it awaits proper scientific and experimental confirmation, from the evidence so far available, it appears most likely that the field rat acts as the reservoir and the infection is conveyed to man by a mite. Mites have been found in large numbers on the local rats. The typical eschar, described in the mite-borne variety of the disease, was seen in a certain proportion of cases.

The Fever—

This presented an almost text-book picture, starting with or without rigors, with intense headache and temperature rising to 103-104°F. It continued with slight daily remissions for 13-14 days, when the temperature came down, usually by rapid lysis, taking about two days. A record of the more important signs and symptoms was kept in 214 cases, and the results are appended below, on a percentage basis—

Headache—99 per cent.
Drowsiness—98·5 per cent.
Congested eyes—70 per cent.
Slow pulse—100 per cent.
Tongue furred—100 per cent.
Rigidity of neck—30 per cent.
Rash—13 per cent. (mostly seen in B.O.'s and B. O. R.'s).
Enlarged lymph glands—24 per cent.
Spleen palpable—60 per cent.

The Leucocyte picture—

Total and differential leucocyte counts were carried out, in 100 cases, in three phases of the disease (1) 3-5 day of the disease, (2) about the 10th day, when the fever usually got complicated by pulmonary or other infection, (3) about the 20th day, when convalescence had set in. With a few variations, which usually had a simple explanation, the results were fairly constant and are represented by the following average figures :

Total W. B. Cs.—

1st count—5—6,000.
2nd count—10—12,000.
3rd count—6—7,000.

A lymphocytosis of over 30 per cent. was found in 40—50 per cent. of cases, in all the three stages.

Blood Pressure—

Daily estimations of blood pressure were made in 80 cases, during the fever. Apart from a lowering in both the systolic and diastolic pressure, no other characteristic changes were noted. The average systolic pressure was between 100—110 mm. Hg. and the diastolic was 75—80 mm.

Complications—

Pulmonary infection, resulting in acute bronchitis or broncho-pneumonia, was found in over 50 per cent. of the cases. Parotitis, which used to complicate 4-5 per cent. of the cases, was completely stopped by improved oral hygiene, carried out under the personal supervision of the dental officer. Amongst other complications, Hepatitis, delirium, mania, coma, pleurisy with effusion and irregularities of the heart, were noted in 2—10 per cent. of the cases.

Diagnosis—

This presented no difficulty in the absence of any other diseases with which it could possibly be confused and, after a little experience, the M. O.s were able to make an infallible diagnosis on sight. The diagnosis was invariably confirmed by getting a rising titre against OXK. No-positives were obtained against OX19 or OX2.

Prognosis—

This was very good in the young and well-nourished. Officers and B. O. R.s stood the fever very well and gave no cause for anxiety. The death rate varied from 2-5 per cent. and most of the fatal cases were from among undernourished I. O. R.'s, usually over 45 years of age. Over 50 per cent. of the deaths were due to a flaring up of quiescent pulmonary tuberculosis with haemoptysis. This was confirmed by post-mortem examinations in each case.

Treatment—

This was mainly symptomatic, special effort being made to keep up the morale of the patient and to keep him cheerful. This was found to be easier in the cases of officers, V. C. O.'s and B. O. R.'s who did well in any case, but was very difficult in the toxic, undernourished type of I. O. R., who was apt to become very depressed. The patients were encouraged and coaxed to drink water and, except in the very ill, it was usually possible to keep the fluid intake at a reasonable level. At the earliest appearance of dehydration, saline was administered intravenously. This had to be resorted to in about 10 per cent. of the cases. The cases showing signs of pulmonary tuberculosis were treated with a short courses of M & B 693 (15—20 Gms. given in 4-5 days), with very satisfactory results. Cases which developed cardiac irregularity were treated with tinct. digitalis. Patients with severe headache and neck rigidity appeared to obtain relief from lumbar puncture and the removal of 10—20 cc. of fluid. This sometimes had to be repeated two or three times.

Convalescent serum therapy—

An attempt was made to try the effects of convalescent serum in treatment. Suitable convalescent cases were selected and the possibility of syphilis was excluded, by means of the Kahn test. Blood was drawn from 4 or 5 such cases and pooled in a dry, sterile flask. After the clot had separated, 10 cc of the serum were injected intravenously in normal saline, daily for three consecutive days in an early acute case. This line of treatment was tried in about 10 cases and, in two of them, it appeared to cut down the course of the fever, the temperature coming down on the 8th and 9th days. Three of the others appeared to benefit to some extent and, in the remaining five, it did not produce any effect on the course of the disease. The work is being continued and no definite conclusions can be drawn until it has been tried more extensively.

Immunity—

In the twelve months during which the hospital had been functioning in the station, there is no case on record of any individual getting a second attack of the fever. In my experience, there was no cases of relapse after the temperature had come down to normal. The occasional rise of temperature during convalescence was invariably due to some other cause, which could be easily found and treated. The local inhabitants appear to have acquired immunity to the disease. In spite of the fact that they are usually ill-nourished and suffer from chronic malaria and anaemia, I could get no evidence of the incidence of typhus fever among them.

Recommendations—

(1) A team of experts should be sent, to establish the reservoir and the vector beyond doubt.

(2) Scrub should be cleared from the vicnity of the military areas. As far as possible local Maldivian labour should be employed for this purpose.

(3) Old and low-category personnel should not be sent to this station.

(4) Protective clothing (shirts with long sleeves, gloves, slacks, boots and short puttees) should be worn by men who have to come in contact with scrub.

37. MAJOR KAUL—"*Tick Typhus in Arakan*" **(D)**

I. These observations were made from a Hygiene Officer's point of view. Quite a number of cases of typhus occurred in the Arakan last year. My contention is that these cases were mainly tick typhus and I offer the following observations in support:

(1) The cases came from the jungle.

(2) As we all know, epidemics of typhus are closely associated with periods of economic distress, presumably old infections, after remaining dormant, have been lighted up by malnutrition. Such conditions existed in the Arakan last year, and several cases of typhus did occur. But, in spite of the favourable conditions for its spread, starvation of civil population, refugee movements, and susceptible troops, some even with malnutritional defects due to lack of fresh meat, vegetables and fruit, the typhus cases remained sporadic.

(3) Routine and special medical inspection of troops from infected units, did not reveal louse infestation in troops.

(4) The serological reactions gave varying degrees of positives, with all three groups, OX19, OXK and OX2 in each case.

(5) Lastly, in two cases, a hard tick, similar to the Dermacentor variety, was picked off the patient. In addition, pariah dogs, infested with dog-ticks, were numerous and suffering from starvation, came in close contact with men, during their search for food, which was available in the vicinity of military camps. I have no proof that these dog-ticks were infected, but it is a possible suggestion that these may have acted as reservoirs of infection.

II. Another aspect of typhus cases in the Arakan, was the lateness in reporting cases; it seemed that diagnosis was made on agglutination tests, rather than on clinical grounds. The probable difficulties in the early diagnosis may have been:

(1) Typhus was not looked for often enough.

(2) The sudden onset, in previously healthy persons, associated with rigors, in some cases with palpable spleens, were apt to be considered as malarious, in a highly malarious area like Arakan, until repeated blood examinations proved negative.

(3) A group of typhus cases, showing, in the first week, predominately lung symptoms (lobar and broncho-pneumonic type of consolidation, especially during the autumn months, when the largest number of cases of typhus were seen in the Arakan) were diagnosed as pneumonia, until sulphonamides failed to cure and a fuller investigation was undertaken.

(4) The rash, in Indian patients, was difficult to observe and easily missed. In any case, the tick typhus rash is very faint.

III. At the time, I was mainly interested in the preventive means, and medical inspections for louse infestation and large-scale disinfections were carried out, wherever possible. But, due to late reporting of cases and movement of troops, these were not always satisfactory. In spite of this, no real epidemic developed in any area, again suggesting that louseborne infection was probably not involved.

In tick typhus, such preventive measures are not helpful. Would a prophylactic vaccine prove to be a better preventive? Cox's vaccine is used on a large scale by U. S. troops. Its use is also reported in the German Army. South Africa and China also prepared and used this vaccine. Three doses, at intervals of 7—10 days are followed by a single dose every 4—6 months. I am not aware if any vaccine of this nature is available in India.

DISCUSSIONS

38. MAJOR-GENERAL MARTIN pointed out that the Army was worried about the problem and enquired about the hygienic measures that could be taken. He emphasised the importance of immediate investigation by a team from the U. K., America or India. The pathologists should clear the question of serological diagnosis and the milder cases must be followed up.

39. MAJOR LYN GREENING enquired regarding the present position of the vaccines.

40. LT.-COL. BOSE mentioned that successful treatment with Mepacrine had been reported in the Bulletin of War Medicine.

41. COL. TAYLOR replied, "Vaccines of XI95 of the undetermined (R. M. S. F.) groups of typhus are of proved value, 'Whether or not, mass immunization is adopted (by vaccines), it is recommended that all medical personnel should be fully inoculated with the most efficient typhus vaccine available.' (Medical Directorate India Bulletin No. 14, March 1943). This quotation applies to epidemic louse-borne typhus of the X19 type. With K strains, efforts by Malayan and Japanese workers, to produce a vaccine, has as yet failed: Kawamura (1937 and 1939) working on a living avirulent K strain, produced by its injection into man, a mild illness, followed by immunity to a strongly virulent strain. I have no evidence as to how long this immunity lasts, but the Weil-Felix reaction in the K strains is raised for a few months only. There is little evidence that cross-immunity exists between the different types of typhus. No good effect has been observed with Mepacrine therapy."

42. COL. SCHLESINGER described an epidemic of louse-borne typhus which he had seen among the coolies working along the Persian L. of C. and over the borders of Persia. He stated that it was only due to the strict de-lousing precautions, undertaken by the Medical Staff of N. W. Army, at Stages along this route, that had probably prevented a serious spread of the infection into the N. W. of India.

43. LT.-COL. PASRICHA: From what has been said, it is apparent that the work in India on this somewhat heterogenous group of fevers, is not widely known. The existence of typhus in India was first established by Megaw in 1917, who, ever since, has been deeply interested in this subject. A number of cases of typhus has been reported from various parts of India. Boyd published a large series, and more recently, workers from the south of India have reported cases.

A fair amount of experimental work has been carried out, under the auspices of the Indian Research Fund Association, but, as sufficient material was not available, there is not sufficient epidemological data, but we know that all the major types occur. For about six years before this war, we carried out, at the School of Tropical Medicine, Calcutta, work on Rickettsial infection of rats, as a preliminary to the study of typhus in man. Because of the paucity of the material during peace time, progress was slow. Now that typhus, the inseparable companion of wars, has made its appearance, there is material, plenty of it, and those who, before the war, were studying scraps and odd bits, would welcome this opportunity of investigating this disease. An outbreak such as we have heard from our Director, are outbreaks which a research worker on this disease would regard as places of pilgrimage. There are plenty of workers, both in the Military and in the great hospitals and research institutes of India, to start a satisfactory research team at once.

In discussing the question of vectors in typhus with several medical officers, I have been surprised by the appalling ignorance, of some M. O's, on the appearances of these vectors. On being asked as to the size of the mites implicated in typhus, I have frequently been told that they are about a centimetre in length. As regards entomologists, there are experienced entomologists in India, men who have worked on this particular animal and who can identify the various species.

44. LT.-COL. ROGAN stated, with regard to the possibility of strains of typhus undergoing mutation in the host reservoir, Lewthwaite, in his experiments in Malaya, observed the mutation of one strain from the OXK type to the X19 type, during passage through guinea pigs.

45. MAJOR CRADDOCK said, "Regarding vaccines, the original Weil's vaccine, as prepared in Peking from a carbolised emulsion of the guts of infected lice, was effective, but produced rather severe reactions. Accordingly, an attempt was made to produce a more refined vaccine and this was ultimately made from cultures of R, Prowazeki. I was protected by this vaccine, which produced no unpleasant effects, while working in endemic areas in China, and the consensus of opinion was that it had definite value. It is unlikely, however, that it would be of use to the Army in India, except in the limited areas where louse-borne typhus is likely to be encountered, e.g. in Kashmir, or in the event of troops operating in endemic areas in China. Its possible value against tick-borne typhus in India has already been mentioned.

Regarding vectors, the incrimination of larval mites is difficult, owing to their minute proportions, but ticks are relatively large creatures and when they parasitise human beings, their burrowing is less likely to pass unnoticed by the patient, and a very careful questioning should be of value, at least in suspected tick-borne outbreaks among European troops. Rattus rattus has been mentioned as the reservoir host of larval mites, but all murine species should be suspect and, in any serious investigation, field mice, voles and all rodents should be collected.

There are many anomalies, the solving of which the proposed investigating team will find of facinating interest. Why, for example, does the same strain of rickettsia appear to produce, at one time a simple two-week fever, and at another a malignant fatal illness, with different clinical features? Mutation of strains has been known to occur and, in explanation of other anomalies, it has been suggested that the same strain may be conveyed by different vectors, at different times, in the same locality. But, undoubtedly, one of the most urgent problems to be tackled, is the identification of the Indian vectors and their hosts."

46. COL. CAMERON, in winding up the discussion, said, "Typhus is a place disease; undoubtedly one of the places is the jungle country. It is likely, therefore, that typhus will increase in importance as the campaign progresses. Vaccine protection is desirable, but the doubts cast on the efficiency of vaccine for OXK are upsetting. Protective measures against the mite will require investigation. There is, at present, no therapeutic remedy."

(The meeting adjourned at 17.30 hours)

8th FEBRUARY, 1944—MORNING SESSION

The Conference commenced at 09.00 hours, with **MAJOR-GENERAL CURSETJEE** in the Chair.

MEDICAL TRAINING

47. MAJOR-GEN. CURSETJEE—"*Opening Address on Medical Training*"

Ladies and gentlemen, I have been asked to open the subject of Medical Training at this Conference. I am not a Scientific Physician. I pride myself on being in ordinary average Army Medical Officer. If I am not average, I am what I consider the average should be.

I have had some experience of training medical personnel since 1919, but chiefly in connection with training subordinate medical personnel, whom some of you regard as inferior personnel' but whom I have always regarded as Comrades in Arms, and some junior medical officers on first appointment. I have also been inspecting hospitals and medical units, since the last fifteen years.

When I go round hospitals, I am told by C. O's that their medical officers know very little or nothing. In the wards, I am told by the M. O's. that their Nursing Sepoys are inefficient. In some hospitals I am told that they cannot train Nursing Sepoys because they have no nurses. C. O's. and M. O's. do not admit that they have made little attempt to teach, or that they do not know how to. Medical Officers and Specialists consider it an insult to their scientific learning, if they are asked to teach a Nursing Sepoy how to make a bed. Unless they have had the good fortune to be seriously ill and to lie on a badly made bed, they cannot appreciate the advantages in treatment, of a comfortable bed.

I had the good fortune to be introduced to Clinical Medicine at Addenbrooke's Hospital, Cambridge, by Sir Thomas Clifford Allbut, Regius Professor of Physics, Cambridge University and President of the Royal College of Physicians—the greatest physician in England and therefore the greatest in the world. He did not consider it beneath his dignity, but a proud privilege, to introduce humble clerks to their first day at the bedside of a patient in a hospital ward. His equipment was a thermometer and a stethoscope. The stethoscope was a simple tube, it had no elaborate chestpiece with amplifier, with which, I understand, the makers guarantee to hear the stealthy footsteps of a tubercle Bacillus crawling down a bronchiole. Yet the modern physician-Specialist requires at least one expensive X-ray film, to confirm his diagnosis. Clifford Allbut had just three sensitive, subtle fingers, with which he felt the pulse, but he was able to detect the nature of the vessel, the calibre of the pulse and its

tension, in addition to its rate. The modern physician applies a black bag round the arm and inflates it, while he watches a column of mercury travel up a graduated scale, or he applies for an Electro-Cardiograph tracing.

I read all Medical Case Sheets that come to me. Granted, they are those of Medical Boards and Fatal Cases, but such are a very useful guide to the standard of work turned out by the hospital; after all, they indicate cases which the hospital has failed to cure. As a rule, they are badly scribbled, and most of the essential details of examinations and findings are lacking. "Patient admitted for fever and cough. Blood for M. P." or "Patient is seriously ill. Specialist sent for". In large letters, "Patient placed on D. I. List". There are no notes of what the M. O. saw, palpated or heard. There are no details of treatment, except M and B four-hourly, or routine anti-malaria treatment. There is no note as to whether the M and B or Quinine was retained, vomited or absorbed. What diet the patient had, the quantity of fluids he imbibed, or the amount of urine he passed—the latter, of course, would be left to the sweeper to measure and estimate from his more practised eye. Finally, "Patient developed a grave turn. Case ended fatally". Remarks C. O. "Death NOT attributable to military service".

I hope that I have not been too destructive in my criticisms, but I would urge you all, when you go back to instruct your officers, to attend to what you scientific physicians may consider elementary details. You must appreciate that elementary details are the foundations of learned findings. Do not consider that teaching your Nursing Orderlies and Ward Staff is a duty unworthy of your scientific learning; after all, the results of the learned scientific treatment you will prescribe, will depend on how that treatment is carried out by your subordinates.

To conclude, I cannot do better than quote Prof. Ryle, late Regius Professor of Physics, Cambridge University, now Nuffield Professor of Social Medicine, Oxford. "The sciences and techniques have come to dominate medicine, to the exclusion of the most important technique of all—the technique of understanding man. Science without humanism may work with atoms, but it will not work with man. 'Mechanised' medicine has, perhaps, captivated the thought and action of doctors. The highest ideal of Hippocrates was 'Where there is love of man, there is also love of the Art' (of medicine). In those early days, the Art of Medicine prevailed, the Science had hardly begun."

48. COL. CAMERON—"*Army Medical Training Centre*" **(P)**

The Army Medical Training Centre owes its origin to the foresight of our D. M. S., Lieut.-General Gordon Wilson. It has been in existence since April, 1942, but has not yet become stablised. During 1942, two trial courses were run at Kirkee, mainly with a view to testing teachers and subjects which were thought to be desirable. At that time, it was expected that the centre would serve as a post-graduate school, providing refresher courses to officers who had been out of touch with recent developments. The refresher courses were envisaged as alternating with more elementary courses, to introduce new officers to Army medicine. The initial two courses gave satisfaction as refresher courses and, with building progress at Ganeshkind, it was hoped to get into regular operation in the first quarter of 1943.

With the visit of the Mission from the U. K. in January, 1943, came a change in policy, and it was evident that refresher courses had to be abandoned. All new I. A. M. C. officers were to enter the A. M. T. C. immediately on enlistment, while all new drafts of R. A. M. C. officers were also to pass through the A. M. T. C.

So much for policy; what was the aim behind it? The A. M. T. C. was intended to produce military medical officers from the material presented to it. There has been much criticism of the Centre and its results, but, if this point is emphasised, criticism should dissipate—the fault does not lie with the Centre, which can only work on the material with which it has been provided. The earliest I. A. M. C. courses contained a high proportion of graduates of the Indian Universities, later, the admission of licentiates to commissioned ranks, resulted in a decrease in the number of graduates and a big increase in the number of licentiates. A fall in the standard was inevitable and, as a lower standard prevailed on entrance, naturally it was proportionately lower when the students were posted to Units. You doubtless blamed the poor work of the Centre; my own opinion is, that the Centre did a very good job, considering what it had to work on.

A few charts of progress, indicating the standards attained by different courses, will focus the above thoughts. View then, in association with the other charts, in which the standards attained by the graduates and licentiates of different Indian schools and note how the standard has fallen, as the number of licentiates has increased. Note too, the apparent rise in the graduate standard, but this is false rise and merely evidences the lower standard, which has to be accepted as average, since the advent of the large number of licentiates. Major Tredgold has carried out psychological tests, in parallel with the observations from which these charts are constructed. He is present at this meeting, and so absolves me from touching on this point.

All those who enter the Centre, have had medical training (to make them Doctors) at some University or College. The Centre sets out to adapt that training with its resultant knowledge, to Army service. The teaching is thus not medicine, but the application of medicine to the needs of war. We must attempt to assess their previous knowledge, build up obvious defects in that knowledge and introduce subjects not normally sufficiently dealt with in the civil schools. In so doing, we aim to turn out G. D. O's, not specialists. If specialists unfortunately arrive at the Centre, they will find the teaching is not pitched to their level, though an attempt has to be made, because of the diversity of knowledge, to teach different grades in one and the same lecture and clinic, starting at the level of poor 3rd year under-graduate teaching, and reaching above the heads of most, by the end of the lecture. Only by such means can a reasonable catering be done for all. And this all has to be done with 1½ months clinical teaching.

How is the Course planned? The complete course lasts 3 months. The first month is devoted to military training—an essential for the type of recruit we secure. During this month the recruit is introduced to military etiquette, mess etiquette, military law, hygiene, field training, map-reading and army administration, organization and procedure. Only the outlines of these can be given, but, as the students proceed straight from this course to Field Units and hospitals, they must be given at least sufficient training to allow them to enter these units with the obvious rawness removed—able to take their place in Mess and to act as officers before the men.

The second month of the course comprises lectures and clinical instruction in Medicine and Surgery in War. I have indicated that the intention is to direct all the clinical teaching into war channels, but I fear, in all cases, a large amount of fundamental teaching has to be undertaken, before war medicine and surgery can be reached.

The third month is a divided one, part being given over to the special subjects, not with a view to disseminating knowledge, but to show the students the elementary conditions and the procedure to adopt with them, in forward lines. During part of this month, too, they are attached to hospitals and units, to accustom them to the routine of these—in hospitals they serve much as clinical clerks and should obtain some insight into the Army's maze of forms and papers.

Towards the end of the third month, comes the examination. but let me emphasise that the examination has been going on all the time—the student has been under the eye of 9 different instructors and each of these has to report on all the students of the course. To these nine reports is then added an oral examination, directed not so much to their medical and surgical knowledge, but towards ascertaining their suitability for army work and the type of work for which they should be recommended. These collated reports then pass to G. H. Q. and the student is posted to a temporary unit, but he is not lost sight of. All reports are circulated to the various sections and, eventually, in most instances, the student is posted to the unit for which he is considered most suited. The A. D. M. S. (T), with us today, may have further observations to make on this point.

How is the teaching done? The staff of the Centre consists of Commandant, Asstt. Commandant, etc. and eight clinical instructors, not doing the clinical, teaching, but tutoring the students after the lectures and clinics. The lectures and clinical teaching are undertaken by the "honoraries"—the consultants of S. Army and the specialists of the hospitals in the Poona/Kirkee area. There are 150 students per month, and they have to be divided into groups of approximately 20, for clinical instruction, which goes on all days of the week and all hours of the day. And when one month ends, another group of 150

immediately enters on instruction. All honour to the specialists of these hospitals, who, in an entirely honorary capacity, have to undertake this teaching in addition to the heavy hospital duties of the Poona group of hospitals and then, in addition, smile as they are called to the Review Board's extra penance.

The Centre does not turn out furnished G. D. O's. They have been put on the lines, but are far from completely trained for this job. Their training must be continued. The specialists of the hospitals take up the task at this stage; we look to you to continue the training which we have started. We present you with better material than we of the A. M. T. C. got, yours will be the success or failure of the I. A. M. C. in the future. This training has not been proceeding satisfactorily in most hospitals, but it must do so. G. H. Q. has found it necessary to issue definite instructions, which you must carry out. 'Go to it', the task is worthy of you and you will not fail.

(The graphs, showing the comparative merits of graduates and licentiates, were demonstrated by epidiascope).

49. LT.-COL. S. N. HAYES—"*The Role of Medical Colleges in War*" **(P)**

The functions of Medical Colleges in war, are similar to those of a factory in Commerce, i.e. to produce large quantities of specialised articles, at the earliest possible moment. As it is our usual custom to be unprepared for war, and we more or less cheerfully embark on these enormous and complicated enterprises with very scanty equipment and organization, complications usually arise. In commerce, complications and deficiencies are fairly easily rectified, and production can soon comply with demands.

In our profession and particularly in this country, it is not quite so easy to arrange for production to satisfy demands. The time-lag and lack of preliminary organization, are important factors, but there are also many other difficulties which mitigate against speedy expansion, on the production side, and which need not be discussed at present.

For war, the Army requires and demands, specialists of all kinds, and also large quantities of unskilled M. O's. In fact, the Army is always demanding specialists and large numbers of M. O's. It has become a habit, and a most inconvenient one for me. As Principal of this College, I am in a somewhat similar position to the Magician who produces rabbits out of a hat. I have done my best, but must inform you that I am now getting somewhat short of rabbits. As, however, more are required, it is as well to take stock and review the situation, at least in so far as this particular production centre is concerned. Probably what I say will apply to other Indian colleges—when I speak of Colleges, I mean the combination of a College and its associated Hospitals, comprising an Educational Centre.

The Organization of the College in Peace-Time

It is well to appreciate that we are not so well organized as similar Western Colleges. I say this without prejudice. The country is large, Colleges are few, and, consequently, the number of graduate doctors is small. Western Medicine is not universally accepted and, consequently, public opinion and support are almost nil. Specialisation is as yet in its infancy. The continual cry of financial stringency imposes restrictions on development that are regrettable, as it is impossible to make do with one anna—when one rupee is needed. There is, however, ample first-class, keen-brained human material available and awaiting the time for the tempo of progress to be accelerated. When this occurs, I shall have to modify my statement so far as this College is concerned, and shall be glad to do so. The materials are available, only waiting to be utilized.

In peace-time, we are equipped with the barest minimum of staff, to train 75 students yearly,—we actually admit 90. There are today, 540 students on our rolls. The response of these boys to war demands has been admirable, and each year, 60 per cent. of our graduates are joining the Army. You probably require more.

Unfortunately, we do not, in our training, progress beyond the graduate stage. This is a defect that, I hope, will shortly be remedied. It is however present and, if we do not train post-graduates, where are the Specialists for the Army to come from? You can, perhaps, tell me.

I think that I am right in saying that post-graduate specialist training is neglected in all colleges in India. If I am correct, it is a regrettable fact and requires rectifying. In

the West, the foundations of specialised professional knowledge, lie in the group of people between the Resident and Professional grades. In this body, lies the strength of the profession, when it organizes for war. It provides for expansion and elasticity and gives us our war-time Specialists and Consultants. I have stressed our deficiency in this section, for definite reasons, i.e.:

(*a*) To point out that, if Specialists are required for the Army or even Civil purposes, then the organization must be made available for their training.

(*b*) To stimulate interest in this important subject.

I have mentioned that, in peace-time, we work with the barest minimum staff. Take, for example, a Medical Unit—we have two such units in this College. The staff per unit consists of:

One Professor.

One Clinical Assistant.

Four Residents.

They control approximately 100 beds and are also supposed to control medical out-patients. It is a very small staff for this purpose, particularly from the teaching point of view. I always describe it as 'all head and limbs with very little body'—in the form of Registrars, post-graduates, and others in the post-graduate stage.

What are the effects of War on our Peace-time Organization?

The effects on an organization with the barest minimum staff, cannot be expected to be good.

Those of the staff, who were seconded from the Army in peace-time, except those in reserved appointments, were recalled. Five out of 10 experienced Professors and teachers have left for the army and this is a large number. Their places were taken by experienced Juniors—whose places, in turn, were occupied by inexperienced juniors. In addition, some senior Assistants have joined the Army.

Formerly, residential appointments were for a period of twelve months and an endeavour was made to obtain these residents for the Army. They were valuable material. The scheme was not a success, and the appointments have been reduced to six months and the incumbents may or may not join the Army. Very few do. The value of our post-graduates to the Army is thus small.

The demands of war have weakened us considerably, from the teaching point of view and, remember, a good deal of routine work has to be carried out by the staff, in addition to teaching work. At present I feel that we are rather like a sausage machine, turning out sausages—many of which are of rather inferior quality. If Teaching Institutions are weakened too much, something is bound to happen, and does. It may be, and has been, argued—"Look what we did in the last war" and "What do we do in England". Well, this is not the last war, nor are we in England. Conditions are very different.

If the Army still requires Specialists and non-specialised Doctors, and, I gather, it does, I feel that it would be wise thoroughly to investigate the ways and means of production.

The Requirements of the Army, so far as I am aware, are for:

(1) Specialists of all grades and types. The better trained and experienced they are, the more they will be welcome, but those of various grades are apparently acceptable and fit in with the Army organization.

(2) Graduates, or non-specialized men.

(3) Facilities for training selected non-specialized men in special branches.

This College and its associated hospitals can provide quite considerable facilities for providing the above, and if required, a group of Experts—if possible unhampered by official procedure—could very quickly produce the facilities we do not possess, and, by co-operative effort, we could establish a very fine training school, which would be of considerable benefit both to the Army and to the Civil.

We have magnificent buildings, unsurpassed clinical materials in abundance, a lot of equipment and a very considerable amount of keenness and desire to help. Many of our

students are really first-class material and only require care, to make them into Doctors and Specialists of the first class. Many of those who qualify and join the Army, wish to receive post-graduate training. It can be and should be given to them. An additional 6—8 months, as Residents, for selected men, would pay good dividends. It is my opinion that it is unsound policy to absorb into the Army, *all* newly-qualified men. Let the best have 6—8 months post-graduate training. They will benefit, so will the Army.

A small example of satisfactory and productive co-operation, is the presence of the Army Anaesthetic Class in the Mayo and Lady Willingdon Hospitals. This scheme is working well, to the mutual benefit of both sides. We provide far better and more material than the Army can provide. They give us something which we lack. I see no reason why this cannot be extended to all clinical or non-clinical departments.

In a war of this magnitude, it is, in my opinion, unwise to deplete the centres of production. No sound business man would do so with his business. Why should we? If increased production is desired, the centres should be strengthened, not depleted.

If Specialists are required, there is only one way of procuring them, i.e. by training. Most of the facilities are available—these can be augmented. Why not utilize them?

The Army requires well-trained graduates and lots of them. The quantity must necessarily be limited, unless the present methods of education are thrown on the scrap-heap for the duration of the war, and the system of training re-organized and shortened. We have, in fact, reduced our course from 5 to 4½ years, and, by various means, pepped up production to its maximum—but more still could be done. The result is that a boy can fail in his professional examination, but can still qualify in 4½ years.

Quality will necessarily depend upon (1) selection, and, (2) the quality of the teachers. If policy dictates that Colleges must be denuded of experienced teachers, then you must be satisfied with what you get. The fault is not ours. If quality, however, is required, then experienced teachers are an absolute necessity and must be provided. I view with the greatest concern, the removal of experienced Professors from teaching Units to appointments such as Medical and Surgical Specialists in the Army. Such a procedure may be a matter of expediency, but it is not one of logic. It has been said that good class G. D. O's. are required. There is only one way of obtaining them, i.e. proper training.

Let me give an approximate and theoretical estimate of the minimum that could be done by the Colleges of this country if they were deliberately organized for one purpose only, i.e. the post-graduate training of Specialists, for a period of 6 months, taking the example of Medicine at this College and Hospital. I do not, of course, for one moment think that a real Specialist can be trained in 6 months.

Unit	No. of Units	Post-graduates in training for Army	Period of training	No. per annum	No. of Universities	No. per annum	Years of War
Medicine ..	2	4	6 months ..	8	8	64	4

War period = 4 years. Total = 256.

These figures should give sufficient food for thought.

I personally feel that this College is not playing its full part in a war of this magnitude. True, we have sent a large number of men, but have we really got down to facts and considered how best we could utilize our resources, either alone, or in combination with others. Have we, like Industry, planned for periods of various lengths? The answer is, I feel, we have not. We have depleted our machinery of production, while still required to produce more, and our standards are falling.

Perhaps my somewhat disjointed remarks may be of value in the next war, or, perhaps, as there is little likelihood of an early cessation of hostilities, and the complications of demobilisation will take some time, they may be of value in this. In any case, I can assure you that this College and its associated Hospitals, will readily co-operate in any scheme that is likely to benefit the Medical Services of the Army, and improve the training of Medical Officers.

50. COL. WHITE—"*I. A. M. C. Training*" **(P).**

In my opinion, no matter how brilliant and painstaking Military Medical Officers may be, the best, or even good results in treatment of sick and wounded, will not be obtained unless subordinate ranks are efficient and reliable, as so much in medical work depends on routine and detail, which the medical officer cannot possibly carry out himself.

To produce an efficient and reliable subordinate medical service—or any other service for that matter—it is essential that two main conditions be fulfilled. These are:

(*a*) The right type of material must be obtained; this, under conditions of voluntary enlistment, as in India, can only be obtained by offering attractive terms, in the way of pay and/or rapid advancement.

(*b*) Training arrangements must be adequate.

Prior to April, 1943, neither of these conditions was, in my opinion, fulfilled. Basic pay had, I admit, been increased to that of the Infantry soldier, but Trade Pay was very low and prospects of promotion, particularly to V. C. O. rank, negligible compared with other Arms. The result of this was, that the then I. H. C. had to accept material which was more or less unacceptable to any other Arm, or go without. Also, Training was not anything like as efficient as it should have been. The main defects being lack of adequate Instructors Establishments and Training equipment, particularly at Training Centres.

This was the state of affairs at the end of 1942 and it was obvious that, unless something big was done, there was a definite danger of a breakdown in the Medical Services in India. Drastic action to improve the subordinate Medical Services was initiated, with the publication of A. I. I. 114/43. This Army Instruction went a long way to solving the problem of attracting suitable material, in that it greatly enhanced the prospects of promotion, demanded a higher standard of education of recruits for the Nursing Section, and more than doubled this Section's pay. It also provided for the recruitment as V. C. O's, of men of a high technical and educational standard, for duty as Male Nurses, Radiographers and Laboratory Assistants.

From early 1943, the question of Training has been given very serious attention by the Medical Directorate and very definite advances have been made. These include:

(1) A substantial increase in the Officer, V. C. O. and N. C. O. establishment of Training and Depot Centres.

(2) Authorization of additional equipment for Technical Training, such as complete equipment for a 26-bedded ward for training of Nursing Section, and typewriters for training of Clerks, and equipment for an Ordnance Museum for training Storekeepers.

(3) Selection of certain large hospitals, in each Army or Command, at which definite courses of instruction for Nursing Section personnel, cooks, etc. are arranged.

(4) Definite courses of instruction under the appropriate Specialists, in X-ray, Laboratory Work, mental nursing, special treatment orderlies, etc.

Training

As you know, all recruits enlisted for the I. A. M. C. are sent to one of the three Training and Depot Centres, where they receive preliminary training, for periods which vary for each section. At the conclusion of this preliminary training, they are posted for practical training in their particular branch, to large Garrison Hospitals.

Training at Training Centres consists of basic Military training and elementary technical training. Basic Military training includes Education, both general and welfare, P. T., foot-drill, anti-gas, first-aid, hygiene and sanitation. In fact, with the exception of weapon training, it is exactly the same as that of an Infantry soldier. While basic military training must remain the main task of Training Centres, a considerable amount of technical training is now being carried out, with the result that nowadays a recruit, on completion of his preliminary training, should be disciplined, fit, well nourished, sufficiently educated to absorb instruction, and possess an elementary knowledge of his trade. Time and the supply and demand situation, do not permit of Training Centres turning out a finished article, therefore Officers Commanding and all Medical Officers attached to medical units to which these men are posted, must ensure that the post-Centre training is taken seriously and run on definitely organized lines. As I have already pointed

out, technical training is now receiving much more attention than heretofore, and I should like to describe briefly what is being done. in this respect, in each of the various sections of the I. A. M. C.

Clerical and Store

Clerical and stores sections do a period of 12 weeks, in Training Centres. Men of these sections are not trained or experienced clerks or storekeepers on enlistment. The only qualification necessarily, is that they must be of a certain educational standard, which includes a good knowledge of English. They must therefore be trained at both Training Centre and medical unit, in Clerical and Store duties. At the Centres, educational training is concentrated on English, with the object of bringing them up to the I. A. 1st Class English standard. Clerks are taught the elements of office procedure, introduced to the various Army Forms, Books and Regulations, and a considerable time is devoted to instruction and practice in typing. Similarly, storekeepers are introduced to office procedure, Books, Forms etc. and have the various items of equipment and stores in use in Medical Units explained to them, in a Stores Museum.

On conclusion of Recruits' training, they are posted to large hospitals and count against the authorized establishment. These men are not fully trained clerks or storekeepers when first posted, but they do know the elements of their trade, and, if O. C. units to which they are posted, take a little trouble to see that they get definite instruction in their duties, they can, in time, be turned into quite efficient clerks and storekeepers. They are no better, but certainly no worse, than the clerks and storekeepers of other Arms, and when O. C. Medical Units complain of the inefficiency of this type of personnel, it is often a reflection on himself that he has made no effort to have them trained.

Nursing Section

Since April, 1943, the Nursing Section has been completely re-organized, as regards selection of recruits and their training—the object being to attract an intelligent type of man, and train him up to a high standard of efficiency, with a view to raising the standard of nursing in medical units and to compensate, to some extent, for the shortage of Nursing Sisters. There are three categories in this section, (i) V. C. O. cadets, (ii) Specialist Improvers, and, (iii) Nursing Orderlies.

V. C. O. cadets are, after training, employed as Male Nurses, Laboratory Assistants or Radiographers. To be eligible for enlistment as a V. C. O. cadet, the recruit must, in the case of male nurses, hold a Civil Nursing Diploma and be registered with one of the Provincial Nurses Registration Councils.

In the case of Laboratory Assistants and Radiographers, the recruit must be a Graduate of Science in one of the recognized Universities. V. C. O. cadets spend four weeks at Training Centres, where they are given basic military training only, after which Male Nurses are posted to Training Centres or large Garrison Hospitals as Instructors in nursing duties. Laboratory Assistants then undergo a four months' course of intensive training at selected District Laboratories, and Radiographers four months at the X-ray department of selected hospitals, after which, they are employed in laboratories and X-ray departments, but continue their training and are not graded as Class I until they have had a total of 19 months' experience and have passed the prescribed examinations.

The minimum educational standard now required of a Nursing Orderly recruit, is Anglo-Vernacular Standard VI, which means that, on enlistment, he has a fair knowledge of English. Nursing Orderly recruits are given a period of twelve weeks training at Training Centres and a further period of 8 weeks at specially selected Garrison Hospitals, before they count against authorized establishments. At Training Centres, education is concentrated on English, and all recruits must pass the third class I. A. English examination, before leaving the Centre. Non-Urdu speaking recruits are taught spoken Urdu, up to third class Roman Urdu standard. The last six weeks of the recruit's stay at the Centre, is almost entirely devoted to elementary training in nursing duties. This nursing training is under the direction of the Technical Training Officer, who is assisted by a Nursing Sister and a staff of male nurses and specially selected N. C. O. instructors. Training is carried out in a fully-equipped ward of 26 beds and consists of elementary Anatomy and Physiology, bed-making, taking and recording of temperature, pulse and respiration rates, taking of laboratory specimens

such as blood-slides etc., giving of enemas, bed-pans etc., administration of medicines etc. In fact they obtain an elementary knowledge of all the duties which they will have to perform as nursing orderlies in a medical unit. At the end of this, an examination is held and only those who are considered up to standard, are sent to hospitals for further training. Certain large Garrison Hospitals in each Army or Command, have been selected as training hospitals for nursing orderlies, male nurses, and N. C. O's, additional to the authorized strength, are provided for whole-time employment as Instructors. The course of instruction at training hospitals lasts for 8 weeks and consists of instruction and practical demonstration of all branches of the nursing orderly's ward work. At the end of this training, another examination is held and those who are up to standard are graded as Nursing Orderlies Grade III, after which they may be employed in large hospitals against authorized establishment but must remain in large hospitals and receive further training, until they have passed the second Grade Nursing Certificate. Matrons have now been posted to each Army and Command, for whole-time duty in connection with the conducting of Nursing Certificate Examinations and this should have a very marked effect on the standard of training.

The minimum Educational standard of Specialist Improver recruits, is Anglo-Vernacular. IX. The object of introducing this class was to attract well-educated young men, who would be suitable material for training as Male Nurses, Laboratory Assistants and Radiographers. The preliminary training of these recruits is carried out at the Poona Centre and is similar to that of Nursing Orderlies in the early stages, and thereafter to that of the V. C. O. cadets.

General Section

General section personnel do a period of six weeks training at Training Centres and are then posted against the authorized establishments of medical units. The supply and demand situation does not permit of their being posted surplus to Establishments for training. The technical training of the general section, particularly of cooks, has recently been given attention by G. H. Q. and special courses of training for cooks have been introduced. Cooks I. T. must now, while at Centres, be given regular instruction in cooking the ordinary Indian Troops' ration, and are not allowed to leave the Centre until they have passed the Trade Test for Cooks I. T. They are then posted against the establishments of the selected large hospital, where they receive further instruction in cooking, particularly hospital cooking, until they pass the appropriate Trade Tests for hospital cooks. Similarly, B. T. cooks are posted from training centres to large British hospitals and are trained in B. T. cooking.

In my Centre, I have managed to beg, borrow or steal a certain number of Instructors and some equipment and I am running definite courses of technical instruction for all categories of the General Section, e.g. Ward servants are taught how to lay tables, serve British food, dust wards, etc.; washermen how to wash and iron; water-carriers to fill basins, lay out towels, soap nail brushes, etc.; sweepers how to make and use brooms, give urine bottles and bed-pans, etc., in addition to ordinary latrine work.

With the new terms of service and training facilities as they are now, I am convinced that we can turn out a really good subordinate medical service, provided that every Medical Officer, whether he is specially charged with training or not, takes an interest and makes a definite effort to maintain esprit de corps, and instructs them in their duties. They are very willing to learn and react well to any efforts made on their behalf but they also react to lack of interest. Medical Specialists can, I think, do a great deal in the matter of training of other ranks, and a little extra time and care devoted by them to it, will help greatly to raise the standard of nursing, feeding, etc. in Medical units. I could say a lot more about lack of interest in our subordinate ranks, but I dare not do it.

51. COL. CAMERON—"*Training of Medical Specialists*" **(D)**

The training of and reporting on potential physicians, is another call that is made on Medical Specialists. The present shortage is approximately 40 per cent. Few are to be available from the U. K. in the near future, and so they must be trained from resources in India. We must beware of grading physicians who do not reach standard. Better a shortage of physicians than 'physicians' who do not fill the part. I am of the opinion that training by attachment to different types of medical wards and visits by O. i/c Divisions, to these wards, accompanied by the trainee, are of greater value than examinations of the trainee's knowledge. We want practical clinical physicians not those laden with academic knowledge.

The War Officer desires the discontinuance of the terms 'recognised and graded specialists'. They will now be classified as 'medical specialists' or 'graded physicians'. Medical specialists require a minimum of 7 years graduate qualification, 5 years in the speciality, and a higher qualification. Graded physicians require 3 years graduate experience, of which 2 must be in their speciality, but a higher qualification is not a sine qua non, though desirable. Graded physicians will still be eligible for advancement to medical specialists, after one year, but it must be emphasised that this is by no means automatic and will be strictly reserved for those showing distinct ability and progress as physicians.

52. LT.-COL. G. T. HAYES—"*Training*" (D)

You have heard a very excellent résumé on Medical Training, from Colonels Cameron and White. It is not possible, in the time available, to give you a detailed exposition on training. I do, however, wish to mention some aspects that need to be stressed.

The D. M. S. gave us certain figures, i.e. 1,700, now swollen to 6,000, and the I. A. M. C. other ranks are over 120,000 strong, and a large part of this expansion has taken place in the last two years, and existing establishments are still not complete. What does it mean?

A minimum training period in Training Centres, and, at the same time, give the man a sound basic training, so that he can undertake his ordinary duties in a unit. But his post-recruit training must be continued in whatsoever unit he may be posted to, if we are to maintain that tradition which the Medical Services have set in the Great War and in this war and of which we are justly proud.

I travel hospitals in every Army. I appreciate that their job is not an easy one, owing to shortage of staff, shortage of equipment, etc. But there is still a lot that can be done in hospitals and field medical units by the staff, to improve the training of the personnel. By improving the training of the Nursing Section, ward servants, cooks, etc., you lighten your own burden. Your standards of treatment are raised and your patients are discharged more quickly. And, you have another responsibility, in fact, I would say, a duty. These personnel will, in the near future, go as reinforcements to operational areas, and it is our duty to give them that training, technical and military, which will enable these men to carry out their duty efficiently and, at the same time, protect their own lives. Now you, as specialists, are in a position to give invaluable help in the training of personnel. How are you going to do it?

(1) You travel from hospital to hospital and are in a position to assess the standard of training of the personnel in the various hospitals.

(2) You can see various methods of implementing the syllabuses, as laid down.

(3) You can discuss with the O's. C. concerned, the various ideas and advise them.

(4) An important part of the training, of Nursing Section particularly, is the co-operation given by medical officers, in regard to lectures, demonstrations, etc. You can make enquiries in this respect and encourage medical officers to do so, and on your visits enquire as to what extent they are co-operating.

Medical Officers

Again, we have the same difficulties in regard to shortage. On completion of their course at the I. M. T. C., officers are recommended for various duties, i.e. R. M. O., Field Ambulances, C. C. S., Field Hygiene Sections, Hospitals, Combined Operations. These recommendations come to G. H. Q., they are then sent to the section concerned, viz. Field Hygiene Section, to D. M. S. 5. As and where such officers are required, they are taken from these lists. So, even though the officer who is recommended for Combined Operations is first posted to a hospital, it does not mean that he is lost sight of. Other things being equal, he will end up in the unit to which he has originally been recommended.

Tactical School recommendations: These should, if possible, be posted for a period of duty with a Field Ambulance in the Army/Command, then sent to the Tactical School.

Combined Operations: H. M. S. Salutte, Bombay and Karalevasala. As far as possible, we endeavour to give the young officer that training which will enable him to undertake his duties from the day he is first posted to his new unit.

Liaison

This seems, in the past, to have been sadly neglected. It is somewhat difficult for those who have not been in jungle, to appreciate to what extent the principles of training can be

varied in operational areas, particularly Burma and Assam. Though the principles remain the same, the methods of implementing them vary. Proposals are being considered whereby those connected with training will visit other training centres, see how they work, and also visit units in operational areas. They can then see to what extent the basic training, given to the man, has fitted him for his role, and what modifications or elaborations are required. And, I know that I speak with the consent of those concerned, when I say that, should you in your tours, be in the vicinity of a medical training centre, a visit from you will be welcomed. You can then see and appreciate training centre difficulties, the recruit on his first day at the centre, and on his final day. Only by such co-ordination and liaison on the part of all concerned, will we achieve what we mean to achieve.

53. MAJOR KILOH—"*Medical Training*" (D)

My first suggestion, in regard to Medical Training, is that this Conference must be repeated sooner or later. In connection with the publication of a 'Proceedings', I would say that this is an absolute necessity and the professional side of the Conference will be largely wasted, if such a publication is not available. I was fortunate enough to attend the Eastern Army Conference last year, and the Proceedings of that Conference is a veritable mine or information of a practical nature. I draw the attention of the Editorial Committee to the paper shortage and the consequent difficulty in persuading the authorities that such a publication is in the interests of the war effort.

From the point of view of clinical meetings and lectures, these must be kept up. In my experience, there does appear to be a considerable degree of apathy and lack of interest, which, unfortunately in my opinion, is largely basic, fundamental and inherited, and if these meetings were not compulsory, I fear that the attendance would frequently be very small indeed. There are, of course, excuses for this state of apathy, too, such as small staffs and consequent overwork, people being on duty at the time or on tour, and perhaps also the fact that, for weeks on end, the medical population remains constant and the same voices and faces tend to become, after a while, boring. I feel, however, that the Specialists in general are at least partly at fault and it is up to us to introduce variety into the meetings and ring the changes. I would suggest that symposiums be held, for instance, the subject might be "Headache", given from the various Specialists' points of view, and so on. One great difficulty is to get officers to rise spontaneously and express their thoughts or even ask questions. To combat this, I would say that, in general, Specialists should never show cases, but should reserve their energies for organising the Meeting and summing up the discussions. If necessary, officers should be prompted to say a few words on a certain case, or to ask pertinent questions in respect of it. One of the great advantages of combined meetings of hospitals in an area, is that the staffs of the two hospitals can meet in social intercourse and get to understand one another's difficulties, and, from the professional aspect, study the vastly different material that each hospital has to show. To further this, I have been in the habit, whenever there is an opportunity, of conducting M. O's. of one hospital round the wards of the other. Finally, in this respect, I would humbly suggest that, when Consultants from G. H. Q. or the Commands visit hospitals, they should lecture to the M. O's. or take part in a clinical meeting. Recently, Colonel Schlesinger visited my Station and took an active part in a clinical meeting. The presence of a strange face and, if I may say so, the face of authority, was like a breath of fresh air. In my Station I plough a lonely furrow and whatever I say, goes. To have a Consultant examining cases, is not only good from the M. O.'s point of view, but is excellent for me too.

Now, to divert to the problem of the graded Specialist. It must be accepted that, in future, the standard required for grading will be lower than hitherto, for obvious reasons. This makes it all the more important that officers graded, should be even more carefully selected and vetted than hitherto. I am in the unfortunate position of having candidates sent to my Station, for my observation and report on their suitability. In my opinion, this is neither fair to the candidate or to myself. I suggest that it would be preferable if teaching centres were set up in base hospitals, where there are teams of medical specialists and better supplies of clinical material. Lectures and demonstrations could be arranged for their benefit and, at the time, attention paid to instruction in elementary Radiology, C. S. R. methods, etc. The final verdict would be given by three Specialists, comparable to a Board, instead of by one. In giving such an opinion, I would stress, too, the character and personality of the candidate. In giving an opinion, recently, I said that, although the candidate's professional ability was probably up to standard, I thought that he should not be graded, because his personality was

bad and his character was suspect. I was told by a senior officer that I was only asked to report on his clinical acumen. This, in my opinion, is not in the interests of the service.

In conclusion, I would mention the Nursing Officers. These Officers arrive in this country without any training whatsoever in tropical diseases and nursing. I have never met one who has been given any training, although I am aware that, rarely, this is given by Specialists. I feel that it is our duty to organise courses for them in the hospitals in which they work. It is obvious that, if they know why it is so important to take blood smears, send stools to the C. S. R. quickly, and generally have a knowledge of tropical medicine, they will take more interest in their work and this will go far to make a well-knit hospital team.

DISCUSSIONS

54. MAJOR-GENERAL MARTIN congratulated Major Kiloh upon his paper and appreciated the difficulty of getting up regular clinical meetings. He pointed out the responsibilities of officers of high rank, Lt.-Colonels and Majors, acting as officers in charge of Medical Divisions and Medical Specialists, corresponding in a Unit to O. C. Battalion and 2nd in Command in peace-time organisation, and the importance of keeping acquainted with elementary rules and regulations.

55. LT.-COL. DAUKES gave his experience at a hospital, where he found the blood-slides being taken badly. He, personally, gave two lecture demonstrations to all the Ward boys, using such Urdu as was at his command. It resulted in a very great improvement at the time and showed that ward boys could be trained, if a personal interest was taken by officers and the matter was not just left to the V. C. O's. Owing to frequent changes of staff, these lecture demonstrations needed to be given at frequent intervals.

56. LT.-COL. PASRICHA commented on the poor type of medical candidates in the present-day Army, and said that, out of 40,000 medical men in India, 6,000 had joined the Army. He agreed with Lt.-Col. S. N. Hayes, regarding depletion of experienced teaching staff from the Colleges and its consequences of inexperienced subordinates having to take their places. With regard to the clinical meetings, he said that there was a tendency among the Specialists to quote authorities, syndromes, and to demonstrate rare neurological conditions, and suggested, instead, the holding of symposiums.

57. LT.-COL. PRESTON said, "The Conference should be grateful to General Cursetjee, for his condemnation of 'Mechanical Medicine'. So many X-rays are asked for, not because it is thought that they will really add useful knowledge, but because the M. O. feels that he must 'cover himself',—a result of years of civil practice, when the doctor is liable to be mulcted for damages. It is hoped that the Chairman's views are shared by the Medical Directorate.

58. MAJOR-GENERAL CURSETJEE agreed with Lt.-Col. Daukes, regarding the importance of personal attention to minor details and cited a case of a British sergeant who, with no knowledge of Hindustani, trained Indian stretcher-bearers, entirely by personal demonstration. Commenting on Lt.-Col. Preston's remarks, he said that the control of X-ray films was being exercised by the administrative officers, but, in certain cases, such examination became necessary for complete investigation.

59. LT.-COL. KELSALL said, "I agree, very largely, with Lt.-Col. Preston in his criticism of 'mechanical medicine', but wish to make one point in regard to X-ray examinations; practically every British soldier whom I have seen in India, with Pulmonary Tuberculosis, was suffering from advanced bilateral disease, when the condition was first diagnosed. In my opinion, there would be a real danger of such tragedies being multiplied, if the use of X-rays was discouraged in chest cases, since, any physician must admit that X-ray provides the only method of diagnosing Pulmonary Tuberculosis in the early stages."

60. BRIG. McALPINE said, "Major Kiloh has raised an important point, namely the further training of Nursing Sisters, more particularly, those freshly out from the U. K. At one hospital in the Fourteenth Army, the O. C. Medical Division gives lectures to the Sisters of the hospital on the principles underlying the medical treatment of the commoner diseases met with in India. Cerebral malaria, water with salt balance, and malnutrition, are among the subjects which he talks about. These lectures are populuar and have resulted in the best standard of nursing that I have seen in any military hospital in India. The Chief Principal Matron has approved of similar lectures being held at other hospitals, whenever they can be arranged. A

syllabus is being prepared, which, after approval by the D. M. S., will be distributed. You, as Medical Specialists, will be called upon to do this teaching, which, if properly carried out, should make the average Sister's work more interesting and more efficient."

61. LT.-COL. S. N. HAYES, in reply to Lt.-Col. G. T. Hayes, (G. H. Q.), pointed out that, in any question concerning the war effort, the Punjab Government was willing to co-operate in any way possible, in accordance with its policy of a 100 per cent. war effort.

He advised those who were new to India, to pay personal attention to the training of ward boys. When such attention was given, it was surprising how quickly an uneducated boy picked up technical work. Without personal attention, they were inclined to lose interest.

62. MAJOR-GENERAL CURSETJEE emphasized that the Nursing Officers should be encouraged to pick up the language and agreed with Lt.-Col. S. N. Hayes. He added that the officers should ensure that their orders were carried out, especially with regard to the feeding and clothing of patients.

63. MAJOR TREDGOLD: Col. Cameron very kindly mentioned work done on I. A. M. C. officers at Poona. This is, so far, very incomplete, but does support the contention that candidates should be seen by a Selection Board (as for other arms), which, by a combination of psychological tests and interviews, could assess the man's military as well as medical value. The rejection rate would, of course, be governed by the supply and demand, but, in any case, it would be of value to know for what type of work the officer is most suited.

64. LT.-COL. CAMERON: Many points of importance have been raised during the discussion.

It is apparent that, for satisfactory observation of gradees, only selected hospitals can be used. Steps will be taken to ensure that this is fully considered. It is unfair to specialist and to gradee that the report should be based on a single opinion. An opinion as to the personality etc. of the candidate is of the greatest value. It is likely that the form of report for trainees will be radically altered, and the new form will call for an expression of opinion on such vital points.

General Cursetjee's remarks on "mechanised medicine" cannot be overstressed. We must continue to be a clinical service and keep mechanisation in its proper place, as an adjunct to clinical medicine.

We must grasp all opportunities for training of personnel. Lectures to Nursing Officers are eminently desirable; no less desirable is the instruction of nursing orderlies.

DYSENTERY

65. LT.-COL. COBBAN and **MAJOR HARVEY**—"*The Management of a Dysentery Ward*" **(P)**

Dysentery is probably the commonest disease—apart from malaria—with which we have to deal, and if not diagnosed early and treated correctly, it produces prolonged invalidism. It would be as well, at this juncture, to apologise for the apparently elementary nature of this paper, but we have found in the hospitals we have visited, that there is often little attempt at accurate diagnosis and rational treatment.

Dysentery Wards are, in our opinion, the most difficult to manage of all wards in a medical Division, especially in an Indian hospital. They call for painstaking care, much of the work is unpleasant, and results are often slow to materialize.

The management of a dysentery section can be considered under the two main headings of (*a*) Diagnosis, and (*b*) Treatment. This section of a hospital should take not only frank dysenteries but also diarrhoeas.

(*a*) Diagnosis

The essential points to be considered in ward organisation are arrangements (1) for stool examinations, macroscopic, microscopic and by culture, and (2) for sigmoidoscopies. The personnel here concerned are the sweeper and the medical officer, but the co-operation of the patient is, of course, also important.

(i) *Stool Examination*

The patient must be told how important is the examination of a fresh stool, and he must be instructed to pass the stool into a clean bed pan, free from antiseptics and not to contaminate

it with urine, and to inform the sweeper or orderly as soon as it has been passed. The bed pan should be labelled with the patient's name and bed number. The ward sweeper plays a most important part in the organisation of the ward, and a well trained and intelligent sweeper is essential in a dysentery ward. He must be made to understand the necessity of taking all specimens from fresh stools and he must be taught which is the best portion of a stool for culture and for microscopical examination. It is his duty to pick out such a portion and put it into glycerinated saline, to be sent to the Laboratory for culture. He should obtain a specimens from the first stool of any new patient and put it into one bottle and thereafter take specimens from several more stools, passed during the ensuing 24 hours, putting them all into a second bottle, kept labelled with the patient's name and bed number. He is further responsible for keeping stools in labelled bed pans, for macroscopical examination by the Medical Officer and for taking fresh stools to the clinical side room for microscopical examination. It is perhaps worth while mentioning that the sweeper should take the shortest route to the clinical side room—it has been known for him to take 2-3 hours and even to lose the stool on the way. In a big dysentery ward, it is worth while having one sweeper detailed for the above duties only.

The Medical Officer should himself carry out the macroscopical examination of all stools For obvious reasons, the best time for this is between 8.30 and 9.00 o'clock, before his morning round. He should of course make a detailed note of his observations, for use on his rounds. Stools of new admissions and of special cases may be examined at other times. For this macroscopic examination, there should be a room in, or adjacent to the latrine, fitted with fly-proof doors and shelves, fly-proof cupboards, and where also the sweeper can take and bottle the specimens for culture. The microscopic examination has to be made in the clinical side room, but should be carried out by the Medical Officer in charge of the ward. It is vital in the diagnosis of protozoal dysentery, that this should be made while the stool is hot, and it follows that the clinical side room must be situated near to the dysentery wards and that stools must be examined there as soon as they are received. This is the greatest difficulty in the accurate diagnosis of dysenteries by stool examination and is one reason for supplementing it by sigmoidoscopy.

(ii) *Sigmoidoscopies*

We consider such an examination to be an essential part of the diagnostic procedure and that it should be carried out on all cases of chronic or recurrent diarrhoea or dysentery, and, under certain circumstances, on acute cases also. It should also be done before discharge on all cases with amoebic infection. Sigmoidoscopy should be performed by the Medical Officer i/c of the Ward, who should be trained in the use of this instrument. It should be carried out in a special side room of the ward, equipped with an examination table, a steriliser, a microscope, slides, saline solution, which can be warmed, and glycerinated saline bottles. The numbers of positive results which result from immediate examination of fresh stools of specimens obtained by sigmoidoscopy and the difficulties of getting stools examined in the clinical side room, while still hot, make us consider that the dysentery section of a hospital should be equipped with its own sigmoidoscope and microscope and that the microscopic examination of all stools should be carried out in the sigmoidoscopy room of the dysentery section, instead of in the C. S. R. Furthermore, such a system should allow of a more central situation for the C. S. R. and, at the same time, diminish the risk of the spread of infection by flies.

(*b*) Treatment

In the management of dysentery wards, the two aspects of treatment which need consideration are (1) the provision of an adequate fluid intake in the acute stage, and (2) the provision of suitable and adequate diet in the recovery and convalescent stages. Here again, the co-operation of the patient himself must be obtained, both in the taking of fluids and in adhering to the diet ordered. The personnel concerned are the Sisters and orderlies, and the success of the treatment depends on their efficiency and hard work in carrying it out.

(1) In all cases except the mild ones, an intake chart should be kept, if only to impress on the nursing staff, the necessity for an adequate quantity of fluids, given at frequent intervals. Bed head reservoirs of salt and water, with rubber tubing and stopcocks are useful, as the quantity can be easily measured. Bottles containing glucose and saline, and intravenous equipment, should be kept in the ward, ready for immediate use.

(2) Diet, adequate in calories, protein and vitamins, suitable in type and taken over a sufficiently long period, is of vital importance, to obtain complete healing of the damaged mucous

membrane. This involves the preparation of a series of diets for use in the dysentery ward, otherwise the food given will almost certainly be unsuitable or inadequate. Some variation must be introduced by the use of alternative items, especially for chronic cases. If such patients do not get adequate and varied food, they are tempted to obtain it from other sources, and often thus undo the work of weeks of careful dieting. We have found it unsatisfactory and uneconomical to have these special diets prepared in the wards themselves, and consider that they should be prepared in a central diet kitchen, by cooks specially trained in invalid cookery.

There are various other points which we might have raised, from such matters as flyproofing and the provision of a sufficient number of bed pans, to the rather Utopian ideal of an adequate staff, with some permanency, which a specialised ward really requires, but we have endeavoured to bring out what we consider to be the most important points in the management of a dysentery ward.

66. LT.-COL. HINDS-HOWELL—"*Relapsing Amoebiasis*" **(D)**

I was extrordinarily interested in Col. Cobban's paper, because we in Karachi have been working along the same lines, although not so swiftly.

The first point I want to mention is amoebiasis of insidious origin. That this occurs, we all know or we would not be required to treat cases of amoebic hepatitis, and so on, in patients who stoutly deny ever having had dysentery. But, about that we can do nothing. More serious is the fact that amoebiasis is occurring right under our noses, and we are missing it. The majority of cases which I have to deal with, are chronic ones from Paiforce, who have had several courses of treatment, not always adequate, for reasons outside the control of the doctors, but nevertheless, several courses. Yet, still they relapse, so they are sent back to Karachi. We treat them for two or three months and, although sometimes we get the stools negative, sigmoidoscopy shows either chronic ulcers or a diseased mucous membrane. Eventually we send them to Poona. Paiforce cannot cure them, we cannot cure them and Poona cannot cure them, at least so I judge, from a scrutiny of my 'returned empties' from the Review Board. I went into the histories of these patients and found the following sort of thing. Dysentery started in 1942 in Burma or at Ranchi, just ordinary diarrhoea. A large number of these cases show no blood or mucus—and there is the rub. The treatment of these cases has been oil and salts, recovery for a month or so, relapse and more oil and salts; in fact, a trail of oil right across India, through Iraq, up to Persia. It is up to us to stop this trail of oil, by diagnosing these insidious cases earlier. That, then is my first point.

My second point, and I make no apology for labouring this point, is that stools must be examined hot—piping hot. Tradition in India dies hard, and it is a tradition that there is no amoebiasis at Karachi. Well, that just is not true. The reason why there was no amoebiasis, was because our technique was not adequate to find it. When I tell you that the technique involved the carrying of the stool in a bottle, by a sweeper on a bicycle, a distance of over two miles, I think I shall have said enough. Anyway, I have now instituted a 'from the patient to the microscope' service, which has improved matters. Soon I hope to get a 'from the ulcer to the microscope' service, but I have not yet been able to get the microscope.

Over my last point, I need your help. I have noticed that, in a few cases—fresh cases—even after a course of Emetine, vegetative forms of E. H. are still found in the stool. And they are still there at the end of the course of Carbarsone or E. B. I. What are we to do? Are we to repeat the courses of these drugs and ignore the effects of such poisons on the body? I have found that these particular Entamoebae resist all forms of combined treatment, they are in fact, 'Emetine Fast'. I would ask the meeting whether this is a common occurrence, and what to do about it?

67. MAJOR CHANDRA—"*Amoebiasis in the Gold Coast*" **(D)**

Amoebiasis is endemic in the Gold Coast colony, especially about the warm and humid coastal towns. Upcountry in the bush, 500 miles from the coast, where the climate is comparatively drier, I did not meet many cases. This report is based on 96 cases of amoebic dysentery and diarrhoea, under my care in a W. A. G. H. on the coast, covering a period of six months, during 1942. More than half the cases admitted with signs and symptoms of dysentery, were diagnosed amoebic dysentery. The diagnosis was based on the finding of vegetative E. H. in the stools, by the hospital pathologist.

I was asked to find a standard treatment with available drugs—Emetine and E. B. I., which would reduce the patients' stay in hospital, from an average stay of about 45 days.

The test for cure was prescribed as six stools negative to E. H. and its cyst, three negatives per week and sigmoidoscopy before discharge. This was in the hands of the pathologist and the surgeons.

24 cases were treated by each of the 4 following treatment:

(1) One lot of 24 were given Emetine gr. 1 a day for 12 days. Average stay in hospital in this series—40 days. Two relapsed within 3 months.

(2) Another series were given E. B. I. alone, 2 grs mixed in jam every night for 10 nights. Average stay in Hospital—42 days. One-third had to be given two courses of 20 grs each, as one course did not clear the infection.

(3) A third series were given Emetine gr. 1 a day for 12 days, followed by E. B. I. gr. 2 a night for 10 nights. Average stay in hospital—40 days. No relapse within 6 months.

(4) The fourth series had Emetine gr. 1 a day for six days, followed by E. B. I. gr. 2 a night for 10 nights. Average stay in hospital—33 days. No relapse within 3 months. This was adopted as the standard treatment for all subsequent cases.

Almost one-third of all cases were mixed infections, mostly with T. saginata, some with ascaris, schistosoma haematobium and ankylostomes. The average stay in hospital, in these mixed infections, was 52 days.

Five cases had complications—two developed hepatitis, one had persistent hiccough for 4 days, one persistent vomiting for 3 days, and one had extensive necrosis and separation of the rectal mucous membrane.

Chinioform or Yatren were not available. All cases had 2 per cent. soda bicarb. colon wash-outs every morning during the Emetine course and Stovarsol tablets for one week after the Emetine course.

68. MAJOR KILOH—"*Amoebiasis*" **(D)**

Yesterday afternoon, Brigadier McAlpine stressed the importance of the elicitation of a proper history and careful examination of clinical material. Today, Major-General Cursetjee quoted the words of that very great physician, Professor Ryle, on the same lines. One of the great benefits to medicine in the Army today, is that, owing to the shortage of X-ray apparatus and other methods of investigation, our clinical sense has been sharpened, a thing of the greatest value, which will benefit us long after the war has ended. In few diseases can it be said that the history and clinical appearances are more important than in amoebiasis.

We are all aware of those cases of amoebiasis which, although text-book in character, the E. H. cannot be found in the stools, even by an expert. Then there is the case of diarrhoea, who spends a varying period in hospital and who eventually clears up, only to be admitted at a later date with undoubted amoebic dysentery—this has, of course, been amoebiasis all along, and amoebiasis with a remission of symptoms. Thirdly, you have the case of amoebic hepatitis, who stoutly denies any history of dysentery or even diarrhoea, yet he must have had it. It was so insidious and so mild that no treatment was given, and, because of this, hepatitis developed. The absence of blood and mucus in certain cases must also be stressed. Finally, we are all aware of those cases where the diagnosis can only be confirmed by the use of the sigmoidoscope. For these reasons, it is apparent that amoebiasis is not always easy to diagnose and many cases must be missed, with consequent delay in instituting treatment.

In my touring, I find that many cases are missed, for the following reasons: (1) The lack of knowledge of the symptomatology and importance of clinical findings. (2) Too much importance is attached to the absence of blood and E. H. in the stools, a prevailing idea being that, in these circumstances, it cannot be amoebic dysentery. (3) Stools are not sent to the C. S. R. quickly enough. (4) There is naturally some inexperience in C. S. R. methods and the importance of the type of exudate is not realized; one hospital which I visited, had not had a case of amoebic dysentery for a whole year. (5) In many hospitals there is still no sigmoidoscope. (6) The pernicious use of salts, which I will refer to later.

Amoebic dysentery, in ordinary circumstances, can be a very difficult diagnosis to make, and is even more so, if a careful routine is not impressed on the G. D. O. and all the evidence carefully collated. Since I have been in this country, I have seen five cases, believed by me to be amoebic dysentery, in Medical Officers. These officers were intelligent, able to co-operate and give a reliable history. The usual investigations were negative. Had they been B. O. R's and possibly less reliable in their statements, and also because they were doctors and receiving

a little more careful enquiry than would otherwise have been the case, it is possible that specific treatment would not have been given and the good results achieved would not have been obtained.

I would, therefore, urge more attention to the history, coupled with a careful clinical examination, and it is our duty to teach G. D. O.'s the importance of this. We must, then be prepared to treat cases clinically, with a little more readiness than we do now. In this way, less will be the tendency to chronicity, to relapse and to complications.

To turn to a related subject. I would like to say a few words on the closely-related subject of the use of salts in dysentery and diarrhoea. This is a medical fallacy, comparable to the value of expectorants in bronchitis. In many hospitals, that I visit, I find that it is a routine treatment, given to anyone claiming to have diarrhoea of any sort, from the moment he is admitted, and I have the greatest difficulty in breaking this long-established habit. This does not appear to be a rational method of treatment, it distorts the clinical picture, tends to increase dehydration and alters the sigmoidoscopic appearance. From the point of view of amoebic dysentery, the amoebae do not like salts, they pre-cyst and become difficult of recognition. Amoebiasis thus tends to be missed in such cases. Apart from the fact that, if anything, an inflamed organ should be rested, I find that medical men themselves avoid salts in treating their own diarrhoea. The ordinary mild case is very satisfactorily treated by rest, fluids and an easily-assimilated nutritious diet. More severe cases require sulphonamide in some form, and those are the only two methods of treatment. The indiscriminate and routine use of salts in the treatment of diarrhoea, could usefully be banned by G. H. Q. There is no advantage, and many disadvantages in its use and, in these days of scientific medicine, it is nothing less than malpraxis.

69. MAJOR KAUL—"*Intestinal Amoebiasis*" **(D)**

I wish to emphasise three observations, one each in the (*a*) diagnosis, (*b*) treatment, and (*c*) prevention, of Intestinal amoebiasis.

(*a*) *Diagnosis*

It is quite well known that there are quite a number of cases of Latent Amoebic Dysentery. These are mild infections from the first, and only show themselves in chronic diarrhoea or, perhaps, diarrhoea, alternating with constipation. It has also been pathologically proved that many cases of amoebic ulceration of the large bowel, produce diarrhoea, without definite dysenteric symptoms. In diagnosing such cases, I have found the method described by Megaw, of washing stools, of real assistance. This consists of pouring water over the whole fresh stool and letting it stand for some time, after which, the supernatent faecal fluid is drained off, if mucus is present, it is found in the residue. In several cases of repeated diarrhoeas, where no exudate was found and stools seemed to contain no mucus at all, this process separated the mucus with ease. Smears taken from such samples usually bring a positive report of cysts amoeba, where previously, repeated examinations had failed to detect anything pathological, because of the fact that previous samples contained nothing beyond faecal matter. Such complications as amoebic hepatitis and liver abcess, are too well known to need mention, but I feel that minor grades of amoebic hepatitis are the direct result of latent amoebic dysentery. We have all met with cases of simple hepatitis, with mild grades of persistent pyrexia, in which all investigations have been negative and yet they have responded dramatically to emetine treatment. This was forcibly impressed upon me when, in the Middle East, I had 9 such cases under my care at one time, but, in spite of very careful history, taking, only 3 gave a history of previous attacks of dysentery. Of these cases, 8 responded to emetine treatment and recovered, one died of liver abcess, confirmed later by post-mortem. I venture to make another statement, that these days we see large numbers of Indian troops with large spleens and palpable or enlarged livers; the majority of these cases are undoubtedly chronic malaria, but there is a certain number of these cases who are both chronic malaria with chronic amoebic hepatitis. In support, I may quote Megaw, who has described amoebic cirrhosis of the liver in Indian patients.

(*b*) *Treatment*

Many physicians now consider the old established purgative treatment of dysenteries as scientifically irrational. I have seen it being abandoned in the Middle East in 1941, and again, last year, in at least one hospital in the Arakan. To irritate further the already inflamed and

ulcerated bowel, seems against the principles of rest that we advocate in all acute conditions. The idea of washing away the toxins or the infective organisms out of the bowel, does it really help? In amoebic dysentery, the toxins are not severe and the amoebae are already imbedded in the mucosa when the symptoms are developed; salines do not dislodge them. In my experience, the patients have done equally well, if not be better, on specific anti-amoebic treatment. In many cases they have recovered more quickly, without the aid of purgatives.

(c) Prevention

A small point that I observed in the Arakan, was the greater susceptibility of British troops, as compared with Indians, to amoebic dysentery. This may, perhaps, be explained by the fact that Indian troops probably acquire some degree of immunity, from subminimal doses of infection, which they continually meet all over India. In the Arakan, amoebic dysentery is endemic and more common than bacillary dysentery, particularly in British troops. In one General Hospital for B. T., out of 100 cases in a month, 62 cases were amoebic. The main source of infection appeared to be cyst-infected water supplies. I made an attempt to investigate the effect of chlorination on cyst-infected water, particularly the effect of super-chlorination on the analogy of effective superchlorination of water supplies for schistosomiasis, that we used to practise in the Middle East. Unfortunately, the facilities available at the Field Laboratory whose assistance I sought, did not prove adequate and so had to be abandoned. However, in my search for information on this subject, I found very good material in the work by Brady Jones and others, on the effects of chlorination of water and viability of E. H. cysts, the results of which may be summarized as follows: Chlorinated water in the usual concentration (1-2 pp. million), usually undertaken for sterilising drinking water when contaminated with viable cysts, with a contact period up to 150 minutes, gave positive cultures in nearly half the tubes. With water superchlorinated 15 times the usual concentration and cysts exposed in such water for periods of 15 minutes or less, nearly all the tubes gave positive cultures. Cultures made after over 15 minutes period, gave only 9 positives out of 150. It would thus appear that cysts cannot be killed by chlorine in any practicable concentration, unless exposure is well over 20 minutes. Obviously, more research is wanted on this subject and I would mention especially, electric current sterilisation of water supplies, which has been introduced in Assam on certain airfields for American forces. I do not think that this Conference will be interested in the details of this method. All I might say is that an electric current from a 6 volt car battery is all that is needed, with special electrodes. Research is required to know if this method, which kills all coliform organisms most effectively, can also kill E. H. cysts.

70. LT.-COL. BOSE—"*Treatment of Bacillary Dysentery with M and B* 693" **(D)**

Satisfactory results have been obtained in the treatment of bacillary dysentery by sulphapyridine, in small and frequent doses. Owing to the limited supply of sulphaguanadine and the massive doses which one has to administer to reluctant patients, trials were made with readily available and perhaps more acceptable M and B 693. The response to such treatment became manifest in 18 hours and the clinical signs and symptoms disappeared within 48 hours, in the large majority of patients.

Well over 100 patients—both British and Indian, including women and children—have been treated by this method during 1943, but records of observation are available for only 73 adult Indian cases. This batch of 73 consisted of: Flexner—58, Sonne—5, Schultz—5, and Exudate—5. There were 13 controls, to whom routine treatment with saline or Ol. recini, or both, was given; 14 semi-controls, who started with routine treatment, but, after 2—5 days, terminated with M and B 693; the remaining 46 received M and B 693 from the start. Notes of clinical observations were made on the frequency and character of stools, the presence of blood and mucus, abdominal pain (tormina), tenesmus, temperature, coated tongue, and on the general feeling of the patient.

Dosage and Diet

(*a*) Each dose was composed of ½ tabloid sulphapyridine crushed into powder, one teaspoonful of liq. paraffin or Ol. recini, and a pinch of soda bicarbonate mixed with an ounce or two of water or diluted lime juice. One such dose was repeated three times, at an interval of one hour, two, three, four and six hours. The patient thus received a total of 15 doses in 48 hours, i.e. 7½ tabloids and 15 teaspoonfuls of Ol. Recini or Liquid Paraffin.

(*b*) The diet during 48 hours of treatment consisted of rice, barley, sugar water, with fresh lime or orange juice, soup; 10—12 ounces were given every 2-3 hours, up to a total quantity of about six pints in 24 hours. More solids were added after 48 hours and convalescent diet prescribed after 72 hours.

Results

(*a*) Controls—showed improvement in 2—4 days; but tongue did not clear; anorexia, general depression and, in some cases, mucus in the stool persisted for some days; flatulence was not infrequent.

(*b*) Semi-controls—failed to show any marked improvement after 2—4 days' routine treatment, but responded to M and B 693.

(*c*) M & B 693—produced appreciable improvement in 12—18 hours; signs and symptoms cleared up in 30-48 hours. There was a general sensation of well-being after 24 hours; tongue became clear; great desire for food was expressed after about 36 hours. No relapse was observed while in hospital.

Failures

Seven patients out of 46 or roughly 15 per cent. failed to respond to 48 hours treatment:

(i) 3 cases, one each of Flexner, Sonne and Schultz, were very severe and required more than 48 hours to clear up.

(ii) 2 cases persisted with abdominal pain and mucus in the stool, which subsequently proved to be E. H. infection as well.

(iii) 1 case, with spasmodic abdominal pain, showed round-worm ova in the stool.

(iv) 1 case, in which fever and frequency of motions continued, showed B. T. malaria and hookworm.

It is strongly suggested that a similar line of treatment may be tried in certain selected hospitals.

71. MAJOR DAVAR—"*Treatment of Bacillary Dysentery with M and B* 693" (D)

My first experience of M & B 693 in bacillary dysentery was gained in the first Military hospital to which I was posted, at a time when Burma evacuation had begun and a large number of patients came to the hospital at Luckuow, with dysenteric infections. They were largely dehydrated and emaciated, due to long marches and want of food and water. It was not thought advisable further to purge and dehydrate them, by giving salines. As sulphaguanadine was not then available, they were treated with M & B 693, and, since then, the results have been so consistently good, that I have not thought it advisable to change over to sulphaguanadine, excepting for checking up the results. In a very few cases, where M & B 693 failed to produce the desired result, or could not be tolerated (10 per cent.), sulphaguanadine was used.

The treatment generally given in acute and subacute cases, is as follows:

A preliminary dose of castor oil or saline is given, if necessary and when griping and tenesmus are marked. 4—6 hours after the opening dose, the patient is given 2, 3 or 4 tablets, according to the severity of the symptoms, and this is followed by 2 tablets every 4 hours. In less severe cases, 6—8 tablets a day have been found quite sufficient. Even in very severe cases, it is not necessary to exceed 6 grams or 12 tablets a day and this dose is continued on the 2nd and 3rd days. In all cases, the drug is continued in reducing doses for at least 5 days, as, otherwise, mild relapses become more frequent.

Other adjuvant treatment is as below:

(1) Pot. permanganate bowel wash, followed by retentive enema when necessary.

(2) Mist. bismuth or astringents with Kaolin.

(3) Large quantity of hypotonic saline to drink freely, 5-6 or more pints every day.

(4) Soda bicarb. grs. 45, 3-4 times a day.

(5) Saline transfusion of 1 or 2 pints are given when indicated.

The beneficial effect is noticed in 24 hours (50 per cent. diarrhoea checked) generally, and in 48 hours at latest. If the diarrhoea is not controlled in 48 hours, it is not advisable to push the drug any further, as some of these cases, which did not respond, turned out to be of double dysenteric infection.

Immediate effects noticed in 24 hours are, that the number of stools is greatly reduced (50 per cent.) and are slightly formed; toxaemia disappears rapidly and the patient experiences a feeling of well-being; blood generally disappears from the stool in 48 hours. Though the drug is so effective in acute and subacute stages, it has no value in chronic dysentery.

Advantages of treatment—

(1) The drug is available in large quantity. Treatment is quite short and the effects rapid.

(2) Dosage is much smaller than that of sulphaguanadine.

(3) No serum treatment is necessary.

Disadvantages—

(1) The drug is more toxic (10 per cent.), though I have not seen many severe toxic effects, in spite of using it in large numbers of cases of different diseases.

(2) Produces nausea, vomiting and abdominal pain.

(3) The greatest danger is haematuria or haemoglobinuria; this can well be avoided by giving alkalies and large quantity of fluids to drink, and, in severe infections and dehydrated cases, by giving 1-2 pints of saline intravenously at the same time. Any severe generalised acute abdominal pain is a very important symptom in a patient undergoing treatment with M & B 693 for any disease and immediately on the occurrence of this symptom in the last few cases, the drug was stopped and the urine examined, haematuria was diagnosed in the earliest stages and successfully treated. Considering the large number of cases treated with M & B 693 for different diseases, the incidence of haemoglobinuria is very low. It occurred in two of my cases of pneumonia, after as few as 6—8 tablets only.

(4) Agranulocytosis—in cases in which blood counts were done, there was no appreciative fall in W. B. C. count.

(5) Recently, cases of sulphapyridine anaemia have been described.

The following dosage is recommended, which, from the study of 400 cases, is considered quite sufficient—

Severe acute or subacute cases—

1st day—Initial 3-4 tablets, followed by 2 tablets 4-hourly.

2nd day—2 tablets 4-hourly.

3rd day—2 tablets, 4 times a day.

4th and 5th days—2 tablets T. D. S. or B. D.

In severe cases, 20-23 grams, i.e. 40—46 tablets.

Mild acute cases—

1st day—2 tablets T. D. S.

2nd day—2 tablets T. D. S.

3rd day—2 tablets T. D. S.

4th day—2 tablets B. D.

In mild cases, 10—12 grams, i.e. 20—24 tablets.

72. LT.-COL. SNELL—"*Treatment of Bacillary Dysentery*" **(D)**

I have no special facts or figures to bring forward, most of the ground has been covered by the previous speakers, so I will confine myself to emphasising one or two points. As shown by Lt.-Col. Cobban, the adequate treatment of dysentery is dependant on the satisfactory organisation of the dysentery ward. Daily naked-eye examination of the stools, by the M. O., is essential, and to this end, the top line of the temperature chart may be used as a place to record the type of stool passed each day. Stools may be classified into 5 types and recorded as follows :

(*a*) No faecal matter, but entirely blood and/or mucus—I B and/or M

(*b*) Fluid faecal matter, with blood and/or mucus—II B and/or M.

(*c*) Fluid faecal matter without blood or mucus—III.

(*d*) Semi-solid stool—IV.

(*e*) Normal stool—V.

In a similar way, the daily diet can be recorded on the second line from the top of the chart :

Diet I—Fluids (without milk), jellies, plain biscuits, some fruit juice and glucose.

Diet II—with the addition of milk (½-1 pint), egg in milk, bread or toast and a little butter.

Diet III—a convalescent diet, including steamed fish, boiled or poached eggs, blanc-mange, milk puddings, but without vegetables.

Diet IV—an ordinary diet, but with vegetables only as a puree.

Diet V—Ordinary diet.

In this way, it is easy to see at a glance the effect of an increase in diet and the progress of the disease.

My experience of dysentery has mainly been of mild cases. These respond to rest in bed and careful dieting. I agree with Major Kiloh that saline treatment is unnecessary, but I do not feel as strongly against it as he does. In more severe cases, with dehydration, I believe saline treatment to be contra-indicated. The relatively few cases with appreciable pyrexia and toxaemia, I have treated with M & B 693 (in the usual doses of 4 tablets, followed by 2 four-hourly or of 4, 4, 2, 2, etc.), and they have done very well. I have had little opportunity to use sulphaguanadine and when I have used it, it has not appeared to be much more effective than M & B 693. Serum, intravenous saline and blood transfusion, I will only mention as necessary in the severe cases, with much toxaemia and dehydration.

73. LT.-COL. MURTHI—" *Chronic Entero-Colitis* " **(D)**

The object of this paper is, (i) to bring to your notice that a large number of cases, diagnosed as malarial cachexia, inanition, malnutrition, dyspepsia, etc. have nothing to do with malaria or malnutrition primilarly, and (ii) to outline the aetiology, symptomatology, treatment, prognosis and prevention of cases of chronic enterocolitis. The material was derived from casualties evacuated from the Eastern Army area to the Base General Hospital at Moradabad, and concerned Indian troops of approximately four thousand admissions during the period commencing from 15th August to date.

Symptomatology—

The cases with the above diagnosis, were in varying states of generalised wasting, with or without symptoms of avitaminosis and also with varying grades of anaemia. These patients were having looseness of the bowels for quite a long time, with several acute exacerbations. This looseness of the bowel or chronic enterocolitis became a predominant symptom in several cases, and, in cases that became seriously ill, was directly responsible for a fatal termination, when it unhappily occurred.

Investigations and Observations on 50 cases—

(1) The motions (microscopic appearances for a continuous period of seven days), were two to eight in number, six to eight ounces in quantity, acid in reaction, generally whitish or pale brown or pale yellow in colour, occasionally containing grossly undigested food particles, remarkably devoid of smell and not at all frothy of abundant in the real sprue sense and containing blood and mucus in widely varying amounts and intervals. Microscopically—Indefinite exudate was seen at one time or another and, occasionally, no exudate at all. During acute exacerbations, vegetative forms of Eptamoebae were found in 20 per cent. of the cases, while an equal number if intestinal flagellates were seen.

Culturally—In 20 per cent. dysentery group of organisms were isolated, while in another 20 per cent. organisms of the Proteus and Paracolon group were isolated. The District Laboratory figures are, from 350 stools of all sorts, showing any blood and mucus, in 4 per cent. dysentery organisms and in 15 per cent. secondary organisms were isolated.

(2) The anaemia in 75 per cent. of these cases was not grave when followed even to a fatal termination, but in 25 per cent. of the cases it was really significant and remained a symptom of considerable importance, in treatment and prognosis, in addition to the enterocolitis. The agglutination reaction of the blood against dysentery and paradysenteric group of organisms in these cases, showed no significant response. For this aspect of the investigation, I am obliged to Capt. Magner, Officer i/c District Laboratory, Moradabad.

(3) A hundred per cent. of these cases gave a definite history of having passed blood and mucus in their motions, 2-3 months prior to hospitalisation and, in some cases, were victims

of large-scale epidemics that had occurred in the individual's unit. It would appear that a number of epidemics occurred in the units stationed round about Dimapore and Manipur, during the period from March to October, 1943. The patients that had the attack, were treated and returned to duty. The strange story that could be gathered from them was, that though they had apparently recovered from their dysentery and were ambulatory, they had not however become normal with reference to their appetite and bowels. Lack of appetite, flatulence after meals, heartburn and looseness of the bowels, were the common features of their lives after the attack, for a priod of 2-3 months during which time they became steadily weak and debilitated and, finally, sought admission for the same, or for an intercurrent malarial infection. This is how they came to be stamped as malaria or malnutrition, a diagnosis which keeps the original cause out of the picture.

(4) A careful investigation was made into the diet of these individuals. These men were recruited to the army within the last three years and were in field service conditions for varying periods of three years to six months, before their illness. They were quite satisfied with the army diet compared to their diet in civil life and had not noticed any particular deficiencies during the period immediately preceding their illness. The drinking water, according to them, was not, however, at all satisfactory. In short, the sources of infection through drinking water were immense; epidemics of dysentery occurred, and, after the attack of dysentery, it was not deficiency of diet but defective assimilation that brought these men to a state of chronic invalidism, characterised by grave symptoms of malnutrition and avitaminosis.

(5) From the treatment point of view, a good number of these cases showed a prompt response to sulphonamide group of drugs, particularly sulphaguanadine, thereby showing a therapeutic evidence of its dysenteric or a similar infective aetiology. The response to these drugs is, however, definitely lacking, if the cases fall under the group of pure intestinal amoebiasis or complicated by a terminal intestinal tuberculosis.

(6) Six post mortems showed extensive dysenteric ulceration of the bowel.

(7) Prognosis in these cases depends on the degree of emaciation, the extent of the anaemia and the waking up of a tuberculous focus.

Conclusions—

From the above observations, it is reasonable to conclude that :

(1) Dysentery, both bacillary and amoebic, is one of the worst handicaps for our troops operating on the Eastern front, as it always has been with all armies on active service.

(2) Life under field conditions is not suitable for post-dysenteric convalescents.

(3) The value of good nutrition and protective foodstuffs is beyond doubt, in safeguarding against infection, but it must be said that nutrition is not the primary factor, at any rate in the cases under review, though malnutrition, following the dysenteric attack, has considerably vitiated the clinical picture. Further, the supply of nutritional and vitamin elements, alone, will not cure the cases under review, if the infective factor is not eradicated.

(4) Post-dysenteric ulcerative colitis, according to Napier, and diarrhoea following chronic bacillary dysentery, characterised by emaciation, weakness and anaemia, according to Sir Arthur Hurst, are well known clinical entities, which have not received sufficient attention and recognition by our medical officers in the forward areas. If a soldier is returned to duty after dysentery, without proper convalescence, he is likely to slip into this clinical state, besides proving a source of infection to his comrades.

(5) The malnutrition and inanition following dysentery, must be deemed as the charred remnants of the original dysenteric explosion and, in dealing with these, it is much better to control the primary bombardment, rather than to rehabilitate the burnt-out after effects.

Suggestions—

(1) All unit and regimental M.O.'s should be strictly enjoined to hospitalise all cases of dysentery, acute or chronic.

(2) All C. C. S.'s M. D. S.'s, or staging sections, should be given full facilities for the diagnosis and treatment of these cases in their acute phase. Sulphaguanadine in the treatment of bacillary dysentry is, in my opinion, the remedy ' par excellence ' and should be made available right in the front line.

(3) Post dysenteric convalescence is of supreme importance and should receive sufficient attention from the attending M. O. Advanced convalescent depots are ideal, but may not be practicable. Convalescence may, however, be watched by the M. O., by observing the weekly record of the weight of the soldier, if the M. O. is provided with the implement to do so.

(4) Finally, I appeal that the diagnosis of primary malnutrition should be discouraged, as it does not occur in the army and the prevalence of such a diagnosis gives a wrong orientation to all concerned. Dysenteric infection is, on the other hand, far-reaching. It should be prevented and, if despite precautions, it occurs and if the soldier on active service dies of its immediate or ultimate effects, his death is certainly attributable to military service.

Demonstration of the X-ray films of two cases of liver abcess, which opened into the pleural cavity, with collapse of the lung.

The findings in the above cases were :

(1) Both gave definite history of dysentery.

(2) One case showed vegetative E. Histolytica in an acute exacerbation.

(3) Pus aspirated from the pleural cavity as well as from the liver.

(4) Both responded to Emetine treatment in addition to aspiration.

74. COL. SCHLESINGER wished to emphasise that sulphaguanadine still remained the drug of choice for severe bacillary dysentery, and that sulphapyridine and others of the group of sulphaguanadine was not available. He also pointed out the negligible risk of toxic symptoms arising from sulphaguanadine, as compared to other varieties of this group of drugs.

DISCUSSIONS

75. LT.-COL. DAUKES gave it as his opinion that it was not the ward boys' function to choose the portions of the stool to be examined; his function was to get the stools to the laboratory, as quickly as possible, notifying the officer in charge of the laboratory or clinical side room, directly they arrived, so that they could be dealt with promptly. He reminded the Meeting of the necessity for washing away all traces of disinfectant out of bed pans before use, and of the help that warmed bed pans can give, over the diagnosis of amoebic dysentery. He also described an easily made warm stage, using two microscope slides, separated by a ring of plasticine, through which two thin glass tubes went. One glass tube was connected to a douche can of warm water and the other to a bucket or dish of some sort, for the waste.

76. MAJOR LYN GREENING agreed strongly with Lt.-Col. Daukes, in emphasizing the necessity for specimen selection of stools by the M. O. i/c ward and not by the sweeper. The alternative was to send the bed-pan direct to the C. S. R., where selection could be made by the officer in-charge of this room, or by the Lab. Assistant V. C. O. who was being turned out in increasing numbers, after training which rendered him competent to under-take this work. The early miscroscopical examination of stools in amoebic dysentery, was already provided for by a D. D. M. S. order, that such examinations would take precedence over all other examinations in the C. S. R. or laboratory. It was pointed out that, in the cases of two hospitals in Central Command, which were separated from the laboratory by 2½ miles and a few hundred miles respectively, isolation rates from cases with bacillary exudate, differed by 44 per cent. It was for consideration whether, in such cases where the hospital was situated a long way from the laboratory, it would not be better for actual plating of stools to be done in the C. S. R. and the inoculated plates sent to the laboratory. Before adopting the term 'Emetine-fast' Amoebae, as used by some speakers, Major Lyn Greening would like to be reassured as to the quality of the drug supplied for use nowadays. The Electrolytic sterilisation of water, or use of the Catadyn reaction, had not been shown to destroy E. H. cysts, as far as the speaker was aware.

77. LT.-COL. HAVILAND-MINCHIN asked whether the tendency was not to treat laboratory findings, rather than patients suffering from dysentery. He pointed out that, as far as the physician was concerned, all that mattered was whether the disease was amoebic or not, and that the complicated classification of the type of organism was of no clinical importance and took up a great deal of laboratory time that could be far better utilised. In amoebic dysentery, he considered that far too much faith was placed in reports of amoebic cysts. These he did not consider required treating, as, firstly, he did not believe that there was more than one or two pathologists whose views on the nature of a cyst was really worth taking, and, whatever treatment was given, the object was to drive the vegetative forms to a cyst form.

From the point of view of infectivity, he did not consider cysts to be of any importance and, in certain parts of India, over 10 per cent. of the local population had cysts in their stools. The object should always be the treatment of the patients and their disease, and never the treatment of Laboratory reports.

78. LT.-COL. BOSE emphasized the importance of careful clincal examination in cases of chronic colitis of amoebic origin. The length of the colon approximately corresponds to the individual's height; any part of the colon may be affected; the posterior surface of certain parts is not covered by the peritonium, so that pathological changes from ulceration in those parts, involve some adjacent organs and tissues, with clinical manifestations, which are not ordinarily suspected of amoebic origin, namely, peri-duodenitis, gall bladder dyspepsia, etc. The palpation of (i) the caecum, with postural contraction of ilio-Psoas muscle, (ii) the entire length of the colon, (iii) the renal angles and the retro-colic areas, will help in the diagnosis of pathological colon.

If sigmoidoscopy is not available, high proctoscopy will yield, in many cases, satisfactory results. With regard to persistent cyst-passers, without any clinical signs or symptoms, the question of non-pathogenic E. Histolytica may be borne in mind and a reference to Dobell's work is recommended. The identification of the cysts is not difficult with Lugol's iodine staining, if diligent practice is undertaken under experienced workers.

79. LT.-COL. DAUKES, in reply to Lt.-Col. Haviland-Minchin, stated that, as according to Regs. A. M. S., the first duty of a medical officer was the preservation of health and the prevention of disease, while the healing of the sick was secondary, that cultures for the type of organism did little or no good for the dysentery patient, yet it might be useful for tracing the source of any epidemic, and telling if it was an epidemic due to one carrier. He was therefore of the opinion that they should continue to be done. As regards the diagnosis of E. H. cysts, he advised the review of many cysts and the keeping of the cysts in the incubator overnight and re-examination next day, to see if any had developed eight nucleii.

80. MAJOR CRADDOCK said, " May I enquire whether the ban on the use of sulphapyridine for diseases other than pneumonia, meningitis and gonorrhoea, has now been lifted? It appears, from the widespread experiments in the use of M & B 693 in bacillary dysentery, recorded by various speakers, that I have been unnecessarily conscientious in observing this ban."

81. LT.-COL. PASRICHA said that, (i) cyst-passers were not necessarily suffering from amoebic dysentery, (ii) emetine was not good for cyst-passers, (iii) stovarsol had not been found effective in many cases, (iv) amoebiasis of liver was not infrequent among British troops, and agreed with Lt.-Col. Bose regarding the importance of clinical diagnosis—tender colon.

82. COL. TAYLOR stated, " Amoebiasis in Eastern India is a major problem. It is estimated that 15,000 cases were treated in medical units of Eastern Army (now Eastern Command and Fourteenth Army) in 1943. Related to this large number of cases, the figures produced by Dr. Das Gupta, the Director of the School of Tropical Medicine, Calcutta, and of Lt.-Col. P. Sayers, are important. The Bengal famine has interrupted this work, but 311 B. O. R.'s who have been in India for about a year, taken from a British unit in Calcutta, have had one stool examination. 83, or 26·7 per cent., were found to have infections of E. Histolytica in their stools. Of these, 23 or 7 per cent. had present both active vegetative forms and cysts of E. Histolytica, 6 or 2 per cent. had vegetative forms, and 54 or 17 per cent. had cysts only. Some figures collected from British troops in Calcutta, who had developed amoebic dysentery, showed that 67 per cent. developed the disease in the first 6 months of service in India. With the prospect of much larger numbers of British troops in Eastern India when the German war is over, the situation is still more serious. My impression is that more officers (often the most senior) than B. O. R.'s develop amoebic dysentery.

You are familiar with the defects in the water supply of Calcutta. I was fortunate enough to go round the waterworks with General T. O. Thompson, the then D. D. M. S., when the supply was described as ' clarified Hoogly '. Evidence was given that, during the hot weather, seepage into water mains had taken place, because of old defective pipes, possibly from neighbouring sewers. Practically all British troops in Eastern India pass through Calcutta, either on duty or leave. Major Kaul's observations that amoebic cysts may not be killed by normal chlorination of water supplies, needs urgent consideration.

The whole problem of prevention of infection, and the significance of the amoetic cysts, need urgent attention. Kitchens are often dirty and may have no fly-proof netting. The system of examining food handlers and cooks for amoebic carriers has been abandoned. I should like to see lettuce forbidden for all officers and men. In 1916 or 1917 a much less extensive problem in Egypt led to a special commission on which I think, both Wenyon and Dobell served.

Captain Willatt in Calcutta, has produced some first class work on this subject. His first 50 ' resistent ' amoebic cases, who had previously been treated by at least 3 courses of emetine injections without success, gave the following results, after one or two Courses of E. B. I., chiniofonum enemata and carbarsone (or ambiarson) :

Cases presumed cured (with follow-up of 3 months) 21 or 24 per cent., cases not cured—29 or 58 per cent., disposal, returned to unit—2 or 4 per cent., evacuated to Central Command or elsewhere—46 or 92 per cent., category C—2 or 4 per cent.

E. B. I. could not be used earlier because it is very short in world supply. The cases were evacuated either because of their general debilitated condition or because of ' resistant amoebiasis '. Captain Willatt is finding M. & B. 693 and sulphaguanadine of use in treating double infections of both amoebic and bacillary dysentery, combined of course with emetine and other antamoebic treatment, and also in treating the secondary infection of amoebic ulcers."

83. LT.-COL. S. N. HAYES considered that little advance would be made, from the curative point of view, until the Dietetic treatment was placed on a sounder basis. Personal practice had been to treat acute cases as soon as possible with an adequate diet. As a rough guide, anything that would pass through a fine sieve would suffice.

8TH FEBRUARY, 1944—AFTERNOON SESSION

The meeting closed at 13.35 hours and was resumed at 14.30 hours, with **BRIGADIER McALPINE** in the Chair. The chairman expressed sorrow at the unavoidable absence of Brigadier Bennett, whose regrets were conveyed to the Conference.

PSYCHIATRY

84. MAJOR TREDGOLD—" *Hysteria* ". **(P)**

I must begin by apologizing for the absence of Brigadier Bennett and Major Galbriath,, both called away on urgent work. I regret that their default has caught me on the wrong foot, as it was not until I got to Delhi that I heard that I was expected here. As I was quite unprepared both for the climate and for giving a paper of the calibre obviously required here, it was rather a chilling intimation.

The original subject chosen for this afternoon was " Common Psychological Diseases in the Army in India." These are, of course, "anxiety" and "hysteria" and, as the former is more generally referred direct to the psychiatrist from the R. M. O. and therefore passes less often through the hands of the medical specialist, I propose to deal solely with hysteria, which, in all conscience, is a large enough subject and seems so often to be a happy meeting-ground for the physician and the psychiatrist. I shall try to deal with the aetiology, symptoms, diagnosis and treatment in turn. I shall refer only to British troops, as my experience with Indians has been too small; but I suspect that, although there are some obvious superficial differences, the fundamental problems are the same. No doubt there will be some of you here who will be able to discuss this from a wider experience.

Before I begin to talk about aetology, I should like to digress for a moment, on the subject of malingering. Hysteria, of course, may be briefly defined as the unconscious production of symptoms without any adequate organic basis; while in malingering, the production is conscious. The confusion is, no doubt, partly due to the fact that both may be directed towards some gain. But there are several strong reasons, legal and medical, why the essential difference should be kept clearly in mind. I hope you will forgive my stressing this point, but I can remember a medical division C. O. who used the term hysteria and malingering as synonymous. It was all the more unfortunate, in that he was referring to a patient who, later the same day, exhibited a condition generally accepted—in my experience—to be due to organic conditions, to wit, death. Having tried to stress the need for differentiation, I must, I fear, now be paradoxical and confess that there are cases which show both conditions and also that they are,

at times, very difficult indeed to distinguish. But, the point is, that an accurate assessment must be attempted. Obviously it depends for its success on a very careful study of what is going on in the patient's mind.

Aetiology—

In all cases, there are two factors to think of, the predisposing and the precipitating. A particular type of factor may, of course, be of one kind in one man, and of the other in another. So, I propose to run through various factors together.

(1) The constitutional : It is generally held that a tendency to hysteria is a longstanding personal defect and the result of an inborn weakness, together with an inadequate or faulty upbringing. There is no doubt that both heredity and environment are important, though the precise degree of each will vary. Also it is clear that some people—the chronic neurotics as they are rather loosely called—exist in civil and military practice. They are generally as easy to diagnose as they are hard to treat, and of little military value, so that I propose to say little of them now.

(2) I should like however to put forward as a point for discussion, the theory that hysteria also occurs in people previously normal enough to have adapted for the army, and to suggest that there are periods which most of us pass through, when we are potential hysterics and might develop overt symptoms where there are other factors also present. You may remember some work done—I fear I have had access to no papers—when it was demonstrated that a number of people who had been involved but not injured in a railway accident, showed an increased suggestibility, most marked, several hours later.

We should, then, consider what these occasions of heightened suggestibility may be. I think there are several that concern us directly.

The first is after action, and has been described in soldiers and sailors. It is obviously of prime importance in the diminution of psychatric casualties. Reports so far obtained, suggest that this increase occurs a day or so after release from action and not immediately. It is therefore essential that all cases should be treated as early and as adequately as possible and, as you probably know, such a system produced very excellent results in N. Africa, where about 90 per cent. of casualties were returned to duty within a week, from certain psychiatric centres. Further, such cases were labelled " exhaustion" and not "N. Y. D. Mental", or any such terrifying term and this undoubtedly produced a positive suggestion in the patient's mind. Exhaustion is a condition which he expected to disappear.

The second occasion is when the patient is in the presence of a doctor—under which term I include the surgeons, and even, if I may, the psychiatrists. I think we are all apt, no doubt from a natural modesty, to forget just how much our words and even more, our behaviour, can affect a patient. I should like to quote a case as an example of this. I recently saw a man at Poona, who had sustained a not very severe accident of his right knee, some four years earlier. He had been bruised, but nothing more. His doctor looked gloomily at it and told him that he was afraid his knee would trouble him for years. It did. Cases are often turning up with considerable fears about the state of their hearts, due to the fact that some doctor in the past has not had the courage to say firmly that there was nothing wrong, but had merely shaken a gloomy stethoscope over them. In Dr. F. M. R. Walshe's words, "With the increasing range of diagnostic procedure, there are few of us who could hope to escape the discovery of some imperfection, were we so unwise as to submit ourselves to inquisition on these lines". Caution is all very well, but there are times when it is far better to take a broad view; anyhow to let an occasional invalid drop dead unexpectedly is surely better than to produce an annual crop of chronic hysterics.

Then there are the pre- and post-operative states, whose importance, the surgeon, acclimatised as he is to the theatre, is apt to minimize. Spinal anaesthetics are, in my opinion, a fruitful source of hysterical symptoms, but far less so when the anaesthetist takes the trouble to reassure and explain to the patient all that is to happen.

The other way in which hysteria is produced, I think, by the doctors, applies especially to the army. A patient who has had a real but slight headache is accused by the M. O.'s behaviour, if not his words, of malingering. His response is very natural : his headache gets worse; it must do so, to save his face. A vicious circle is easily set up by the resentment which then occurs on both sides.

It is a great help to a psychiatrist, if he gets a patient who has been authoritively told he has no serious organic disease, but only a minor functional upset, which the psychiatrist will rapidly cure. So often we get the opposite, a thoroughly bad start. I should like to put in a protest about a man who recently came to Poona, having been told by a medical specialist that he could not do nothing for him, but his subconscious must pull itself together.

You may well say, "If the patient is as stupid as this, can we really do much?" and this is the next point—his intelligence level. It affects the onset of hysterical symptoms in several ways. Firstly, he has not the ability to understand what the doctor really means; and secondly, he has not the control of his doubts and fears; and thirdly, in the army in particular, he is often inadequate at his training or at his job, so that he is driven to find an excuse, subconsciously to save his face, and it is easier to complain of a headache, when asked to learn how to drive a tank, say, than admit his incompetence. Even a small amount of insight into our own feelings, when we are 'unlucky' enough to fail in exams, should make us sympathise. It is a very human reaction. Thus, any inadequacy heightens our suggestibility. Fourthly, in any physical illness or fatigue, one can readily understand increased suggestibility and can see examples. Meningitis and head injury are both very often followed by hysterical symptoms, which, as might be expected, take the form of headache and dizziness. Cerebral malaria is similar. The type of symptoms may be due to the common deep fear of head injury, to combat which, as well as splinters, a tin hat is designed. Fifthly, is the emotional shock of bereavement, or domestic trouble, which is a commoner cause of anxiety, but is also of hysteria. Last, but certainly not least, is the sense of frustration, so often seen in young and enthusiastic officers in unsatisfying jobs.

The precipitating causes may be any of the above, except, of course, the constitutional factors. Besides, there are the injuries or threatened injuries, which determine how or when a symptom develops. Blindness after a flash of an explosion, or shoulder paralysis after a blow on the arm, are examples. Organic heart disease in the family produces cardiac neurosis.

Symptomatology : I do not think we need say any more about this except that hysteria is surely on a par, if not ahead of syphilis and malaria, in being the great imitator. Sometimes it is easier to find out why hysteria has produced a particular symptom, than why syphilis has. The mind is, at times, though not always, more open to inspection than the brain. But we must remember that the condition corresponds with the patient's idea, rather than with any lesion. Thus, a paralysis is of an arm or leg, not of the area supplied by certain nerves. A man was recently seen, who showed almost true narcoleptic phenomena; it was difficult to understand how this could have arisen, until one heard he had been in a ward with two true narcoleptics.

Diagnosis : again can be skirted over as unnecessary in this audience, but I will remind you how often hysteria arises as an exaggeration of an organic lesion. Your opinion, then, as to the exact amount of disability due to the latter, will be of value to the psychiatrist to whom you refer the case. Again, the differentiation from malingering may be difficult, as I have said, but, in doubtful cases, it is safer, for reasons I will give, to treat them as hysterics.

Treatment : has already been largely indicated, if not in great detail, at least in broad outline, from our discussion on aetiology. The history-taking will have produced a very firm basis of confidence for the psychiatrist to work on. If you can ensure that the M. O.'s you teach, act accordingly, you will have halved our work and doubled our recovery rate.

I should like to quote a last case, to illustrate the treatment I would recommend for malingerers. This, as I have tried to indicate, will depend entirely on the reasons for their malingering, and one who is merely avoiding military service, must be punished by discipline. Others will recover, like hysterics, once their underlying reason has been found and removed. A man was seen, who had a talipes deformity of his foot, regarded by the surgeons as a malingerer, but by us as a hysteric; they wanted to send him for detention, but we somehow managed to treat him. After a month, he was cured and was fit for duty; he then admitted that, though he had a minor real (hysterical) disability, he had consciously exaggerated it, in resentment of his M. O.'s original attitude. Although the laugh was, in a sense, on us, it seems to me that we did right, in so far as we cured his disability and returned him to duty, where I think he still is, whereas a return to detention would merely have forced him to continue with his complaints.

Malingering seems to me to be as common for such reasons, as from a desire to avoid service. To correct our ideas, we should remember that it can be entirely patriotic, e.g. to escape from a Prisoner of War Camp.

85. MAJOR ST. JOHN BROOKS—" *Neurosis in Iraq* **" (D)**

I have been asked to give a brief account of cases of neurosis, seen in the medical out-patient department of a C. G. H., during three hot weathers in Baghdad, Iraq. We saw no "battle neuroses", though these were, in a wide sense, "war neuroses" as, in Iraq, there were rather special conditions obtaining. First, there was an extremely trying climate and, secondly, there were, except very early on, no active operations, but merely steadily increasing boredom and a feeling that one was in a backwater and serving no useful purpose. This was reflected in the increase in out-patient attendances, for neurotic and psychosomatic conditions. In 1941, they were not numerous, but in 1942 they formed 60 per cent. of all new attendances by I. O. R.'s, who were beginning to become homesick, and about 30 per cent. of B. O R.'s. During this year, we were reinforced by a large number of low category men, who became the G. H. Q. personnel of the newly-formed Paiforce, and in 1943, the proportion of functional conditions in B. O. R.'s increased to 60 per cent. while among I. O. R.'s it was unchanged. These numbers were about equally divided between pure neurotic conditions, mild and severe, i.e. anxiety neurosis, hysteria and an occasional case of obsessional neurosis, and psychosomatic states, such as functional dyspepsia, effect syndrome and many cases sent for such conditions as headache, fibrositis and sciatica.

As time went on, especially in the summer of 1943, a common type of case was recognized, of a condition which came to be known as " Paiforcitis". This became almost epidemic in some units. It affected chiefly officers and B. O. R.'s, rather than I. O. R.'s and attacked many men with a previously stable background. The symptoms covered a wide range, though common to most were depression, headache and inability to concentrate. The universal cause, at most only slightly concealed, was the desire to get out of Paiforce. In one British unit, which had previously been in action and was now being rather unimaginatively handled by its O. C., there was a small outbreak of hysterical paralysis.

Treatment was simple—varying degrees of explanation, firmness and encouragement. We looked on it as very important to keep these cases out of hospital. Co-operation by unit M. O.'s and vigilance at the hospital reception centre, were largely successful in this, though it swelled the number of out-patients. Most cases gave no further trouble, even to their unit M. O.'s. Of about 10 per cent. who had more than one O. P. attendance, a residue were chronic neurotics, who attended regularly, often for months. Their symptoms waxed and waned, but meanwhile they managed to carry on with their work—generally clerical. A still smaller number were referred to the psychiatrist, usually at the first attendance, but sometimes after complete failure. It was felt that the vast majority of these patients should be kept away from the psychiatrist if possible. Best of all, of course, is for them to be dealt with by their own unit M. O.

Finally, one observation on the occurrence of effects of heat. I felt strongly that neurotic conditions predisposed to heat exhaustion and to the milder grades of heat intolerance, which we called "subacute effects of heat." I think one could pick out, on psychological grounds, men who would not stand the hot weather well, and that neurotic subjects are unsuitable for extremely hot climates. Conversely, men who had suffered from severe heat-stroke or heat exhaustion, often later showed neurotic manifestations.

86. LT.-COL. BHALLA—" *Neurosis in the Middle East* **" (D)**

To begin with, I wish to point out that neither am I a Psychiatrist, nor have I any statistical data to support my remarks, which are based merely on my impressions formed while working in General Hospitals in the Middle East, for over three years.

The incidence of neurosis among Indian troops, appeared to be remarkably low in the earlier days of the war, but, of late, it appears to be on the increase. This is because of the direct or indirect effects of the duration, stresses and strains of the war, because of the size of the Indian Army having grown considerably, and also because the type of individual that has come to be enrolled, is not as suited for these stresses and strains. This includes those individuals who were enrolled to do jobs that they had never done before and could not reconcile themselves to do now, because of their being against religion and caste traditions, e.g. a high-caste person becoming enrolled as a sweeper. Others found after enrolment that their

job was not in keeping with the dignity of their former trade, e.g. a fitter or a driver having to work as a dhobi, or a tailor as a water-carrier, and so on.

The types of cases and the manifestations of neurosis that one came across were :

Firstly, those in whom neurosis became superadded after a trauma or a sickness occurring while on Field Service. These commonly developed hysteria, manifesting itself as paralysis with anaesthesia, as deafness, blindness, or headaches and other localized pains at the site of any trauma.

Secondly, those who developed neurosis without any such 'excuse' as trauma or sickness were hysterics as well as anxiety states. Common manifestations of hysteria were fits, blindness, tremors, backaches and sciatic pains, i.e. the part affected usually being the one that will make the individual unfit for his job. Manifestations of anxiety states were dyspepsias, tachycardia and praecordial pain, frequent seminal emissions, headaches, attacks of dyspnoea with no abnormal physical signs, frequency of micturation, hyperidrosis involving palms (in clerks).

Thirdly, those in whom neurosis was implanted by the medical officer's unduly drawing the patient's attention to some or other organ, e.g. to his heart, because a murmur was heard, or to his spleen, because it was enlarged, and because they were sent down to base hospitals through different medical units for investigations. These patients showed anxiety states, referable to the organs on which their attention became focussed.

Regarding disposal of cases of neurosis, the policy followed was to evacuate cases with established neurosis to India, as and when advised by the local psychiatrist; while the others were returned to duty after assurances etc., to see how they would fare, and these eventually found their way back to India as a rule. One would like to hear what eventually happened to the cases that were evacuated to India.

To ensure early detection and consequently more effectual and early treatment of the cases of neurosis, I feel that we should put all M. O.s through at least an elementary course in Pschiatry, say at the Army Medical Training Centre.

DISCUSSIONS

87. LT.-COL. BHATTACHARYA: I want to say a few words about the mode of production of functional diseases in the Army. Hysteria and neurosis the part of an escape mechanism—escape from the firing line, from field service, from military service. In face of danger, a person may react in two ways :

(i) He may fight—this is the normal reaction,

(ii) He may flight—(*a*) either bodily, (*b*) or mentally.

In the latter reaction, the patient may only feel—the result is neuroses : the manifestations are tremor, sweating, tachycardia, palpitation.

The patient may dramatise—the result is hysteria; the manifestations are paresis, anaesthesia of various types, fits, deafness, blindness, etc.

In my experience, of more than 1000 cases of functional diseases in Indian troops, hysteria is more common than neurosis, namely 10 : 1.

88. MAJOR CHANDRA opined that, in operational areas, the most important thing for the regimental M. O. was to differentiate between those who were malingering from those who were suffering from anxiety states or hysteria, in order to keep up the front line fighting strength; he enquired whether all malingerers or hysterics should be evacuated to the psychiatrists for investigations, as he was told at Jullundur. He further opined that the number of malingerers or neurotics in the Indian Army, was very few, considering the present strength.

89. MAJOR GUEST commented on the very great proportion of simple hysteria cases in the general medical wards and at the Medical Specialists' out-patient departments in certain I. M. Hs. Many of these patients produced symptoms within a few days of reporting for training. The proportion was often so great that it was suggested (in jest) by him that the psychiatrists should first see all patients and after refer any patient with organic disease to the Medical Specialist.

90. MAJOR KILOH agreed with Major Guest regarding the extremely high incidence of hysteria and of malingering in the I. O. Rs., and expressed his views that many patients were told too many details about their condition, e.g. enlarged heart, a murmur, etc. He thought that this was bad medicine.

91. LT.-COL. BOSE stated that the percentage of malingerers in the Indian units was relatively very small and that it became incumbent upon the R. M. Os. and upon the Specialists to investigate the genesis of his mental state when such patients came under observation. He further opined that a careful and painstaking clinical examination, with sufficient ability to understand the patient's language and to appreciate his point of view, would enable us to come to a correct diagnosis. Some of them had been found intrinsically unfit to perform the required duties.

92. LT.-COL. BINDRA: In my experience, the Commanding Officer of an Infantry Battalion fussed so much about his men in hospital (he spent several hours daily in the wards), that I am sure he was responsible for retarding the cure in a certain type of case. In spite of the fact that action had to be taken to keep him out of the wards during working hours, and to stop him from reading case notes of the patients, he remained a menace. Was there any provision for bringing this type of C. O. before a psychiatrist?

93. MAJOR-GENERAL CURSETJEE emphasized the importance of observing men in their units. Such practice made Regimental M. Os. better general practitioners.

94. MAJOR TREDGOLD: Malingering must be investigated and its cause found. On this depends the treatment, medical or disciplinary. It may be a desire to avoid service, and if so, should be punished, but it may merely be a protest at neglect, and if so, it is easily removed. Punishment here is likely to exaggerate it. Similarly, the true psychopath, who cannot co-operate, must be differentiated from the man who just won't co-operate. The whole question of neurosis is, of course, closely bound up with morale. The R. M. O. and combatant officer, thus have a great responsibility, to collaborate with the psychiatrist, and to concentrate on building it up by positive encouragement, as well as by negative avoidance of harmful factors. Raising the prestige of the army and unit would help. We have let this sink, in peace time and are now paying the penalty. Everything we can do to combat this, is essential.

95. BRIGADIER McALPINE, in closing the discussion, said; "One of the most interesting medical developments of this war, is the way in which the Army has become increasingly alive to the necessity of help and advice from the psychiatrist. Brigadier Bennett, in a recent lecture to officers from G. H. Q. on morale, stressed the importance of good morale to an army in the field, and he showed the principal factors responsible for lowering it. Major Tredgold has mentioned several points of importance. M. O.s are apt to forget the effect on the patient, of their examination and of words said at the bedside. I wish some of our surgical colleagues could have listened to his words on the importance of a simple explanation to the patient, both before and after operation. Patients should not be told precisely what is the matter with them, but they should be reassured. Major Tredgold has mentioned the exaggeration by some patients of symptoms due to organic disease; this is not uncommon. Here again, the M. O. must make up his mind about the case and must obtain the co-operation of the patient, by explaining in simple language the meaning of his symptoms, and by reassuring him as to the results of treatment. Finally, I entirely agree with him about the medical officer who tells the patient there is nothing wrong; this sort of statement certainly does lead to malingering in some cases.

SPRUE

96. LT.-COL. HINDS-HOWELL, "*Sprue*" **(P)**

My object in reading this paper is to draw attention to a condition which, in the past, has been dismissed as a rarity, and of which the early stages are easy to miss. But, before I go further, let me say straight away that, for the purposes of this paper, I use the term Sprue in its broadest sense, including post-dysenteric steatorrhoea, Para-Sprue and non-tropical Sprue. As you know, originally Sprue was used only to denote a stripping of the tongue. Wherever there are doctors gathered together, there will, I suppose, always be differences of opinion as to the precise significance to be attached to any set of signs, and this

is definitely so in sprue. What then is sprue? Should it be classed among the anaemias, is it a bowel disease, is it an infection, is it a disease at all? I think that it will be best if I first described my cases briefly and then set out my conclusions.

There is at Karachi, quite a brisk little Sprue unit, which has developed as an off-shoot of the dysentery wards. Patients are admitted to this unit, either because they complain of a sore mouth, or because they are found to have a smooth tongue. Altogether, in recent months, I have collected 34 cases of this sort, and, for purposes of tabulation, they can be divided into groups.

Group I: The first group, as it happens, may not be sprue at all, but you shall judge. It consists of patients who have had prolonged treatment for dysentery, particularly amoebic, and who have developed a flatulent dyspepsia as a new and recent symptom. Investigation shows that they have achlorhydria. Hydrochloric acid will relieve the symptom, but a little nicotinic acid will cure the condition completely, and the acid curve returns to normal. Controls, by the way, showed hyper-chlorhydra. This group has not been treated in the sprue unit.

Group II: In this group there has usually been a long history of diarrhoea, not necessarily of proven dysenteric origin, but the complaint is of "sore mouth". The tongue is usually bright red along the edges and tip and there may be aphthous ulcers, either on it or on the buccal mucous membrane. Stools are of normal composition and there is achlorhydria. Nicotinic acid is the cure.

Group III: This group is the same as Group II, except that there is a constant diarrhoea, which may be absent in Group II. Any diarrhoea, if it be severe enough, will give a high fat content in the faeces and this is so in this group, but the point to note is that, however high the fat be raised, the proportion of split to unsplit fat remains in normal proportions, that is 1, 2 or 3: 1.

Group IV: The history and signs are as before, smooth tongue, achlorhydria and and diarrhoea, the total fat of which may not be increased, but in which the ratio of split to unsplit fat is increased; ratios of 5, 6, 7 : 1 becoming apparent.

Group V: In this group, the content of fat in the stool has become grossly excessive, with marked disproportion in the two fat fractions. This is, in fact, the sprue of the Story Books.

Great loss of weight, two to three stones, was noticeable in the last three groups, and variable degrees of macrocytic anaemia were present in all of them, but becoming severer in the later groups. The duration of the diarrhoea did seem to have some bearing upon the severity of the condition, but the state of the tongue did not necessarily reflect the condition of the bowel. Glucose tolerance curves were not done, as the flattened curve found in sprue is also found when there is a high carbohydrate intake, as in these particular patients. Nor were barium meals attempted, because of shortage of contrast medium, so I do not know whether the feathery appearance of the duodenum disappeared, as, according to Kantor, it should in sprue.

The routine treatment adopted was nicotinic acid, mgms 100 daily; much too small a dose for the later groups, but all that I could procure: adexolin, when available; calcium lactate, hydrochloric acid, and a fat-free diet containing 20 gms of fat, 600 gms of carbohydrate and 120 gms of protein. It was sometimes necessary to start the patients on a diet of frequent feeds of skimmed milk, until the diarrhoea was controlled, but we found that once that corner was turned, they did better on our fat-free diet than on the regulation sprue diets. All the cases except three have done well, gaining weight at an average of 9 lb. a week, the tongue clearing up rapidly, the gastric acidity rising and the stools returning to normal, so that, at the end of three or four months, the majority are able to take a full diet containing 120 gms of fat daily. The three cases which are not doing well, are two of text-book sprue and one of para-sprue. In these cases, liver extract parenterally has been added and a pint of blood each to the two most chronic cases, and at last they do seem to be responding, at least, the loss of weight has stopped.

Fat-free diet by itself does have a beneficial effect, for I found that a patient who, on ordinary diet, would be showing, say, 30 per cent. of fat in the stools, with a ratio of 8 :1 split to unsplit fat, would, when put on to the fat-free diet, show from 12—15 per cent of fat, with a ratio but little above the normal, suggesting that more of the fatty acids were now being

absorbed. This shows how useless it is to give data on fat estimations in stools, without first stating how much fat is being ingested; yet, how often does one hear that in sprue there must be more than 50 per cent. of fat, and not a word about what diet is being given. I made the same mistake by doing my early fat estimations on a fat-free diet. Apart from the effect of a high protein, low fat diet, there is no doubt that nicotinic acid and liver do have a curative effect. Some writers maintain that the liver must be crude and must be given parenterally, but, in the past, I have found cooked liver quite efficacious.

Personally, I do not regard sprue as the distinct entity of the books, but rather as a syndrome, and it is my contention that all the groups that I have described, are merely stages of one disease process and that text-book sprue is the end state. The proof lies in seeing one particular patient pass through all the different groups, and while, owing to shortage of Nicotinic acid, I have seen patients pass from each group to the next, I have not yet witnessed any one single patient pass through all the groups.

Now let us look for a moment at the theories of the aetiology of sprue. As you know, there are certain houses in Ceylon and Cochin, known as sprue houses, because all the inmates develop sprue. There are two theories about this: the first is that rotten woodwork is the agent, and the second that, in some way, the faeces of whiteants are responsible. As far as my cases are concerned, there can be nothing of this sort, as the majority have only been at Karachi.

Then there is the infection by Yeast's theory. Monilia Psilosis was isolated as long ago as 1901, in Costa Rica; then Monilia Rotunda was held responsible for true sprue and Monilia Asteroides for pseudo-sprue; but I think it is universally accepted that these are merely secondary invaders. The strepto-coccus, too, has been blamed, but without much foundation.

Also there is the belief that faulty calcium metabolism is the precipitating factor, and it would certainly account for the tetany and cramps found in advanced cases, but surely it must be a secondary disturbance, rather than a primary one.

Another theory is a mechanical one. The suprarenal cortex is supposed to produce a hormone called Villikinine, which acts upon Meissner's Plexus and is responsible for the pumping action of the intestinal villi. The secretion of this hormone is dependant upon Vitamin B2, and is supposed to be absent in sprue.

To my mind, the most satisfactory explanation is the one put forward at the end of 1942 by Stannus, and based on Frazer's 'partition' theory. According to this scheme, you remember, when fats are broken up by the pancreatic ferments, the unsplit moiety (glycerides) is absorbed as an emulsion through the lacteals of the intestinal villi, to the thoracic duct and thence, by the systemic circulation, to the body fat depots. The split moiety (glycerol and fatty acids) in the presence of phosphoric acid and choline, either in the intestine or in the mucosa, is "phosphorylated, that is to say, the trivalent phosphoryl molecule is added with the production of phospholipines. These are carried off by way of the portal circulation, to the liver. According to Stannus, sprue is due to a breakdown in phosphorylation. In order that this reaction may take place, an enzyme is necessary, but what this enzyme is, is not quite clear. Part of the Vitamin B2 complex may be the enzyme or form part of it. Riboflavine, nicotinic acid and pyridoxin (B.6) all have a marked therapeutic value. There can be no doubt that all our cases were suffering with vitamin B defiency, but, presumably, so were the other patients in the dysentery wards. One must therefore search for an additional factor. Well, the only other thing of real therapeutic value is liver, so I think it is reasonable to imagine some factor stored in the liver. This seems to me all the more reasonable when one compares the blood pictures of sprue and pernicious anaemia, to which there is a close resemblance, except that abnormal forms of cells are rare in sprue. It may well be that there is some factor stored in the liver, closely related to Castle's H factor of P. A., which is necessary, together with vitamin B2 complex, for the phosphorylation of fats and the prevention of sprue.

Diarrhoea, in any form, whether it be post-dysenteric, Hill diarrhoea, Karachi tummy, or too much fruit at Durbar, will tend to produce an avitaminosis, varying in intensity between different people. Avitaminosis may, of course, be present without diarrhoea at all, an important point in the aetiology of sprue. It then only requires some fault in the liver factor, for sprue to develop.

Differences in composition of the fat content of the food in different parts of the world, according to Stannus, accounts for the geographical distribution of the disease, but I would also

suggest that it is to these differences that we owe the differences between text-book sprue, para-sprue in Indians, post-dysentery sprue and even idiopathic steattorrhoea or non-tropical sprue.

As I have already said, I regard all these diseases as being different stages of one disease process, brought about by deficiency of an extrinsic factor, B2, and an abnormality in an intrinsic factor, allied to Castle's H factor.

In conclusion, I would say this; that the close inspection of tongues, together with gastric analyses and the fat estimation of stools in patients suffering from diarrhoea, who complain of dyspepsia or a sore mouth, has proved worthwhile and may well have saved men from developing the full picture of sprue.

DISCUSSIONS

97. COL. TAYLOR: In the Chittagong area, during the summer of 1943, there were over a hundred cases of sprue. All stages of severity were encountered, from early, fatty diarrhoea, larval or para-sprue of Napier, to the fully developed 'true' sprue cases. The cases were all British, mainly R. A. F. and A. A. Gunners. Many of these cases began with a sudden explosive diarrhoea and early sprue symptoms developed after some weeks. Some cases began with dysenteric symptoms, with blood and mucus in their stools. No evidence exists as to any causal organisms, but laboratory facilities were not good. There was nothing special about their treatment. They recovered with correct and adequate feeding and with administration of vitamin B2 complex.

Medical specialists in this area, considered the following possible causes for this outbreak:

(1) Infective.

(2) Abnormal fats due to 'blown' tins of butter, cheese, milk, meat, etc. A large quantity of 'blown' tins were condemned in all ration stores.

(3) Excessive quantities of abnormal fat used in cooking.

(4) Infection from canned foods. A recent investigation which has only just begun, showed Morgan's bacilli in tins of food, I think in cheese and bully beef.

My own view is that the probable cause was some intestinal infection, as yet unisolated, because of the explosive onset of the intestinal symptoms in many cases. Men in all areas of Eastern India had rations which included tinned food which was often 'blown' and rancid. But the outbreak was mainly confined to one area. It will be of interest to see if a further outbreak occurs in this area this year.

98. COL. SCHLESINGER described an interesting epidemic of sprue with severe anaemia, which was occurring at Yol—near Lahore. The Kangra valley had always been notorious for hill diarrhoea, but more recently, a number, of cases of definite sprue had been reported. Indian troops, Italian P. O. W. and a few British troops, were all present at Yol, and the incidence of sprue occurred in that order, although not many British troops appeared to have suffered from it, despite the fact that they did not escape the hill diarrhoea. It was difficult to exclude diet as a factor in the aetiology. The Indian ration included only the bare minimum of vitamin B2—containing foods and the diet of the rather static P. O. W. had been unbalanced voluntarily, by the inclusion of appreciable quantities of macaroni—thus diminishing his appetite for other more valuable articles of food. The rations of the British troops left nothing to be desired and contained large reserves of vitamin B complex, thereby withstanding any loss that might be incurred through repeated attacks of diarrhoea.

96. LT.-COL. PASRICHA: Sprue is the 'ugly duckling' of tropical medicine; it is like one of those ill-kept dustbins, which has less inside and more rubbish littered outside. The very name—Sprue—deserves to be thrown deep down into the dustbin. It is often loosely applied; the public know of it and about strawberries and milk. The name frightens and often undermines the will to recover. As far as Indians are concerned, sprue does not occur—certainly not frequently. I would remind you that, for many years, work has been carried out on bowel diseases in India. I have been associated with this enquiry for more than 12 years. There are not many workers who admit the occurrence of classical sprue in Indians. What we get is a macrocytic anaemia, with diarrhoea due to nutritional defects. The aetiology of this is distinct from that of sprue. Before the label sprue is applied to a diseased condition, it is worth considering whether we will not get a truer picture by labelling it Nutritional Anaemia, or, perhaps, more correctly, Anaemia specifying the type. This would certainly lead to better treatment, for, with sprue, we are apt to think of special sprue diet and vitamins.

DECOMPRESSION CHAMBER

100. FLIGHT-LIEUT. PRICE—"*The Decompression Chamber*" **(P)**

In modern aerial warfare, flying personnel are subjected to physical and mental stresses on a scale previously unknown. In addition to facing enemy air and ground opposition, aircrews may be exposed to low atmospheric oxygen tension, extreme cold, high centrifugal force and rapid changes in air pressure, to mention but a few of the stresses.

It is essential that all flying personnel should understand the elements of aviation physiology. During training, lecture-demonstrations are given by a medical officer and sound films have been prepared by Air Ministry, to illustate the use of oxygen in aircraft, and the effects on the human body of centrifugal force. Extensive use is also made at flying training units, of mobile decompression chambers. They do not reproduce the cold experienced at high altitudes, but the pressure of the air in the chamber can be altered at will, enabling three main effects of decreased atmospheric pressure to be demonstrated—anoxia, decompression sickness (commonly known as "bends") and problems of the ears and sinuses. The insidious effects of anoxia can be demonstrated so vividly in the chamber, that the official ruling that oxygen must be used "at all heights above 10,000 feet, and from ground level when the rate of ascent is 2,000 feet a minute or more" is seen to be no peculiar whim of a distant official, but a wise and necessary precaution.

The decompression chamber is a welded steel tank, mounted on a road trailer. It is cylindrical in shape, 7 ft. 6 ins. in length and 5 ft. 6 in. in diameter, with an entrance sealed by an airtight door placed at the rear. Inside, there is seating accommodation, oxygen equipment and intercommunication telephones, for six people. During an instructional test, three people sit on upholstered seats on each side of the chamber, each seat being numbered, so that the medical officer conducting the test can speak to each person without delay, calling him by number over the intercommunication telephone, from his observation cabin at the front of the vehicle. Along each side of the chamber is a bank of steel cylinders, containing the oxygen supply. The chamber is decompressed by means of a vacuum pump, driven by a petrol engine. Pump and engine are housed on a platform at the rear of the trailer. In the observation cabin, are valves for controlling the inlet and exhaust of air and instruments for checking the altitude and rate of climb or descent at any given moment. Having ascertained that no candidate is suffering from Eustachian obstruction, the test is commenced.

The chamber is exhausted at a rate equivalent to an ascent at 1,000—2,000 feet a minute, up to 25,000 feet. Three persons use oxygen correctly from ground level, observing and guarding the other three, who remain without oxygen. During the ascent, the main features developing anoxia are described, increasing depth of respiration and the onset of cyanosis being observed, and spelling, writing and mathematical tests carried out by the persons without oxygen. Card games are popular, persons with oxygen playing those without oxygen. At 25,000 feet, signs of grose anoxia rapidly appear. The anoxic person becomes drowsy and apathetic, localised or generalised epileptiform twitchings begin, and unconsciousness rapidly ensues. Oxygen is gradually turned on to the requisite rate of flow and the person usually recommences his previous occupation, unaware that he has been unconscious or that his oxygen supply has been turned on. After discussion of the observations and symptoms, the second three persons are rendered anoxic, much to the delight of the previous victims.

When all are convinced of the absolute necessity for oxygen, ascent is continued, with all six using oxygen, to 35,000 feet or more, to demonstrate (1) the effects of low atmospheric pressure on intestinal gases, and (2) decompression sickness. During descent, methods of equalising pressure on the two sides of the tympanic membranes are demonstrated.

Apart from its educational uses, the decompression chamber is of value in assessing the fitness of persons to fly, after recovery from various ear and sinus diseases, and also in differentiating between headaches due to sinusitis and other causes. A typical sinusitis pain is accentuated at high altitudes and at rapid rates of descent, whereas most other types of headache are not so definitely affected.

At present, there are two decompression chambers in South-East Asia Command, one situated at Base Headquarters, Royal Air Force, Calcutta, and the other at Headquarters, No. 223 Group, Royal Air Force, Peshawar. Appointment may be made at either place, for educational or investigational tests on any Service personnel.

(The following photographs were demonstated by epidiascope:

(1) Four types of aircraft now in service with the Royal Air Force, to illustrate some of the conditions under which operational aircrews fly.

(2) The Mobile Decompression Chamber, including photographs taken of persons undergoing tests in the chamber.

(3) Specimens of handwriting of persons undergoing educational tests, to demonstrate the deterioration accompanying onset of anoxia.)

DISCUSSIONS

101. LT.-COL. KARAMCHANDANI enquired whether high-altitude flying tended to produce hypotension. He had recently examined a pilot officer, who complained of headaches while flying; the only sign of note was a blood pressure of $\frac{S}{D} = \frac{100}{60}$ mm. Hg.

102. MAJOR GUEST: (*a*) Do observations with the decompression chamber bear out the contention of Prof. Christy in his article on Dyspnoea (Quar. J. Med.) that pure anoxia never causes dyspnoea of itself, but only coma?

(*b*) Are pressure cabins in use, and in what type of aircraft?

103. FLIGHT-LIEUT. PRICE, in reply to Lt.-Col. Karamchandani, said, "There is no evidence that hypotension or hypertension is produced by high-altitude flying."

In reply to Major Guest, (*a*) "No, people in the decompression Chamber do feel dyspnoea and are seen to have laboured breathing." (*b*) "To ascend much above 40,000 feet, it is essential to maintain the pressure inside and outside the body above that of the surrounding atmosphere. Otherwise, owing to the high partial pressure of carbon dioxide and water vapour in the lung alveoli, in relation to the atmospheric pressure, oxygen will not diffuse into the blood stream. Hence, pressure cabins are fitted to aircraft capable of ascending above 40,000 feet. Alternatively, pressure suits may be worn by the occupants of the aircraft."

TROPICAL EOSINOPHILIA

104. MAJOR GUEST—"*Tropical Eosinophilic Asthma*" **(P)**

Under the name 'Tropical Eosinophilia', Weingarten (Lancet Jan. 23/43), has described an illness in which a state of fever and general malaise heralds the onset of acute spasmodic bronchitis; this acute phase is succeeded by a chronic tendency to dyspnoea, with paroxysmal exacerbations of cough and bronchial spasm. The degree of freedom from symptoms between the bouts of asthma, appears to vary considerably. Two features give distinctive character to the disease the presence of a very high eosinophilia and the complete clinical and haematological regression to normal, which follows intravenous medication with organic arsenic. Another feature is the constancy with which a history of prolonged residence by the sea is obtained.

A case of this syndrome occurred in a young Indian woman under our care. It is recorded, as observation of the blood picture during the course of treatment was close, and interesting changes in the character, as well as the number of eosinophilic cells were seen.

History: Miss S., Sgt. W. A. C. (I.), age 21, lived most of her life in the Punjab, but was resident in Calcutta, within three miles of the sea, for 4 years (1939—41) and illness started towards the end of this period. Was quite well until August, 1941, then had "pneumonia" and pain in the right side of the chest was severe. This illness lasted 20 days. 3-4 months later, the patient developed a slight cough and brought up sputum "like pieces of rubber" or "sometimes like boiled rice" and, on opening out the pieces, "like threads". After this state had lasted 8-9 days, severe pain in the right side of the chest recurred, cough grew worse and she took to bed. Ten days later, she felt well enough to get up, but cough continued and she felt abnormally breathless on exertion. She gradually recovered a good deal, but slight cough and mild dyspnoea on exertion continued. In early September, 1942, patient got wet and developed a high temperature, with cough and breathlessness. She was admitted to a British Military hospital and stayed there about 25 days. An X-ray taken at this time was reported as showing "bilateral chronic bronchitis with signs suggesting early emphysema". No other notes are obtainable. On discharge, her cough was slight, but she was breathless when she played games or cycled. About June 1943, attacks of breathlessness, accompanied by a feeling of suffocation

commenced. They occurred when at rest, almost every day, and often "once during the day and once during the night." The attack was relieved by coughing up sputum of the type described above. During the attacks, a wheezing sound could be heard. No other symptoms were complained of. Bowels were normal, there had never been any skin trouble, and no tendency to migrainous headaches. No family history of any similar trouble.

Examination at this time (11*th September*, 1943) A girl of good physique and rather slim. Chest expansion moderate. Rhonchi and rales could be heard all over both lungs, otherwise no abnormality detected. Total white cell count 15,000 per c.m., 73 per cent. of which were large eosinophils. The stools had been repeatedly examined; no worm eggs, cysts or other abnormalities being found. The patient was admitted with a view to treatment with neoarsphenamine injections.

Abbreviated summary of progress: Following the second injection of N. A. B., vacuolation of the eosinophils was seen, later the granular matter became condensed, the eosinophilic staining of the cytoplasm becoming more or less even. About 2 grams of N. A. B., given over a period of about 5 weeks, resulted in a fall of eosinophils from 11,000 per cmm to under one thousand per cmm. Clinically, paroxysmal dyspnoea ceased, breathlessness no longer occurred too easily on exertion and the lungs were clear on auscultation.

105. MAJOR LEHMANN—"*Investigation and Treatment of Tropical Eosinophilia*" **(D)**

Major Guest has outlined the syndrome, which is, briefly—Asthma—with an increased absolute eosinophil count—cured by N. A. B. The lowest in Weingarten series having 7,200 eos. per cmm. (normal approximately 200). I first met this syndrome in a case shown to me at Dehra Dun, early in 1943, but had not seen Weingarten's paper then. Later, I saw two articles, one entitled 'Pseudo-Tuberculosis' and some notes in the I. M. G. by Simeon, and decided to follow up any further cases, Major Guest, at that time, very kindly lent me Weingarten's paper. Since then I have seen 12 cases.

In November, 1943, I arranged with the Medical Specialist, Meerut, that all cases of asthma should be sent to the Dist. Laboratory for W. B. and Differential counts and that he should take case histories of these, with special reference to this syndrome. By this method, 6 cases were discovered with Absolute Eosinophil counts varying from 6—66,000. The following points from these cases may be of interest:

Environment—Only one lived near the sea. But all had lived near a big river or canal.

Haemoptysis—2 gave a history of haemoptysis.

Eosinophils—These differed from Weigarten's description.

He stated that they were normal in size and shape, fully mature, with a shift to the right (Arneth count). The ones seen by us were, on the whole larger than normal—some up to 20 Micron. These were not myelocytes as one might have expected from their size, but were nucleotoid. If we consider Ponder's work on the degenerative Index—classification from:

(i) large size of the cytoplasimc granule,
(ii) amoebicity and vacuolation,
(iii) pyknosis of the nucleus,

it can be seen that these cells showed these features and were high in the degenerative scale.

Sternal punctures were done in two cases, but, apart from the increased number of eosinophilic granular cells, the ordinary differential counts of bone marrow were found.

X-rays—These were taken, but, as was to be expected at this advanced stage, they showed only the changes of chronic bronchitis and not the generalised symetrical mottling, reported in early cases.

While these cases were having their differential and total counts done daily and being treated, arrangements were made for 250 recruits to come to the District Laboratory and differential counts were done on these, to see if this condition was prevalent. Of these 250, 11 cases (4·5 per cent.) showed eosinophile cells between 20 and 50 per cent. of which 6 cases (55 per cent.) gave an asthmatic history. These are being followed up and treated. Careful examinations were done and histories taken, to exclude other causes of eosinophilia, e.g.

allergy, skin conditions, helminthic infection, drugs, etc. Apart from allergy (asthma), none of these were discovered.

I feel that this syndrome should be widely known, as all asthmatic cases are boarded out of the army, often after months of training. It will be of value if even a small proportion prove to be this condition and can be cured.

I was once shown a case of idiopathic eosinophilia, 26,000, 30 per cent.—46 per cent. which, when treated with quinine grs. 10 T. D. S., for 10 days, returned to normal at the end. For this reason, one case was treated in this way. It is of interest, in as much that, although this case had a comparatively low count, he rapidly returned to within normal limits. Unfortunately, he was discharged while I was away and his unit had left and I could not follow him up. I think, however, if quinine can be spared, that further cases should be treated in this way.

(Four diagrams of abnormal eosinophiles and five graphs, showing blood counts, treatment and after effects, were demonstrated by epidiascope.)

DISCUSSIONS

106. COL. CAMERON: This condition has been known in India for several years. My attention was first drawn to it by Dr. Frimodt Moller, at Madanapale sanatorium, some months before the appearance of Weingarten's paper. Under the title "Eosinophilic Lung", Frimodt Muller and Barton had reported numerous cases in the *Indian Medical Gazette* (1940, 75, 607). They arrived at the sanatorium, diagnosed as T. B., because of the snowstorm appearance of the radiogram, suggestive of miliary tuberculosis. Designating the condition, Weingarten's disease, or syndrome, is therefore to be deprecated.

We have had quite a large number of cases in the hospitals of Southern Army. All but one were from near the sea, the exception had never left Bangalore. Two of the cases appeared before the Review Board, "Recommended Category E—Asthma". Both returned to service after treatment.

Major Lehmann's case showed a white count of 100,000. The highest I have seen was 84,000 with 80 per cent. eosinophils. When discussing the condition with Dr. Berger, who had a large experience of the disease in Bombay, he impressed the need for at least 8 injections of N. A. B., if recurrence was to be prevented. Three injections might give apparent cure, but 8 were required to ensure against relapse. The aetiology of the condition might be the pulmonary circulation of larval parasites, prior to their settlement in the intestine, e.g. ascaris, ankylostoma, strongylodies.

107. COL. TAYLOR: The condition was described in German, about 1923, as Benign Eosinophilic infiltraion of the lung, in a paper to which Dr. Frimodt Moller referred, in the *Indian Medical Gazette*. I happened to see two such cases—one in Lahore and the other in Barrackpore, before I had heard of the treatment by arsenic injections. They were acute cases, in which the radiologist had suggested military tuberculosis. The eosinophilia count was up to 70 per cent. These two cases cleared up in 3 months, without arsenic of any sort and with no other specific treatment. One, a nursing orderly, is fit and working, a year after this acute attack.

108. LT.-COL. KELSALL mentioned that (i) one case was treated at 3 I. B. G. H. (B. T.), with Stovarsol by mouth and the result was as good as those obtained by intravenous N. A. B. in six other cases; (ii) One speaker at a Southern Army Medical Conference had claimed that the mottling of the lung fields, sometimes seen on X-ray in these cases, could be seen on the screen to disappear, following an injection of adrenalin, suggesting that they had an allergic basis; (iii) one of his seven cases showed strongyloides stercorales in the stool and that a pathologist had informed him that he (the pathologist) had never seen a case without strongyloides in the stools; (iv) He had never seen a case except in Indians and Anglo-Indians.

He therefore enquired (*a*) whether anybody at this Conference could confirm this statement (ii); (*b*) if anyone had any evidence regarding (iii), which might throw light on the possible relationship of the condition under discussion to strongyloides infection, since the latter infected the lungs; (*c*) whether anybody had seen the disease in non-Indian races.

109. LT.-COL. PASRICHA replied that strongyloides infection was common in Bengal, but associated eosinophilia had not been observed. In addition to the two well-known

helminths—ascaris and hookworm, which have a developmental sojourn in the lungs, there were two others—Strongyloides stercorales and Paragonimus various species. The latter was of particular interest; it was the parasite that developed in raw crab or crayfish eaters, in certain areas. It was worth keeping in mind, in obscure cases of rusty sputums.

110. BRIGADIER McALPINE remarked that nothing had been said regarding the aetiology of this condition, except a suggestion from Lt.-Col. Fasricha.

111. MAJOR GUEST agreed that the condition could not be regarded as Weingarten's syndrome. He remarked on the spontaneous recovery and vacuolation of the Eosinophiles.

112. MAJOR LEHMANN, in his reply to various questions, said,

(i) It is known that these cases do have remissions. In my opinion, it is very probable that, if the case had been followed up, he would have been seen to have further attacks (Para. 107).

(ii) A careful history was taken and examinations made, to exclude all known causes for increased absolute eosinophilia counts, including, naturally, stool examination (Para. 108).

(iii) Regarding the length of history, one of the cases in Meerut gives a history of 10 years, without treatment; the disease seems to be progressive.

(iv) With regard to prognosis, it has been stated that, of less than eight injections are given, relapses will occur. Our cases have not been followed up for a sufficient length of time for any authoritative statement to be given on this point."

(*The Meeting adjourned at* 17·30 *hours*)

9TH FEBRUARY, 1944—MORNING SESSION

The Conference commenced at 09.00 hours, with **COLONEL WHITE** in the Chair. The Chairman regretted the absence of Brigadier Covell, the head of the Malaria Institute in India, and Consultant Malariologist, India Command, who had recently toured America, and whose paper would be read by Lieut.-Col. Lamprell.

MALARIA

113. BRIG. COVELL—"*Recent Developments in the Treatment of Malaria*" **(P)**

Standard Treatments

Prior to and during the war of 1914—1918, it was customary to prescribe quinine, in massive doses and over long periods, with the object of diminishing the number of relapses. The argument was, that, since quinine has a specific action on the malaria parasite, its failure to prevent relapse must be due either to insufficient dosage, or too brief a period of treatment. In one of the systems of treatment, used in the Macedonian campaign, no less than 1,180 grains of quinine were administered, over a period of 29 days, and up to the year 1930, the courses usually recommended in various countries, consisted of 600 to 700 grains of the drug, given over a period of 1 to 2 months.

In 1939, Sinton published a paper, based on the treatment of 3,700 cases of malaria, advocating a standard treatment in which 30 grains of quinine were administered daily, for 7 days, followed by 0·015 gramme of plasmoquine daily, for 7 days, a total of 210 grains of quinine plus 0·105 gramme of plasmoquine. From this time onward, it has been generally recognised that the administration of massive doses of anti-malarial drugs, over long periods, is no more effective in producing a permanent cure, than short courses with moderate dosage, and may, indeed, be actually detrimental to the patients' general health.

The course recommended by the League of Nations Malaria Commission in 1937, consisted of 15 to 20 grains of quinine, or 0·3 gramme of mepacrine daily, for 5 to 7 days, followed by 0·02 gramme of pamaquin daily, for 5 days. The standard treatment for the Army in India resembles this, the chief difference being that quinine only is given during the first 2 days and mepacrine only during the next 5 days of the course.

Methods of Administration

There is still some divergence of opinion regarding the parenteral administration of anti-malarial drugs. It has been claimed that intramuscular quinine has the advantage over the oral method, in respect of rapidity of action, prolongation of action, prevention of relapses and greater therapeutic value. The first three of these supposed advantages

have been definitely shown to be non-existent, whilst the last is probably due, in the main, to the fact that intramuscular quinine is usually administered by the medical officer himself, and the patient actually does receive and retain the correct dosage, which is often not the case when it is prescribed by the oral route. The generally accepted opinion regarding intramuscular quinine, is that its adoption as a routine method of treatment is not justified. It is true that, with patients whose general health is otherwise good, the proportion in which abscess occurs is very small; and it is possible to give a very large series of injections without apparent ill-effects. No one, however, who has actually seen a few of the unfortunate results which occur, is likely to prescribe quinine by this method, if it can be taken by mouth. The risk of untoward results is greatly increased, in the case of debilitated patients, and in those suffering from wounds. If treatment by intramuscular injection is decided upon, it would seem preferable to give mepacrine methane-sulphonate (atebrin musonate), which causes considerably less necrosis, rather than quinine.

The intravenous method of giving quinine, has no greater effect in preventing relapse, or producing a permanent cure, than when the drug is given orally, but it is of the greatest value under certain emergent conditions, e.g. (1) where the patient cannot swallow quinine or mepacrine, (2) Hyperinfection (where more than 4 per cent. of the red blood cells contain parasites: where more than 5 per cent. infected red blood cells contain 2 or more parasites, or where pigmented assexual forms of P. falsiparum are present in the peripheral blood), (3) Acute pernicious malaria (cerebral, cardiac, algid), (4) Hyperpyrexia, (5) Persistent vomiting, (6) Where, despite oral quinine in adequate dosage, asexual parasites persist in the blood or the temperature fails to fall to normal in 3 or 4 days (in such cases, however, it usually transpires that the drug is not actually being taken in the dosage prescribed).

Mepacrine versus Quinine in the Treatment of Malaria

The relative therapeutic value of mepacrine and quinine has been the subject of some divergence of opinion, owing, chiefly, to the fact that different strains of malaria parasites vary in their reactions to the two drugs, in certain respects. Some observers have reported that malignant tertian infections are more rapidly controlled by quinine than by mepacrine, whilst others have recorded a diametrically opposite conclusion. It is probably true that relapses are fewer on the whole, following a 5-day treatment with mepacrine, than with a 5-day treatment with quinine. The disappearance of malaria parasites from the blood, is usually effected somewhat earlier by mepacrine than by quinine.

Quinine has an essential antipyretic action of its own, and it is for this reason that some clinicians prefer to treat malignant tertian cases with quinine for the first 48 hours, followed by 5 days treatment with mepacrine.

The Dosage of Mepacrine

Recent investigations in the U. S. A. have shown, (1) that where mepacrine is given in the usual therapeutic doses, the maximal level of the drug in the blood, is not reached for several days, and, (2) that there is a very wide variation in the height of blood level attained by different individuals who are taking the same dosage of the drug. It is presumed that the therapeutic effect of mepacrine depends on its concentration in the blood plasma, and it has been suggested that a higher dosage should be administered during the first day of treatment, so that the maximal blood level may be attained with the least possible delay. The U. S. Army have recently adopted a regime in which 1 gramme mepacrine (in 5 doses of 0·2 gramme each) is administered on the first day, followed by 0·1 gramme thrice daily for the next six days. 10 grains sodium bicarbonate is given with each dose, on the first day. I understand that experiments are now in progress with a still higher dosage, in which 1 gramme of the drug is given on each of the first three days of treatment.

The delay in reaching the maximal blood levels is still more prolonged with suppressive than with therapeutic doses of mepacrine. It is advisable, therefore, to commence suppressive treatment at least a fortnight before the troops enter a malarious area, or else to prescribe a higher dosage (e.g. 0·2 gramme daily) during the first 4 or 5 days, and to recommence with a similar 'boosting' dosage, if there is at any time a break in the continuity of treatment.

The Status of Pamaquin (Plasmoquine) in the Treatment of Malaria

Pamaquin has a marked destructive effect on the gameto-cytes of all three of the common species of human malaria parasites. It has practically no effect on the trophozoites of P.

falciparum, but has considerable schizonticidal action on infections with P. vivax and P. malariae. Its principal value in the treatment of malaria, however, is its effect in reducing the relapse rate in chronic infections of benign tertian and quartan malaria.

The effect of pamaquin in combination with quinine, in reducing the relapse rate in chronic benign tertian infections, was demonstrated at the Malaria Treatment Centre, Kasauli, some years ago. The subsequent introduction of a combined plasmoquine-quinine treatment for malaria in the Army in India, resulted in the closure of the Centre, for lack of patients. Similar results were obtained among the U. S. troops in Panama, some years later, the relapse rate in chronic benign tertian infections being reduced from 45 to 9 per cent, by the administration of a 3 days course of pamaquin (0·01 gramme tds), following a 4-day's course of mepacrine (0·6 gramme daily). The value of pamaqun in reducing the relapse rate in similar infections in Mid-East, has been recorded during the present war.

Whilst there is no doubt of the value of pamaquin in chronic relapsing P. vivax infections, however, its effect on the early (first four months) relapses of acute infections, is less evident. Such relapses have been so frequent in the campaign in New Guinea, that pamaquin therapy no longer forms part of the routine treatment of U. S. troops in this area, its use is being left to the discretion of individual medical officers.

The Sulphonamide Group of Drugs

Some years ago, the discovery that some of the members of this group, acted as causal prophylactics of certain species of avian and simian malaria, gave rise to the hope that sulphonamide derivatives might prove equally effective in human malaria. A number of these have been tested recently in both the U. K. and U. S. A. and some of them have given promising results. S-diazine and S-merazine have been recommended in U. S. A. Claims have been put forward that the former is a true suppressive in P. falciparum infections and may turn out to be a causal prophylactic of this species. The general opinion is, however, that any action these drugs may have is curative in the very early stages of the parasite, rather than truly prophylactic of infection. A certain number of field tests have been conducted among U. S. A. troops, with S-merazine, but the results have not been very promising. As suppressives, the sulphonamide drugs in general, act more effectively against malignant tertian than benign tertian infections, while, with mepacrine, the reverse is the case.

In the course of a recent tour in the U. K. and U. S. A., I had the opportunity of meeting most of the workers on antimalarial drugs, in both countries. At the close of each discussion, I put the question, "Do you consider that any of the sulphonamide drugs is likely to prove of practical value as a prophylactic of malaria during the present war?" The answer was invariably in the negative, the reason being that, in order to afford protection for all the troops, the dose administered would have to be too high for safety.

Other Drugs

In the U. S. A. promising results have been achieved on benign tertian and quartan malaria, with a drug known as 204, one of the phenanthrene group. It is less effective in malignant tertian infections, with the same dosage. Tests were incomplete at the time of my visit. The drug is cheaper to produce than mepacrine and more easily manufactured, but it will take some time to get it into production.

Imperial Chemical (Pharmaceuticals) Ltd. have obtained encouraging results with a new drug, as yet unnamed, which has no chemical resemblance to mepacrine, the sulphonamide drugs, or the phenanthrenes. Preliminary tests in England indicated that this is as effective as mepacrine (perhaps more so), but a limited number of tests, carried out on monkey malaria in this country, have been less successful. This drug also, is said to be more easily produced than mepacrine.

114. LT.-COL. ROGAN—"*Relapsing Malaria and War*" **(P)**

The title of this paper, relapsing malaria and war, has been chosen advisedly. In time of peace, the faculty possessed to a unique degree by the malaria parasite, of causing recurring illness in the infected individual, is responsible for much ill-health and consequent social and economic distress among the communities affected. In war time, however, the significance of relapsing malaria is enormously enhanced. During a short campaign in malarious country, such as our invasion of Sicily, or the Japanese invasion of Malaya, heavy casualties may be suffered from fresh attacks of malaria. On the other hand, when protracted operations have

to be conducted in areas where malaria is hyperendemic, such as the Eastern Frontier of India, the additional problem of relapsing malaria becomes acute, as the fighting efficiency of the troops may be gravely reduced, by a high incidence of relapses among the military population. It will thus be apparent that every aspect of relapsing malaria deserves careful study by the military medical services.

"Relapsing malaria" is a convenient term for that condition, in which a patient who has been exposed to malaria, suffers from repeated attacks thereafter, in spite of treatment and in the absence of re-infection. Before considering relapsing malaria in detail, it may be helpful to discuss, briefly, the cause of a true malaria relapse, which may be defined as a recurrence of clinical malaria, due to a reappearance of the strain of parasite which caused the primary attack.

Many theories have been offered, to explain the phenomenon of the malaria relapse, such as:

(1) That schizonts are derived from gametocytes. This theory was advanced by Schaudinn, who observed both a gametocyte and a schizont in one red cell and concluded that the schizont was developing from the gametocyte. It was later pointed out that the association of gametocytes with schizonts in single cells, is purely fortuitous.

(2) That relapses are due to strains of parasite specially resistant to treatment. No such strains have been identified.

(3) That relapses are due to a persistance of the schizogony cycle, as a low grade of afebrile infestation. This theory, once very widely held, was advocated by Ross, Bignami, James and Knowles. When one considers the enormous reduction in the number of parasites in the blood, brought about by a few doses of quinine, it is difficult to understand why a complete course of quinine treatment fails in many instances to kill out the infection, if we assume that the schizogony cycle continues unaltered.

In spite of extensive speculation, it is still unknown why relapses occur. An indication, however, is provided by the action of pamaquin, which has a definite effect in reducing relapses. This effect is unlikely to be due to the action of the drug upon the schizogony cycle, which is feeble, and it is reasonable to suppose that pamaquin acts upon the malaria parasite, in some phase of the parasitic cycle as yet undiscovered, which does not take place in the peripheral blood. The fact that this phase has not yet been found, is no evidence against its existence, particularly when it is remembered that almost complete ignorance still prevails about the parasitic cycle during the incubation period. During recent years, the existence of a phase of development of the malaria parasite in the reticulo-endothelial system and outside the erythrocytes, has been found in birds. This phase of the parasite is resistent to quinine and can give rise to the schizogony cycle, if it is inoculated into a clean bird. Raffaele has claimed to have discovered a similar phase of the malaria parasite in man, but his claim has not been generally accepted.

Returning to the consideration of the clinical condition, relapsing malaria, it should be borne in mind, that this condition, as it is encountered in war time, is seldom simply due to a succession of true relapses, following a single primary infection. It is usually the result of one or more of the following causes:

Cause (*i*)—*Multiple Infection*—Fighting conditions on the Burma front are such, that troops operating in the front line are frequently unable to use mosquito nets, for days and even weeks at a time. They are manoeuvering in areas where malaria is hyperendemic. It is, therefore, exceedingly likely that a large proportion of the force, thus continually exposed to malaria, acquires more than one infection. It will be obvious that this has occurred when troops, admitted to hospital with B. T. malaria, develop M. T. malaria, soon after or during treatment, or vice versa. What is not quite so obvious is, that an early recrudescence of malaria, due to the same species as that causing the primary attack, may not be a true relapse, but may be due to a different strain. In such a case, both the first and second attacks of malaria may be primary attacks, arising from different strains of the same species of parasite, in a patient who has been doubly infected. Experimental work in artificially induced malaria, has provided some interesting information regarding multiple infection. It has been shown that, when a multiple infection occurs, immunity is interfered with, to some extent. It has also been shown that, when a patient is infected with two different species of

parasite simultaneously, e.g. M. T. and B. T., one of the infections tends to become dominant at the end of the incubation period, causing clinical malaria. The other infection usually remains latent for a variable period, causing clinical malaria, after the attack due to the dominant infection has subsided.

The practical importance of multiple infection is this. Soon after it occurs to a considerable extent, early recrudescences of malaria after treatment may be numerous and may erroneously be regarded as true relapses. Later, a large number of true relapses must be expected, as a sequel to the original primary attacks.

Cause (ii)—Inefficient Treatment of Primary Attacks—This cause has, in the past, been a potent source of relapsing malaria. The necessity for ensuring that every case of malaria actually swallows all the drugs prescribed in the standard treatment, has been heavily emphasised, and it is hoped that this cause of relapsing malaria may diminish in importance. It should be realised, however that, in the conditions prevailing in the forward areas, it is sometimes exceedingly difficult to ensure efficient treatment.

Cause (iii)—Deficient Immunity—There is a considerable individual variation in immunity to malaria. Without entering into a discussion on the very extensive subject of immunity, it may be stated that certain individuals tend to relapse repeatedly, after one or two infections with malaria. The evidence available suggests that such individuals comprise only a small part of the population affected by malaria. Deficient immunity is suggested by frequent relapses after a short exposure to infection, and by frequent relapses outside, as well as within, the time limits when relapses may be expected. The time limits within which relapses commonly occur, are as follows: in M. T. malaria, within 6 months of the primary attack and in B. T. malaria, within 3 months of the primary attack and later from 6—9 months after the primary attack. Severe constitutional disturbance accompanying each relapse, also suggests deficient immunity.

Realization of the complex aetiology of relapsing malaria may help to explain the sometimes disappointing results of treatment.

Two graphs may be of interest to the Meeting. It will be appreciated that, soon after the height of transmission in a hyper-endemic area, an increased incidence of relapsing malaria, due both to short-term relapses and to the maturation of latent infections, may be expected. It is difficult to obtain relevant statistics in the field, owing to the difficulty of distinguishing fresh attacks from relapses. Figures can, however, be obtained from convalescent depots. The graphs show the percentage of convalescent malaria cases returned to hospital, each month, with a recrudescence of clinical malaria, and also give an indication of the transmission rate for reference. It will be noted that the graphs show a steep rise soon after transmission has reached its maximum. Graph I deals with British cases, admitted to a convalescent depot near the Arakan and indicates, incidentally, that the relapse rate has been modified by suppressive treatment. Graph II deals with Indian cases, admitted to two convalescent depots in Assam and includes a large proportion of L. of C. troops, who had not been on suppressive treatment.

(*The graphs were demonstrated by epidiascope*)

Turning to the treatment of relapsing malaria, the first principle of treatment is prevention, and application of the most complete anti-malaria discipline in the field will do much to prevent the acquisition of multiple infections. This is hardly the role of the clinician, which, however, commences once the infections have been acquired and the patients develop primary attacks of malaria. Then the utmost efficiency in treatment must be achieved, in order to kill out as many infections as possible. However efficient this treatment may be, a certain proportion of the troops operating in hyperendemic areas, sooner or later become debilitated as a result of repeated attacks of malaria and require evacuation to the rear, where they present the hospitals receiving them, with the considerable problem of their treatment. Here, the aims of treatment should be :

(1) Sterilization of each infection as it becomes clinically manifest. The standard treatment is the best treatment, so far discovered for this purpose, suitable for general use.

(2) Provision of a generous diet. It is generally agreed that immunity to disease is seriously interfered with by malnutrition. There is no reason to suppose that malaria

is an exception to this rule, and the diet of cases of relapsing malaria should be sufficient to make good the pre-existing dietetic deficiencies. In relapsing malaria, repeated blood destruction occurs and, in this connection, it might be apposite to mention recent work by Madden, Whipple and others, who have shown that a diet rich in amino-acids, aids appreciably in the regeneration of erythocytes.

(3) Correction of anaemia. The proper treatment of malarial anaemia is the treatment of malaria, and remarkable improvements in the blood picture of patients suffering from relapsing malaria have occurred, after the efficient administration of one course of standard treatment. Iron and liver should be given, when they are indicated. All cases of severe anaemia should be investigated for hookworm and amoebiasis, which may be present as complicating factors.

(4) Provision of facilities for recreation and rehabilitation. Patients who have suffered from repeated attacks of malaria, are not infrequently depressed and anxious about their physical condition. Every opportunity should be sought to reassure them and to provide them with recreation, to counter introspection. It is tactless to talk to patients about their relapses. It is better to tell them that they have picked up several infections, which will gradually be killed out as they appear and are treated. The rehabilitation of patients with relapsing malaria, is an important part of treatment. When they reach rear hospitals, they have frequently suffered considerable deterioration in physique, so that physical re-education may be necessary, before their return to the forward areas.

So far, this paper has dealt with the clinical aspects of relapsing malaria. In conclusion, it may be of interest to attempt to apply clinical knowledge to problems of strategy in the South East Asian theatre of war. As we advance south-eastwards, the climate becomes subject to little variation throughout the year, and the monsoon season is less well defined. In consequence, the transmission of malaria remains high, all the year round, and is subject to less seasonal variation than in the North. Into those areas will be deployed troops of the Allies. If they remain fighting in those areas, month after month, not only will casualties from fresh malaria be heavy, but, after a few months, the casualty rate will be considerably increased by relapsing malaria, although suppressive treatment may mask this increase to some extent. The casualties from fresh malaria will, it is hoped, be largely unavoidable. The relapses will, unfortunately, be inevitable, unless some revolutionary improvement in treatment is discovered in the near future. If, however, the forces sent into hyperendemic areas were divided into two, and half the strength of each force employed alternately in those areas for 3 months at a time, the periods when the majority of relapses would occur, would be spent while the troops were withdrawn out of the fighting zone, to remote areas with the lowest possible incidence of malaria. This plan can be more easily explained by means of a diagram. Objections to the operation of such a scheme as this can easily be foreseen, nevertheless, when it is remembered that, up to the present, casualties from malaria in the forward areas have exceeded battle casualties by more than ten to one, it would seem reasonable to give it serious consideration.

(*The diagram was demonstrated by epidiascope*)

115. LT.-COL. HINDS-HOWELL—"*Malarial Relapses*" **(D)**

I have had two opportunities of studying malaria, the first in Polish refugees, and the second in an English Infantry regiment, which was in Burma with Wingate's expedition. They both show very nicely, two of the characteristics which lead to chronic malaria; malnutrition, and delayed or inadequate treatment.

The Polish refugees, mostly women and children, came to us from Russia, via Persia and Iraq. They had been living on grass, and so on, and had all got malaria, for which the treatment had been extremely casual, and were relapsing on every stage of their journeyings. Even by the time they had arrived at Karachi, they were still emaciated in the extreme, and went down with malarial relapses in rows. We gave them the routine 2.5.2.5. treatment, which stopped the acute attack, but did not stop the relapses, which occurred with monotonous regularity at the end of the treatment. In the end, we resorted to prolonged quinine therapy, with fair success, but their stay with us, about six weeks per batch, was too short to allow of any conclusions to be drawn. If it be true that the parasite prefers young blood cells to the mature erythrocyte, then these Poles should have been a fertile field for them, as, in fact, they were.

Turning now to the infantrymen. Their position in Burma can have been little better than that of the Polish refugees. I do not know how many went into Burma, but only a few over three hundred came out of Burma at the end of May, mostly through Imphal, went to Bombay and Jhansi and arrived at Karachi towards the end of September, so that, by then, all traces of malnutrition had disappeared, but I gather that it had been severe. Now, all these were supposed to have had suppressive and blanket treatment, but this did not prove practicable, and many were unable to get it. To give a few figures, if I may: There are four groups. The first group of 42 had neither suppressive nor blanket treatment, but only 11 of them developed malaria. The second had no or inadequate suppressive treatment, but did get blanket treatment; of the 134 men in this group, 94 developed malaria and 40 avoided it. Then there is a funny little group of men, 19 in number, who, having had their suppressive treatment, somehow or other missed their blanket treatment. Only 8 of these got malaria. Finally, there is the group of those who had the complete treatment, 88 out of 123 developing malaria. Put in another way, of the 176 men who had no suppressive treatment, 105 developed malaria, and of the 152 who did have suppressive treatment, 99 got malaria. A curious feature was the number of men who showed malaria, either whilst on blanket treatment or immediately afterwards, no less than 71 men going down with malaria during this period. The rest of the story of these men is continual relapse. As with the Poles, 2.5.2.5. is of no value in reducing the relapse rate. The Tropical School, Calcutta, has been trying out N. A. B. and, although 606 was tried in 1920 and rejected, I decided to try N. A. B. on these men. I gave 0·3 gms, once a week for three weeks, to 28 men who were relapsing at the rate of once a month. 23 have now been free for two months, but five have relapsed.

DISCUSSIONS

116. COL. TAYLOR gave some figures to indicate the extent of the malaria problem in the Eastern Army in jungle warfare and referred to the interesting researches that were carried out by Lt.-Col. Rogan. In 1943, out of about half a million cases admitted into medical units, over 200,000 were due to malaria and N. Y. D. fever. During the spring of 1943, in the Arakan, two-thirds of the effective fighting troops in one British infantry brigade and over 30 per cent. of two battalions, were evacuated because of malaria.

117. LT.-COL. KARAMCHANDANI: I wish to draw the attention of the Conference to two adjuncts, which have been lost sight of. One is the use of alkaline mixture, consisting of soda bicarb. gr. 60, Pot. Citras gr. 40, Cal. Chloride gr. ii, in one ounce of water, administered one hour before each dose of quinine. Sinton has proved that quinine is seven times more efficacious in the presence of alkalies. Secondly, in those cases where intravenous quinine, for some reason, cannot be administered, quinine acid hydrobromide by deep subcutaneous injection, gr. 5 in 3 cc of aqua distillata, may be borne in mind. This route of medication was worked out by me, after research in the malaria institute with Col. Sinton in 1932, and published in the records of *Malaria Surevey of India*, Vol. II, No. 3, June 1933, pp. 456 and 470. Quinine acid hydrobromide, when injected hypodermically thus, is free from all risks attending intramuscular injections of quinine acid hydrochloride.

118. MAJOR GUEST quoted some statistics of malarial incidence and of response to treatment with a routine course, supplemented with arsenical injections.

A battalion with strength of about 800 men, went into the Comilla-Chittagong area in May, 1942, and into the Arakan in November-December, 1942. A review of figures after one year, i.e. on 12th November, 1943, showed that only 53 out of the original 800 had escaped malarial infections. The battalion came out of front area in May, 1943; some went to Ranchi, some to the Hills, and the remainder to Fyzabad. They started to move to Lucknow in mid-August, 1943. Battalion questionnaire on parade, to about 800, on 25th January, 1944, regarding the number of attacks since May, 1943 (all such attacks being, presumably, relapses):

Number of attacks	Number of men
8	17
9	8
10	3
11	1
13	2
14	2

It is obvious that men who admitted to more than 8 attacks in the period of 8 months, were referring to the number of attacks they had altogether.

Follow-up of 28 men, after special course with arsenic

Course : Quinine (2 days), Mepacrine (5 days), 1st N. A. B., Pamaquine (5 days), then N. A. Bs. in the following week.

Relapse after this treatment (by 28th January, 1944).. 15

No relapse 13

Interval between the last N. A. B. injection and the relapse:—

11 weeks. 4 weeks. 4 weeks. 10 weeks. 3 weeks. 9 weeks. 5 days. 3 weeks. 19 days. 7 days 6 weeks. 7 weeks. 23 days.

119. LT.-COL. HAVILAND-MINCHIN suggested that G. H. Q. instruction should be given on the treatment of the patients, evacuated from the Eastern Army, who had had only a part of their malaria course. He had given plasmoquin only when the atebrin treatment was completed and had repeated the whole course of atebrin and plasmoquin when the atebrin course was stopped half-way through. In patients who had relapsed during or soon after a course of treatment, it was his practice to give one or two injections of adrenalin, with the idea of driving the parasites out of the spleen and the other internal organs, and permitting blood, rich in the drug, to be drawn into these areas. The results seemed to have been good. He further enquired whether solution of mepacrine was stable.

120. MAJOR KILOH: While I was at 47 B. G. H., an experiment was commenced, in giving suspensions of mepacrine and plasmoquine in mucilage. Could Col. Taylor give any indication of results of this treatment? I was posted a few days after it was commenced.

121. CCL. TAYLOR replied that it was stopped and not proceeded with.

122. LT.-COL. PRESTON: All medical officers are seriously perturbed at the high relapse rate. Practical experience with mepacrine and pamequin does not seem to come up to the optimistic picture painted by the standard text-books. Reasurance would be welcomed, that the drugs we are receiving, have been standardised and are of guaranteed potency.

123. LT.-COL. PASRICHA: As the compiler of the Blue Book, on the standard treatment of malaria, I would like to draw attention to the fact that intravenous use of mepacrine methane sulphate is not recommended. Its use by this route is dangerous. Mepacrine and atebrin are the same, except in their proprietary names. Experimentally in animals and clinically in man, mepacrine is as active as atebrin. I am one of those who is against the use of quinine by the intramuscular route. The experimental work of Acton and Chopra has been confirmed by a host of observers.

124. LT.-COL. MURTHI: In view of the large-scale use of mepacrine in the preventive treatment of malaria, and in view of the possible dangers of malarial psychosis, information is sought whether any worker has studied the retention of mepacrine in the various viscera, either experimentally or otherwise. So far, the American literature gives the liver as the organ that retains most.

125. MAJ.-GENERAL MARTIN said that initial treatment with atebring musonate injections had given good results in Waziristan, especially over relapse rates.

126. LT.-COL. ROGAN, in reply to various questions:

(*a*) There is no evidence of a new malignant strain of malaria in the forward areas. It is true that there are many cases of severe M. T. malaria, but there is also a very large amount of malaria, and the incidence of severe cases should be kept in proper perspective. During the height of transmission of M. T. malaria, there may be a temporary exaltation in the virulence of strains.

(*b*) Mepacrine can be prepared in a stable solution. If, however, the drill for the administration of tablets has been efficiently organised, it seems unnecessary to give mepacrine in solution, in which form it has a highly unpleasant taste.

(*c*) The use of adrenalin, as an aid to treatment, is not advised. No clear evidence of the value of adrenalin in treatment has been obtained.

(*d*) One speaker has questioned the potency of mepacrine. Experience in every theatre of war and careful biological tests have vindicated the efficacy of mepacrine. The American forces have now dispensed altogether with quinine, in the routine treatment of malaria, and rely solely on mepacrine.

127. COL. SCHLESINGER emphasized the need for more frequent check of quinine and mepacrine absorption, by urine tests. He advocated the more liberal use of the intravenous

route, early in the course of treatment, if the temperature persisted. In that case, the fact that anti-malaria drugs were known to have been swallowed, was not sufficient, and use of the above-mentioned tests should be made.

128. LT.-COL. KELSALL: Is there any evidence for the statement, sometimes made, that the liability to relapse in B. T. malaria is less, if patients are deliberately allowed to have several rigors, before treatment is started?

129. LT.-COL. ROGAN replied that there was no convincing evidence to that effect.

130. COL. CAMERON: Brig. Covell's paper referred to the use of sulpha drugs, including sulphadiazine, in the treatment of malaria. An effectively controlled experiment on sulphadiazine in malaria was carried out by Coxon and Hayes at 3 I. B. G. H. This showed conclusively, the low value of sulphadiazine as an anti-malarial drug. Most of the cases so treated, relapsed within 6 days of arrival in a hill station convalescent depot.

131. COL. TAYLOR: Atebrin musonate has been short in supply and so has not been used in a large number of cases. Obviously, the suggestion of General Martin will be of great value, when supplies are adequate in forward areas.

132. LT.-COL. LAMPRELL—"*Suppressive Treatment of Malaria*" **(P)**

Suppressive treatment is the regular administration of a small dose of anti-malaria drug, over an extended period. It does not prevent infection with malaria. Its object is to reduce the risk of the individual succumbing to an attack of fever, so long as the treatment is continued.

Almost a year ago, I was asked to speak on this subject, at a meeting of medical specialists in Eastern Army. On that occasion, the findings of the League of Nations Commission, regarding this measure, were considered in some detail, the results of suppressive treatment with quinine during the war 1914-18 and the possible reasons for the wide discrepancy in the assessment of the value of suppressive treatment under controlled experimental conditions and in actual practice in armies in the field. At that time, there was little reliable data to hand, as to the value of suppressive mepacrine in this war. Suppressive mepacrine had been given on a fairly extensive scale in the Arakan in 1942-43, but it was difficult to obtain reliable statistics for estimating its value in this rather confused campaign, the failure of which has been attributed in no small part to malaria.

Since this time last year, considerable reliable data has been collected and the value of suppressive mepacrine, in an army in the field, has become much less a matter of speculation. A brief resume of an analysis of suppressive treatment, administered to two formations in Central Command, last malaria season, will be given in this paper, stressing certain points regarding the indication for, and value of, this method of control of malaria, which can be well illustrated from this analysis.

Before turning to these results, I would like to remind you of the experiences in the 1914-18 war, and of the findings of the League of Nations Commission on treatment of malaria. Quinine was given on an extensive scale in the last war, in a number of campaigns in malarious countries, and by several combatant nations to their armies. Short, in Mesopotamia and Wenyon, in Macedonia, both expressed doubt as to whether suppressive quinine, even when given in large daily doses, under careful supervision, played any part in reducing the incidence of malaria. Christophers, in reviewing the subject of suppressive treatment, in his presidential address to the Royal Society of Tropical Medicine, in 1939, stated with reference to the Mesopotamia campaign—"In regard to the value of quinine prophylaxis, the only thing that can be said is, that as in Wenyon's experience in Macedonia, the incidence might have been even heavier if it had not been carried out." Now turn to the League of Nation's findings, and more particularly to the contribution by Field, Niven and Hodgkin from Malaya. The population in which the controlled experiment was carried out, consisted of 420 persons, who were an isolated community. Amongst them there was extensive breeding of the vector A. umbrosus. The community was divided into three sections, as equal as possible in every respect. To one section 0·2 grms of Atebrin was given, on two successive days each week; to the second section 0·4 grms. of a quinine salt was given daily; and the third section was given a neutral dummy tablet. The effects were a substantial reduction of malaria cases in the quinine group, an even greater and a more immediate reaction in the atebrin group as compared with the controls, but on discontinuance of the suppressive treatment, there was

a rise in incidence of cases in the quinine group to well above the control group and an even greater rise in the atebrin group. Field et al, summarizing their findings, stated:

> "It would appear that, in many cases, the effect of clinical prophylaxis in the presence of infection and superinfection, was to suppress the clinical evidence of infection—even, in many cases, to suppress the primary attack—so long as administration of the drug was maintained, and, in effect, to produce a prolonged incubation period, the primary attack occurring within a variable but short period of prophylaxis being suspended."

This work was imitated in Assam, and results published by me, in the *Indian Medical Gazette* for May, 1940. The differences were that A. minimus was the vector, the population was substantially larger, being a total of 2,173 persons, the dosage of atebrin and quinine given was smaller, and the treatment was deliberately discontinued at the peak of the transmission season. The findings were substantially similar to those in the Malayan experiment, but some evidence suggested the possibility of the suppressive treatment interfering with immunity or tolerence to infection. A particularly interesting feature is, that although after eight weeks of treatment, the degree of reduction of case incidence in both the quinine and the atebrin groups was considerable, being greater in the atebrin group, subsequent to this, the case incidence in the treated groups rose steadily, until, by the twentieth week, when the case incidence rate was very little lower than in the control group. It is with a view to demonstrating this feature, that I have referred to this experiment.

To digress for a moment on this subject of acquired tolerance, the old controversy of large versus small doses of quinine, some months ago boiled up again in India and overflowed into the lay press. When there are two such opposed schools of scientific thought, each supporting its opinion with a mass of evidence, the probability is that both are right, and an explanation or framework can be found, into which both views will find a place. In this case, the difference can be resolved by this factor of tolerance or acquired immunity. The same Assam strain which may cause such severe and intracticable malaria in imported troops, is the cause, in the resident population, of comparatively mild attacks, which usually readily respond to a few grains of quinine, given for one or two days. It is, in fact, very difficult to get them to receive treatment for more than three days. The usual procedure with Nigerian troops, who have been heavily infected from birth, is, I am informed, to give them a few hours or a day off duty, when attacked with fever, or, at most, to give one or two days quinine. The explanation of, on the one hand, success of suppressive treatment in indigenous poulations in Malaya and Assam, and, on the other hand, failure in troops in Mesopotamia and Macedonia, can, I think, be ascribed largely to this factor of acquired tolerance, which was present to a much greater extent in the former than in the latter. A good deal is said about immunity being not only species specific but also strain specific, but there is some evidence of group immunity and it will be interesting to see how the West African reacts to infection on the Assam-Burma frontier.

Now, after these introductory remarks, let us consider the data recently obtained from two formations under training in Central Command, which were placed on suppressive treatment. Full records are obtainable, over a period of thirteen weeks of daily admission for malaria, unit by unit, the species of parasite, whether cases were diagnosed as fresh or relapse. The rather tedious procedure of collecting this data from several hospital registers, was done by Lt.-Col. Snell, R. A. M. C., at the request of Command H. Q.

The administrative area, in which these formations were located, is, on the whole, fairly highly malarious. Figures from the static formations, within whose area they were situated, show an increase in the incidence of malaria in August to a peak of 92 per thousand strength in September, the highest incidence being in the second half of September, falling slightly through October and more steeply in November.

Formation A, consisting of British and Gurkha troops, with British predominating, was located for training, within this area, adjacent to most prolific breeding areas for A. culicifacies and within effective range of flight from a village with 100 per cent. spleen rate. There was a steep rise of malaria incidence during July and throughout August, when the average weekly admission rate for malaria had reached 56 per 1,000 strength, and on 7th September, suppressive treatment was started for the entire formation, the treatment consisting of 0·2 grms of mepacrine daily, on three successive days each week. Immediately on commencing treatment, the incidence of malaria fell sharply, although the incidence was rising in static units, in the area, that were not undergoing treatment. After falling for two weeks to a weekly admission rate of 21 per 1,000 strength, it again rose to a fairly high level, between 50 and 55 per 1,000 per week, at which it remained until the seasonal epidemic season terminated.

Formation B, consisting of British and Gurkha troops, with the Gurkhas predominating, was, during August and the first twelve days of September, fairly healthily located in the area, and the incidence of malaria was, considering the nature of training, satisfactorily low. Between 12th and 23rd September, the entire formation, in a series of strenuous marches, moved to an intensely malarious site, arriving between 17th and 23rd September During the third week of September and more especially at the end of the third week, there was a rapid increase of malarial incidence. The admission rate for the third week was 48 per 1,000. By 22nd and 23rd September, reports by telegraph and telephone, of daily admissions to hospital, were alarming and Command H. Q. and G. H. Q. went to investigate the outbreak. Steps were immediately taken to make sufficient drugs available, and, by 2nd October, suppressive treatment was started, consisting of 0·2 grms mepacrine daily for three days, followed by 0·1 grms daily thereafter for six days in the week. The case incidence was at its peak 10 to 14 days after the arrival of the troops at the new site, and fell from 141 per thousand strength for the week ending 5th October, to 24 per thousand in the week ending 12th October, and thereafter remained at a satisfactory low level.

Points of special interest in this outbreak are that a Gurkha unit, which had been previously subject to fairly heavy infection, had a crop of 88 cases, over 80 per cent. of which were B. T., in the three days following the termination of the march, and a further crop of 64 cases, 47 per cent. of which were M. T. and 53 per cent. B. T., on the 10th and 11th days after arrival It would appear that the first of these crops was largely due to recrudescence of old infection, precipitated by fatigue, whereas the second crop was largely the result of infection on the day of arrival.

Both formations concluded suppressive treatment at the end of October and were at once put on a course of Blanket Treatment, which commenced on 30th October. No subsequent rise occurred and the formations were in excellent condition at the end of December.

Several points regarding suppressive treatment of these two formations are deserving of special comment.

(i) Suppressive treatment with mepacrine gave, on the whole, satisfactory results. This is in marked contrast to the results reported with suppressive quinine in Mesopotamia. The chief causes of this contrast are, I suggest, the superiority of mepacrine over quinine and the fact that most of these troops had been in India some time and had probably a higher degree of acquired immunity than the troops in Mesopotamia. Other possible factors are that the strain of infection was more responsive, or supervision of administration was of a higher standard.

(ii) A second point of special interest is the second rise or break-through of infection in Formation A, three weeks after suppressive treatment was commenced; a closely similar break-through occurred in the Assam experiment, attributable, probably, to inadequate dosage of drug.

As to whether or not suppressive treatment can maintain good results, is, I suggest, provided full dosage is given, dependent mainly on two factors, namely, the degree of fresh infection, and the degree of acquired immunity of the population. In the case of Formation A, the majority were British troops and had certainly not a high degree of acquired immunity and they were in a site where they were submitted to a high degree of infection, that was rising daily until the latter part of September and maintaining a high level throughout the greater part of October. Formation B, however, with Gurkha troops predominating, had a higher degree of immunity and was subject to a lower rate of infection and for a shorter period, commencing treatment a month later, after the peak of the seasonal epidemic and showed no break-through.

I would like to stress the importance of one or two points in administration: first, the importance of proper supervision in administration. Section, platoon and company commanders must be personally responsible. The drug should be conveniently packed in small parcels, suitable for daily distribution. Officers and other ranks must be drilled in the procedure, before it is carried out in actual operations. Another point which cannot be too heavily underlined, is the risk of a calamitous outbreak, if suppressive treatment is withdrawn and not followed by blanket treatment. All men should be warned of the danger of discontinuing the treatment. I suggest that an analogy may be drawn between a formation on suppressive treatment and a ship that has been holed. If the ship is not taking water too fast, then, so long as the pumps are working and weather conditions are not unfavourable, she may cover a considerable journey and arrive safely in port for repairs. If the pumps break down, or the ship meets rough weather,

she will probably founder. A formation on suppressive treatment may function satisfactorily for a considerable period, until it can be withdrawn, or the malaria season ends and blanket treatment can be given, but if, in the stress of active operations, the drug is not taken regularly or rations are in short supply and there is excessive fatigue and exposure, a large part of the formation may suddenly succumb, with complete disaster.

In conclusion, in my opinion, suppressive treatment should never be given as a method of choice, for the control of malaria. It should be resorted to only when troops must, for tactical reasons, be located in a malarious area, where malaria cannot adequately be controlled by measures directed against mosquito breeding, against adult mosquitoes and by protection against bites, or, alternatively, when, owing to these measures not having been effectively adopted, a serious outbreak has occurred. When, however, suppressive treatment is indicated, for one of these reasons, the treatment with mepacrine, in the dosage at present advised, is an excellent method of control for a short period, but, as to whether or not any substantial degree of reduction of case incidence will continue after the first few weeks, will depend primarily on the degree of super-infection and the acquired tolerance of the troops, and also, possibly to some extent, on the strain of the parasite involved.

(The graphs, showing experiments in Malay and Assam, were demonstrated by epidiascope)

133. LT.-COL. PRESTON—"*Possible Dangers of Blanket Treatment*" **(P)**

Between 9th November and 17th November, 1943, four cases of Toxic Haemoglobinuria (Blackwater Fever) were admitted to No. 21 B. G. H. Jhansi. All four patients were Burmese subjects; none had a history of recent malaria (one stated that he had never had malaria, one thought he had had it in childhood, two had had it in 1942). Each of them had completed "Blanket Treatment" on the day prior to admission. There was no record of unusual exposure, etc. during the period of blanket treatment in any case. Three of the patients responded well to treatment and ultimately made a good recovery; one died.

In the light of these four cases, three questions arise: (1) What are the possible toxic effects of the synthetic anti-malarial drugs? (2) Are these toxic effects more prone to occur during blanket (or suppressive) treament, than when used therapeutically in the treatment of an actual attack of malaria? (3) Are Burmese subjects more susceptible to these drugs than British or Indian subjects?

With regard to the first question: the consensus of opinion among experienced observers, is that toxic effects can and do occur, both after mepacrine and pamaquin, but that these effects are so rarely serious that there is no reason for restricting their use.

For the production of blackwater fever, three factors seem to operate; (*a*) An interior haemlytic factor—commonly the result of malaria, (*b*) A lowered blood-cholesterol, so that its protective effect is diminished, (*c*) An external poison. Quinine has often been incriminated as factor (*c*) and the synthetic mepacrine (or atebrin) is generally considered to be less dangerous in this respect. Foy and Condi (1), however, record two cases beginning on the first and third day respectively, after a full course of atebrin, and a third case in a boy of five, on the fourth day of atebrin. Manson-Bahr (2), states that plasmoquine is liable to produce blackwater fever. Martindale (3), states "Pamequin is usually well tolerated, but may give rise to cyanosis, epigastric pain and methaemoglobinuria."

Various other toxic effects have been described by a number of observers. Thus, Turner (5), describes an atebrin psychosis, characterised by mental exhilaration and insomnia, followed by a maniacal stage; this merges into a somnolent delirium, and, on regaining consciousness, there is mental confusion and disorientation, which usually clears up in a few days. Bispham (4), Kingsbury (6) and Allen (7), describe similar cases. Ayala and Bravo (8), describe four severe cases during or following administration of Atape—a tablet combining atebrin and plasmoquine. Fischer and Staupendahl (9), report a case of generalised dermatitis, with an enlarged liver, bili-uria and haematuria. Pigmentation of the skin has also been described; and I have myself seen a case of peeling of the hands and feet, which, on two occasions, coincided with the exhibition of mepacrine. Other recorded symptoms are: epigastric pain, mild diarrhoea, and a yellow discolouration of the skin and conjunctiva. James (10), states that pamequin may cause cyanosis, fatigue, profuse perspiration and cardiac symptoms, but usually when the daily dose has exceeded ·06 gramme. A number of my own patients have complained of transitory abdominal discomfort whilst on the normal daily dose of ·03 gramme.

The second question, "Are these synthetic drugs more liable to produce toxic effects during blanket or suppressive treatment." Apart from my own four cases of blackwater fever, I have no direct evidence, but I would bring to your notice the following observations:

(*a*) Manson Bahr (2), states that he has not infrequently observed blackwater fever occurring in persons on leave in the United Kingdom from malaria-infested countries, who admit no history of malaria. He suggests that a latent malaria infection may be aroused into activity by exposure, chill, alcoholic excesses, etc. It is suggested that, unless special care is taken, a soldier is more liable to be exposed to these factors during blanket or suppressive treatment, than when he is in hospital, undergoing treatment for malaria.

(*b*) Slowness of excretion favours toxicity. Attention to the regularity of the bowels and a liberal fluid intake needs emphasis.

(*c*) It is agreed that mepacrine and pamequin combined, are especially toxic; it is suggested that, in the field, an orderly might confuse the two types of tablet and a man be given one tablet of mepacrine and one of pamequin in the same day. There is no evidence that this occurred in the cases under my own care, but it is suggested that the danger should be borne in mind.

The third question, "Are Burmese subjects especially susceptible?" I have been unable to obtain any further evidence on this subject and so far, enquiries as to the number of Burmese troops who received blanket treatment have not produced any figures, but, certainly, the four cases recorded, must form a percentage sufficiently high to be not insignificant; no similar cases were seen among the large force of British troops in the area.

References—

(1) Foy and Condi, quoted by Manson-Bahr, Tropical Diseases 1941.
(2) Manson-Bahr, P. Tropical Diseases 1941.
(3) Martindale, Extra Pharmacopoeia 1941.
(4) Bispham, Amer. Jnl. Trop. Med. 1941—21—455.
(5) Turner, Trop. Dis. Bulletin 1937.
(6) Kingsbury, Lancet 1934—2—979.
(7) Allen, J. M. A. Georgia 1937.
(8) Ayala and Bravo (Rev. Clin. Espanola 1942).
(9) Fischer and Staupendahl, German Med. Klin. 1941.
(10) James, Col. S. P. Br. Med. Jnl. ii/1933, 928.

134. CAPT. WALLACE—"*Complications of Treatment*" **(P)**

The purpose of this paper is to call attention to the possibility of blackwater fever occurring as a complication during the latter part of the routine Army treatment of malaria, and also following the blanket treatment. It is my impression that pamaquin was the precipitating factor in seven cases seen by me. All these occurred in Indians, Burmese, or Anglo-Indians, and I would suggest that this toxic action of pamaquin is of much greater importance in dark-skinned races. I would also like, briefly, to discuss some new conceptions of the cause of impaired renal function in blackwater fever and their bearing on treatment. Seeking information, I suggested to Colonel Schlesinger that this toxic action of pamaquin might be a suitable topic for discussion at this Conference, only to be instructed by him that I was to read a paper on the subject myself. I am very conscious of my limited experience and of the fact that this paper has been prepared without access to the literature on the subject, or to the records of the cases I have seen.

The case that first called my attention to the dangers of pamaquin, was a thin mal-nourished Anglo-Burmese boy in the 3rd I. B. G. H. Poona. On the fourth day of the pamaquin course for malaria (0·1 grm. t. i. d.), he was transferred to my ward, as a case of infective hepatitis. After a rapid response to quinine and mepacrine, he had been quite fit until the day previously. Then, his temperature had risen again, he had passed dark-coloured urine and became icteric. When seen, he was ill, vomiting repeatedly and deeply jaundiced. His finger nails showed some degree of cyanosis and his spleen, which had not previously been palpable, was two fingers down and tender. The urine was almost black, with a heavy granular deposit and showed the bands of methaemoglobin. The haemoglobin percentage of the blood was only 50, with a corresponding red cell count. After two days' illness, he recovered remarkably quickly, the spleen disappearing and the icterus fading within a day or two.

In the Ranchi area, I had under my care, four cases of blackwater fever in Indians who had just completed the blanket treatment. In the 21st B. G. H., I saw two Burmese with similar attacks. Although most of my work has been among British and American troops, I have seen no serious toxic effects of pamaquin in any of them, though some cyanosis is common. There seems to be no useful purpose served in trying to distinguish clinically between methaemoglobinuria due to pamaquin, and blackwater fever. Both may be either mild or severe and several deaths have been attributed to this action of pamaquin. References to twelve such fatal cases are given in the last edition fo "Stitt's Tropical Diseases".

The only clinical difference I detected was this : on making the urine alkaline in blackwater fever, by giving alkalies by mouth or intravenously, the colour of the urine usually changed to red. This would be expected, as methaemoglobin is produced more readily from oxyhaemoglobin in an acid medium. In cases due to pamaquin, there was no such change of colour. In ordinary blackwater fever, there is said to be no methaemoglobin in the plasma, the pigment that was thought to be this having been found by Fairly and Bromfield to be a substance which they named pseudo-methaemoglobin and later methaemalbumin, which is not excreted in the urine. The methaemoglobin present in the urine, is probably produced in the kidney from oxyhaemoglobin or reduced haemoglobin. Many problems remain to be solved with regard to the production of cyanosis and of haemolytic reactions by drugs like pamaquin and the sulphonamides. I note that, in the Bulletin of War Medicine of September 1943, an article is abstracted, describing a fatal case of haemoglobinuria in an Indian treated with sulphanilamide, and another, from America, describing haemolytic reactions due to sulphanilamide, many of which had occurred in negroes. Sunlight eruptions, following sulphanilamide, occur and it may be, that, with pamaquin as well, the pigmentation of the skin affects, in some way, the reactions of the body to this drug.

Until recently, it was generally accepted that the failure of renal function in blackwater fever, was due to a blockage of the renal tubules by insoluble acid haematin. Rapid alkalisation of the urine would prevent this deposition. The evidence for this, has been examined by Foy and others, in relation, also, to the renal failure of crush injuries and incompatible transfusions. It seems certain that the main factor is of extra-renal origin, some upset of the acid-base-electrolyte-water balance, resulting in dehydration, diminished blood-volume and glomerular filtration pressure. They state that a toxic alkalosis may be produced and the urine will still be acid, and that the degree of renal failure bears no relation to the acidity of the urine.

The effect of this new theory on the rational treatment of blackwater fever is of interest, and I would be glad of your advice or suggestions. An adequate fluid intake is most important, a urinary output of 1½ litres daily should be aimed at, and three or more litres of fluid daily will have to be retained to achieve this. It is also reasonable to give glucose intravenously. To me, it seems that the administration of alkalies is still indicated, taking care not to give them in doses that might produce a dangerous alkalosis. It will reduce the tendency to haemolysis, according to Smith and Evans, which is greater with a lowering of the pH, and will counteract the acidosis which exists. At the same time, it will reduce the tubular depositions of acid haematin, the effect of which, though by no means the whole story, may still play a part, especially in the degree of eventual recovery. I would suggest that the treatment recommended by Whitby, for mis-matched transfusions should be given. He used sodium citrate, as sodium bicarbonate is difficult to prepare in sterile solution without the production of toxic sodium carbonate, which may itself produce a haemolytic reaction. Intravenous fluids will usually be necessary, because of the vomiting. 150 cc of 3 per cent. sodium citrate in 5 per cent. glucose is given initially, followed throughout the 24 hours by 45 cc of 3 per cent. sodium citrate in 2,400 cc of 5 per cent. glucose. Thus, a total of 18 gms. of sodium citrate is given daily. If there is no vomiting, then 8 gms. of sodium citrate can be given by mouth, followed by a daily maintenance dose of 35 gms. I would be grateful for opinions on the efficacy of alkalies in blackwater fever.

In conclusion, I would suggest that there is a case for discontinuing the use of pamaquin in the treatment of troops of the dark-skinned races, especially in cases of M. T. malaria. Its chief value in M. T. malaria is as a gametocidal drug and, in the field, in an endemic area, with an infected local population and numbers of carriers, it is unlikely to have any appreciable effect in reducing the rate of mosquito infection. Even as a gametocidal drug, Ciuca has found that, after treating gametocyte carriers with five daily doses of 0·02 gms (which is the dose given to Indian troops), 25—33 per cent. still showed the presence of gametocytes in their peripheral blood. The difference between the therapeutically effective dose and the toxic

dose is too small. Its use increases the number of days unfit for full duty, of the men receiving it. There may be adequate statistical evidence to disprove entirely my thesis, and I would be glad to receive your comments. Finally, I would point out that, in the blanket treatment, Indian troops are given the full dose of three tablets of Pamaquin daily, for three days. Perhaps this is too much.

DISCUSSIONS

135. MAJOR-GENERAL MARTIN, in commenting on the above papers, said that the West African method was to carry on for sixty days with the suppressive treatment.

136. COL. CAMERON: In West Africa, malaria is treated without pamaquin, except in British troops. West African troops in India, come under the Standard Treatment Order and the exhibition of pamaquin has been associated, in a number of cases, with haemoglobinuria. They are treated as for blackwater fever. A number of similar cases of pamaquin haemoglobinuria (with a number of fatalities) are reported from the Middle East. These cases occurred in several races, African, Indian, Arab, French and Pole. It would thus appear to have no relationship to body weight in proportion to dosage.

137. LT.-COL. LAMPRELL: I think there is a risk of over-emphasizing the dangers of the toxic effects of pamaquin. In 1937, in an analysis by the League of Nations Commission, of the literature involving the treatment of many hundreds of thousands of cases, the toxic effects from plasmoquine were recorded in only a very small proportion and most of these were comparatively mild and of a transient nature. There were, however, a few instances recorded of haemoglobinuria with fatal results. Simeons, 1936, observed four cases of haemoglobinuria, two of which were fatal, amongst 5,650 cases treated with 0.02 grms. of plasoquine for three days, following two injections of atebrin musonate. Misseroli and Marina, 1934, amongst about 1,000 cases treated for ten days with 0.02 grams. plasmoquine, recorded one case of blackwater fever as a complication. It must, however, be admitted that the increasing number of reports of haemoglobinuria with a high mortality rate, is somewhat disturbing. Capt. W. N. Mann, R. A. M. C., in a communication to the Royal Society of Tropical Medicine, published in their Transactions September, 1943, reports a case of haemoglobinuria in a Bantu patient, which he attributes to the toxic effects of pamaquin Brig. Sidney Smith, Consultant to M. E. F., commenting on this report, instances fourteen similar cases occurring on the Middle East, six of which died. He stresses the fact that all of these cases had lived for a considerable period in areas where malaria was endemic and he appears to consider that they were true blackwater fever, precipitated by pamequin. The cases reported today among Africans and the four cases among Anglo-Burmans, with one death, do suggest very strongly the possibility of pamaquin acting as a "trigger" in precipitating true blackwater fever, under certain circumstances, as distinct from methaemoglobinuria. The condition obviously needs further investigation.

138. LT.-COL. ROGAN: Regarding toxic reactions to pamaquin, the more frequent reports of their occurrence in Indian and Burmese troops, may be due to the fact that Indian and Burmese troops are, on an average, lighter in weight than British troops. Toxic reactions to pamaquin are much more likely to occur when patients are not properly rested during the exhibition of the drug, which explains why they occur more often during blanket treatment than in the treatment of clinical attacks of malaria. It is important to distinguish between blackwater fever and pamaquin methaemoglobinuria, if only to maintain correct epidemiological data.

139. MAJOR GUEST said that sodium lactate solution should be available for intravenous injections in haemoglobinuria of blackwater fever, pamaquin poisoning and of incompatible blood transfusion reaction, as it is said to be superior to sodium citrate and soda bicarbonate solution, for producing a large volume of alkaline urine.

140. LT.-COL. ROGAN: Sodium lactate is not yet available, but its advantages, it obtainable, are being kept in mind.

141. COL. TAYLOR: Two sets of figures, collected from men supposed to be taking suppressive mepacrine, are available. Capt. Roberts examined the urines of men admitted with fever, from units on suppressive mepacrine; over 50 per cent. had no mepacrine in their urines. Capt. MacMartin examined the urines of over 1000 such men, but including men in units doing ordinary duties in units. Over 20 per cent. showed negative results.

I understand, this Spring, that the test for mepacrine in urine will be used extensively in the 14th Army and that the numbers of men found with negative urines, will be reported to the A. D. M. S. and the Divisional Commander.

Suppressive mepacrine may be dangerous, if morale and discipline are poor. Up to the present time with lack of beds in forward areas, if a man desired to get out of the fighting, he could do so, often with evacuation back to Eastern Command, merely by omitting to take his tablets of suppressive mepacrine for a fortnight.

There are two other points which have come up in discussion, the use of subcutaneous and intramuscular injections of quinine and the use of N. A. B. May I remind you of the orders of the D. M. S.? Intramuscular and subcutaneous injections of quinine are forbidden, and N. A. B. can only be used in investigations, sanctioned by H. Q.

142. MAJOR KILOH: Many have suggested that mepacrine tablets should be impregnated with methylene blue. Some four months ago, I wrote to high authority on this point and, so far, have received no answer. It would seem a simple method of detecting man who do not take the drug. Could Col. Rogan give any indication as to why this has not been adopted.

143. LT.-COL. ROGAN, in reply to Major Kiloh, said that, so far, experiments have been conducted on six volunteers, to determine the number of hours in which methylene blue was excreted.

144. LT.-COL. DAUKES—"*Clinical Side Room Diagnosis of Malaria*" **(P)**

Last year, there were about 250 deaths from malaria, in Central Command (actually, I was surprised it was not more). I read all the fatal case sheets and while, with a large number, one realized that they could not humanly have been saved, there were a large number where one felt, "Oh, why did they not take earlier and more slides, they might have saved 24 to 48 hours in diagnosing the case, which might have made all the difference."

The Officer Commanding one of the large hospitals in this Command, wrote on the fatal case sheet of a patient, who had died undiagnosed after four or five days in hospital, having had one blood slide taken—"Malaria was excluded by the negative blood slide". Seeing cases in hospital, one frequently finds that the medical officer does not know the result of a blood slide, 24 hours after it has been taken.

A consideration of the foregoing, shows that a review of the points of malaria diagnosis is desirable. These points which I shall mention, should be pointed out *ad nauseam* to medical officers, because, though you may get fed up with saying them so often, you may be telling them to the medical officer for the first time, since staffs are changing so frequently. (1) First, let us consider when and how many blood slides to take. One should be taken as soon as possible after admission, in all cases with fever or a history of fever, unless you are certain that the full extent of the fever can be accounted for by the physical signs. Subsequently, at least two a day should be taken, until the case is diagnosed, or for at least three days. These are best taken between 9 and 10 in the morning and at the beginning of the afternoon session: the morning results should all be in by mid-day and the afternoon ones well before the medical officer leaves, if they are not, he should go and find out why they are not, getting them himself, if necessary. These are the routine slides, others should be taken if there are any indications, such as rise in temperature. Slides should be taken from any case which is under treatment for some other condition and is not responding as he should, or which gets an unexplained rise of temperature. In cases which are seriously ill and where malaria is strongly suspected, I am in favour of taking a thick drop and a thin smear on the same slide, at once, and then proceeding with the treatment, as laid down in the G. H. Q. pamphlet, in anticipation of the result. If a patient is seriously ill, malaria is not particularly suspected but you want to exclude it, adopt the same procedure and mark it urgent.

(2) The type of slide to take. There is no question but that the thin smear, well taken, gives the best picture for diagnosis that you can get, but, if there are not many parasites, they are liable to take a long time to find, and a reasonably confident negative cannot be given under about 20 minutes searching. This means that, if more than about 25 slides are being received by the C. S. R. in a day, they cannot be adequately examined by one man, since, to examine 25 slides properly, takes about 8 hours work if they are negative. This leaves semi-thick and thick drops. The semi-thick drop has the advantage that it dries quickly and does not need de-haemglobinising (though, in my opinion, it is better for it) and also it is much quicker. The thick drop has the advantage that it is very rapid of diagnosis, though it is slow in drying and needs de-haemoglobinising. Both have the disadvantage that the parasites are liable to distortion, which is especially the case with young rings and trophozoites, but less so with medium rings and crescents. Possibly the best thing, as a routine is to take a semi-thick and a thin smear on the same slide. This can easily be done by putting a medium-sized drop of blood near one end of the slide, touching the spreader against it until the blood spreads out,

lifting it clear and putting it down about a quarter of an inch away and making a thin smear in the ordinary way. The corner of the spreader is then used to spread the remainder of the drop out, until the figures of a watch can just be read through it. Ward boys *can* be trained to take good blood slides, but, with the frequent changes in staff, they all need a five-minute lecture-demonstration about once a month, not less, telling them the importance of using grease-free slides, only handling them by the edges, etc. Of course, once the medical officer in charge of the C. S. R. is really experienced, he will not need the thin smear, but I am of the opinion that the routine should not be changed, as this may lead to misunderstandings.

(3) What stain to use? Here I would say that, if the medical officer in charge of the C. S. R. is used to one particular stain, this should not be changed, even for a better one, if possible, and certainly not during a rush period. A good Leishman stain has not yet been beaten by any and gives a lovely picture for diagnosis. It has the drawback, however, that it is essential to check the pH of the distilled water every day, and, if necessary, adjust it. This can be got over by the use of buffer solutions, but sometimes these are hard to get and may need to be made up by a District Laboratory. In addition, we are not getting quite as good Leishman stain as we did in peace time, and some has been frankly bad. If there is a good laboratory available to make it, Field stain gives just about as good results and is quick to use, but it is rather complicated to make up and needs a stain difficult to obtain, though this can be got over by the use of a home-made substitute. In these days of Alcohol shortage, and shortage of rare stains, Simeons modification of Boyes stain is good; it is prepared locally and is quick to use. It does not give as good differentiation as Leishman's or Field's stain, the chromatin being purple and not pink. The other day, I was shown parasites stained by Simeons stain, where the staining of the parasites was every bit as good as the best Leishman; it had been done by washing in acidulated water after staining, but, unfortunately, they were not able to reproduce the result while I was there. I have tried several times since, and although I have had improved results, I have not had any as good as that one. If any of you have any success in that line, I should be glad to hear of it. Major Jaswant Singh, at the Malaria Institute, is experimenting with a new method of polychroming his methylene blue, but at present it is rather complicated for any but a District Laboratory.

(4) Finally, we come to one of the most important points, the returning of the slides to the wards and the informing of the medical officer. Each hospital must decide for itself, what routine it wishes to follow, but, broadly, we can say that, in rush times, when many wards are sending several or many blood slides at a time, they should include all the names in one nominal roll, giving each patient a number on the nominal roll, to correspond with a number put on the slide by grease pencil, or, preferably, since grease pencil can be rubbed off, scratched on to the thin smear with a pin. This obviates the confusion caused by the use of many little bits of paper, which are always liable to get lost, especially in the hot weather, with a breeze blowing. As each batch of slides is disposed of in the C. S. R., it is entered in the register and in the despatch book of the C. S. R. perhaps only the ward number, the date and the serial number for the report, would do here, together with the time of despatch. These are taken to the wards by the C. S. R. ward boy or sweeper, or, if necessary, a special orderly detailed for rush periods. A responsible person in the ward will sign a receipt for the reports and the time of receipt. It is then up to the person who received the reports, to inform the medical officer of the results, with the least possible delay. But the medical officer's responsibilities do not end when he says, "Take a blood slide". It is part of his duties, and a very important part too to see that he gets his reports in reasonable time, say, two hours for routine results and half an hour for urgent ones. One of the important causes of lack of knowledge of diagnosis, and, with this, Major Greening and Major Lehmann will agree with me, is lack of interest and lack of conscientiousness on the part of some medical officers; there are, of course, many shining examples of what *should* be done, but the attitude frequently is, "I have taken (or given orders to be taken) the blood slide (though I don't know if it has been taken) and my responsibility is ended." Please do everything in your power to combat this appalling outlook, it is the outlook of the bazar doctor, who, if his patient dies, says, "Hamara koi qasur nahin, Khuda ka marzi hai."

145. MAJOR CHANDRA—"*Adrenalin in Malaria Diagnosis*" **(D)**

On arrival in Hyderabad, in October last, I found that two-thirds of the cases in hospital were in the malaria wards. I was asked to take over the malaria wards and to find out why the malaria patients were coming in over and over again, and if there was anything wrong with the standard anti-malaria treatment. Here is a summary of records of 102 patients, treated during November and December, 1943, and the methods adopted to solve the problem.

(1) All cases in the malaria wards were blood positive.

(2) The administration of the anti-malaria course was personally supervised by me or by another officer in charge of the cases.

(3) On the day after the course finished, 8 ms of 1 in 1000 adrenalin was administered. 52 men were given intramuscular injections and had 8 ms of adrenalin in 10 cc of saline intravenously; 10—15 minutes after the injection, thick and thin blood slides were taken and closely scrutinised for any gametocytes.

Out of 102 cases, it was found that 71 were fresh infections (64 B. T. and 7 M. T.), 29 genuine relapses all B. T., and two recrudescences. Apart from blood pictures, any attack of fever during the last 6 months was taken as criterion of a relapse. Spleen was palpable in 8 out of 71 fresh infections and 6 out of 29 relapses. Gametocytes were found in only two cases, after the standard anti-malaria course, in this series of 102 cases. The problem was partially solved by going into details of anti-mosquito precautions, carried out by the garrison.

I used adrenalin in the above 102 cases, for a different purpose, and, as far as the results are concerned, the draw has been almost a blank, but I trust even negative results have their value. A few cases of malarial splenomegaly, with hard and caky spleen, were treated by modified Ascoli method; that is, 5 minims of adrenalin in 5 cc. of saline intravenously, followed by ten grains quinine by mouth for 7 days. This was followed by a 7-day course of Fowler's solution in increasing doses. The numbers treated are too small, so far, to be dogmetic as to its efficacy, but I consider it worth a trial in selected cases.

146. MAJOR BATHGATE—"*Malaria in Ceylon*" **(D)**

Ceylon is, generally, a highly malarious country, with, here and there, islands of freedom and with considerable tracts of hyperendemicity. Its history has been considerably influenced by malaria and its people are alive to the dangers and its administration to the need for prevention. Since 1905, when Chalmers first studied the Vector mosquito, up to the outbreak of war, they have built up a comprehensive organization, and, since the epidemic of 1934-35, much has been done to control breeding grounds, to treat the malarious patients, and to extend education. Therefore, when troops were sent to the island, they came to an area in which the hazards were known and in which some measure of control was immediately possible, unlike conditions on the Eastern Front.

The Vector mosquito is Anopheles Culicifacies, a breeder in clean sunlit waters, river pools and any small collection of water. Hence, jungle clearance for camps or aerodromes, without drainage or spraying, immediately raised the number of Vector mosquitoes locally. These were infected from the neighbouring villages, which contained a reservoir of infection never completely controlled, and a high initial incidence of malaria was produced, especially in aerodrome staffs, anti-aircraft gunners and troops encamped and training in jungle areas, in many cases areas of hyperendemicity, whose civil population was scanty.

In the six months from January to June, 1943, to a five-hundred bedded hospital, serving about 5,000 troops, 1,912 cases of malaria were admitted. The types were:

B. T. (F) 890 R 492 72 per cent. of the total.

M. T. (F) 298 R 106 21 per cent. of the total.

Q. (F) 47 R 4 2 per cent. of the total.

Two distinct peak periods were noticed—April to June and November to January. Even during these periods, B. T. was by far the most prevalent type and Quartan the least. In the civil population, Quartan is said to occupy an intermediate position, figures up to 39 per cent. being quoted. The difference noted in the military figures, is attributed to the prevalence of Quartan among the child population. Despite the presence of African troops, no case of Plasmodium Ovale was diagnosed.

Two deaths occurred in this series, in one of whom, M. T. schizonts were present in the peripheral blood, in large numbers, on admission.

In dealing with large numbers of malaria cases, the value of the rapid methods of staining thick films is very great. Field's stain was used almost exclusively and gave excellent results. By its use, the Reception Officer was able to stain and examine a blood film in the Reception Room, and many cases went to the ward diagnosed. This, or one of the other rapid thick film methods, is essential to speed and accuracy of diagnosis. Speed in diagnosis is emphasised, because it was our experience that the cases did best and gave least anxiety, who came under

treatment within 48 hours from the onset of the first symptoms. Delay in sending in men with temperatures was deprecated, especially if a long ambulance journey was necessary. Cases of M. T. malaria, especially, travelled badly. It would appear that the further forward malaria can be treated, the better is the response.

Complications and untoward happenings were not common. Vomiting was the most troublesome feature in cases of B. T. In the M. T. cases, the complication causing most anxiety, was circulatory failure of varying degrees of severity. In primary cases, with continued fever, a rising pulse rate was viewed with concern and occurred frequently in the absence of any considerable fluctuation in temperature. Blood pressure fell, and the first heart sound became feeble and shortened. Many of these cases also developed moist sounds at the base of the lung. The severity of the symptoms did not necessarily correspond to the number of parasites per field of the film, but a heavy infection of M. T. parasites always made one consider the possibility of this complication. Vomiting was a frequent concomitant. The treatment of this type of case is difficult. Vomiting was regarded as a symptom of the complication and it was usually found expedient to treat such a case with intravenous quinine, immediately it was evident that oral medication was not being retained If treatment was delayed, dehydration and further collapse was encountered, and at that stage, intravenous quinine had to be used with caution, and given extremely slowly, preferably in a small intravenous drip infusion of, say, 10 oz. of glucose saline. Few cases of cerebral malaria and a few cases of hyperprexia were encountered.

Routine treatment was normally adequate, a purgative always being given on admission and alkalies administered before the quinine, a modified Sinton method. Quinine dihydrochloride in solution was used for all M. T. cases. Treatment by mepacrine alone, without preliminary quinine, was unsatisfactory and, in M. T. cases, dangerous, the fever taking longer to control and convalescence being longer. In a short series of cases, original atebrin was available and gave better results than the mepacrine, both being used without preliminary quinine. In a short series of cases of M. T., treated by quinine dihydrochloride, 30 gr. daily for ten days, and pamaquin, the immediate relapse rate was reduced. Late relapses were unaffected.

DISCUSSIONS

147. MAJOR LYN GREENING stated that it appeared necessary to draw attention to the fact that the exhibition of quinine was no contraindication to the taking of slides for malarial parasites. Experience of Clinical Side Rooms would suggest that the system of serial numbering of slides by the Wards, was likely to lead to mixing. It was recommended that such a policy should be rejected, in favour of writing the name and number of the patient on the dried slide, on the blood smear, with a pin, needle, or even a pencil or uninked pen; a permanent record was thus obtained and mixing was impossible.

148. LT.-COL. MURTHI: Capt. G. R. Chandran, at my instance, carried out a comparative study of blood smears, in malaria cases at Moradabad, taking smears before and ten minutes after a subcutaneous injection of 5 m. of adrenalin. Out of the 50 cases, only in one case the post-adrenalin smear showed M. T. rings, while the pre-adrenalin smear was negative, and this is not of much significance. The value of adrenalin, in producing contractions of the spleen, of sufficient importance to be of advantage in blood smear examination, is doubted. In chronic malarias, where spleen has undergone profound histological changes, the action of adrenalin on the spleen is probably even less significant.

I made a comparative study of the Field's stain and the Simeon's modification thereof, with the routine Leishman's stain, and found that the last is the best. The defect with the Field's and the Simeon's stain would appear to be with its basic part. The protoplasm of the parasite is not well brought out and the parasites, particularly M. T. rings, can easily be missed.

149. LT.-COL. BOSE: I had tried, six years ago, intravenous adrenalin treatment and deep X-ray therapy, at the Patna Medical College Hospital, on a few cases of Splenomegaly of apparently malarial origin, without encouraging results. During 1942, at an Indian General Hospital in Poona, 22 cases of splenomegaly, in whom other causes than malaria were excluded, were treated on Ascoli lines by Dr. Jug Rao, under my personal supervision; a few cases showed moderate reduction in the size of the spleen, but, on the whole, the results were not promising. In a certain number of cases, the immediate reaction after the injections was so great, that further continuation of treatment had to be abandoned.

150. MAJOR DAVAR: I can say that I have tried Ascoli's treatment of splenomegaly in about 12 cases of malarial splenomegaly, but the results have been quite disappointing. As Ascoli's treatment consists of intravenous injections of adrenalin, in increasing doses, for twenty consecutive days, it is generally easy to give the injections for the first 5-6 days, when the dose is well tolerated, but, when the of 8 to 10 minims or over has been reached, due to the very unpleasent reactions produced, such as severe headache, tachycardia, discomfort, etc. the patient dreads the injection treatment and strongly objects to it thereafter, and it is very difficult to continue the full treatment, as recommended by Ascoli; but it was continued and the maximum tolerable dose was injected every day, but no appreciable diminution in the size of the spleen was noticed. Further to augment the effect of adrenalin, two patients were given, throughout the treatment, a mixture containing quinine, arsenic and iron. Though this treatment has been highly spoken of, my experience, like the other speakers before me, has been disappointing.

151. LT.-COL. LINDSAY—"*A Note on M. T. Malaria*" **(D) (by post)**

As M. T. malaria is liable to be the major medical problem of the Asiatic war, a brief reference to it may be of interest. Already, the disease seems to have caused a certain amount of trouble in minor operations in Burma. The malaria of Arakan and the Chin Hills is, however, of a mild type and should not be regarded as a sample of what will be found in other parts of Burma, in Yunnan, or in Siam.

The most important thing to remember about M. T. malaria, is to remember it. When practising in an endemic area, one must never let the disease out of one's mind. It is desirable that this should be explained to every M. O., because, to remember malaria always, is not easy when the features of a case strongly suggest another diagnosis, such as renal colic, bleeding piles, etc. Apart from cholera, there is no infection which may kill a man so quickly. An apparently healthy person may drop unconscious and be dead in four hours. When a man collapses suddenly in that way, it is natural to think first of malaria. But when pernicious malaria appears in someone already seriously ill with another disease, one is apt to forget malaria, and, if one forgets, the patient dies.

All M. O.'s should be taught the various manifestations of malaria and they should understand that many of the worst cases have no fever. They should know, for instance, that a man who suddenly attacks a companion without provocation, may be a case of cerebral malaria, that an intravenous quinine may restore his sanity in two hours and that, if quinine is not given, he will die in a day. They should realize that a person whose malaria shows no sign except epistaxis, is probably bleeding into his gut and that he may be beyond hope in a few hours, if he does not get quinine. The rate at which haemorrhages develop, may sometimes be seen in the skin. One may watch an intracutaneous haemorrhage appear in a pin-head spot and grow to the size of a rupee in five minutes and, in an afternoon, there may be little normal-coloured skin left. Such a person, unlike the haemorrhagic yellow fever, is greatly collapsed. If the cause is diagnosed early, an intravenous quinine stops such haemorrhages and does so with an abruptness that is hard to understand. Early diagnosis is essential.

The surgeon must also be very much on his guard. A man repairing a roof gets an attack of splenic colic, falls to the ground, and becomes unconscious because of the fall. The dangers of the fractured base may be small, compared with those of the malaria which caused the fall. A lorry-driver becomes giddy and crashes. He is removed unconscious, with a cut head. He is observed, perhaps operated on, as a case of intercranial haemorrhage; the coma or restlessness, the blood-stained C. S. F., the signs of localised haemorrhage, may all be due to the malaria, which made him giddy. An intravenous quinine may restore him in an hour; without it, he will die. A pilot may lose his finer judgment, land badly and just be able to escape from his blazing plane. In a week he dies unexpectedly from burns, from which he would have recovered, had not his vitality been lowered by an undetected malaria—the same malaria that spoiled his landing.

There remains, in spite of Army Orders on the subject, a tendency for quinine to be withheld until the temperature falls (or rises), or until a positive blood report is received. Such a practice is not very dangerous in the malaria of garrison towns in India, but, in the disease which will be seen further East, a delay of an hour may mean the death of the patient. The practice in a civil hospital, receiving 10—30 pernicious malarias daily, was that every (?) acute abdomen and every case of coma or shock, which was not due to assault injuries, was given intravenous quinine before any detailed physical examination was made. It was not scientific medicine, but we believed that it saved many lives. On the treatment of various malaria

emergencies by quinine (intravenous or intrathecal), morphia, methylene blue, lumbar puncture, chloroform, venesection, saline infusion, etc. one need not dwell. It is enough to say that a malaria emergency is very much an emergency. It must be diagnosed at once and it must be treated at once.

152. LT.-COL. LINDSAY—"*A Note on the Sweating Stage in Malaria*" **(D) (by post)**

The ordinary attack of malaria is usually considered to have three phases—the cold, the hot and the wet. Two explanations of the third phase have been suggested, namely, (1) a physiological reaction to reduce the temperature by evaporation, (2) a direct effect of the toxin, just as the first two stages are thought to be. It seems hardly likely that it is a physiological reaction to the fever, because sweating does not occur in diseases such as plague, pneumonia, smallpox, etc. which produce an equivalent temperature, and also because the profuse sweating of dengue does not reduce the fever. The question is, whether or not the sweating stage is part of the disease: one is inclined to think that it is not, and that, on the other and, it is the result of treatment.

When the shivering begins, it is usual to cover oneself with blankets, which are generally retained, even at the height of the fever and are discarded only when the sweating stage is on and one gets up to put on dry clothes. One gets the impression that, if blankets are not used in the second stage, sweating does not take place. It may be that the third phase is a physiological reaction to a high external temperature, resulting from the use of blankets.

Once, I had occasion to trek for 5 days with untreated malaria. Shivering began at noon, fever reached 105° and fell to normal before we camped. The third phase was always absent and sweating occurred only when the path was steep or the sun hot. This suggested that sweating might not be a normal feature of malaria and that, if it could be prevented, the prostration produced by great fluid loss could be avoided. In ten subsequent attacks, a series of experiments were made. It was noticed that, if one went to bed with blankets, one sweated and felt very washed out as a result, whereas, if one sat by a fire, or walked around in the sun, a fever of 105° produced no sweating and one felt reasonably fit afterwards. I mentioned the idea and the experiments to a non-medical friend, with vast experience of malaria. He cast his mind back over 16 years and some 70 separate attacks of M. T., B. T. and Q. T. He recalled that those attacks which he had had on the march, never produced sweating or real weakness, whereas in any attack which he had had at home, when he was given hot-water bottles and blankets, he invariably soaked through the mattress and was unable to work on the next two days.

Being no malariologist, one would like to have the views of experts on this subject. It is not a matter of great importance in the army, but in civil life, where officials and business men have to carry on, in spite of malaria, a simple means of reducing post-fever weakness would be useful.

9TH FEBRUARY, 1944—AFTERNOON SESSION

The Meeting closed at 13.30 hours and was resumed at 14.30 hours, with **COLONEL SCHLESINGER** in the Chair. The Chairman felt that some indication should be given to the Conference, regarding the policy and the activities of the Review Medical Board in Poona.

REVIEW MEDICAL BOARD

153. COL. CAMERON—"*Review Board*" **(P)**

Colonel Cameron, after briefly sketching the origin of the Review Board, gave as its aims:

(1) Conservation of Man-Power.

(2) Creation of uniform standard of boarding.

(3) Facilitation of evacuation.

During the year, 3,261 cases were reviewed, and of these, 327 were placed in Category C 1 or above, i.e. 10 per cent. rescinded. The change over the year was shown by graph, which indicated a rise in the number of cases reviewed, associated with a fall in rescind rate. In the early months of the Board, this rate was over 20 per cent. recent rate was under 5 per cent.

Comparison of officer and other rank boards showed that 1 officer was boarded for 4 other ranks, and that the year's average rescind rate for officers was 12 per cent. as compared with 9 per cent. for other ranks. The figures suggested that too many officers were being recommended for evacuation.

The evacuation situation was now very satisfactory. Long delays had been eliminated. Other ranks were evacuated very shortly after approval by Review Board. The slight delay occasioned to officers was entirely due to the need for the Military Secretary's sanction for evacuation. Officer evacuation was also now very much speeded up.

All cases before the Review Board were given full investigation and, if need be, further treatment. No recommendation was rescinded without very full consideration and discussion. Cases were, on occasion, tried on duty in a lower category. If such a trial failed, it was the duty of hospitals to re-board these cases and return them for further review. In this respect, the attitude of the Review Board appeared to have been misinterpreted by the hospitals.

The wording of form MRB 10 was unfortunate, especially as it was misinterpreted by some hospitals. Col. Cameron assured the Meeting that nothing sinister was implied by the bald statement "We disagree". The words were used as laid down by headquarters and were not the choice of the Review Board.

The standard of boarding had improved immensely since the Review Board was inaugurated. A few boards still failed to provide full information. A few medical specialists were abruptly brief and non-informative in their reports. The Review Board could not, in all cases, correctly interpret what was in the specialist's mind, if he did not make a statement. The improvement in the standard of boarding was the most useful contribution of the Review Board.

(The above is an abstract, only, of Col. Cameron's paper. The annual report of the Review Board is about to be published and will provide the data which could not be included in the above abstract.)

154. LT.-COL. KELSALL—"*Review Board Details*" **(P)**

(1) *Composition*—Consulting Physician, Southern Army, as President, all Os. C. Medical Divisions and Medical Specialists from British-staffed hospitals in Poona-Kirkee area, as members. Neurologist and Dermatologist to advise on special cases, and two Psychiatrists. The Medical Section of the Review Board is divided into three sub-sections, in order to get through the large number of cases, as follows:

I. President + two Specialists. Sees all officer patients and some B. O. R. medical cases.

II. Officer-in-charge Division + two Specialists. Sees B. O. R. medical cases.

III. Officer-in-charge Division + two Psychiatrists. Sees psychiatric cases.

(2) *Procedure*—Cases accommodated in 3 I. B. G. H. (neurological cases now in 126 I. B. G. H.), in special wards, with specially picked M. O. in charge. No cases are submitted to the Review Board until the M. O. is satisfied that reasonably thorough investigation has been performed. Even so, the Review Board often defers cases for further investigation, observation or treatment.

(3) *Policy*—(1) General—

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Correct recommendations are still not understood in many hospitals; for Officers and O. Rs. of British Service, the only recommendations are:

C.2 Unfit for service in the tropics, but fit for service in a temperate climate. Implies fitness immediately on reaching a temperate climate, not requiring further treatment, and does not necessarily involve return to the U. K.

Evac. U. K. Unfit for service in tropics, may become fit for service later, but still requires further treatment.

E Permanently unfit for military service.

For British Officers of the Indian Army, C.2 is not applicable, nor is 'Evac. to U. K.', but 'Leave to S. A. or U. K.' can be recommended.

Policy—(2) (Special diseases)—

(*a*) *Malaria*—Extreme caution is exercised before evacuating for malaria, because of (1) Tendency of disease to burn itself out in absence of re-infection and impossibility of of forecasting when relapses will cease (2) Difficulty of drawing any arbitrary line as to the number of relapses qualifying for evacuation (3) Danger of slackness in personal protection and extension of treatment-dodging, if it became known that a sufficient number of relapses meant a ticket to the U. K.

Difficulty of checking accuracy of history as to number of relapses. Therefore, initial policy was to evacuate no malaria cases, unless gross anaemia or splenomegaly were present Now the criteria are slightly less stringent and, though no arbitrary rule can be made, men who have been relapsing regularly and done virtually no useful duty for a year or more, or who have had blackwater fever or an acute haemolytic crisis, are evacuated.

(*b*) *Chronic Dysentery*—Nearly all amoebic. Again, no arbitrary rules, but, in general, men are evacuated who have been continuously in hospital under treatment for 6 months or more, or who have had recurrent relapses over a year or more, provided that full treatment has been given and proved ineffective. Many cases have been sent to the R. B., who have never had really adequate treatment; some have been sent merely as cyst-passers.

(*c*) *Steatorrhoea and Sprue*—Most violent criticism of the Review Board has arisen in connection with this group. 20 were rejected out of 75. Many of these were boarded as Sprue, without any evidence of steatorrhoea and many more without evidence of mal-absorption of any other constituent of diet. A pale stool does not necessarily mean steatorrhoea. Apart from these, difficulty arises from definition of 'Sprue' and disagreement as to aetiology and prognosis. In Europe, steatorrhoea may occur in a variety of conditions, e.g. mesenteric T. B., gastro-colic fistula, and may be associated with avitaminosis and nutritional anaemia. Why should 'sprue' be a syndrome with a single aetiology? All grades can be observed in India, from single transient steatorrhoea, up to full steatorrhoea-avitaminosis-malnutrition syndrome, some occurring spontaneously, others following bacillary dysentery, amoebic dysentery or infective hepatitis. Our experience is that many of these simple steatorrhoeas, even if associated with some degree of hypo-vitaminosis and anaemia, will recover completely in India, if early and efficient treatment is given, and very few relapse if kept under reasonably good living conditions. No case was returned to duty in India, in any category, by the Review Board, unless he had been observed for several weeks, on a full diet and was maintaining normal weight, passing stools of normal fat-content and had a normal blood-count; all cases returned to duty were placed temporarily in a lower category (usually C). Several M. Os. of my acquaintance have had mild or moderate grades of 'sprue syndrome' (including one in the present audience) and have made complete recoveries in India. Policy is thus to give thorough treatment as early as possible, retain in India if full recovery occurs, (this may take 4—6 months), but evacuate if response to treatment is unsatisfactory or if relapse occurs.

(*d*) *Peptic Ulcer*—2 rejected out of 181—diagnosis not considered to be correct. Policy—proven cases Category E, except officers and key W. Os. and N. C. Os., who may be made C.1 at the discretion of Boards. Very heavy responsibility of radiologist "X-ray dyspepsia" cases are not evacuated, unless there is very strong clinical or pathological evidence of an organic basis, or unless evacuable on purely psychological grounds.

(*e*) *Organic Heart Disease*—With symptoms—Category E. If symptomless, discovered on routine examination—if severe, Category E; if slight, B.1 or C.1 unless history of recent active rheumatism.

(*f*) *Effort Syndrome*—Many cases are sent to the Review Board labelled "Myocarditis" or "V. D. H." E. S. superimposed on mild disabilities also often unrecognised. Minor degrees of E. S. very common among British Troops in India and tend to be "infectious'. Policy therefore is not to evacuate any E. S. cases unless they are completely useless psychologically, in which case a frankly psychological diagnosis should be attached.

(*g*) *Chest Diseases*—Pulmonary Tuberculosis; 246 cases sent, 6 rejected as showing no clinical, radiological or pathological evidence of active P. T. Definite active P. T.—Category E. Chronic Bronchitis: do badly in India; if severe, genuinely incapacitating

or liable to frequent recrudescences—C.2, evac. U. K. or Category E.; but many milder cases are capable of duties in B.1 or C.1; total cases 90, rejected 29.

Asthma: if genuine severe or frequent attacks—evac. U. K.

Bronchiectasis: unless very slight—Category E.

Pleural Effusions. if large or persistent—transfer to 9 I. B. G. H. (B. T.) for disposal as for P. T.; if small, resolving rapidly and with no X-ray evidence of disease in underlying lung—convalescence + temporary downgrading to C.1 and X-ray again after 3 months.

(*h*) *Epilepsy*—Definite epilepsy in O. R.—Category E. Officers can be B or C.1 if fits are infrequent and there are no personality changes.

(*i*) *Hypertension*—If diastolic pressure high, symptoms marked, hypertensive retinopathy present or evidence of impaired renal function—Category E. Otherwise can be B.1 or C.1. (War Office ruling).

(*j*) *Skin Diseases*—In general, any chronic skin disease which has involved continual or repeated hospitalisation for 6 months or more, in spite of best available treatment—C.2 or Evac. U. K.

(4) *Conclusion*—The Review Board is composed of men who have had an important, difficult and thankless task thrust upon them; they get plenty of kicks and no extra ha'pence for it. "Don't shoot the pianist—he's doing his best."

DISCUSSIONS

155. LT.-COL. PASRICHA: Are officers of the Indian domicile entitled to have their cases put up to the Review Board?

156. LT.-COL. HINDS-HOWELL: Part of the trouble arises out of lack of information. Until very recently, there were no instructions on policy, and one had to judge for oneself from the Poona returns. Furthermore, when instructions were issued, there were, in them, instances of medical inaccuracies. Our Dermatologist was upset because one group of diseases (I believe it was Epidermophytosis), was not being boarded until it had received a course of superficial X-ray, and that, he assured me, is universally accepted as extremely bad treatment. Later on, the Adviser in Dermatology explained that the cases so treated, turned out, in the end, not to have had Epidermophytosis at all, but that is the sort of error which leads to ill-feeling. I suggest that when an authoritative statement is issued for guidance, as in this instance, it should be accurate in every respect.

It has been our practice, when boarding border-line cases, in which there is a likelihood of relapse, to put them into Category C for three months, an unpopular practice, but the only method of ensuring that the man is re-examined at the end of that period. Is it true that men in temporary C are held at Deolali and not employed? If this is true, it is of no value in assessing the man's capabilities when at work and must be extremely bad for morale. Imagine sitting in a rest-camp for three months.

Finally, there have been one or two regrettable incidents, in which the Board has changed the diagnosis. As an instance, recently there was an officer in our area, who had what I diagnosed as a Cardiac Neurosis. I treated him with graduated exercises and encouragement and eventually found him a job at the local headquarters. He was then re-admitted to hospital with Paroxysmal Fibrillation. My colleague saw him in this attack and accordingly boarded him E. The attack, of course, stopped, as these attacks do. However, the report came back from Poona, "Auricular Fibrillation not proven, Cardiac Neurosis, evacuate." Now that sort of thing will not do! One can only suppose that the Board had not read the case notes, which, I am sure, is unlikely. And, in any case, if they could find no evidence of organic disease, why on earth did they evacuate him?

157. LT.-COL. PRESTON: Colonel Cameron may remember that, at a previous Conference in the Southern Army at Bangalore, it was proposed that a recommendation be made that 'Category C' men should be employed in hospitals. Also that Medical Boards should have the power to recommend that an officer was psychologically unsuited for service as an officer, but not necessarily unfit for any form of military service. Were either of these recommendations accepted?

158. LT.-COL. CARRIER enquired whether or not there was any member on the Review Board who had extensive experience of Medicine in the Tropics. By this, he did not imply that such was essential only in cases of tropical disease, but that it would be highly desirable also in cases of Europeans whom it was proposed to invalid, who might be suffering from other than purely tropical diseases and whose prognosis might be especially affected by a tropical climate. Sprue, he pointed out, was not always continuously symptomatic and intermissions were often a feature. He considered that cases with a definite sprue history might show deceptively few signs on arrival in Poona, after months of hospital treatment, and that, in such cases, as in others, a more correct assessment of the disability, for the purposes of the Review Board, could be made if at least one member of the Board had some years experience in the Tropics. Regarding a point made by Col. Cameron, that, in some cases, Medical Specialists appeared to wish to defeat the Board, rather than get the patient invalided, he thought the position may have been misunderstood, but that there may have been instances where Specialists were quite certain that the patient should be invalided to a temperate climate, in the interests of his own health, as well as in the interests of the Army, and that, in order to make reasonably certain of achieving their object, full emphasis had been used.

159. LT.-COL. COBBAN suggested that one of the faults of the present system lay in the failure of the army to employ men placed in Category C. They, not unnaturally, spent their time in and out of hospital and were a drain on the resources of the medical services. Many Category C jobs, that in India were done by Indian personnel, were done by British personnel at home, and, if these men could not be employed in India, it would be better to send them to England, where they could be made use of and not burden the Army in India with useless men. Another point was that the cases that the Review Board rejected, but would re-consider later, if they failed to carry out their duties in Category C, were not usually seen again by the same hospital.

160. MAJOR DAVAR: It is said that the patients, when they come before the Review, Board, were found, many times, not to have been thoroughly investigated, nor have had any laboratory findings done. If such is the case, it is not the fault of the hospitals, nor of the Specialist, that such tests were not done, nor is it that the Specialist did not know the tests, but that they had no facilities for doing them. I can mention a case. I sent an officer, with high blood pressure, to another hospital, for certain investigations of his kidney functions, as I did not consider it safe to diagnose it as essential hypertension, without investigating his kidney conditions. I mentioned the particular tests that had to be done. After a time, the officer returned to me, without any of the tests having been done. As his blood pressure was at a high level, it was not considered advisable by me to keep him and detain him until these tests could be done somewhere else, so a Medical Board was held and I had to mention on the Board Papers, that these tests could not be done, due to want of facilities, and probably that officer will appear before the Review Board, without the tests having been done, and I hope that they will be done at the Review Board.

Sometimes, by the time a patient appears before the Medical Board, a long interval may have passed between the time when the patient was first seen by a specialist and the holding of the Review Board, and it is likely that the physical findings may not always be the same, as the patient's condition may have deteriorated, or may have improved temporarily, in the meantime. I would like to know how long it takes for a patient to get admission to the hospital at Poona, to appear before the Review Board, after he has been boarded out at one of the garrison hospitals, and whether beds are always available for such patients.

161. COL. CAMERON: 'Skin' cases, appearing before the Review Board, are dealt with by the Command Dermatologist, Southern Army. Facilities are available for superficial X-ray therapy, at the J. J. Hospital, Bombay. Cases which might be retained in India following such treatment, are treated there, prior to final consideration by the Board.

Amoebiasis: If there are residual evidences, the patient should be evacuated. The Indian Category C2 is useful for chronic cases. If the patient is suitable for retention in India, give him a permanent Category—B or C1—rather than a temporary one, in which he is not likely to be employed.

Efforts were made to obtain Category C men, for employment in hospitals, but these were, unfortunately, unsuccessful.

Indian officers do appear before the Review Board, but only in special cases. The Board was instituted for British personnel recommended evacuation ex-India; more recently, it has come to be the "Appeal Board", and so, most important cases have been referred to it.

162. COL. SCHLESINGER, in closing the discussion, advised all officers not to talk too much in the presence of the patients, as it was not in the best interests of the patients. Instances had occurred, when the rejected officers had got hold of the Board Papers.

The Meeting re-assembled in the Pathology Theature, K. E. Medical College, at 1630 hours, with Lt.-Col. S. N. HAYES in the Chair, for demonstration of clinical cases, by the Staff of the Mayo Hospital.

CLINICAL DEMONSTRATION

163. A CASE OF PELLAGRA—*Khan Bahadur Dr.* YAR MOHAMMAD KHAN, *Professor of Medicine.*

Mrs. A. B., aged about 35, is demonstrated, to lay emphasis on the following points:

(i) that the ingestion of maize is not the sole cause of the disease, because, in this patient, there is no history of maize having formed part of her dietary;

(ii) that the disease has got a great relapsing tendency and the patient in question was diagnosed as such, on clinical features only, ten years ago, in this hospital, in the absence of a Biochemist at that time: further, the patient completely recovered at that time, by nicotinic acid treatment. This time, the diagnosis was confirmed by biochemical tests, carried out by Mr. B. D. Kochhar, Biochemist to the Pharmocological Dept. His findings are:

Nicotinic acid excretion in urine

(*a*) 24 hours excretion of the case=0·27 mg (Normal=3—11 mg). After a test dose of 400 mg of Nicotinic acid by mouth, three samples of urine were collected and estimated.

(*b*) 3 hours after test dose—0·154 mg (Normal 5—15 mg).

(*c*) 6 hours after test dose—3·6 mg (Normal 5—10 mg).

(*d*) 24 hours after test dose—0·87 mg (Normal 5—10 mg).

Normally, 5 per cent. of the test dose is excreted in the first three hours, whereas, in this case, only 0·154 mg was excreted. In the case demonstrated, the 24-hour amount of nicotinic acid is very small, as compared with the normal, and in the first 3 hours specimen after the test dose of nicotinic acid, the amount excreted is also a fraction of the normal.

(iii) That there must be some other factor responsible for the production of the disease and, if Chick's views be admitted, then this toxic substance or factor, derived from maize or occasionally from other cereals, produces the disease, but only in the absence of sufficient quantities of nicotinic acid and deficiency of essential amino acids.

Other points of interest in the case are:

(1) Glossitis with angular stomatitis.

(2) Gastro-intestinal disturbances, resembling dysentery (3-4 motions daily).

(3) Achlorhydria which is histamin—fast.

(4) Hyperchromic macrocytic anaemia.

(5) Dermatitis of the bridge of the nose, nape of the neck, dorsum of the hands and wrists and dorsum of the feet.

(6) Irritable temper with tingling and numbness; hysterical fits.

(7) Lungs—clear on X-ray examination.

(8) Heart—normal on physical examination and X-ray examination.

(9) Gasto-intestinal tract—on opaque meal and screening, showed rapid passage of meal through colon; nothing else abnormal.

(10) No enlargement of liver or spleen.

(11) Leucorrhoea and dysmenorrhoea.

(12) On nicotinic acid treatment (200 mg daily), the patient has made a wonderful recovery within a fortnight.

164. ADOLESCENT RICKETS—*Khan Bahadur Dr.* MOHAMMAD YUSUF, *Professor of Clinical Medicine.*

Mr. R., Indian Christian, age 16, has epiphyseal thickening, bony deformities, polyuria and fine tremors of hands; duration seven years.

Clinical and Laboratory Findings—Feminine features, enlarged breasts; no sexual deficiency; fundus and vision normal; urine, sp. gr. 1005—1010 with a trace of albumin; B. P. 130/90; B. M. R. within normal limits. Blood—calcium 10 mgm, Phosphorus 4 mgm, chlolesterol 125 mgm. Polyuria controlled by pituiterin: no improvement in rickets so far.

Diagnosis—adolescent rickets and diabetes insipidus. Special points of interest, (*a*) Interrelation of the two diseases? (*b*) Cause of rickets in a boy whose home conditions provided plenty of sunshine and suitable diet?

Brigadier McAlpine and Colonels Cameron and Schlesinger took part in the subsequent discussion and agreed upon the diagnosis; they made certain valuable suggestions, namely, (1) testing the field of vision, and (2) detailed investigation of calcium metabolism. Interrelation of the two diseases was not explained.

165. INTRACRANIAL TUMOUR—*Dr.* K. L. WIG, M.B., B.S., M.R.C.P.

S. R., Hindu male, age 30, developed headache, mostly occipital, two years ago, which increased progressively; occasional vomiting; diplopia occurred six months ago and disappeared with glasses prescribed by some eye specialist. Vision began to fail two months ago and was completely lost at the time of admission into the hospital.

On examination, there was marked papilloedema both eyes; pupils were dilated and sluggish in reaction; there was a comparative diminution of the sensation of touch; pain and temperature on the right side of the face; conjunctival reflex on the right side was completely lost; there was weakness of the seventh nerve and complete deafness and loss of labrynthine functions on the right side; marked nystagnus on looking to the right; gait was unsteady; dysdiadochokinesis was present on the right side; the skin showed numerous patches of pigmentation. The case was diagnosed intracranial tumour in the cerebellopontine angle. X-ray of the skull showed some erosion of the petrous bone; this, combined with the spots on the skin, and the localisation of the tumour in the cerebellopontine angle, suggested the diagnosis of an acoustic nuroma.

Brigadier McAlpine agreed to the diagnosis. Dr. Ramzan Ali objected to the diagnosis, on the basis of absence of Tinnitus. Due to the history of Diplopia and sluggish eye movements which he had noticed and sluggist pupil responses, he thought it was a mid-brain tumour. Brigadier McAlpine pointed out that diplopia could be due to increase of intracranial tension and the sluggish pupil responses were due to post-neuritic atrophy. He, moreover, opined that, due to this atrophy, an operation would not restore the sight.

166. CONGENITAL CYSTIC DISEASE OF THE LUNG—*Dr.* RIAZ ALI SHAH.

Patient, age 42; a skiagram showing polycystic lung (R) or congenital cystic disease of the lung, was demonstrated.

167. DR. WIG, in demonstrating another similar X-ray film, said that, in his opinion, some cases showed the symptoms late, because the latter started when an infection occurred, and that the disease was not rare. Dr. Madan Lal Aggarwal (a radiologist of Lahore), in supporting Dr. Wig, said that he had seen several cases in his practice and thought that the disease was congenital in origin, because cartilage had been found in histological sections.

168. CHRONIC AMOEBIASIS—*Dr.* M. A. PIRZADA.

This case showed the following features:

(1) The intestinal infection proved refractory to nearly five courses of emetine by injection, in a period of ten months.

(2) The first attack of hepatitis, which occurred in March, 1943, did not respond to emetine and had to be treated with two aspirations and another course of emetine by injection.

(3) As no oral treatment was given, hepatitis recurred in August, 1943.

(4) The second attack of hepatitis went on to rupture and abscess formation in the lung.

(5) The hepato-pulmonary amoebiasis did not show satisfactory response to emetine injection, probably due to associated secondary infection and the patient was an invalid for several months.

(6) The expectoration, which was profuse, was of the pyogenic type, throughout his stay in the hospital, although, on the first day or two, it was said to be of a dirty brown colour.

(7) Lack of response to emetine should not imply a revision of diagonsis, because both primary and secondary amoebiasis may show refractoriness, due to secondary infections.

Lt.-Col. Pasricha, in the subsequent discussion, condemned the use of M & B 693, which had been used for some time in this case.

The Meeting adjourned at 18.30 *hours*

10th FEBRUARY, 1944—MORNING SESSION

The joint meeting with the Medical Staff of the K. E. Medical College and Mayo Hospital, Lahore, commenced at 09.00 hours, with **LT.-COL. S. N. HAYES** in the Chair.

TUBERCULOSIS

169. DR. RIAZ ALI SHAH—"*Tuberculosis During War Time*" **(P).**

Until the last world war, sickness always caused more deaths among the fighting nations than battle wounds. For example, American statistics show that, in the Civil War, the number of soldiers lost through disease was almost double that lost through fighting. The large-scale epidemics of acute infectious diseases have recently been conquered, through improved public health measures and more efficient medical services. In consequence, in the world conflict of 1914—18, tuberculosis was revealed for the first time, in bold relief, as a major war problem. Formerly, acute infectious diseases like plague, cholera, smallpox, typhoid fever, dysentery and typhus fever, were taking an overwhelming toll of human lives, obscuring tuberculosis mortality during war time. War and tuberculosis have, in fact, always been close allies.

We are now engaged in another war, which has already produced conditions comparable to those of 1914—18. Fortunately, we have before us the experiences of the last world war, and a quarter of a century of fruitful research, which have led to a far better understanding of the methods for the control of tuberculosis. Thus, we are in a much more fortunate position today, to face the problem, than we were in 1914—18.

In the first world war, our knowledge of epidemiology of tuberculosis and the methods of diagnosis, were very defective. The exogenous mode of infection was not considered important. At that time, all healthy individuals were already thought to have latent lesions, and the advent of illness was attributed to endogenous breakdown under strain. No apprehension was felt of the transmission of the disease from the sick to the healthy in the camp, so much so, that the idea was prevalent in some places, that change of climate was so beneficial, that it might be wise to send patients into the army for their health. Large numbers of soldiers, who developed advanced tuberculosis in service, have had early or latent lesions on admission. The physical examination employed at the time of recruitment, was not adequate to detect early or latent disease. X-ray was seldom used. Incredible as it may seem now, Matson in 1918 wrote: "As compared with the physical examination, the roentgenological examination, even when done by an expert, occupies a place of secondary importance in the diagnosis of tuberculosis of clinical significance."

Lessons of the Last World War

After the last world war, much labour was spent in assembling mortality records of the war years, in order to find the factors responsible for the increase in tuberculosis in Europe. Overcrowding, malnutrition, excessive exposure to infection and strain, have been considered by various investigators as responsible to a different degree. In Germany, Austria-Hungary and Belgium, the nutritional factor was considered most significant and insufficiency of proteins was held chiefly responsible for the rise. In England and France, overstrain was chiefly blamed, but more recent studies have revealed that there too, nutrition played a most important part. Overcrowding certainly had an adverse effect, but perhaps not to the same extent as malnutrition and strain. The experience in Denmark was studied by Faber, and his findings are interesting. Denmark shared in the tuberculosis increase during the last world war.

Until the submarine blockade of 1917, Denmark exported food to England and Germany. Consequently, the prices soared and there was great shortage of food, particularly of meat and fish, for local consumption. With the advent of the blockade, Denmark retained much of her meat produce. Within a year, and definitely before the drop in other European countries, tuberculosis fell rapidly. Subsequently, a housing shortage developed, which led to overcrowding and increasing exposure to tuberculosis. The decline in tuberculosis mortality in Denmark appeared to be unaffected by the housing shortage. From this, one concludes that protein deficiency was the major factor in the tuberculosis rise.

Advancement in the Knowledge of Tuberculosis

Since the last world war, great strides have been made in the understanding and management of this disease. Two fundamental facts, which have come to full realization, are.

1. Exogenous re-infection plays an important part in the spread of tuberculosis; and
2. Without the help of X-rays, a large number of early, arrested and latent lesions, cannot be diagnosed.

Alongside, the science of nutrition has made phenomenal progress. In the matter of treatment, in 1918, artificial pneumothorax was practised on a few cases, even in the foremost countries of the world, and other surgical methods were almost unknown. Now they are being universally employed, with conspicuous success.

Application of Methods During the Present World War

The present war differs from the last war in some ways. It is total war. Whole populations are engaged, either through the army or through nation-wide food restrictions. The military and civil problems are fused together, more intimately than ever before.

The high pressure industry, leading to serious overcrowding, favours exogenous infection, which was discounted as a menace in the first war. Apart from industrial crowding, the war has created other opportunities for wide spread of infection. The mass migrations and evacuations have created huge refugee problems. In some European countries tuberculosis sanatoria have been evacuated, to make room for war and air-raid casualties, thus liberating open cases among the population. Air-raid shelters and black-out conditions are very likely promoting a spread of infection. Overcrowding in homes, due to destruction of residential property by enemy action, and food rationing, are other important factors. In short, it may be said, that the present war has created conditions which have lead to an increase in the risk of infection, and to a decrease in the resisting powers of the individual, whether he is in the fighting forces or a civilian.

The nations who feared the ultimate outbreak of hostilities, recognized tuberculosis as a serious war time menace, and prepared accordingly. Tuberculosis service have been developed. The defects, inherent in the system of recruitment to the army in the last war, are being avoided as far as possible. An increasing use of the X-ray is being made, to keep the tuberculous out of the army and this, incidentally, is leading to the discovery of a large number of patients who, with due care, can be prevented from being a menace to others.

Nutrition is now universally believed to have an important bearing on the problem of resistance to tuberculosis. In all national programmes, nutrition is occupying a front place.

In England, the public health has been surprisingly well maintained, and all diseases show a decline, with but one exception, that of tuberculosis, which is on the increase. France, also, has found tuberculosis markedly more prevalent, and the disease is again number one in rank, among all causes of death from disease.

From Germany, in spite of censorship, comes a report that Hitler has announced that all tuberculosis cases, as well as healthy workers, must do whatever they can, until finally the terminal breakdown puts an end to their productivity. No better device could be produced, to maintain and spread tuberculosis.

A terrible famine is reported to be raging in Greece and some other Balkan countries. It is said that the people are dying by the hundreds, from tuberculosis and famine.

In Japan, the tuberculosis death rate in 1936 was 206·6 per 100,000 population, according to a report of the League of Nations. This was the second highest tuberculosis death rate, among the nations of the world. In 1939, after three years of war with China, the death

rate had gone up to 225·0 per 100,000, according to a personal communication to me by Dr. Nobechi, in Tokyo. More recent figures are not available.

The Present War and Tuberculosis in India.

India has not endured the hapless fate of the occupied countries, or the devastations that have taken place in some of the fighting countries. Our privations are, as yet, trivial, as compared to theirs. But even before the war, tuberculosis was considered, by the best authorities in the land, to be on the increase. Sinister influences are now at work, seeking to lower further, our standards of health and to weaken our resistance to disease. Furthermore, thousands of physicians and public health workers have been taken for necessary service in the army. Health protection of the home front lessens, while health risks are increased.

In the present total war, it has been realized that the home front and the firing line are both equally important. To keep one man in the field, twelve men are needed at home, to keep him supplied with mechanised weapons of war and food. The maintenance of health of these twelve men is as important as that of the men under arms. Tuberculosis, thus, has a double significance. It offers a constant threat to the fighting forces, as well as to the home front. Very aptly, we can call tuberculosis a master saboteur during war time.

The following measures seem to be of fundamental importance, in order to prevent or cut down the harm done through this menace, at the two fronts:

(1) Subject to the availability of apparatus, frequent and, if possible universal use of radiography at the time of recruitment, to the fighting forces and to the war industries. Extended use of radiography, among certain urban groups, in which the disease is known to be more common. Miniature films are economical and time-saving. In England, preference is given to the 35 m.m. Cine films, and, in the U. S. A., to the 4×5 inch films. Tuberculin testing will lead to further economy.

(2) Tuberculosis institutions are urgently required, to treat and isolate cases. There will be the greatest difficulty in finding building material, equipment and trained staff for these institutions, but it will not be insurmountable. As pointed out before, tuberculosis weakens our war effort. It should be treated as a major war hazard and not just as a nuisance that can await solution until peace is restored. The developing of tuberculosis institutions should be dealt with as a war industry.

(3) Even in peace time, the level of nutrition in this country is not very high. Malnutrition, and particularly protein deficiency, should be guarded against, as far as possible. Prices of principal foodstuffs, especially of milk, meat and eggs, should not be allowed to become prohibitive, as they are at present. With high prices, the poorer people are not only unable to purchase them in adequate quantities, but also, if producers, the people are tempted to sell all their produce.

While mass radiography, development of tuberculosis institutions and food control may appear to be of herculean proportions, careful planning and mobilization of all our resources should make these feasible, to a considerable extent.

Fortunately, we have an infant national organization for the prevention and control of tuberculosis, namely, the Tuberculosis Association of India. It should be developed to organize a wholesale attack on tuberculosis, on the lines indicated. Mere defence will not do. The Maginot Line, Dunkirk, Pearl Harbour and Singapore have shown that the defenders may lose the battle. In tuberculosis, as in war, the attacking forces carry the day.

170. MAJOR VAUGHAN—"*The Development of Lung Cavitation in Tuberculosis*" **(P)**

It is always a matter of the greatest interest, to any doctor, to try and co-relate the pathology, physical signs and X-ray appearances, in any disease with which he is confronted. This is particularly true in regard to diseases of the chest. I have heard it suggested, that such a study is in the nature of a jigsaw puzzle, and the successful treatment of the individual case, goes to the doctor who best can fit together the living pathology, physical signs and symptoms and the X-ray appearances, at the time of diagnosis. Obviously, not everyone fits the pieces together in the same way. Some claim ability to find the answer with only a limited number of pieces and some even despise certain sets of pieces. For some years now, we have all read articles or heard discussions on the value of mass radiography. There are some, to this day, who profess to be independent of X-ray aids, in the diagnosis

of tuberculosis. Such people say that, to them, a very careful clinical history and stethoscopic findings, are all that is necessary; whereas some have gone to the other extreme, and have relegated the stethoscope to the scrap-heap? Now, both classes of individuals, as such, may be entirely truthful in their statements, the first having, as it were, ears to hear, and the second, eyes to see. But the real truth is we still require every possible aid, pathological, radiological and stethoscopic, in the diagnosis of chest disease, and happy only is the physician who makes use of all these aids, without over-emphasising the importance of any particular one.

Bearing the above remarks in mind, I should like to try and show how these principles affect the discovery of Cavitation, throughout its development, in what is unfortunately still a relatively common disease, viz. pulmonary tuberculosis.

But, in order fully to understand the abnormal, it is all-important to have a good working knowledge of the normal. Therefore, a few words on the finer anatomy of the lung, in order to refresh your memories, may not be out of place to begin with.

The Lung has been described as "a hollow tree, ramifying almost to infinity, whose numerous branches are the Bronchi, whose ultimate twigs, or Alveolar canals, widen themselves and form Alveoli and change their structure, in order to assume the character of respiratory surfaces." Following the branching of the Bronchial Tree, we eventually reach the terminal Bronchioles. Here, the structure of the bronchiole changes from that of the large bronchi, viz. it loses its cartilage and mucus glands, but it now possesses abundant elastic tissue and its muscle walls are more highly developed than those of the larger tubes. The Bronchiole is continued as the Alveolar Duct and this is the last division of the bronchial tube. The Duct then dilates into an antichamber, called the Atrium, from which open several alveolar saccules or Air Cells. This whole unit, comprising the terminal bronchiole, alveolar duct, atrium and a number of aveolar saccules, is sometimes called the Primary Pulmonary Lobule or Acinus. As we shall mention later, the acinus is the pathological unit in Pulmonary Tuberculosis. These air cells, as we know, are in intimate relationship with the surrounding capillaries, for the interchange of gases. The highly-developed musculature of the bronchioles, ends at the alveolar duct and it is not continued into the walls of the Atria. Hence, it acts as quite an efficient sphincter, which goes into spasm in bronchial Asthma conditions and thus closes the exit of the Atrium. It has been shown by X-ray studies, that the bronchi are not rigid, immobile tubes, as we are often apt to picture them. Secretions, to a large extent, are brought to the main bronchi by a series of peristaltic contractions, as well as by the natural sweeping movements of the cilia. It is only when the secretions reach the main bronchi, that the cough reflex is excited.

Pulmonary Tuberculosis

It is not within the scope of this paper, nor is it my intention, to discuss, in any detail, the various theories held, as regards infection or immunity in this disease. As yet, among those engaged in such research work, there is no concensus of opinion on the mechanisms of either natural or acquired resistance. However, broadly speaking, in adult life, there are two main theories as regards infection, viz. (1) Endogenous, and (2) Exogenous. That is, either the disease is due to reactivation of a childhood infection in the lungs or elsewhere, or the disease may be caused by a recent re-infection by inhalation. This later, or exogenous infection, is probably the more usual occurrence, as shown by the evidence of contact infection, and the laboratory investigations, which demonstrate that, in the vast majority of cases, the organisms are of the human type, whereas, in the glandular infections of childhood, the tubercle bacilli are of the bovine variety. The tubercle bacilli may reach the lung by one or more of three routes, (1) by respiratory tract, (2) by the blood stream, (3) by the lymphatics. The infection having taken place, the key to the understanding of all changes leading up to tuberculous cavitation, lies in the study of the behaviour of the Acini. Until their caseous content breaks down, they are quite silent areas, although they may impose an appreciable lack of movement on the affected side. Once they begin to break down, Nature immediately attempts to evacuate the caseous material into its corresponding bronchiole. The first clinical sign of active tubercle is now audible, in a very fine sibilus, or a very fine crepitation or moist sound. This is heard most clearly and loudest, in the last phase of inspiration, thus differing from the sounds of ordinary bronchitis, which are always most marked in the early or 1st phase.

An X-ray, taken before the breakdown of the Acinar lesions, is generally of one of two main forms:

(1) The acute, non-fibrous, or exudative type of infiltration. Here we get various areas of shading or loss of translucency, not unlike a broncho-pneumonia, but they differ from the latter in that their edges appear more woolly and that there are more clearly defined translucencies between them, than is the case in a broncho-pneumonia. Also, the broncho-pneumonic distribution is nearly always in the lower lobes, as against a T. B. condition, which is generally more marked, or confined to the upper and mid zones.

(2) The fibro-caseous, or productive type of infiltration. Here, as a rule, we find a rounded area of shading, usually in the subclavicular region, or near the apex of the lower lobe, and, most commonly, on one side of the chest only, most usually the right. This is sometimes known as Assmann's focus. In children, the primary focus is frequently seen in the periphery of the lower lobe and it is known as Ghon's focus (who first described it). In the adult, evidence of this early infection can sometimes be recognized as a healed calcareous nodule. If the initial infection has been heavy and the patient's resistance poor, these two types of disease will progress towards established cavitation, somewhat differently in each case.

Acute Non-Fibrous Tuberculosis

This type of disease very seldom produces a large cavity, but several individual cavities may appear at or about the same time. This is because the disease lacks organization—acinar collapse and consequent increase in the intrathoracic negative pressure, which is characteristic of the fibrous type. The inflammatory exudate, filling the acini, caseates and undergoes central necrosis and liquefication. This is uncontrolled by fibrous tissue and its round cell infiltration and giant cells. No barrier, therefore, is set round it, to protect the surrounding lung tissue, and, as a result, the exudate fills the bronchi and the tubercle bacilli flood the lymph nodes, which in turn, causes them to caseate rapidly. At first, the cavity is small, but it may coalesce with other cavities, until, finally, through ulceration of the interstitial tissues, a large cavity is seen. In some cases, this cavity thus formed, may closely resemble a pneumothorax on the X-ray film, but it can usually be recognized as a cavity, by having a somewhat crenated edge, whereas, in a pneumothorax, the lung edge is very sharply defined. Heavy hilar shadows will also be seen, somewhat indistinct and confused, but occasionally the right tracheo-bronchial group of glands may show up as a separate and well-defined shadow. As there is no fibrosis, there is no shift of the heart or mediastinum to either side.

Physical signs: There will be slight early clubbing. Such clubbing is quite different from the usual finding in an established case of bronchiectasis, where the enlargement is 'septic' in type and the terminal phalanx is 'drumstick' in appearance. In tuberculosis, the enlargement is 'dorsalpalmar' in type—claw-like, tapered and refined-looking, sometimes referred to as parrot' beak or puffin bill. P. N. Somewhat impaired but no true dullness. Lung movement is poor. The greater part of the area infiltrated may be silent, but, here and there, the diagnostic sign of active infiltration will be heard, viz. persistent sibilus or fine crepitation towards the end of inspiration. Over the site of the cavity, local pleural friction may be heard, but the diagnostic sign of active cavitation is a limited circumscribed bronchitis—this is actually a true tuberculous bronchitis.

Chronic Fibro—Causes Tuberculosis

Here, as I have already mentioned, the earliest lesion is usually subapical in the upper lobe, most commonly on the right side. This is also usually the site of its cavity, but it is rather slow in its formation, due to the fact that fibrosis attempts to keep pace with infiltration. If the Assmann focus breaks down fairly rapidly, as it occasionally does, then it shows up on the X-ray film as a well-defined Annular shadow, rather thin in outline, and very often there is a fluid level present. The average case, however, shows a slow disappearance of its regular outline, its edge becomes hazy and it gradually merges into shadows made by the increased blood supply of draining bronchi, tracking down towards the hilum. As there is some degree of fibrosis present, there is a slight but definite shift of the mediastinum towards the affected side. As the tuberculous granulation tissue empties into the bronchioles, many of the empty acini collapse; others, which are unable to rid themselves of their exudate, proceed from caseation to fibrosis. In time, the affected bronchial wall weakens, it dilates and loses its

power of elastic recovery on expiration and is consequently pulled upon by the negative intrathoracic pressure. Eventually, depending on the degree of resistence put out against the disease, fibrosis gets, as it were, the upper hand, the surrounding affected acini combine to form a more or less solid surround, and the cavity is squeezed or even obliterated by the contracting fibrous tissue. As a result of all this, the pleura overlying the diseased area becomes inflamed, adherent and often thickened, to a considerable extent.

Physical signs: are usually well marked. Tuberculous clubbing is nearly always present. If the case is very advanced, and long standing, super imposed secondary infection may make the clubbing 'septic' in character. There is flattening of the chest wall and displacement of the mediastinum, to the affected side, or, in bi-lateral disease, the shift is to the more affected side. Depending on the stage of the disease, there will be the signs of fibrosis and active infiltration, to a greater or leaser degree. As a rule, the amount of lung tissue involved is always greater on the X-ray film, than is found on physical examination. A good working rule appears to be, that signs heard *above* the clavicle, only mean that at least $\frac{1}{3}$ of lung is involved; signs heard *below* the clavicle, mean that at least $\frac{1}{2}$ of lung is involved. If laryngeal T. B. is present, the true extent of involvement is about three times that of the stethoscopic findings.

Until recent years, the whole pathology of tuberculous cavity formation has been considered as directly due to actual and continued destruction of the lung tissue. But opinion is now gradually changing, largely due to the original work of Monaldi (1938) on closed-suction drainage of T. B. cavities, and, more recently, quite favourably reported upon, as a result of their own experience, by Sellors and Maxwell (1943). As a result of this work, it is now considered that, although tissue destruction is initially responsible for the commencement of a cavity, its growth and persistence are more influenced by mechanical factors, viz. (1) Thoracic wall traction during respiration, and (2) Inflation by air entrapped distal, inflamed and partly stenosed bronchus. In other words, a checkvalve action is set up and every inspiration drives air past the valve, to distend the small initial cavity into a large and still larger cavity. The denser tissue in its wall is due, then, in part, to inflammation and fibrosis, but also to the compression of the surrounding lung tissue.

The chronic tuberculous cavity shows up on X-ray examination, as no other cavity does. It has usually a well-defined wall, which, on close inspection, may be hazy in its distal borders, where changing densities show how the scattered nodules of organized and collapsed acini are lying in a background of fibrosed lung and covered by thickened pleura. If viewed from a short distance, it throws these various densities into relief against the background, thus producing a picture quite distinctive and diagnostic of fibro-caseous tuberculosis.

Such, then, is a very brief attempt to try and correlate the living pathology with the signs and the X-ray findings, in an all-to-common chest complaint. Of necessity, it must be an individual attempt, and the relative value placed on the various findings, is also, to a large extent, dependent upon personal experience. Others may set their own values, but arrive at the same answer in the end. Although this paper has not touched on treatment, yet no one would deny that correct diagnosis and proper evaluation of the pathological cavitation present, is of primary importance and is, in fact, the keystone to all rational therapy.

171. MAJOR CRADDOCK—"*Pleurisy with Effusion in Indian Troops*" **(P)**

The object of this paper is to describe two cases of special interest, to make certain general observations on cases observed during a 12-month period and to discuss the correct disposal of such cases. Symptomatic effusions, sulphapyridine effusions and protozoal effusions, etc., are excluded.

Pleurisy with Effusion followed by Miliary Tuberculosis

The rapid development of miliary tuberculosis following pleurisy with effusion, has been described, but is of sufficient interest to merit description of the following two cases:

Case 1. *Havildar aged* 29, *service* 6 *years*

C/o pain in all joints, headache, fever and pain in left chest. Developed pleural effusion (left). Aspiration was carried out on 3 occasions and a total of 700 c.c. withdrawn. The fluid was reddish in colour but otherwise typical. The signs of effusion gradually disappeared, the patient improved and became afebrile. For the first two weeks the fever was remittent, rising to a maximum of 104 and was then intermittent for a further 10 days, before settling to normal on the 30th day after admission. After remaining normal for a week, the temperature

suddenly rose to 103 within 24 hours, reached 104 on the following evening and thereafter remained mostly remittent for a period of 85 days before death. The diurnal range was considerable, up to five degrees, the peaks being recorded in the evening. The patient began to complain of cough and sputum and pain in both sides of the chest. Loss of weight was rapid. The liver and spleen became palpable and two weeks before death, meningism was present, and, later, signs of a pyramidal lesion appeared.

C. S. F. globul in increased. Cells 26 lymphocytes/cmm.

Blood. 1375 eosinophisll/cmm.

Sputum, persistently negative for T. B.

X-Ray showed increased density on left side, due to pleural thickening. Nil else.

Autopsy: Extensive pleural adhesions on left side of thorax. None on right. Both lungs showed miliary tubercles scattered uniformly and densely throughout the lung substance.

Pericardium adherent to left pleura and studded with miliary tubercles. Heart: Tubercles seen along the blood vessels on the posterior surface of the left ventricle. Section showed miliary tubercles in the heart muscle.

Liver: a few tubercles seen on the anterior surface of the right lobe.

Spleen: enlarged and studded with miliary tubercles.

Kidneys: numerous miliary tubercles.

Brain: tubercles seen along the course of the blood vessels on the external surface of both hemispheres.

Other organs: N. A. D.

Case 2. *Signalman, aged* 30, *service* 10 *years.*

C/o pain in chest, fever, cough and breathlessness for 15 days. Signs of pleural effusion (right). Aspirated Fluid clotted on standing. 22 lymphocytes/cmm. The initial fever was remittent, rising to a maximum of 102 and falling to normal by lysis, 22 days after admission. After remaining normal for 5½ days, during which the patient felt better, the temperature began to rise intermittently with evening peaks up to 100, and the fever then continued being mostly remittent, with a small diurnal range up to the time of death, 91 days later. The highest rise during this period was 102. The liver and spleen became palpable, and, owing to double rises in the 24-hourly temperature, he was suspected of Kala Azar. Formal-gel test was, however, negative. Lymphadenitis then appeared. Developed clinical and X-ray signs of thickening of left pleura, but no radiological signs of pulmonary disease. Finally, an acute abdomen supervened 'with signs of a ruptured viscus. No surgical interference was possible. Sputum throughout was negative for T. B. and Mantoux tests were also negative up to dilutions of 1 in 100.

Autopsy: Chest dense pleural adhesions on both sides. Fine miliary tubercles studded throughout both lungs. No tuberculous focus found in the lungs, but the bronchial glands were caseous.

Abdomen. generalised purulent peritonitis, due to a perforated tuberculous ulcer, circular in shape and 1" in diameter, in the ileum, 6 feet from the ileocaecal junction.

Liver. much enlarged, no tubercles.

Spleen. studded with very numerous large tubercles, ½—¾ cm. in diameter, giving it an 'almond toffee, appearance.

Omentum. infiltrated with the tubercles. All other organs, including brain. N. A. D.

(*The temperature charts of these two cases were demonstrated to the Conference*)

In both these patients, the temperature settled to normal, after 3-4 weeks, and then rose again for the final illness, lasting approximately three months. It seems probable that the blood dissemination occurred when the temperature rose for the second time. All cases of pleurisy with effusion, should therefore be watched, for the onset of a miliary complication after the initial fall of temperature, or, if the temperature should fail to settle as expected.

General Observations on 19 *Cases*

These cases were observed over a period of 12 months, and were distributed among the different sects as follows: Gurkhas—6, Sikhs—4, Hindus—4, Mussalmans—4, Karen—1.

The average age was 24, and the average service 3½ years. Of the 19 cases 4 died; 1 was invalided out of service; 6 are still in hospital; 8 were returned to duty, 2 to garrison battalions in forward areas, and 6 to peace stations, in Category C.

The 4 patients who died were all Gurkhas; 3 died of miliary tuberculosis, confirmed at autopsy, and the fourth was suspicious of this termination.

Of those patients who recovered, only one has since developed pulmonary tuberculosis. He was repatriated from the Middle East, following an idiopathic pleurisy with effusion and tubercle bacilli were found in the sputum 9 months later. He was then invalided.

The only other complication encountered was pericarditis. This patient had pleural friction sounds over the left upper lobe on admission, but no effusion. Acute sero-fibrinous pericarditis then supervened. This subsided after one month and was followed in six weeks by a large left pleural effusion. Three months after, there were no signs of effusion and the lungs were radiologically normal. The Mantoux test, with a dilution of 1 in 10,000, was strongly positive.

Of the eight who returned to duty, 4 had X-ray evidence of thickened pleura on discharge, and are still at duty, 5 months later in one case, and 2½ months later in the other 3 cases. Of the remaining four, one had thickened pleura on admission from a forward area; since discharge, 6 months ago, he has remained symptom-free, has played games, and an X-ray just taken shows a very small collection of fluid in the right pleura and visualisation of the interlobar fissure; he is now very anxious to go back to the forward area. Another has remained symptom free, 4 months after discharge, and the seventh is a jemadar who had an effusion with a typical fluid and with X-ray evidence of consolidation in the right lower zone. Mantoux tests were all negative.

In all the above patients, repeated sputum examinations were carried out and were all negative, as many as 15 examinations of concentrated sputum being done in many cases. In one, the prolonged course and the presence of cervical and axillary adenitis, and in another haemoptysis, made the suspicion of a tuberculous origin very probable. If these two cases are grouped with the one complicated by pericarditis and with the invalided case and the four fatal cases, a total of 8 patients, almost definitely tuberculous, results. The figures are too-small for the proportion to be significant.

Out of 448 cases of pleurisy with effusion, admitted to the London Hospital, between 1926 and 1936, only 70 (16 per cent.) were tuberculous effusions, i.e. associated with evidence of active tuberculosis at the time of effusion. Of the remainder, 70 were symptomatic, and 308 were primary cases (Vaizey & Parry 1940). Into this group, fall the 11 non-tuberculous cases of this series. Of these, a proportion will develop tuberculosis in after years. Trail (1943) notes, "If we are to believe after-histories, the disease is seldom tuberculous under the age of 15. In the age group 15—25, however, the diagnosis of tuberculosis must be to the fore." Borelins (1932) followed up 230 patients, 10—20 years after their effusions and found that 23 (10 per cent.) had died of pulmonary tuberculosis. Kallner (1937) in a series of 690 cases, found a morbidity of 39 per cent. from tuberculosis in those followed for the longest time, i.e. for 20 years or until earlier death. I am well aware, of course, that these figures may not apply to India. Perhaps our Indian colleagues can contribute indigenous figures.

It is evident, therefore, that more than half of such cases may remain free from after-effects due to tuberculosis. The remainder are possibly tuberculous, but live long enough to die of something else.

Disposal

I am frequently asked by my medical officers, "Should this case of pleurisy be invalided or not?" As far as I am aware, there is no official policy which lays down definite guidance in this problem. I have no doubt that the advice of Medical Specialists varies very considerably. One noted, "This effusion is probably tuberculous and the patient should therefore be boarded out." Presumably he recommended this in all such cases. Some A. Ds. M. S. approve board papers without question, others return the papers unapproved. I feel it would be a considerable help to medical officers and a saving in man-power to the Army, if some guidance were officially given. No hard and fast rule can be laid down; each case must be considered on its merits, but, at least, a medical officer can be advised that the policy is to retain such men in service, during the present emergency, where reasonably possible. Hutchison (1942) says, "The patient recovered from tuberculous pleurisy. should have a period of regulated living, fresh air, good food and periodic medical observation; such is supplied to the full, by service in the Army in one of the lower categories. A good rule would seem to be, that the man with a small effusion, which completely absorbs, should be retained after an adequate convalescence (2 to 3 months off duty), whereas the large effusion, or one which takes a long period to resolve, leaving much pleural thickening, should be considered a bar to further service. In future, I shall advise the retention of a higher percentage of cases than in the present series." We have unwittingly followed this policy.

Briefly, our attitude is summed up in the following principles, and I should like to hear if they agree with official policy, if any:

(*a*) If the patient's usefulness to the Army is small, for example, if he is an untrained recruit, recommend invaliding.

(*b*) If there is strong suspicion of underlying active tuberculous disease, recommend invaliding. The sedimentation rate may be a guide to activity in doubtful cases.

(c) In all other cases who are symptom-free, even if there is evidence of thickened pleura, return to duty in a lowered category (after an adequate period of convalescence), provided that the patient can be of useful service to his unit in such a category.

(d) After 6 months, check clinically and radiologically, and consider return to higher category, if warranted. Should the patient be upgraded and take part in active operations and break down in consequence, the disease to be considered due to service.

Sanatorium treatment need not be recommended, as the results have not been shown to be significantly improved thereby.

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172. COL. SCHLESINGER—"*Generalized Tuberculosis*" (D).

Different races have varying degrees of resistance against disease. Years of contact with smallpox, for instance, has given the Indian a certain degree of immunity against it, so that when he catches the infection, severe though it may be, he can usually put up a better fight than the European, who has to rely almost entirely on vaccination as protection. Measles has been known to take on serious and often fatal epidemic proportions in countries where the infection had not been previously encountered. On the other hand, malaria is apparently not such a menace to the local population of some parts of the tropics, such as the West Africans, who have developed a high immunity to the disease.

The development of active immunity against tuberculosis is not such a cut and dried process. Thus, despite the widespread incidence of the disease in this country, the Indian generally possess little resistance against it and we meet with the most amazing instances of rapid and extensive tuberculosis. In fact, the Indian, in many ways, reacts in the same manner to the infection, as the child does in Europe. This is well demonstrated by the following brief case histories, for many of which, I am indebted to the Medical Specialists of North-Western Army and Central Command. They also reveal mistakes that so often arise in the differential diagnosis.

Nature, Course and Location of the Disease

Many young doctors are apt to picture tuberculosis too much as a disease of the lungs, and forget that various other parts of the body can become involved.

A sepoy aged 24, was admitted to hospital, complaining of pain in the lumbar region and difficulty in walking freely, following a history of a fall. Previously, he had been diagnosed as lumbago, in several hospitals. An X-ray finally revealed the true nature of the case—caries of the spine, for which he was treated. A few months later, however, the infection spread to the lungs, the abdomen and the lower part of the rectum, where a cauliflower type of growth was discovered. For several months, a most hectic remittant pyrexia was present. Finally he died.

The temperature may, however, remain quite normal thoughout, and, in cases of chronic diarrhoea and grave anaemia, resistant to all forms of treatment, the presence of tuberculosis should be borne in mind, even in the absence of pyrexia.

A sweeper aged 37, with a history of frequent motions, containing blood and mucous, was discovered to have a red cell count of under two millions per cmm, and haemoglobin of 35 per cent. There was a suspicion of enlarged abdominal glands and yet a diagnosis of dysenteric cachexia with secondary anaemia was made. At autopsy, soon afterwards, tuberculous broncho-pneumonia was found with cavitation, and the mesenteric glands were also involved.

The rapid course which this disease can take, is well illustrated by the case of:

A nursing sepoy aged 20, who developed pleurisy, with scattered rales over the lungs. Despite repeated examinations tubercle bacilli were never discovered in his sputum. He died two months after the onset of his symptoms and a post-mortem revealed a small cavity at the apex of the left lung and a miliary spread in almost every organ in the body.

The extent to which the disease can progress, before some Indian troops will report sick, is shown in the following case:

A Lance Naik in the Gurkhas developed hoarseness and difficulty in speaking one morning and was admitted unconscious to hospital. He died soon after arrival, and at the autopsy was found to have a tuberculous mass of glands lying behind the sternum and attached to the pericardium, the visceralpleura of the left lung and spreading ound to the oesophagus. Miliary tubercles were found all over the peritoneum, the spleen and the liver.

Complete reliance must not be placed on sputum examination, since absence of tubercle bacilli here, will not exclude miliary tuberculosis of the lungs.

A recruit aged 26, was admitted to hospital with pain on swallowing and pyrexia. Blood slide failed to show malaria parasities nevertheless, an initial diagnosis of clinical malaria was made. Anti-malarial treatment was instituted, but failed to influence the condition. Soon, catarrhal signs developed in the lungs, but repeated examinations failed to reveal tubercle bacilli in the sputum. The clinical course proved rapidly fatal, with cerebral signs, and at antopsy, a tuberculous ulcer was found below the vocal chords and miliary involvement of the lungs, meninges and most of the abdominal viscera.

In Europe, short of a miliary spread, a tuberculous infection tends to remain limited to one part of the body. I except here the development of lesions in areas over which a stream of tubercle bacilli is constantly flowing, such as the larynx and the intestines in cases of pthisis. Thus, pulmonary tuberculosis affords some protection against tabes mesenterica and vice versa. Joint and bone tuberculosis are good examples of isolated infections. The source of infection and portal of entry, also, naturally, influence the nature of the disease, human types of infection being largely confined to the lungs, and bovine to the abdomen. In India, bovine infection is apparently rare, and even if cattle were widely infected, the custom of boiling milk before consumption would serve to sterilise it. On the other hand, the universal habit of spitting, and close association with the ground, at all ages, lays the Indian constantly open to human infection. It also appears that the individual's defence mechanism is more easily overwhelmed. Here are three cases, illustrating this point.

(i) A Lance Naik, aged 28, fell a distance of 4 feet and developed pain in the right knee. It soon became obvious that this was a tuberculous infection. Not long afterwards, marked kyphosis developed and caries of the 4th and 5th lumbar vertebrae was detected, unhealing sinus developing in the left groin, above Poupart, ligament. He ultimately succumbed when the disease spread to the lungs.

(ii) A sepoy, aged 21, with two years' service, reported sick with diarrhoea and irregular pyrexia. Physical signs pointed to tabes mesenterica, but spread to the lungs was not long delayed, the sputum became positive, the spleen greatly enlarged, and at autopsy, tuberculosis of the lungs and tuberculous peritonitis were discovered, with a final miliary blood stream infection.

(iii) Another recruit gave a four months' history of an intractable fistulo-in-ano. Diarrhoea and broncho-pneumonia supervened and death occurred three weeks after admission. Post-mortem showed a tuberculous ischio-rectal abscess, tuberculous broncho-pneumonia and tuberculous peritonitis.

Tuberculous Broncho-Pneumonia

Pneumonia, not responding to sulphapyridine, should always arouse the suspicion of tuberculosis and prompt investigations along those lines.

A Lance Naik aged 28, was such a case. A full course of sulphapyridine had no effect on his broncho-pneumonia. He developed marked dyspepsia, diarrhoea and meningeal symptoms, and a diagnosis of generalised tuberculosis was made. Post-mortem revealed miliary tuberculosis of the lungs, with a few small cavities tuberculous enteritis and meningitis.

Acute Abdominal Symptoms

Abdominal tuberculosis, arising as an acute abdominal diagnostic problem, is encountered in all countries and at all ages. In Europe, it is a common mode of onset in children. In this country, this type of clinical picture is often seen amongst Indian soldiers and is apt to run a particularly virulent and fatal course.

A recruit, aged 28, was first seen with a history of abdominal pain, diarrhoea and pyrexia of two months' duration. His condition suggested some acute abdominal condition. A tender, smooth, palpable mass was felt in the right iliac fossa, but the case was deemed to be tuberculous and he was not operated upon. At autopsy, shortly afterwards the lungs had several small cavities and were studded with miliary tubercles. Tuberculous peritonitis was also present, and there was a pericaecal abscess associated with a perforation of an ulcer in the caecum.

A signalman, aged 30, with ten years' service, came into hospital with a pleural effusion, a hectic temperature and marked enlargement of the liver and spleen. Symptoms of an acute abdominal catastrophe developed, and the diagnosis was further complicated by great enlargement of the liver and spleen. Sputum was persistently negative and the martoux reaction was negative up to a concentration of 1/100, not an infrequent finding in an overwhelming infection. It soon became clear that this sepoy was suffering from peritonitis, and at autopsy this was proved to be tuberculous and there was a perforation of an ulcer at the ileco-caecal junction. The spleen presented a "Hard bake" appearance.

Hepato-Splenomegaly

The discovery of an enlarged liver and spleen, with no other associated physical signs, is not an uncommon early clinical picture of generalised tuberculosis in children, in Europe. X-ray of the lungs, at once reveals the miliary nature of the infection, and at autopsy, the liver and spleen are found to be studded with large white tuberculous nodules, resembling

the morbid anatomy of Hodgkins Disease. Here, in India, it is the first time I have met this type of tuberculosis in adults, and with such diseases as malaria and Kala Azar in one's mind, the diagnosis is likely to go astray.

A sepoy of the Punjab Regiment, aged 27, was admitted with frequency of micturition and pyrexia. Marked enlargement of the liver and spleen was discovered and a blood slide showed the presence of B. T. malarial parasites. The fever continued, despite anti-malarial therapy. Crepitations developed in the lungs, no tubercle bacilli were found in the sputum, but X-ray revealed miliary tuberculosis of the lungs. Persistent hiccough pointed to the possible involvement of the diaphragm, and post-mortem later confirmed the diagnosis. Here, the most marked initial sign was hepato-splenomegaly, and, as so often in the tropics, malaria complicated the diagnosis.

Obviously, enlargement of the liver, with fever, can easily give rise to the mistaken diagnosis of amoebic hepatitis.

A soldier aged 40, came to his doctor with pyrexia and tenderness and enlargement of the liver. In addition there was a small basal pleurisy, but no X-ray evidence of anything else. There had been a history of dysentery and it was difficult to decide whether the pleurisy was due to a lesion above or below the diaphragm. Emetine was administered without any beneficial effect, and at post-mortem, an extensive tuberculous condition was discovered in the right lower lobe, with large caseous masses in the liver and spleen.

In another case, a Gurkha aged 17, admitted for dysentery, a mass resembling the liver was found to extend down to the umbilicus. Fever was present, which did not react to a course of emetine. Small painful lumps developed subcutaneously on both arms, and the glands became enlarged in the left iliac fossa. X-ray showed nothing beyond some enlargement of the hilar glands. Death rapidly supervened, and at post-mortem, the supposed enlarged liver was found to be grossly thickned omentun—such a common diagnostic pitfall and miliary tuberculosis was also present as a terminal event. Histological examination of the nodules of the skin, disclosed that they were tuberculides, a generalised spread, which I have, so far, only seen on rare occasions in children.

Summary.

The cases that have been briefly described, demonstrate, without a doubt, the frequent incidence of generalised tuberculosis. They also show the overwhelming nature of the infection, that so often occurs among Indian people. In many ways, the clinical picture bears a close resemblance to certain types of tuberculosis, encountered in children in Europe, and thus suggests an almost completely lack of resistance against the infection, in many Indian adults.

DISCUSSIONS

173. DR. RIAZ ALI SHAH said that about 20 to 30 cases were examined daily in the Out-Patients Department; in the diagnosis of cavitation, the screen examination yielded better results than the physical examination. The Metropolitan Insurance Company of New York, appointed a Committee of experienced physicians and radiologists, to examine known tuberculous patients; in their findings, X-ray examination showed more positives than clinical examination. Ghon's focus and Assmann's focus were seldom seen.

He enquired how the pleurisy cases were treated and how to deal with moderate and small effusions; and whether such cases should be tapped. He mentioned that, in civil practice, the pleurisy cases were treated on conservative lines, owing to late development of tuberculosis; about one third developed tuberculous signs; only a small percentage developed tuberculosis, if four months bed-rest were given, after the patients became afebrile.

He further stated that, (i) attempts were being made to classify child-type of tuberculosis, about 10 per cent. were of exudative lesions; (ii) the resistance was high in white races: (iii) acute onset was much more common than usually believed.

174. MAJOR CRADDOCK, in reply to Dr. Riaz Ali Shah: Aspiration was done, only for diagnostic purposes, but we did not hesitate to evacuate the effusion if any indications demanded it. Air replacement was not done. It was not possible to evaluate the merits of the treatment.

175. LT.-COL. HAVILAND-MINCHIN: I am convinced that 90 per cent. of these cases have a tuberculous origin, and I consider that every case should be boarded out of the Army. Exceptions might possibly be made in the case of key-men, or fully-trained N. C. O's, who could be used for base duties. In no case do I consider that any of these cases should be Categorised A, and definite instructions should be given to Unit M. O's, that they should never be upgraded from C, under one year.

176. DR. WIG: So-called idiopathic pleural effusions are, in a vast majority of cases, really tuberculous. Instances of cases, where such patients later developed clinical

tuberculosis in the lung or elsewhere, are not lacking. Some of these effusions can be proved to be tuberculous, by culture or by guinea pig inoculation.

Tuberculosis is not new to India. Populations, even in the villages, show a high percentage of tuberculin-positive individuals. While in certain parts of India, a very large percentage of the cases is of the acute type, in the Punjab, more than 50 per cent. of the cases are of the chronic type. The reason why a fairly large percentage of the cases are of the acute type, probably lies in nutritional factors and unsuitable living conditions, and does not mean that India is virgin soil.

The subject of the P. U. O. of a low type, which is a very common entity in the Punjab, is, I think, somewhat allied to the question of generalised tuberculosis. If the lungs are found clear on X-ray examination, it should not at once be concluded that the patient is non-tuberculous. Tuberculosis can obviously occur in other situations also; there are several instances where cases of P. U. O. ultimately and after prolonged observations, turned out to be tuberculosis of other organs, like the spine, testes and lymph glands; some developed tuberculous meningitis ultimately.

Bovine tuberculosis is responsible for a large percentage of non-pulmonary tuberculous cases in Europe, but this is not the case in India. Investigations done in various parts of India (by Dr. Goyle in Lahore, Major Mallick in Amritsar and Ukil in Calcutta), show that bovine tuberculosis plays practically no part in the prevalence of tuberculosis in India. Bovine tuberculosis does occur in a certain percentage of cattle in India. Taylor, in 1917, found its incidence to be 3·3 per cent. in the cattle slaughtered in Ferozepur, in the Punjab, but he was rejecting most of the cattle of poor quality for slaughter purposes and so the incidence of tuberculosis, in his investigations, naturally turned out to be very low. Soparkar and Dhillon, in an investigation in Lahore, done much later than the one mentioned above, found the incidence of tuberculosis in cattle to be 22 per cent. All investigations done amongst human beings, however, show that bovine tuberculosis is extremely rare, if it exists at all, though I would advocate that further investigations may be done on this point.

I am advocating an investigation of this problem, in spite of the work already done, because intestinal tuberculosis of the primary type—not secondary to the lung—is, in my opinion, common in this Province. It is not seen commonly in its hypertrophic form, as is the case in Europe, but in its ulcerative form. This form, in Europe, exists only as secondary to lung lesion, but here, the primary cases of this type are fairly common and the problem needs investigation.

177. MAJOR LYN GREENING emphasised the necessity for repeating the sputum examination, where acid fast bacilli are reported as being present in the sputa of patients on only one occasion, other signs and symptoms of pulmonary tuberculosis being absent. Under such circumstances, a diagnosis of pulmonary tuberculosis is not justified, when based on one positive slide examination only. Too much reliance should not be placed on the examination of pleural effusions by culture, on Loewenstein and Jensen's medium, and animal inoculation, as the low numbers of positive results obtained in the District Laboratory, Lucknow, from such specimens, was out of proportion to the percentage of cases of "sterile pleural effusions", which other speakers have shown to be proved subsequently cases of T. B.

178. MAJOR KILOH: I would say that all cases of pleural effusion, in which the fluid is an exudate, with the lymphocyte as the predominant cell, must be considered tuberculous, if no other cause can be found. This can be demonstrated by cultural or animal inoculation methods, in 70—80 per cent. It has been said that 30 per cent. of cases of pleural effusion, later develop physical signs, and these people were living under much better conditions than the troops on the Burma front, where the incidence must be higher. The term "idiopathic" is not a desirable one, and, if we consider any other cause than tuberculosis, then we must postulate a new disease and that a very common one. Taking tuberculosis as the cause, then the interpretation is that active thoracic tuberculosis is present and we know that this condition cannot be overcome for years. It is wrong, then, to return men in Category A to the Assam Burma border, where we know that the stresses and strains are great and the food situation not always of the best. I myself have been in the habit of recommending these men to be boarded out of the army, except in those cases that do well and have some high technical or educational qualification, when they may be placed in Category C for at least a year, during which, B. S. R. and X-ray observation should take place.

179. DR. HASSAN: Were the idiopathic cases subjected to Mantoux test?

180. MAJOR CRADDOCK: Only since we have received a supply of tuberculin. No case was considered to be non-tuberculous if the Mantoux test were positive (though it was not considered that the pleurisy was necessarily tuberculous on that account). On the other hand, a negative Mantoux test was not held to exclude the possibility of a tuberculous cause.

181. DR. PIRZADA: It is abundantly clear from the discussion, that there is no lack of specific immunity against the tubercle bacillus, amongst the Indian population. The incidence of generalised tuberculosis among the troops must, therefore, be attributed to a breakdown of general immunity, due to such factors as increased stress or strain of military life, or nutritional factors in personnel derived from a community in which the level of general immunity was never too high.

It has been pointed out during the discussion, that the bovine type of tubercle bacillus plays little part in human pathology in India. This is supported by recent reports, which indicate a very low incidence of tuberculosis among cattle in India, including the Punjab.

I do not agree with the remark that primary intestinal tuberculosis is very common in the Punjab. In children and in adolescents it does occur, but it is an uncommon event amongst the adult population. The final proof of the incidence of primary intestinal tuberculosis lies in postmortem data. Tribedi, Gupta and Ukil report an incidence of only 5 per cent. in Bengal, at all ages. Vishwanathan, from Vazigapatam, reports an incidence of 7 per cent. Figures from other provinces, if any, are not within my knowledge. It is significant that, in all cases observed by Col. Schlesinger, in the army, where diarrhoea was a predominant feature, extensive lung lesions were also present, proving the secondary origin of intestinal tuberculosis, which, indeed is the usual form of intestinal tuberculosis in adults.

182. LT.-COL. PASRICHA: Intestinal Tuberculosis—A great deal of work on tuberculosis has been done in India. The result of this work can be found in the publications of the Tuberculosis Association of India and in the October number of the Indian Medical Gazette. Primary intestinal tuberculosis is uncommon in India; the reason for this is that bovine tuberculosis is rare in our cattle.

Bovine and Human Tuberculosis in India

It would be interesting to determine the type of Mycobacterium tuberculosis, found in human infections, particularly in intestinal infections. The mass of material available in the Army is undoubtedly more than any single civil research unit would have at its disposal.

183. DR. GOYLE: Regarding the incidence of bovine tuberculosis in man, I would point out that a research worker, working under the Indian Research Fund Association in my department, isolated 21 strains of B. tuberculosis from cases of non-pulmonary tuberculosis, which were all found to be of the human type. In the Punjab, Hissar is the only District where tuberculosis is common in cattle, and the Veterinary Department is now engaged in an investigation into the incidence of udder infections in that area.

184. COL. CAMERON: In dealing with tuberculosis, India has failed 'to grasp the nettle, danger'—the facilities for treatment have not been parallel with the large number of cases. I am fully aware of the many difficulties etc., which have led to this position and of the improvement that has occurred in recent years.

The Army would like to meet 'the right to treatment' of every sufferer from tuberculosis, but doubts if it is its province to do so. Two interesting developments are taking place now: A. T.B. centre is in course of construction, at which the most modern methods of treatment will be given, including advanced surgical procedures. It will compare favourably with sanatoria in any part of the world. Apparatus for mass radiography will shortly be installed, for the examination of recruits to the Indian Army.

As regards pleural effusion, each case must be treated per se. I prefer the "Ca' canny" conservative policy—tapping only when essential. A large effusion, giving embarrassment, should be tapped; small effusions should be left for natural absorption. Nature can be assisted by graded exercises in the Rehabilitation Centre—early deep-breathing exercises and, latter, more advanced exercises, e.g. rowing, to develop full expansion. These, of course, can only be undertaken after the acute phase.

A thorough investigation is necessary, before tuberculosis is excluded, in effusion cases. I would stress the value of blood sedimentation rate and examination of the gastric fasting juice for tubercle bacilli. in arriving at an opinion.

185. COL. SCHLESINGER said that bovine tuberculosis was not a great problem in India, because of the usual habit of the people, in drinking boiled milk; and suggested, rather, a hygienic drive in this country, with a "DO NOT SPIT" campaign, as in Europe. He pointed out the fallacies of Mantoux test and advised the use of freshly diluted tuberculin.

186. LT.-COL. S. N. HAYES, in reply to Col. Cameron, pointed out that it was incorrect to state that the problem of tuberculosis had not been attacked in India. The facts were, that prior to the war, an organized campaign was inaugurated, sponsored by Lady Linlithgow. A great deal of work had been done and practical progress made. Unfortunately, the war had slowed things down and India being such an enormous country, some considerable time would elapse before the results of the campaign were markedly appreciable.

Regarding mass radiography, it was pointed out that, sometimes, criticism of inactivity was based on inaccurate information. e.g. the Army authorities now proposed to adopt mass radiography in India. Col. G. Taylor, I.M.S. submitted a scheme for mass radiography for the troops in 1939. This scheme was not accepted.

In congratulating Col. Schlesinger on his excellent paper on General Tuberculosis, he agreed that a 'No Spit Campaign' was desirable, but, if such a campaign was inaugurated, he hoped that the appointment of Director would not be offered to him.

TYPHOID FEVERS

187. PROF. GOYLE—"*Enteric Fevers in Lahore*" **(P)**

Epidemiology

Seasonal Prevalence—Enteric fevers are endemic in Lahore. Cases occur every year and throughout the year. The largest number of these cases are admitted to the hospital, usually from May to July, but this is not always the case, as, in some years, the maximum incidence may be in the winter months and may be due to the readiness with which food becomes contaminated by flies, which are then numerous, and to the rapid multiplication of bacteria in articles of diet.

Age—The youngest patient in the series was 2 and the oldest 62. The maximum number of cases is between 11—20 years and next from 21—30 years. The incidence of enteric infections above the age of 40 is extremely low.

Actiology

Enteric fever is a comprehensive term, including continued fevers caused by a variety of organisms, such as B. typhosus, B. paratyphosus A, B. and C. Enteric-like infections are also caused by other members of the salmonella group, e.g. B. enteritidis and its several varieties. Smith and Scott reported 3 cases of continued fever in man, from which the Dubin type of B. enteritidis was isolated.

Relative Prevalence of the Different Organisms of the 'Enteric' Group

During a period of two years, cultures were made with bloods of 450 febrile patients, out of which 114 were positive for one or the other organism. You do blood cultures after some preliminary tests, but the practice here is to send the blood of patients suffering from any type of fever.

The distribution of the various organisms was as follows:

B. typhosus	90
B. paratyphosus	23
B. enteritidis Gaertner	1

That is to say, the proportion of typhoid to para. A infections is 4: 1. There was one case of infection with Salmonella enteritidis Gaertner and this was recently reported by us in the Indian Medical Gazette. The case was of a child, aged 5 years, who was suffering from continued fever of the enteric type. He was an Indian evacuee from Burma, who had been infected on his way to India. B. enteritidis was isolated from his blood. It has been suggested by Topley and Wilson, that such infections are due to the Dublin type of B. enteritidis and not to B. enteritidis. Whilst the Dublin type may be responsible for the majority of cases of continued fever, I wish to point out that B. enteritidis is also capable of invading the blood in children and causing a similar type of fever. Guthrie and

Montgomery described a series of cases, most of which had occurred in the form of small epidemics among infants, with severe catarrhal enteritis, septicaemia, meningitis and purulent cholecystitis, and attended with high mortality. Para. B. was not isolated even once, and I am of the opinion that infection with this organism is not met with, in this part of the country.

Diagnosis

Blood Culture: It is a matter of great importance, from the point of view of control of the disease and of the patient, that an early diagnosis is made. In Enteric infections, positive blood cultures are obtained in the first week of the disease, in the vast majority of cases, provided the conditions for growth are suitable. Bile, containing 1 per cent. peptone and 1 per cent. glucose, has proved extremely useful. A negative blood culture at the beginning of a febrile illness, makes it unlikely that the disease is enteric. After the first week, the bacilli gradually disappear from the blood stream, though, in rare cases, positive results may be obtained, as long as the fever lasts. We have obtained positive blood cultures, in a good many cases, after the first week of illness, and it is a method that should be attempted at all stages of the disease. In one case, the organism was recovered from the blood as late as the 39th day of illness. There is some difference of opinion as to the effect of prophylatic vaccination, on the degree and duration of bacteraemia, in subsequent enteric infection. The matter is of some importance, as you deal with a population which is generally vaccinated. According to Perry, it shortened the duration of bacteraemia, thus reducing the chances of successful cultivation. Other workers, also, had difficulty in recovering typhoid bacilli from the blood, in vaccinated cases. On the other hand, several workers, including Ledingham, who had considerable experience of enteric infections, during the last Great War, is of the opinion that prophylactic vaccination is without effect on the bacteraemia of enteric fever. It is now generally held, that there is no difficulty in obtaining positive blood cultures in vaccination cases.

Blood cultures are of prognostic importance in enteric infections, for two reasons. Firstly, the mortality is higher in infections caused by B. typhosus, than in parayphoid infections. In infections due to B. typhosus, the mortality was 39 per cent. of culturally positive cases, but in infections due to para. A, it was nil. Secondly, in cases in which bacteraemia is prolonged, mortality is high. Blood cultures, after having been negative, again become positive in a true relapse. Some time ago, a patient was admitted with fever, and his blood was cultured on the 10th day of illness. The result was negative. His temperature came down after some time, but went up again. The physician in charge of the case was in doubt about the nature of his illness and consulted me. A second blood culture was done, and B. typhosus was recovered. Relapses are milder and of shorter duration, presumably because specific antibodies are already present.

Widal Reaction: Whilst the most conclusive method of establishing a diagnosis of enteric infection, is the isolation of organisms, the agglutination test, generally known as Widal Reaction, is extensively used in the laboratory. I do not propose to discuss this subject at length. The difficulties in the interpretation of this reaction are known to you. Your difficulties are much greater than in civilian practice, because all soldiers are inoculated with T. A. B. vaccine. Some of the difficulties have been overcome by the use of "O" suspensions. Some cases of enteric infection fail to develop flagellar (H) agglutinins, while developing somatic (O) to a high titre. In rare cases, only H agglutin may be present. For these reasons, among others, it is necessary to use both H and O suspensions for the diagnosis of typhoid fever. It is frequently asked if there is any particular titre which should be regarded as a reliable indication of infection. The fixing of arbitrary diagnostic titres is difficult and is not free from risk. Broadly speaking, we have come to a tentative conclusion on the basis of observations made here, that titres of 1 in 125 'O' and 1 in 250 H for B. typhosus, and 1 in 50 'O' and 1 in 125 H for B. paratyphosus A, may be regarded as diagnostic. But, it must be noted, that these titres should be considered in relation to all other available evidence. A single test is not always reliable. In the vaccinated individuals as well as in cases in which the first test in indefinite, tests are carried out with serial samples of blood from the same patient, at different periods of disease and the rise in titre noted.

188. CAPT. WALKER—"*Diagnosis and Management of Typhoid*" **(P)**

Even in an inoculated community, the enteric group occurs, with reasonable frequency. After the dysenteries, it was the commonest infectious disease seen in the 47th British General Hospital, Calcutta, comprising 0·5 per cent. of all admissions.

In spite of the prominence given to typhoid fever in the standard text-books, it is a disease rarely seen by medical men trained in the United Kingdom. It has to be borne in mind, constantly, when practising in India, for it can be diagnosed clinically in the first few days. Any illness of gradual onset, with fever, lasting over a period of days, with malaise and severe headache, no localising signs apart from bronchitis and showing no response to quinine, should be considered to be enteric group fever, and laboratory examinations initiated, to confirm or correct the diagnosis.

If laboratory facilities are available, the presence of a leucopenia is a strong point in favour of enteric fever. A total white cell count of less than 5,000 is significant. When concurrent malaria is present, the leucocytes may rise to normal figures (7,000 or 8,000). The leucocytes count is of the first importance in the early differential diagnosis of the enteric group from typhus fever and smallpox, both of which generally show a leucocytosis in the first few days. Occasional difficulty arises in a minority of cases of typhus, about one third of which show a normal count. Although the violence of the onset of these two conditions, presents a totally different clinical picture form that seen in typhoid, cases do occur in which the onset is similar and confusing.

In Bengal and Assam, kala azar is a real difficulty. The onset, the early course and blood picture, closely resemble typhoid fever. In the early stages, the soft enlargement of the liver and spleen may be taken for tympanites, and recurrences of fever for relapses. The patient is never so ill as he would be if he were suffering from typhoid fever at the corresponding period. The prolonged course, negative blood cultures and Widal reactions, and progressive leucopenia, will indicate the diagnosis, if the possibility of kala azar is borne in mind.

In an inoculated community, only the 'O' agglutinins are of value for indicating infection. A single Widal test is of no assistance. A series of examinations, made at intervals of a few days, must be made. A rising titre is the diagnostic criterion, not merely a high one. A case usually shows a rise from 160 to 320 to 640, falling rapidly after the febrile period. In paratyphoid A infections, both the typhosus O and the paratyphosus A 'O' rise in the first days of the disease, reaching 320. In the course of the next few days, the anamestic reaction for B. typhosus falls, leaving the other at 320, where it may remain for several weeks. The figure for paratyphoid A infections rarely reaches high figures. Anamestic reactions are common. Both M. T. malaria and amoebic hepatitis may give titres of 320 B. typhosus.

In one month, five cases occurred, which gave some difficulty in diagnosis and taught a useful lesson. They presented all the clinical features of the enteric group, but routine agglutination of B. typhosus and paratyphosus A was present in low titres only. The blood was cultured late in the fever, without much hope of recovering anything, and B. paratyphosus C was grown, in all five cases. These men came in the same convoy from Burma and appear to have been infected in Chittagong. Paratyphosus C can be recovered more easily from the blood and at a later stage in the fever, than the other members of the group. A similar difficulty occurred in one case of paratyphoid B. It is important, in cases of prolonged pyrexia, to ask the laboratory to examine for B and C, where the clinical picture is that of enteric fever and the routine serological findings are negative.

However valuable the Widal reaction is, the diagnostic measure of first importance is blood culture. Unfortunately, in military practice, a large proportion of the cases are seen too late for blood culture to be successful, though it may be so, as late as the fourteenth day. Occasionally, one obtains a second chance, with the onset of a relapse. One should always be alert for this development and if a bacteriological diagnosis has not been made, blood should be taken for culture, with the first appearance of a rise in temperature after defervescence of the initial fever. The technique of obtaining uncontaminated blood for culture, in a humid tropical climate, requires careful attention to detail. By far the most satisfactory method, is by oil sterilisation of the syringe and needle. The causal organism may be recovered from the urine or faeces, during the febrile period, but in 150 cases, only two of these diagnostic examinations were positive. Blood culture offers the greatest prospect of success.

Management

Although typhoid fever may be nursed safely in a general ward, it is desirable, in a military hospital, to isolate all cases. In a civilian general hospital, there is little movement of patients about the ward. In a military hospital, large numbers of men move about freely and assist in the routine work. Although they are all protected by T. A. B., it is wiser to remove a source of infection from convalescents.

As absolute rest is enforced, the enteric ward requires a larger staff than a general ward. Rest is the most important therapeutic measure in typhoid fever. The disastrous effect of moving a patient, is well shown in this series. Five of the six deaths occurred within a period of hours after transfer to the Isolation Ward. Cases in the second week, should be nursed where they lie. In the 47th B. G. H., transfer of cases, diagnosed after the first week, is discouraged. It is a dangerous procedure, which does nothing but harm.

The patients are given as full a low-residue diet as they can be persuaded to take. The diets are graded in four steps, the lowest containing 1,600 calories, of fluids only. This one is seldom used. Usually, a patient can be persuaded to take at least 2,800 calories; the two higher caloric diets are the ones that are used most frequently.

The diets are fairly flexible. When a man refuses one item, it is usually possible to substitute something of equivalent caloric value. Sugar is extremely valuable for supplementing a diet, one ounce giving 100 calories. It is easy to add several ounces to fruit drinks. Chocolate has a caloric value of 150 per ounce. With two ounces of choclate and a few ounces of sugar 600 to 800 calories extra, can be worked into any one of the diets.

When a patient finds that he cannot consume all his food, it is important to reduce the diet to the next stage. The diet used is the minimum. If it is not sufficient to satisfy, more can be added. Accuracy is impossible when a patient is leaving something with each feed, whereas it is possible to calculate the caloric value of extras, added to a basic diet. With extra sugar in fruit drinks, cream and occasional chocolate, the majority of my patients have taken 4,000 calories a day, throughout the illness. At the height of the fever, a few became resistant and unco-operative and great difficulty was encountered in giving sufficient calories. Nasal feeding was resorted to in these cases and, during the few most difficult day of the illness, an intake of at least 2,000 calories was given.

It is important to ensure an adequate fluid intake. If the daily output of urine is over 40 ounces, it can be assumed that the intake is sufficient. When the patient is incontinent, at least 100 ounces of fluid must be given.

Management of Symptoms

Diarrhoea is an uncommon symptom. It occurred in only eleven of my first 150 cases and cleared up in the course of a few days, with a reduction in the diet. 'Pea soup' stools are rare and were seen in three cases only. Constipation is much more frequent and, during the febrile period, is corrected with enemata.

Tympanites may be a very troublesome symptom. At first, my only way of dealing with it was by a rectal tube and reducing the milk and sugar to a starvation level. Later, the use of prostigmin was found to relieve the distension most effectively. The increased peristalsis does not appear to affect the ulcers and probably the prostigmin is less likely to precipitate haemorrhage or perforation, than the persistence of the distension, which only develops in severe cases. The does of prostigmin is ½—1 cc, repeated in an hour if necessary.

Delirium is an important symptom, which tests the ingenuity of one's prescribing. If the patient lies in low delirium with little restlessness, no sedative is necessary. A restless delirium is a much more serious condition and has a bad prognosis. Phenobarbitone, chloral and bromide, paraldehyde, may all be tried in large doses. If these are unsuccessful, one need have no hesitation in prescribing morphia in repeated doses, if necessary. An initial dose of ¼ grain, followed by repeated doses of 1/6 grain, at intervals varying from 4 to 8 hours, may be necessary, to induce quiet. With the use of prostigmin, the possibility of causing tympanites is no contra-indication to morphia. Delirium is not always associated with a high temperature and even when it is, sponging and "water-evaporation" from the skin, has little effect.

Now, a few words about perforation. The symptoms and signs of perforation of a typhoid ulcer, are by no means clearcut. The popular description of severe abdominal pain with collapse, may occur without perforation, and perforation may occur without the classical

symptoms. In the first group, there is no clear explanation. Severe intestinal colic is probably the reason. The symptoms and signs closely resemble those of perforation. When perforation does occur, the patient is already extremely ill and may be too ill to indicate that some serious change has occurred.

189. LT.-COL. PURI—"*Protection afforded by T. A. B. and Its Effect on the Widal Reaction*" **(P)**

I have been asked by Col. Schlesinger, to speak a few words on protection afforded by T. A. B. and its effect on the widal reaction. As regards the protection afforded by T. A. B. inoculation, I can do no better than to relate to you some outstanding facts concerning the incidence of enteric fever, and leave it to you to judge the efficacy or otherwise of this important prophylactic measure.

The incidence of enteric fever, as you all know, is still extremely high among the civilian population of India, and it is very doubtful if there has been any appreciable decrease in this incidence say during the last 50 years. The incidence of this disease used to be equally high among the military population, and, during the S. African War of 1899—1902, no less than 57,684 cases of anteric fever occurred amongst a total of 208,000 men engaged in that compaign. This, roughly, works out at the high rate of 280 per 1,000.

Prophylactic inoculation with T. A. B. had been more or less universally brought into force, prior to the world war of 1914—18, and only 31,011 cases of enteric fever occurred amongst the approximately 6 million British, Dominion and Indian troops engaged in the various theatres of operation. This approximately works out at 5 per 1,000 only.

The incidence of enteric fever in the Army in India, both among British and Indian troops, during the post-war period of 1919—1933, remained between 3 and 4 per thousand. At about that period, however, the quality of the T. A. B. vaccine, which hitherto had been prepared from the old classical strains of bacilli, was further improved by ensuring that only virulent organisms, which had fulfilled certain tests, were employed in its preparation. Since the introduction of this more potent vaccine, the incidence of enteric fever in the army has shown a still further decline, and the figure published for the year 1941 is under 1 per 1,000.

The superiority of this improved vaccine, was quite clearly demonstrated during the N. African compaign of the present war. The incidence of enteric fever among the Axis troops, who were still being protected with the old type of vaccine prepared from the classical strains of doubtful virulence, was much higher than that among the British and Indian troops, and, in fact, assumed almost epidemic proportions, among the large number of prisoners of war taken at El Alamain, Re-inoculation with the captured stocks of vaccine, failed to check the spread of the disease, but as soon as stocks of the British vaccine became available, the epidemic was quickly brought under control. On the other hand, although ideal conditions existed for the spread of enteric fever among the British and Indian prisoners of war in enemy hands, to the great surprise of the Axis medical authorities, very few cases of enteric fever were encountered. The sanitary conditions of these prisoners of war camps were so primitive and flies were so prevalent, that more than half the prisoners contracted dysentery. It cannot be denied that improved sanitation, control of water supplies and care of food, both in cantonments and in the field, have each contributed to lower the incidence of bowel infections, but, from the above-mentioned facts, the conclusion is irresistable, that this remarkable reduction in the incidence of enteric fever, has been mainly, if not entirely, achieved by the routine practice of prophylactic inoculation with the potent T. A. B. vaccine, employed by the army.

So much for the protection afforded by T. A. B. inoculation. Now, let us consider its effect on the Widal reaction, and this, naturally, has to be studied individually in respect of the three antigenic components of the typhoid bacillus namely, H antigen, produced by the flagellae; O or somatic antigen, produced by the body of the bacillus; and the Vi antigen, produced by the inherent factor, responsible for its virulence.

The H antigen is the least specific of the three and evokes a well marked immunological response in the inoculated individual, with the result that an agglutination titre of 1 in 500 and even higher, against an H emulsion, is met with quite frequently. Furthermore, this high titre has a tendency to persist for long periods and even exhibit a pronounced anamestic rise as the result of infections other than enteric group. The results of Widal test, performed with H emulsions, on previously inoculated individuals, are therefore so unreliable, that the use of these emulsions has long been discontinued in the Army laboratories.

The O or somatic antigen is more specific and evokes a less marked response in the inoculated individual, with the result that, following Felix's technique, as is the practice in the Army laboratories, an agglutinin titre of more than 1 in 160, is rarely met with and even tends to decline to lower levels after a period of 6 months. Widal tests carried out with O emulsions are, therefore, more reliable and, although a rising titre must be considered of a greater diagnostic significance, a single T. O. titre of 1 in 320 can be considered as quite suggestive, and a titre higher than that, as practically diagnostic of enteric fever. Unfortunately, however, every case of proved typhoid fever will not show such high titres; according to our present limitations of knowledge and the technique employed, not more than 70 per cent. of such proved cases show such titres, and 7-8 per cent. of non-enteric cases may also show these high titres. The exact evaluation of the Widal test in an inoculated individual is, therefore, a difficult problem. This was one of the subjects recently discussed at a conference of pathologists at Rawalpindi and each pathologist in N. W. Army has now under taken to maintain detailed records and critically analyse the data thus collected. In this, he would naturally need the full co-operation of the physicians and I need hardly mention that undoubtedly, all of you would readily give such help, because this problem interests you, just as much, if not more.

The Vi antigen, which determines the virulence of the typhoid bacillus, is highly specific and, as a rule, evokes little or no response in the inoculated individual and it is claimed that it is unusual to find an agglutinin titre of even 1 in 20, in persons other than those suffering from, or convalescing from enteric fever. But, Vi emulsions have a tendency to deteriorate rapidly on keeping, and perhaps the personal factor plays such an important part in reading the results, that very conflicting views have been expressed by many experienced pathologists. It has been advocated that more uniform, consistent and significant results are obtained, if emulsions made from live cultures of the bacilli are employed, in carrying out the test. That may be so, but how many pathologists would be willing to take the risk of playing with live bacilli, reputed for their high virulence?

In conclusion, one has to admit that the Widal test, performed on an inoculated individual, is only of limited help to the clinician. A negative test does not exclude a clinical diagnosis of enteric fever. You can hope to get some guidance in about 70 per cent. of your enteric fever cases and you should be prepared to be misled by this test in about 7 per cent. non-enteric cases. The question then arises, is it worth while carrying out so many tests and achieving so little? Despite such disappointing results, however, I am afraid I cannot suggest the complete abandonment of this time-honoured test, because further improvements in the technique etc., may increase its value.

190. LT.-COL. HAVILAND-MINCHIN—"*Diet in Typhoid*" **(D)**

In the absence of specific therapy, diet and rest remain the main things that we can do for typhoid cases. A high diet has been advocated in India since 1907, and has been used in the U. S. A. since 1917. Many British physicians still keep to the classical low diet, which is still recommended in the text-books.

A study was made in 450 cases, which were divided into three groups of 150, the first on high diet, the second on high diet and a daily dose of castor oil whenever the bowels did not open, and the third on the classical low diet. The mortality was identical in each group. All the cases of perforation occurred in the starvation group. Other bowel complications, such as haemorrhage and distension, occurred in each group in similar amounts. The period of convalescence in the group to which high diet had been given, was about half that in the other group, which had been starved. In the absence of specific therapy, it is considered that a diet as high as the patient will take, should always be given.

191. LT.-COL. KARAMCHANDANI—"*Observations on Typhoid*" **(P)**

Typhoid Fever—Its Unusual Form and Treatment—

Typhoid fever is recognised to be a continuous type of fever and, if the temperature touches normal during its course, it is said not to be typhoid. Whereas this is true, in that the intermittent, the quartan, the trench fever types of temperature, i.e. touching normal regularly every day, third day or less frequently, are not typhoid fever; cases do occur when the temperature touches normal on more than one occasion during the course of the fever. Such is one aspect which I wish to present before this meeting.

On 3rd February, 1937, a Hindu male arrived in a military station and, the same day, he was inoculated with T. A. B. 1 cc.

On 16th February, 1937, he reported sick with temperature of 99°F., pulse 88 Res. 22. Blood was negative for M. P., while his tongue was clean. He stated that this was the third day of his fever.

On 17th February, 1937, his T. rose to 102° F. P. 100, R. 26. Bowels moved for the first time since reporting sick.

On 19th February, 1937, he was admitted into hospital. His T. was 102·2° F. and pul. 80. T. L. C. 6800; D. LC. Poly 60%, Lymphocytes 35% large mono 3% and eosino 2%. Nothing abnormal was detected in the system.

On 21st February, 1937 (6th day of illness), T. became normal at noon, but rose to 100° F. in the evening. Widal showed a titre of TO 1/25 , rest nil.

On 22nd February, 1937 (7th day), T. became normal at noon, but rose to 101·4° F. in the evening. B. typhosus of Eberth isolated from blood.

On 26th February, 1937 (11th day), at 2 a.m. passed a stool containing six ounces of altered blood. T. became normal at noon, but rose to 99.4° F. in the evening.

On 8th March, 1937 (22nd day), T. normal.

On 9th March, 1937, T. normal up to noon, but rose to 99·4° F. in the evening.

After this, he ran a continuous temperature for 29 days, raising up to 104° F. on two occasions. Temperature came down by lysis on 7th April.

Total period of fever was 51 days. No complications. Patient was discharged on 28th April.

Widal reaction was as follows:

6th day, 21st February, 1937, TO 1/25 rest nil.
7th day, 22nd February, 1937, B. typhosus isolated
10th day, 25th February, 1937, TO 1/250, A. & B. 1/125.
15th day 1st March, 1937 TO 1/250, A. 1/125, B. 1/50.
20th day, 6th March, 1937 TO 1/250, A. & B. 1/35
35th day, 21st March, 1937 TO 1/175, A. 1/35, B. 1/25, T. 1/400.

Points for Discussion

(1) Course of fever was very much unlike typhoid. It was more a bacteriological than clinical diagnosis.

(2) Average incubation period for typhoid fever is 10—14 days. On 3rd February, 1937, the patient was inoculated, on 13·2 first signs of illness appeared. This means that inoculation during the incubation period did not stop the disease.

(3) There was a very protracted course of illness, in spite of priphylactic inoculation. Did the inoculation during the incubation period contribute towards the illness?

Note: This case was published by the author in 1938 (Karamchandani & Miller, *Ind. Med. Gazette*, 73, 4, p. 220).

Another point, on which I wish to comment, is the treatment. During my tropical practice, I have employed empty bowel treatment, in suitable cases, with excellent results. Two items are most important:

Firstly, *diet*, which consists of whey, as long as the temperature has not settled down. Two limes—four teaspoons of juice are added to a pint of milk. Pints four of milk will make pints three of whey. Warm the milk, add the lime juice, bring to the boil, allow to stand until clot forms, then break up the curds and strain. In addition to the above, are given pure vegetable soup, chocolate to suck, fresh lime juice adlib. and glucose water. After the temperature has been normal for at least three days, milk is given alternately with whey. Secondly, medical treatment, which consists of calomel gr. 2 and soda bicarb. gr. 5, to start with, followed by castor oil next morning. Subsequently, calomel gr. 1/6, with soda bicarb. gr. 2 six-hourly, and castor oil, ounce half to one, every morning, depending upon the number of evacuations in 24 hours. These should be three to four, loose, watery, dark and free from curds. Bismuth is added to the calomel, if stools be more frequent. Advantages noted were: Soft, moist, clean tongue; flaccid soft abdomen; no bowel complications and no smell.

In India, certainly, a constipated state of the bowel is the rule. If we take the two main and most feared complications, i.e. haemorrhage and perforation, and consider how constipation and undigested food must, perforce, increase danger of these complications, by the purely mechanical effect of a foreign body rubbing on the necrosed intestine wall, any treatment which attempts to ensure an empty bowel, must be summum bonum of treatment. Again, calomel increases the hepatic secretion, thereby ensuring a constant flow of at least aseptic, if not antiseptic fliud. There is, then, asepsis and rested bowel. Escatin is given when the condition of the pulse warrants.

Whereas it is true that there has been an inexplicable tendency to withhold food of an adequate caloric value from febrile patients, it is also true that pressing food against inclination, has no advantage, i.e. appetite being the best expression of the need for food. There is diminished activity of the alimentary system, as a result of toxins, while high temperature may actually damage the glandular tissues. On the other hand, thirst is an expression of the physlological needs of the body. Desire for water and fruit is but natural response to the needs of the body for fluids, sugar, and vegetable acids, which, in the body, will supply carbonates, assist the balance of water and salt and mitigate acidosis during pyrexia. There is no doubt that the balance of water and salt is disturbed, because sod. chloride is diminished in urine, urine is concentrated, while acidosis is produced by imperfect oxidation of fats. If, therefore, adjustments of variations, on which life depends, be expressed by patients' likes and dislikes (a reflection of expression of physiological needs), is it right to force food, simply because caloric needs must be satisfied? Besides, do we not know that some forms of proteins and even excess of salts, can be factors in the production of pyrexia? If we find fur and debris covering the organs of taste in the mouth, may not the same metabolic changes be affecting the gastric organs, and depressing the digestion? Would not rinsing out be more helpful than gorging? When we know that high caloric diets produce digestive upsets and metabolism, even in amenable patients, is it then suitable for every case of severe attacks with profound toxaemia and defective digestive secretions?

DISCUSSIONS

192. LT.-COL. S. N. HAYES enquired whether British T. A. B. was similar to Indian T. A. B.; the former produced no reaction, but the latter produced severe reaction.

193. LT.-COL. DAUKES reminded the Meeting that the R. A. M. C. type of T. A. B. was manufactured from the original Rawlings strain of Bact. typhosum, isolated in the South African war. In about 1933, doubts were cast on its efficacy and it was thought to have become avirulent, and so the virulency was enhanced by mouse passage, until it took (he thought) one hundred million organisms to kill a mouse in 48 hours. He understood that the Indian T. A. B. was manufactured from other strains.

194. LT.-COL. PASRICHA said that British and Indian T. A. B. vaccine were of the same strain; Kasauli vaccine was all that one could desire. He related an incident in the U. K., when about 200 British troops, who had been recently inoculated against T. A. B., developed typhoid after bathing. He laid stress on the cultural examination of blood, stool and urine, for the diagnosis of typhoid fevers and appealed for the more liberal feeding of patients suffering from any of the enteric fevers. In the past, there had been a tendency to starve in these fevers; the patient needed food, he strongly advocated, in the fight against his infection.

195. BRIGADIER McALPINE: Whenever I have the opportunity, I always ask, when visiting hospitals, if I may see recent cases of typhoid, in order to find out whether any case has tender feet. 'Tenderfeet' is an early sign of nutritional polyneuritis, and always means undernourishment.

196. MAJOR-GENERAL MARTIN said that 'whey' had no nourishment and only contained sugar; its use had been abandoned for many years. He suggested the use of curd instead, which would be better digested.

197. DR. PIRZADA: The speaker (Lt.-Col. Puri), has rightly expressed his doubts with regard to the utility of the Widal test as a method of diagnosis, on account of its many fallacies. If he were more frank, he would probably recommend its complete elimination from the laboratory. From the civil practitioner's point of view, it is a useless test. By the time it begins to give doubtful results, the diagnosis has been made on clinical grounds, or the physician changed.

There is some difference in the view point of the army and the civil medical officer. The army medical officer deals with an inoculated population, which, it has been pointed out, shows a high 'H' agglutinin titre, even normally. There is ample justification for doing away with 'H' agglutination in the army. The civilian medical officer deals largely with an uninoculated population, which, on account of repeated exposures to infection, due to unsatisfactory sanitation, or subclinical infection, shows a much higher 'normal' agglutinin titre, as compared with the population in Great Britain. In such a population, 'O' agglutinins are met with more frequently and in a higher titre, than 'H' agglutinins. As an indication

of active infection, therefore, 'H' agglutination deserves greater respect than it has hitherto received.

The use of prostigmin in the treatment of meteorism in typhoid fever, appears to me to be a dangerous therapeutic measure. The violent peristalsis set up, is likely to dislodge sloughs from ulcers and precipitate haemorrhage and even perforation.

The question of dietetics in typhoid fever, is a very difficult one. Two extreme views have been expressed. The drawback of a high diet is, that the patient who is in the greatest need of it, is also the patient who is probably highly toxic and whose powers of assimilation are poor. Over-feeding in such a case may do more harm than good. The patient who is not too toxic, could easily take a relatively generous diet, but he is also the patient who would do equally well on a lower diet, although he need not be starved on that account. Dietetics, therefore, is largely an individual problem and no hard and fast rules can be laid down. The truth lies midway between the two extreme views.

198. COL. SCHELESINGER said that the use of whey, which contained only ten calories per one ounce, had been given up in pediatrics. He suggested culture from the 'spots' for the diagnosis of typhoid fevers.

199. MAJOR LYN GREENING: The dose of a vaccine should be such, as to produce optimal results with the maximal dose which can be tolerated comfortably by the patient. The India-produced T. A. B., at present in vogue, produces very severe reactions, when given in the officially recommended doses. In my opinion, an initial dose of 0.3 cc and second dose of 0·75cc, with annual doses of 0·25cc, for a man weighing 9 stones, and increase or decrease of this dose, in proportion to body weight, has much to recommend it, in view of the poor physique of many recruits received nowadays.

200. CAPT. FREEDMAN: Medical Officers may experience some difficulty in estimating the caloric values of the diets, owing to the lack of adequate food analysis tables. Very good tables have now been published in 'Health Bulletin No. 23—The Nutritive Value of Indian Foods and the Planning of Satisfactory Diets'. Copies (price 2 annas) can be obtained through most of the larger booksellers, or from 'The Manager of Publications, G. of I., New Delhi '.

201. LT.-COL. KELSALL protested against Lt.-Col. Haviland-Minchin's rather astonishing attack on British Medicine, in regard to diet in typhoid, and assured him that, in all hospitals in the U. K. wth which the speaker was acquainted, the necessity for diets of adequate caloric value and vitamin value, for any ill patients, including those with typhoid, had been recognised and taught for many years. He further stated that, until he came to India, he did not realize that there was still any tendency to argue about this, and that the only doctors whom he had ever met, who still advocated semi-starvation for patients with typhoid, were Indian doctors at the A. M. T. C. at Poona.

202. DR. MEHTA: What is the opinion regarding diet in typhoid during convalescence? I had the privilege of working at the Indian Military Hospital, Jhelum, for a few months, as M. O. in charge of the Malnutrition Ward. Two cases of enteric were sent to me during their convalescence. I put them on high caloric diet, gradually graded up from 3500 calories to about 6000 calories, with the following results; patients were weighed with the same clothes and at the same time, i.e. about 10.30 a.m.

Case 1. *Sepoy S. N., age* 24, *service* 17 *months.*—

3rd May, 1943—7st., 2 lb. 10th May, 1943—7st., 3 lb. 24th May, 1943—7st., 6½lb. 2nd June, 1934—7st., 12lb There was a net gain of 10 lb. in weight in one month, despite the fact that he had malaria (B.T.) fever from 8th May—11th May, 1943. His red blood cells increased from 2.2 million on 29th April, 1943, to 3.2 million on 29th May 1943.

Case 2. *Sepoy, J. S.*—

10th March, 1943—10 st. , 4 lb. 5th April, 1943—11 st., 3 lb. 19th April, 1943—11st., 5½ lb.

There was a gain in weight of about one stone in a month. I think high caloric diet does give good results

203. LT.-COL. KARAMCHANDANI: With reference to the discussions, may I be permitted to answer criticisms and to point out that the diet I have been giving, is not starvation diet? One ounce of whey is equal to 10 calories and 100 ounces mean 1000 C. Then, 200 calories are obtained from 1 oz. of chocolate, 2 oz. of chocolate mean 400 C. 150 calories are yielded by 1 oz. of glucose and 2 oz. of glucose added to sweeten whey, improves the taste and yields 300 C. Thus, there are 1700 C. and the diet is rich in vitamin C, due to lemon juice ad lib. Average Indian basic metabolic needs are adequately met, and this is by no means a starvation diet.

The next question is, the advantage gained from giving meat, fish, etc., to an enteric fever patient. One speaker said that he encountered meteorism, which he treated with prostigmin. In my diet, no serious meteorism was ever encountered. Secondly, in typhoid, there is depression of adrenalin, wherefore the slow pulse. Manson and others, on this account, recommend escatin, to stimulate sympathetic mechanism. The rationale of giving a stimulant of parasympathetic, where there is already compensatory parasympathicotonia, is worth mature consideration. Another speaker said, "If perforation occurs, we shall sew it up and the surgeon should be ready to operate within half an hour." Is it not better to avoid perforation, rather than give perforation and then sew it up. I am speaking from a wide point of view In a military hospital, this may be feasible, but is it workable in civil practice? Besides, if perforation occurs in civil practice, as a result of gorging, the repercussions become extreme. Finally, Lt.-Col. Haviland-Minchin said that his series of cases had been ready for work in three months. If that be so, that line of treatment has no advantage, for my cases became fit after two months. The pendulum has swung right to the opposite side, i.e. over-feeding, as compared to past starvation; via media is best, and that is what my experience has been, since I commenced this line of treatment in 1928. Success should be judged by results. Some day, the pendulum will be steady and my view point redeemed.

204. LT.-COL. S. N. HAYES, in closing the discussions, emphasized that, in the treatment of the enteric group, collaboration between the Surgeon, Physician and Pathologist was essential. As many cases died during the night, said to be from perforation, it was necessary to see that careful watch during the night by the ward staff—particularly in Indian hospitals—be maintained, and that the time to prepare for a surgical emergency, was before the emergency arose.

10th FEBRUARY, 1944—AFTERNOON SESSION

The Meeting closed at 13.30 hours, and was resumed at 14,30 hours, with **PROF. GOYLE** in the Chair.

DRUGS

205. PROF. KHEM SINGH GREWAL—"*Indigenous Drugs*" **(P)**

Mr. President and Gentlemen, I have selected the subject of Indigenous Drugs, for today's talk, but I am fully conscious of the fact that my claim to speak on the subject, before such a distinguished gathering of medical experts of the Army, is a weak one. I wish that you had some distinguished research worker, like Colonel Sir Ram Nath Chopra, to address you. Though my associations with pharmacology are of long duration, and I can claim the privilege of learning pharmacology from two of the outstanding pharmacologists—the late Professor W. E. Dixon and Colonel Sir R. N. Chopra, my acquaintance with indigenous drugs is not very deep. I have carried out researches on the subject during the last six years, but, unfortunately, for many reasons, this has not been a favourable time in India for research workers in the Civil. In spite of the difficulties, some twenty drugs have been partially investigated, and I shall make a few observations, based on the knowledge gained through the work.

Owing to the vastness of the country, variety of climate and soil, the natural vegetable resources of India are enormous. It was but natural, that men living amidst such profuse vegetation, should have learned both curative and prophylactic use, in diseases, of vegetable products. Drugs have been used from very great antiquity in India, as is evident from the old Sanskrit literature still extant. Some of the well known books on Surgery and Medicine (Sushruta and Charak), have many chapters on drug treatment. They are believed, by Western authorities, to have been written about 2500 years before Christ. The knowledge at that time seems to have been well advanced. During Buddhist times, there were medicinal gardens in India. King Asoka took great personal interest in these. The value of genuine medicines in treatment, was a recognised fact. A quotation from Kunja Lal's English translation of Sushruta, may not be iut of place: "The physician, the patient, the medicine and the attendants, are the four essential factors of a course of medical treatment. Even a dangerous disease is readily cured, or it may be expected to run a speedy course, in the event of the preceding four factors being respectively found to be—physician qualified, patient self-

controlled, medicine genuine and attendant intelligently watchful." It is also mentioned that the proper medicine is that which consists of drugs, grown in countries most congenial to their growth.

It is a very common present-day popular belief that there are a great many indigenous medicines which are extremely efficacious. The belief is not only prevalent among the uneducated, but is also strongly shared by educated laymen. The belief is so strong, that, in spite of competent scientific medical aid in great cities, a great many Vaids and Hakims are doing lucrative medical practice. Even in Lahore, there are Vaids and Hakims, who are as popular as some of the best scientific medical practitioners, a fact that cannot simply be ignored. Because of the past claims and the present status of indigenous medicines, attempts have been made to examine these claims scientifically, but, so far, very little has been done in India, systematically to explore this field. The researches on indigenous drugs, have been done at half a dozen laboratories. There are only two laboratories in India, one at the School of Tropical Medicine and the other at Madras, where this work is being done as part of the normal functions of the laboratories, and even at these laboratories, not more than half a dozen workers are engaged on this subject. At other laboratories, the teachers of pharmacology or chemistry, are struggling hard to keep the flame of research on the subject, flickering. The outstanding contribution to this subject, has been that of Col. Sir R. N. Chopra, from the laboratory of the School of Tropical Medicine, where suitable conditions existed. The output of research, naturally, could not be very great from other centres. The research workers in this field, are greatly handicapped, because the problems of indigenous drugs are difficult and intricate, and they require the co-operation of the finest analytical chemists, pharmacologists, bacteriologists and clinicians, and that is not always forthcoming. Further, every research worker is expected to have introduced a drug in the therapeutics which should be superior to the pharmacopoeal drugs. This is a task, the burden of which may crush the scanty attempts at research in this field, and I beg you not to judge the researches on the subject from this point of view alone.

Some twenty drugs have been investigated by us, and, to give some insight into the difficulties, I shall take a few drugs and briefly tell you what has been done on them.

Nelumbium Speciosum

The seeds of Nelumbium Speciora (Kawal Douda), after the removal of the embryo and after parching, are eaten with relish by children and young folks. The embryo is very bitter. According to Kirtikar and Basu, the whole seed is used "to check vomiting and are given to children as diuretics and refrigerants. They form a cooling medicine for cutaneous diseases and leprosy, and are considered as an antidote for poisons." Our investigation showed that the embryos contained alkaloids. They were separated into phenolic and non-phenolic portions. The alkaloids were isolated in as pure a state as possible under laboratory conditions. The alkaloid was found to be pharmacologically active. M. L. D. 50 for frog was 4 mgm. per 20 gm.; M. L. D. 50 for rabbit was 8 mgm.; and M. L. D. dog I. V. was 20 mgm. Before proceeding any further, it was necessary to determine the nature of the alkaloid. After such work as was possible in Lahore, one gramme of the alkaloid was sent to the late Professor G. Barger, at Edinburgh. He evinced great interest in the problem, as, on theoretical considerations, he expected the plants of nymphacace order to show alkaloids of the iso-quinaline group. Professor Barger had been attempting to isolate alkaloid from the rhizome of N. Alba and Luteum, but was unable to crystallize it. The alkaloid sent by us, therefore, greatly interested him. He sent the alkaloid to Dr. H. Rath of Heidelberg, for micro-analysis. From the study of the result, it became apparent that the alkaloid was not quite pure, and required further re-crystallization. From directions sent by Professor Barger, the alkaloid was re-crystallized and two gms. of it were sent to him again, but never reached him, as we were informed by him in September, 1937. Another gm. was crystallized and sent to his Edinburgh address. We learned later that Professor Barger migrated to Glasgow, and died suddenly while on holiday in Europe. We lost 3 gms. of pure alkaloid and got nothing out of it; meanwhile, war started and the problem has got to be shelved for the time being.

Another drug that has been investigated, is Cassia Absus (Chaksu). The chemistry of it has been studied by Siddiqui, who isolated an alkaloid which he named Chaksine. The drug is a common household remedy for purulent infections of the eye, for which it is highly

esteemed, and it is also given to women after child-birth, and is believed to prevent complications such as fever. The pharmacology of it has been studied extensively by us. It has been found to cause stimulation of the cerebral nervous system in small doses; peripherally, it causes partial atropine action. It stimulates, directly, the cardiac muscle and the non-striated muscles. Most curiously, its toxicity is much more in winter than in summer. This mechanism has been studied, and it has been found that it causes dilation of the skin vessels; this results in great loss of heat in winter and the animal dies of shock. To examine its claims in therapeutics, its action on micro-organisms was studied. In 1 in 4000 dilution, chaksine sulphate inhibits the growth of staphylococci and streptococci, but has no action, even in I in 400 dilution, on B. typhosus and B. coli. The effect on experimentally-produced ulceration of the cornea in rabbits, has been studied without conclusive results. The main difficulties in these experiments have been the non-availability of strains of staphylococci and streptococci, of proper virulence and we have not yet succeeded in getting the strain. The problem again found an obstruction. The answer to the therapeutic value, can only be given by laboratory experiments, there is no other way. Clinical trial may be suggested, but, for the results to be convincing, it requires certain conditions; are these conditions available at the Mayo Hospital? If not, can it be expected that these will be forthcoming? I shall not attempt to answer these questions. Clinical trials, under unsatisfactory conditions have already been made in Europe. In an epidemic of purulent ophthalmia, which visited Brussels in 1882, Dr. Harbaur gave a trial to the treatment with Cassia Absus and the results were, on the whole, confirmatory of its alleged efficacy, but the drug did not find favour after this trial in scientific medicine. As for its claim to prevent complications in pregnancy, that also requires laboratory experiments, and it is the co-operation of the bacteriologist that can solve the problem. There is one interesting thing that is noticeable in the method of administration of the drug in the Punjab. The powdered drug is mixed with a cooked preparation of sugar, ghee and atta, and the patient takes it twice a day. In evaluation of the various sulphonamides, the most satisfactory method for keeping a concentration of the drug constant in the blood of a mouse, is to mix the drug with food, and this method is used by all experimenters on sulphonamide. The similarity of the mode of administration of drugs, used for similar purposes, is very interesting.

Rattanjot (Onosma-echoides) is said to grow abundantly in Kangra. It was used for dyeing silk and wool, before the European dyes ousted it completely. It is still being used to give a pleasant colour to cooked meat and vegetables. It is used to hide patches of Leucoderma, by colouring them. It has been used in medicine, for a great many diseases. The root bark contains an oil-soluble dye, which can be separated into two fractions, benzene soluble and alcohol soluble. The benzene soluble is 10 per cent. in weight and has been identified by us as amino hydroyanthraquinone dye. It resembles, in colour reaction and spectroscopically, Alizarin Cyanin G. which is considered a most valuable dye in Germany. So far, our researches have been easy, but when we wanted to make use of it, we came up against a difficult situation. We studied its dyeing properties on wool, silk and cotton; it gave fast colours, with or without mordants in acid baths. A sample of the dye was sent to Dr. Sir S. S. Bhatnagar, he could not suggest any use for it, and he referred me to Dr. Venkatraman, of the department, of chemical technology of Bombay. He informed me " that the consumption of this in the country, is likely to be small ", and could not give any information as to its price in the market. The Industrial Chemist to the Punjab Government could make no use of it. I got in touch with the military authorities, to find out if there could be any use for it in chemical warfare. I was told that there might very likely be a use for it and was asked to supply 5 lb. of the dye, and also an approximate estimate for maximum product. For 5 lb. of the dye. I want 60 lb. of the drug, which cost Rs.60 and, for its extraction, requires 100 lb. benzene, which will cost Rs.200, though the loss of it is only 10 per cent., still, I have to buy the benzene. The approximate cost of the dye will be Rs.100 per lb. There is no money given to the Professor of Pharmacology for such purposes and the permission of the I. G. C. H. Punjab, through the Principal, has got to be obtained for it. My total grant from I. R. F. Association is Rs.2,200, including the pay of the chemist and a laboratory attendant. As for the estimate, here is no agency through which I can get it.

Lana Sueda Fructosa : It is a perennial plant, which grows in saline soils. It is used for feeding camels, or for production of crude alkali carbonates for washing purposes. It is used as a household remedy for pains in joints, and medical men have testified to its usefulness.

We collected the plant, dried it in shade and analysed it. On analysis, it showed to yield the following substances :—Soluble tartrates, which were isolated as Pot. Hydrogen tartrates; the percentage was 2 per cent. Carbonyl diurea, 1·5 per cent. this substance has been synthasised in Germany in 1934, and a derivative of it has been patented; we could find no information, from available literature, as to the use of this substance. Two purine compounds in small quantities; a glucoside which is related to emodines; the crude drug has mild laxative action; the study of pharmacology of carbonyl diurea has not been started; we are still attempting to find it's literature, and unless we can be sure that no literature exists, the work is held up.

Hydrocotyla Asiatica : This plant is called Brahmi Booti, and is extensively used in the Punjab as a tonic, and especially by students, to strengthen their memories. A good many university students believe in its efficacy. It is firmly believed to retard the deterioration in memory, due to advancing age. It has also been used in the treatment of leprosy, Dr. Hunter, of Madras Leper Hospital, found it most useful in ameliorating the symptoms and improving the general health. The drug has been studied by various workers, at the School of Tropical Medicine and also at the Indian Institute of Research, Bangalore. Walle and Katti have reported, in 1936, the presence of a green essential oil, fatty acid, esters, sittosterol and sugar. Our investigations showed it to contain, in addition, Phytosterolin glucoside and a reducing principle, in large quantity, which gave reactions of ascorbic acid; the isolation of it was done according to the method of Schat Gyorgi, for the isolation of ascorbic acid, but, owing to the unavailability and the cost of the solvent-methyl alcohol and acetone, the identity could not completely established. The use of the drug in a small quantity, for a prolonged period, raises many important issues. Physiological researches, vitamin investigation and chemotherapeutic investigation, researches in internal secretion and enzymic actions, have greatly advanced the conception of the action of drugs. The role of the automatic nervous system in recovery from fatigue and recovery from disease, is still not clear. It is conceivable that the automatic nervous system plays an important part in this mechanism. The drug given in small doses, for a prolonged period, may produce decided action through the automatic nervous system.

I have, from my limited experience, tried to show that there is scope for first-rate research in indigenous drugs, and that the problem is so extensive, that many research centres are necessary, and it is too early to expect any practical discoveries. If research is promoted adequately, the time will come when the research in this field will bear fruit.

206. COL. TAYLOR—" *Therapeutics in India* " **(D)**

(Resumè is not available. *Ed.*)

DISCUSSIONS

207. MAJOR LYN GREENING recommended that, in view of the apparent relationship of Rattanjot dye Alizarin, the dye be tried as a bacteriological stain.

208. LT.-COL. THOMAS (*Chemical Examiner, Punjab*) : I can only discuss indigenous drugs from the toxicological point. It is an established fact, that drugs which prove most efficacious when taken as medicine, are also highly poisonous when taken in toxic doses. It would, therefore, seem reasonable to expect many cases of accidental poisoning in India, especially in the villages, by unknown indigenous drugs. This, however, is not the case. During my twenty years experience as a Chemical Examiner, I have come across very few such drugs. I may however mention one rather interesting plant called Illicium Religiosum, sometimes called the bastard anisi, which is the Japanese sacred anisi tree—its fruit in India is called Badyan Khatai. The poison contained in this plant, produces all the typical symptoms of strychnine poisoning, except that unconsciousness is present. Whether it has any properties as a medicine, which are superior to strychnine, remains to be investigated.

India is a land of villages, and ninety per cent. of our population live in villages. The Indian peasant is usually poor and cannot afford expensive western medicines, with the result that he must rely on crude drugs, which are cheap and readily available. Every encouragement, therefore, should be given to the improvement of such crude drugs, until the economic conditions in rural India can be improved.

209. LT.-COL. BOSE stated that, recently in Abbottabad, a number of patients had been admitted into the hospital, for chronic ulcers. It was suspected that these ulcers were being

produced deliberately, by rubbing on some irritants. An indigenous plant, reputed to cause local irritation, was obtained, and was lightly rubbed over the skin of the forearm of a volunteer nursing sepoy. Within a short time, the skin showed erythyma; after about four hours, the part became markedly swollen, angry-looking and exudate began to appear. This was followed by vesicle formation and ulceration, next day. It took over a month for the ulcer to heal. Any information regarding the nature of this plant would be welcomed. Subadar-Major Bhatia, I. A. M. C., conducted the experiment.

210. LT.-COL. THOMAS, in reply to Lt.-Col. Bose, pointed out that such cases had been referred to the Chemical Examiner's department, and that the drug usually found, was ordinary Bhilawa or the Indian marking nut (anacardiaceae).

211. LT.-COL. PASRICHA: As one who has absorbed something of the subject, because of being housed in an institute in which a large volume of modern work on indigenous drugs has been carried out, and as one who is keenly interested in the history of medicine in India, I protest against the use of drugs, just because they are indigenous. Disease in India is not different to disease elsewhere, and, in its treatment, drugs of proven efficacy must be used. True, for minor maladies, many of the "grandmother's remedies" suffice, but who would advicate a drug—' X ' or ' Y ' for the treatment of malaria or pneumonia, or a mixture of ' pearls ' for dysentery or cholera, when we have proven therapeutic agents? Why search back and revive a host of drugs, already confined to the limbo of forgotten memories? Let those who are interested, excavate into the past, but we shall not allow emotion or sentiment to rule our judgment; we shall only accept those drugs which are of proven efficacy, and not hanker after the glories of ancient medicines. I would rather that we resolved to further the progress of manufacture of drugs of standard efficiency in India, and call the work by a progressive and a more inclusive name—the drugs in India. Modern therapeutics is shedding the old ritual, the alchemist; why must we perpetuate what science, the truth, has discarded in other countries?

Regarding the teaching of medical students, a previous speaker has remarked that the Indian student is poor, " he learns by heart and can repeat a whole text-book like a parrot". On behalf of the students, I protest against this. The fault is not his, but it is the fault of his teacher, who, far too often, has not taken sufficient pains to master his subject. If the teacher dishes up a miserable hash from a text-book, how do you expect the student to respond? Like his teacher, a parrot. As a teacher of some years standing, I have grown to have a very high regard for the receptive capabilities of the student mind. I can assure you, that if the teachers take trouble, the results are very satisfactory.

212. CAPT. LUTHRA said that synonimous drugs could be found in India, and that the civil surgeons in the U. P. and in Bihar were using those drugs, at a much lower cost. Dr. Kanya Lal Bose's book contained very valuable information on the point.

213. DR. MANMOHAN SINGH: The main thing to consider, when talking of indigenous drugs and their importance, is the poverty of India, as was stressed by Lt.-Col. Thomas also. It may be that indigenous drugs are not as good as some of the imported ones, but if a thing is not available to an average person, of what use is it to him? The imported medicines are so expensive, that an average Indian can not afford to buy them. In such a case, something is surely better than nothing, and, although an indigenous drug should not be used as a substitute for an imported drug, as was remarked by Lt.-Col. Pasricha, certainly it can be the only alternative, if the imported drug is beyond one's capacity of purchase. If my gardener suffers from dysentery, he may, first of all, not be able to afford to pay for a conveyance to go to the nearest dispensary, if he manages to do that, if the medicine is not given free to him, but he is given a prescription, surely, with his meagre income, he cannot purchase the medicine from a chemist's shop. So, the only alternative for him, is to buy a pice-worth of ' Ispaghula ' from a grocer and use that. It may not cure him of the dysentery, in the true sense, but it relieves him of the symptoms, to a great extent. Further, quinine is not now available, even by purchase, and, under the circumstances, if one gets malarial fever, one has to look to some local remedy, in the form of an indigenous drug, as the only alternative, and some of these drugs have been effective, though they may not be substitutes for quinine.

RADIOLOGY

214. DR. MOHD. YAKUB KHAN—" *Role of Radiology in Heart Disease* **" (P)**

The examination of the cardio-vascular system, by means of the X-rays, is but one among several methods of study of the circulation. Certain aberrations from the normal, in the C. V.

system, can be demonstrated by radiological methods only; a certain number are better diagnosed by X-rays than by clinical methods; while many changes, especially the early ones, can not be elicited except by clinical and electro-cardiographic methods.

The radiological examination of the heart, is likely to help on the following points:—

A. Size and shape of the heart and its different chambers.
B. Condition of the great vessels arising from the heart.
C. Study of the pulmonary circulation.
D. Study of the certain congenital abnormalities of the heart.
E. Diagnosis of certain pathological conditions of pericardium.

By X-rays, we not only study the static conditions of the heart, but also its dynamic activity, i.e. pulsation of the heart and blood vessels, seen on the X-ray screen.

A. *Size and Shape of the Heart*

I think there is no difference of opinion on the point that radiological method, with all its limitations, is the best means of ascertaining the size of the heart. An ordinary fluoroscopic examination gives a fair idea of its size, but, for more accurate determination, either an orthodiagraphic tracing or a telecardiogram, taken from at least two meters (about 7 feet), is essential.

Measurement in the Anterior View: The most commonly used and accepted standard for measurement of the size of the heart, is the cardio-thoracic ratio, i.e. transverse diameter of the heart, compared with the inner diameter of the thoracic cage. Transverse diameter is the sum of the maximum perpendicular distances of the heart shadow, to the right and left of the middle line. If this ration is 1 to 2, or, at the most, 1 to 1·9, the heart is within normal limits. In more, the heart is taken to be enlarged, except, probably, in the case of infants. The converse is not always true; the heart may be enlarged, but still within normal limits. This will be easily appreciated, if we consider the various factors which cause the variation in the shape and size of the heart. The following are the main factors:—

(1) Age: Infant's heart has relatively bigger T. D. than adult's, due to various reasons which I need not discuss. It also shows appreciable variation with respiratory movements, at the end of prolonged expiration, for example as the result of crying, the lungs are very much deflated and the heart shadow appears very big, and a mistaken diagnosis of cardiac enlargement may be made. Incidentally, superior vena-cava shadow is very prominent in infants and has been mistaken for enlarged thymus. After the age of 7 to 10 years, the heart takes one of the adult types.

(2) Body-Build: In adults, the most important factor is the body-build and width of chest. Thin persons, with long narrow chests, have a small, narrow, vertical heart, and stout and sthenic individuals, with wide chests, have transverse type of heart. In the majority of normal individuals, it is oblique in type.

(3) Position of the Diaphragm: This affects the shape and size, materially; during inspiration, when diaphragm is lower in position, the heart becomes more vertical, and T. D. is decreased. On the other hand, T. D. increases during expiration. For this reason, there should be a standard phase of respiration to take heart pictures, but, unfortunately, different authorities advise different phases as being the best. A picture taken at the end of inspiration has certain advantages. For one thing, the base of the heart is more clearly seen with the diaphragm fully descended, and, secondly, since the the size of, the heart is diminished, the error is towards normality; and, thirdly, the patient can hold this breath for a longer time, during this phase. All conditions which raise the diaphragm, for example pregnancy, ascites, etc., make the T. D. bigger, and the conditions which depress the diaphragm, for example emphysema, etc., make it smaller.

(4) Deformity of Spine and Thorax: For example, Scoliosis; it may produce a profound change in the shape.

The Left Oblique View to Determine the Size of the Heart: It is claimed, and verified by autopsy, that measurement in this view gives more accurate size of the heart, than any other view. For this purpose, the patient is screened and the angle of rotation, which gives the smallest size of the heart, is determined, and then a skiagram is taken. This angle of rotation is generally 45 degrees. T. D. of the heart in this view (which is actually the depth of the heart), is normally half the measurement of the chest, from

the front to the posterior articulations of the ribs with the spine. If more than half, the heart is enlarged. This view also gives a fair idea of the relative size of the right and left ventricles.

The Shape of the Heart : depends upon the relative size and position of the different chambers of the heart. It is on this account that certain valvular diseases can easily be diagnosed from characteristic X-ray picture—the so-called mitral and aortic configuration.

Mitral Configuration :—consist of a straight or convexally prominent middle portion of the left cardiac border and poorly visible aortic knob. This is typically seen in mitral disease, especially stenosis, but it may be seen in a certain number of young normal individuals, in thyrotoxivosis, in right-sided scoliosis, and in certain congenital abnormalities. So it does not necessarily mean valvular disease. Similarly—Aortic Configuration : by which is meant, prominence of aortic shadow and left lower cardiac contour, with deepening of the middle portion (the so-called waist of the heart), does not necessarily indicate aortic disease. Besides this disease, it is seen in a certain number of aged normal individuals, in hypertension and in cases of raised diaphragm from any cause. The more important point is to ascertain the size of various heart chambers separately, For this purpose, in addition to the Anterior View, oblique views have to be taken. For enlarged *Left Auricle*, anterior and right oblique views are required. Visualising of the oesophagus with barium gives a more impressive demonstration of the enlargement, but is not essential. The *Right Auricle* can also be well demonstrated in these two views. For ventricles, as already stated, left anterior oblique view is very helpful.

B. *Great Blood Vessels arsing from the Heart, especially Aorta and Pulmonary Arteries*

Even when the signs and symptoms of disease of aorta are very well marked and typical, radiological examination is essential, to find out the extent and nature of the pathological change. To visualise completely the aorta, anterior as well as both oblique views and fluoroscopic examinations are indispensible. This applies to the pulmonary arteries, even to a greater extent, for here the signs and symptoms are seldom clear cut and radiology may be the only means of detecting the abnormality.

C. *Condition of the Lesser Circulation*

Radiology is the best means we possess, of finding the condition of the lesser circulation-congestion and oedema of the lungs. It is now recognized that congestive heart failure, manifests itself earliest in the lungs, much earlier than in the systemic circulation and before there are signs of increased venous pressure, enlargement of liver, or oedema. So, in radiology, we possess a method which, I think, should be used more frequently than at present to detect the earliest signs of heart failure. In mitral disease, there is always a degree of chronic hyperaemia of the lungs, even in a well compensated heart. But, in other conditions of the heart, chronic hyperaemia and oedema of the lungs, indicate a degree of heart failure. In the initial stages, only the pulmonary vein and capillaries are engorged, but, as the right ventricle hypertrophies, the pulmonary arteries also distend. When both sets of vessels are involved, transudation occurs in the lymphatic vessels and glands, and, finally, in severe cases, in the bronchial mucosa. X-ray appearances vary with the degree of hyperaemia and several well-marked appearances are recognized.

(1) In the earlier stages of cardiac failure, and in compensated mitral disease, hilum shadows are enlarged and ill-defined, big branches seen end-on may be as big as peas sometimes erroneously diagnosed as calcified glands). Even smaller vessels are enlarged. It is impossible to get a picture of good contrast, translucency of the lungs is decreased, all the movements seen on the screen are restricted. There may be thickening of inter-lobar pleura. Sometimes, the combination of swollen end-on vessels and alveoli containing heart failure cells, give a miliary appearance in both lungs, something like military tuberculosis or pneumo-coniosis.

(2) Localized Passive Hyperaemia of the lungs may occur in the larger vessels near the hila. This may be an early sign of impending failure, long before failure is suspected from clinical evidence. As failure becomes more marked, large opaque areas are

visible in the lungs near the roots. These opacities are areas of atelectasis, due partly to oedema of bronchial mucosa, and partly to compression by distended capillaries. These occur near the hila, because that is the situation of least elasticity. Pleural effusion may also occur. This oedema and stasis can disappear completely under treatment.

(3) In cardio-renal disease, whether acute or chronic, one occasionally comes across a typical picture. There is hyperaemia, localised to large vessels in the medial and lower parts of the lung fields, accompanied by loss of translucency and by excessive exudation into the alveoli; peripheral parts of the lungs are normal. It would be better described as sub-acute pulmonary oedema.

D. *Congenital Cardiac Abnormalities*

It has been estimated that about fifty per cent of the congenital abnormalities can be diagnosed by X-rays. Some may be quite obvious, like dextro-cardia or right-sided aorta, others might be recognized by the characteristic change they produce in the shape of the heart, like the Fallot's tetrology or pulmonary stenosis. In coarctation of aorta, actual stenosis of the aortic isthmus may be seen, or characteristic changes in the ribs, produced by dilated collateral arteries may give a clue to the diagnosis.

E. *Effusion in Pericardium*

If the effusion is more than three hundred cc. it is recognizable, probably more easily and earlier, by X-rays than by clinical methods. A lateral picture, by showing a bulge on the posterior inferior recess of the sac, has probably a better chance of revealing a small effusion, than any other method. In more advanced cases, the changes in the cardiac shadow and in the cardiac pulsations, give a characteristic picture. Rarer's condition, e.g. pneumopericardium and pericardiae calcification, are of course, easily recognizable radiologically.

To sum Up

Taken in conjunction with other methods, radiology is the best means of ascertaining the size and shape of the heart and its chambers, of revealing the condition of the great vessels arising from the heart, and of visualising the lesser circulation in the lungs. It is likely to give considerable aid in the diagnosis of the pericardial disease and congenital deformities of the heart. It should be used more frequently than at present. To disregard this method of investigation, is to neglect our best, trained sense, the eye.

(X-ray films were demonstrated)

P. U. O. AND DENGUE GROUP

215. LT.-COL. KARAMCHANDANI—"*Dengue Group of Fevers in India*" **(P)**

The classical description of dengue, with a saddle-back temperature and a rash, is so well known to all students of tropical diseases, that they have come to regard these two features as essential to the diagnosis of the disease. The existence of modified forms, first suggested itself to me, when a fever epidemic occurred in 1936 in Madras, and extended to the penitentiary where I was working at the time. This fever has a sudden onset, accompanied by headache and backache. There was congestion on the buccal membrane and soreness of the tongue. Slow pulse and leucopenia, with diminution in the number of polymorphonuclear cells (50—59 per cent.), and relative increase in the number of lymphocytes (40—45 per cent.), were constantly present. Blood slides for malaria parasites invariably proved negative. The epidemic of fever lasted from 10th October to 7th November, 1936, and I studied 110 cases. Analysis of temperature charts showed the following three groups :—

The first group of 54 *cases* : These had a saddle-back temperature. In 31 of these cases, the temperature touched normal before the second rise took place, while, in 23, it dropped to 99 degrees F. The average duration of the fever was 5·5 days in the former, and 5·35 in the latter.

The second group of 17 *cases*: These showed a continuous temperature, lasting, on the average 7·17 days.

The third group of 39 *cases* : These were not associated with a saddle-back temperature and the pyrexia lasted an average period of 3·28 days. Had they not shown other

features, suggestive of dengue fever, they might have passed as sandfly fever. No eruption was noted in any of these 110 cases, but an isolated case, outside the penitentiary, with a dengue-like rash, attracted my attention and suggested that the whole epidemic was, in fact, dengue, and they were published as such (Karamchandani 1937). After that, I began to associate the 5-days fever of Scheer and the 7-days fever of Rogers, as examples of the same disease, and no longer believed that the absence of an eruption precluded the diagnosis of dengue, but still thought that the disease only occurred at the coast, in places like Madras.

During the month of October 1943, cases occurred in the British population of Ferozepore (Punjab), which presented similar features. The limited number, the locality of the epidemic and the absence of rash in the early cases, were at first against dengue, but the character of the fever, so similar to that described in my original series, and the characteristic rash in the later cases, suggested the presence of this type of infection. Indian troops in the district were also similarly affected, but there was not the same opportunity of carrying out a close clinical study among them.

Fever and Rash. (Temperature Charts of 18 cases were demonstrated).

It will be seen that ten cases show a saddle-back type of temperature, duration of fever being 4·8 days. Two cases show a continued type, and one case a short one-phase type of temperature. One other case presents a rarer three-phase saddle-back pyrexia. Four patients reported with a rash, their preliminary temperature not having been noted; they had felt feverish, with pain in the back, lower extremities, head and eyes, but had not come to the hospital until the rash appeared; but for this, they would have been missed. A profuse, maculer, rubeloid eruption, so very characteristic of dengue, was present in 8 cases. I wish to remark here, that there were three other cases who got mixed up in this group, of whom two had rash; but, because the temperature touched normal on the tenth day or later, they were separated; one of these turned out to be a case of para-typhoid, and two of flea typhus. It may, therefore, be stated that fevers with a duration of ten days or more, cannot be regarded as dengue.

The questions which arise for discussion are :

(1) Whether dengue fever is a distinct disease by itself and whether a number of local forms or varieties can be distinguished? If so, is the existence of these special forms due to the influence of local conditions on the vector which conveys virus from one person to another, or is it due to different groups of the culicine tribe of the vector?

(2) Whether dengue fever, noted for its pandemicity, can occur sporadically and exist in central parts of India like Ferozepore?

(3) Whether distinct features, such as rash, saddle-back temperature and a wide epidemic, should be considered essential for the diagnosis of classical dengue fever, or is there sufficient evidence to formulate a dengue group of fevers, which would include subsidiary forms?

(4) Whether sandfly fever, although conveyed by phlebotomus, can also be included in such a group of dengue fevers?

The answer to the first question is, that while saddle-back exanthematous, classical dengue may be a distinct disease, several varieties can be distinguished, not only in different epidemics, but also in the same epidemic. This was the case in my first series in Madras (Karamchandani, 1937). It is also borne out by Dinger and Snijder's experiments. Mosquitoes infected in Sumatra, were brought to Amsterdam and allowed to transmit the disease to volunteers. Although the infection was due to a common virus, various types of fevers were produced in different individuals (Manson Bahr, P. 1940).

Regarding the vector problem, although Aedes aegypti has now been universally accepted as the vector, Aedes albopictus, Aedes taemorhynous and Culex fatigans, continue to be suggested as alternatives (Manson, 1940). I consider that, in Madras at least, Culex fatigans is mainly responsible. The elucidation of Buxton's three classes of insects, in my analytic study in the London School of Hygiene and Tropical Medicine Laboratories, where Culicine was proved to be " Spender " (Karamchandani, 1934), backed by my synthetic field work (Karamchandani, 1937), has inclined me towards Graham's original conception that Culex cannot be excluded, at least in some places (Manson, 1940). In Ferozepore, no Aedes aegypti have, as yet, been identified in the catches of Culicinae.

The answer to the second question, is given by Manson, who states, " Between epidemics, cases do occur sporadically, which means the virus is maintained and forms the nidus of infection for a new epidemic, but, owing to their mild nature, they are frequently not recognized " (Manson, 1940). This is perfectly true. Had it not been for the typical rash, four cases out of eighteen under discussion in the present paper, would not have reported to hospital and would have gone unrecognized. There is no doubt, that the eighteen cases in Ferozepore were cases of dengue. Manson believes that dengue prefers deltas and valleys of great rivers in the interior, which would make Ferozpore, situated on the Sutlej, a suitable locality.

My experiences in Madras deal with the third question. Here, characteristic features of dengue were not always constant; epidemic rash was rare, saddle-back temperature only seen in 50 per cent. of cases, although there was a rapidly spreading epidemic, and thousands of cases occurred in the city during October, 1936. In Ferozepore, the rash was seen in 44 per cent. of cases, and a saddle-back temperature was present in 55 per cent. of cases, but, unlike the Madras series, the epidemic was very limited. In both, the cases occurred in October and November. From the available evidence, I feel that the conception of a dengue group of fevers is justified. Apart from scientific alignment, it would focus the attention of the general practitioner on sporadic mild forms of fever, which, at present, are labelled ' Clinical ' Malaria, and treated with quinine, or, alternatively, diagnosed as Influenza.

The fourth question, embracing sandfly fever, is more debatable, but there are certain clinical and actiological similarities. Both diseases are transmitted by an insect, the infection being caused by a filterable virus in the blood stream. Both are diseases of warm climates and, clinically, have a short incubation period, a brief, rapidly-developed fever, associated with a slow pulse, leucopenia, and relative decrease of poly-morphonuclear cells. Thus, the virus of dengue and sandfly fever have much in common.

Conclusions

I. It is time that we adopted the name of " Dengue Group of Fevers " and modified the classical description, which stresses the most dramatic features of dengue, rather than its great variability. We shall, by this means, ensure a correct diagnosis, by remembering the main group features. These are :—

(1) Sudden and abrupt onset, with generalized pains and no local manifestations of an acute inflammatory condition, such as tonsillitis or a septic infection, etc.

(2) Absence of any so-far known parasites in the blood, absence of leucocytosis and rather a tendency to progressive leucopaenia.

(3) Fever, with a short, self-limited course, the temperature, in most cases, conforming to the forms outlined above, viz. (*a*) saddle-back type, either continuous (average duration 5 days) or interrupted (average duration 5 days); (*b*) continued type (average duration 7 days); (*c*) short one-phase type (average duration 3 days); and, (*d*) larval type (average duration less than one day).

(4) The presence of a rash in some cases.

II. The disease has greater prevalence in coastal, delta, and river areas, as explained by the distribution of the culicinae order of diptera. On the other hand, epidemics in dry, temperate central zones, with relative humidity even as low as 0 per cent., cannot be ruled out.

III. A certain degree of immunity exists, which governs its incidence among communities. Sporadic cases can occur, where there are insufficient numbers of susceptible persons and a sparse population. Epidemics arise when opposite conditions prevail.

IV. Varieties in the disease are not so much due to differences in the strains of the virus, as to the susceptibility of individuals.

V. Sandfly fever should be included in the group of dengue fevers, under the name of Phlebotomus dengue.

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DISCUSSIONS

216. PROF. GOYLE: Lt.-Col. Karamchandani will remember that Col. Shortt, of the King Institute, Gindy, Madras, was trying to grow the virus of dengue and sandfly fever, in the chorio-allantoic membrane of the developing chick. I would like to know if it is possible to distinguish these two viruses, and if he used vaccine made from these viruses, in prophylaxis.

217. DR. WIG: Every year, in the autumn, we get round about Lahore, an epidemic of a group of fevers, lasting, on the average, five days or seven days. After every few years, it comes in severe epidemic phases. We usually call them sandfly fevers, but, in my opinion, they belong to the dengue group. My reasons for this are, that the fever lasts either for five days or for seven days, in some localities keeping to the five-day type, and in other localities, in the same epidemic, conforming to the seven-day type, and is accompanied be severe pain in the temples, inside the eyeballs, in the back and in the extremities. It shows a typical saddle-back temperature chart. The pulse becomes progressively slow during the course of the fever. Not only is the conjunctiva congested, but the throat also is very congested in some cases. Rash is not so common, but, in some epidemics, has been a fairly prominent feature.

In two cases belonging to this group, I have met a complication—haemoglobinuria—which I have, so far, never seen mentioned anywhere. Both were definitely cases of this group. They occurred during the epidemic, and, in the case of one, all other members of the family were down with the same fever. They showed the typical temperature chart and a typically slow pulse and all the other usual features. Haemoglobinuria occurred at the end stage of the fever and was of a mild character. They had received no treatment to which we could ascribe the haemoglobinuria.

It would be worthwhile investigating the vector of these fevers in the Punjab. My impression is, that they belong to the dengue group, and so the vector is likely to be a mosquito.

218. DR. PIRZADA: In the present state of our knowledge, the classification of the dengue-sandfly group of fevers into Sandfly, Dengue,—Mosquito Dengue, and Dengue of unknown vector (Rogers and Megaw), appears to be quite satisfactory. The clinical differences could be explained by different strains of a virus conveyed by different vectors. This group of fevers is common in the Punjab, but in and about Lahore, I have not seen any rashes. Although pains are usually severe and generalised during an epidemic, some cases show only headache. These cases, on account of a slow pulse and a coated tongue, are mistaken for the enteric group of fevers. The temperature, however, drops suddenly in the usual period.

219. COL. TAYLOR: I have been interested in these fevers in N. W. India, for the last 15 years. They vary in clinical symptoms and signs and duration, each year. And, in each yearly epidemic, many stages of severity are seen. It was, I think, in 1937, in the September outbreak, that a large proportion of both Indian and British cases showed a well-developed rash. Rashes were seen in the odd cases in most years, particularly in European cases.

Immunity, resulting from such attacks, varies. Some never develop attacks, but these are rare; others have yearly attacks, or even two attacks in the season.

Until the fever comes down, the diagnosis is often in doubt, infection of the respiratory tract, malaria, and the enteric group, being commonly the main trouble.

KALA AZAR

220. MAJOR KILOH—"*Kala Azar*" (P)

Gentlemen, from the point of view of its incidence in European troops, kala azar does not stand high amongst military medical problems. However, when it does occur, there are two important features which should be stressed, (*a*) it does not lend itself to very early diagnosis, such as, for example, in the case of malaria and dysentery, and, (*b*) it is not a self-limiting disease, and, in the absence of treatment, will progress almost inevitably to a fatal outcome. In peace time it is rare in the European—in spite of its alternative name of Sahib's Disease—and it is seen, almost to exclusion, in the Indian, and, it may be added, for all practical purposes, in the poorest class of Indian. The class of Indian who

contracts kala azar, is almost certain to suffer from chronic malaria also, and, may be, other diseases. He is, in fact, a poor physical specimen and, furthermore, does not complain or seek advice at an early stage. It is, then, rarely seen in the virgin state, but usually in association with other conditions, which may distort the clinical picture at an advanced stage of the disease.

It is not surprising that, at the present time, with large numbers of European troops living under varying conditions in the endemic areas, that its incidence in the European has risen, and I propose to discuss, in the few minutes at my disposal, the findings made in a series of 10 cases, observed in B. O. Rs., in the early months of 1943. I have not the time to confuse your minds with details of each individual case, but will confine myself to general conclusions, and I would point out that these men were not investigated academically and exhaustively, but from the more practical viewpoint of reaching a diagnostic conclusion.

These ten men were all fit, healthy, Category A individuals, practically without Tropical Medical History, who entered the endemic area between 1st March, and 31st May, 1942, and remained there until the diagnosis was confirmed. This period includes the monsoon of 1942, with its encouragement to the proclivity of the sandfly and other pests—a fact of importance in discussing the incubation period of the cases in question. In passing, it is likely that a corresponding rise in incidence will be seen at about this time and corresponding to the last monsoon.

History prior to admission

Three of the men were originally admitted, on account of some other established condition. Two were admitted on account of dysentery, one amoebic and the other bacillary, and the other had a long history of relapsing B. T. malaria, with malarial symptoms and a positive slide, on admission. The remainder, seven in number, gave a history of vague and indefinite ill-health for a varying period, consisting of loss of energy and power of concentration, general lack of well-being, a vicarious appetite and some loss of weight in consequence. This vague history culiminated in ' fever ' and symptoms such as shivering, coldness and sweating, simulating the onset of malaria, and because of this, they were finally sent for admission.

On Admission

These patients were all febrile and, in the majority, the spleen was palpable to a greater or lesser degree. With one exception, the general condition was good and there was inconsistency between this fact and the temperature chart. The investigation relevant to the case were carried out, always with negative result, apart from a definite tendency to leucopenia. In the earlier cases, there was a considerable lag between admission and conclusive diagnosis, and this interval, coupled with negative, investigation and persisting signs and symptoms, effectively ruled out the possibility of the more common and self-limiting infections. In the meantime, these more common conditions were considered, and if one looks at the temperature charts, one can, in some cases, see a resemblance to the ' staircase ' type of chart, seen in typhoid fever, yet the clinical picture and the progress of the case did not, in any way, support this. It is as well to consider this clinical picture presented in the interim period, in association with the relevant investigations that were carried out.

Temperature Chart

A study of the temperature chart does not help very much. In no case was the characteristic ' double ' rise seen, and it was assiduously looked for. In some cases, there was persistent pyrexia throughout, but there was a definite tendency for afebrile periods to alternate with bouts of pyrexia, slowly rising to the fastigium. One case was confirmed after a lengthy febrile period. The pulse rate was not in accord with the temperature and was generally well below 100, which corresponds to the relative absence of toxaemia.

General Condition

In spite of, in many cases, a high temperature, there was absence of that apathy and general mental and physical depression which, for instance, is so characteristic of typhoid fever. These patients remained bright and cheerful, mentally alert, with good appetite, and not, apparently, losing ground. This was a common feature of all the cases, with one

exception that I will quote later. In no case could the individual be considered as anything but adequately covered. It is, then, reasonable to suppose that emaciation and cachexia result only when the condition is of longer standing, aided, possibily, by the presence of other diseases, and is an indication of chronicity.

Spleen

This was palpable to varying extent, with one exception. In this case, the liver was not palpable either, and this man, before the diagnosis was finally made—after a lengthy afebrile period—had obviously had the condition for three months. As this man had no temperature and had no physical signs, I suppose that he must be accounted lucky not to have been discharged! The progressive leucopenia in this case, led one to the clinical diagnosis. In three cases, the liver was palpable and abnormal, but did not approach the degree of splenic enlargement. In no case was there enlargement of glands.

Pigmentation

This was not seen in any case, and presumably is an indication of chronicity when it does occur. In a group of subjects such as this, it would have been easy, obviously, to recognise if it had been present, but I understand it does not occur in the European.

Investigations

Leucocyte Count—It was generally agreed that, apart from the actual isolation of the Leishman-Donovan body, the total leucocyte count was a better guide, than any other investigation, to the probable presence of the disease. All cases presented, on admission, at least a moderate leucopenia, and time showed that this was slowly progressive. The granular series were alone affected, and, in several cases, with an absence of eosinophiles. The leucocyte count was not only valuable as an indication when to carry out sternal puncture, but the progressive rise, following treatment, showed that the treatment given was being successful.

Red Cell Count—In all cases, this was found to be between three and four million, and remained static, in spite of the passage of time. The RBC/WBC ratio—normally 630/1, was found to vary at the time of diagnosis from 1300/1 to 3300/1.

Serum Tests—The formalin test, commonly known as the Napier Test, was negative in all cases, and the Antimony or Chopra Test was positive in one case. This is presumptive evidence in favour of the relatively early type of case seen.

Sternal puncture was the routine adopted for the isolation of this parasite. When this was negative, it was repeated a week later, and if negative again, then splenic puncture was used. Eight cases showed a positive sternal puncture, and in two, splenic puncture was necessary, to confirm the clinical diagnosis. In our experience, it was not always by any means an easy matter to detect the parasite in the smear. In several cases, they were extremely scanty, and in one case, atypical. Without the aid of a senior member of the Staff of the Tropical School, the diagnosis would have been longer delayed. In cases which are strongly suspected of Leishmaniasis, therefore, I suggest that negative smears should be sent for examination by an expert. It may be pointed out that this difficulty was experienced, in spite of the fact that sternal puncture was not used indiscriminately, but only when clinical diagnosis was becoming increasingly certain.

Complement Fixation Test (Witebsky, Klingenstein, Kuhn—The European offers an ideal medium for the investigation of the efficacy of this test. The non-specific W. K. K. antigen is used, consisting of the alcohol insoluble pyrisin soluble and acetone insoluble fraction of the tubercle bacillus in benzolic solution with added lecithin. Experience seems to show that this is positive before the antimony and aldehyde tests, and about a month after symptoms manifest themselves. It was not used in any of the above cases, but has been adopted now by the hospital concerned.

A typical Case

A moment may be spared to describe briefly the most unusual case of the series. This man gave a vague history of ill-health and was admitted with a very high temperature. Examination showed an ill man, with some abdominal distension and marked tenderness and rigidity of the upper abdomen. There was evidence to show that, but for the rigidity, the liver and spleen would have been palpable. Blood slides were negative, and within a few days, his condition became progressively worse and was complicated by severe diarrhoea.

The temperature remained high and the pulse rate rose to 140. He was extremely ill and the immediate prognosis appeared very bad. A white count was done at this stage and was found to be 1000. The RBC/WBC ratio was 3300/1 and the aldehyde test negative. Sternal puncture was negative and, with some trepidation, splenic puncture was resorted to and found to be positive. The result of treatment can be seen from the chart. (Demonstrated—Ed.). This is a case, and that almost an emergency, where the diagnosis could only be confirmed by an expert. Only one parasite was seen by him, and that an atypical one.

Treatment

In all cases, the pentavalent antimony preparation, urea stibamine, was used intravenously, commencing with O.1 gm, O.2 gm the following day, and O.2 gm on alternate days thereafter, until fifteen doses had been given, leading to a total dosage of approximately 3 grammes. A study of the charts shows that the results of this treatment are comparable to that of sulphayridine in pneumonia, and, in the above-quoted acute case, the temperature fell by crisis after the first injection. It is interesting, too, to note that by the third day, the rigidity had vanished and the spleen and liver were now palpable. The results of treatment in general could be gauged by the rapid fall in temperature to a normal level, the improvement in the general condition, the diminution in size of the spleen, and, perhaps above all, by the progressive rise in the total leucocyte count, all of which were noticeable in this series of cases. No toxic effects were observed at all, by the use of this drug.

Conclusion

There was no clinical feature that was common to all 10 cases and which could be called diagnostic or pathognomonic, as witness, for instance, the spleen, which in one case was not palpable. Excluding the atypical case and taking a line through the remaining nine, one can point out the important clinical feature as being pyrexia, a relative absence of toxaemia, with enlargement of the spleen and, may be, the liver. Persistent and progressive leucopenia, with the accent on the granular series, is the most useful of the investigations carried out, apart from sternal or splenic puncture, and this latter is by no means always positive at the first attempt. Treatment in all these cases was strikingly successful. A well-recognised complication of kala azar is dysentery and this occurred in 4 of the present series, one amoebic and three bacillary, one of which almost precipitated a fatal ending. The possibility of relapse has to be considered, and it is difficult to express an opinion about this. One case was discharged to another hospital, with his spleen just palpable and one might not be surprised if relapse occurred in this case. Tht remainder were discharged with no symptoms or signs, clinical or otherwise, and here one would expect the ultimate outlook to be good. I ought to point out that the cases first admitted were some time in hospital, before a diagnosis was made, largely due to the fact that they occurred in a hospital which had but recently arrived in this country. This reminds one of the lesson that a tropical disease can be extremely localised, and one should consider carefully the possibility of disease limited to the locality in which one works. The cases admitted later, undoubtedly benefited from the experience of the earlier ones, so that the latter five admissions were confirmed within three weeks of admission, and one within four days. Kala azar must be considered in a European who has lived in the endemic area (particularly about the time of the monsoon) and who presents a febrile illness, with enlargement of the spleen, malaria being excluded, and who is shown to have leucopenia of a progressive type.

Finally, these cases were all diagnosed in the endemic area. In these days of large-scale troop movements, it is likely that cases will occur in parts where kala azar is normally a stranger, especially when one considers its long incubation period. One must, therefore, be on the lookout for it, in other parts of the country, in Europeans.

221. LT.-COL. BOSE—" *Kala Azar* **" (D)**

Leishmaniasis is not peculiar to India, as it occurs in other parts of the world, but, in its generalised form, Leishmania donovani (Kala azar), is widespread in certain parts of India, producing high mortality in untreated cases and causing much confusion to the unwary in the diagnosis, as it is frequently associated with other infections in the tropics. The object of my taking part in the discussion on this subject is (i) to mention India's contribution towards the cure of this malady, (ii) to emphasise the importance of acquiring

necessary working knowledge of the Epidemiology of the disease in India Command, and, finally, (iii) to acquaint my colleagues (who have recently arrived in this country for the first time and who are situated outside the zone of prevalence, namely, the Punjab), with the salient clinical and laboratory manifestations of the infection.

It may be of interest to note that the causative organism of the disease, as indicated by its name, was discovered in India, during the early part of this century, and the specific remedy—Urea Stibamine—in Calcutta, by Dr. Brahmachari, the founder of the Brahmachari Institute, Calcutta.

The disease is highly prevalent in the Eastern half of the India Command, where the 14th Army and the Eastern Command are at present operating. Assam used to be the death-bed for such patients during the latter part of the last century; Rogers and Bentley have done considerable research work to find out the cause of this black fever, by which name it was then commonly known. The disease existed or seemed to have spread eastwards and began to be known as Dum-Dum (near Calcutta) fever. Now, with improved methods of diagnosis, we find it all over Bengal, Eastern U. P., Orissa and Madras Presidency. The disease is frequently met with among Gurkha recruits, most of whom hail from Nepal. In these days of rapid and mass movement of troops, from one end of the peninsula to the other, and of men having been recruited from these endemic areas, or of having been stationed in such places, the possibility of this infection, in the differential diagnosis of diseases, has constantly to be borne in mind.

Major Kiloh has just given an admirable review of the clinical picture of the disease, as he found it among recently-arrived B. O. R.'s. I do not propose to go into details, but shall mention certain salient points, which may be of some use to you in the diagnosis and treatment of the infection. In the diagnosis of early cases, it is a good practice, when taking case history, to find out the locality of the patient's permanent home and his recent movements in and outside India; also to let the patient describe, in his own way, how and where his disability first started; usually there is a story of physical and mental exhaustion after moderate effort. The incubation period is variable and uncertain. The insiduous onset is frequently ushered in by fever of varying duration and range of temperature; not infrequently the temperature chart resembles that of enteric fevers, or of quotidian type of malaria : bouts of fever, lasting for 10—12 days, are usually followed by an irregular apyrexial interval; sometimes the fever remains continuous. The spleen becomes palpable and no specific cause can be found. This is the period when associated symptoms and signs rouse one's suspicions; good appetite, clean tongue, peculiar absence of toxaemia with leucopenia, are the diagnostic points. In the later stage, when the infection is well established, we observe muddy complexion, coarse and dry hair, pigmentation of the skin and mucosa, haemorrhages—especially epistaxis, anaemia, diarrhoea, debility with splenomegaly, and, not infrequently, hepatomegaly. Later still, appear ascites, cachexia and canerum oris. The disease is commonly associated with malaria, dysentery, hookworm and tuberculosis, which may be a terminal and fatal factor.

The laboratory examinations, at different stages of the disease, are extremely helpful. Leucopenia is diagnostic; there is relative diminution of the granulocytes and an increase in the monocytes. The latter, if malaria can be excluded, is highly significant. Any increase in the lymphocytes or in the eosinophiles, during the course of the disease, is suggestive of super-imposed complications. A positive aldehyde and/or a positive urea stibamine test of the blood serum, is almost pathognomonic of the infection. The coagulation time of the blood is frequently prolonged. A positive sternal or spleen puncture smears, gives the conclusive diagnosis. In several cases, when the smears are negative (probably due to indifferent material collected, or to an unsatisfactory technique), the culture gives positive results.

With regard to the treatment, I advise an initial dose of Urea Stibamine, 00.05 grm., to a man weighing 9 stones, to be followed by daily injections of 00.1, 00.15, 00.2 grm., until the temperature comes down, and then, on alternate days, 00.2 grm., until a total of 3 grm. is reached. It is essential to give specific treatment at the same time, for any associated infections; good results have been obtained by giving vitamin therapy, consisting of A, B, C and D, combined with calcium (intravenous) and iron simultaneously. The response to the treatment is manifested by the fall of temperature to normal, reduction in the

size of the spleen and the liver, general feeling of well-being, gain in weight, and improvement of the blood picture. The patients should be under observation for at least a year, during which period, a record of the haemogram and the weight should be kept, and a modified course of specific treatment repeated, if splenomegaly and abnormal blood picture persist.

(A practical demonstration of 'formal-gel' and 'urea-stibamine' test was given)

DISCUSSIONS

222. LT.-COL. PASRICHA: Whilst my friend, Lt.-Col. Bose, is demonstrating the kala azar tests, I will tell you the "Mystery" of the origin of the Formal-gel tests. I call it a mystery, because a test, of which one had heard nearly 25 years ago applied in medicine, had become one of the most popular and well-known tests (or, better, a pointer) for kala azar. The story is not well-known and will be of particular interest to the Punjab, for, although the disease is not endemic in the Punjab, the formal-gel test for kala azar was first used in the Punjab and later developed in Bengal.

A young I. M. S. Officer, Capt. Spackman (now Colonel Spackman of Bihar), was posted in Dhramsala in 1920-21, and, whilst there, tried out a test, which had then been described as specific for syphilis. This test was called the 'Gate-Papacostas' reaction, or the Formal-gel test for syphilis. This keen young officer tried out the test, and, as every scientific worker should, used controls. In this, it was just accident that he used cases of kala azar. He (Spackman) found that the so-called 'Gate-Papacostas' reaction gave a more characteristic result with kala azar than with his syphilitic patients. He was surprised and he did two things; one, he wrote a letter recording his finding to the B. M. J., and this was published, and, secondly, he wrote to his friend Knowles, at the newly-formed School of Tropical Medicine, Calcutta. And, after this, Spackman seemed to have forgotten this valuable information, until about 1942, about 2 decades later, when he found his observation described as 'Napier's formal-gel test' for kala azar. Spackman had apparently forgotten this period of decades and meanwhile, Napier had worked on it and popularised it. This is one of the little-known facts, and it is mentioned in General Bradfield's 'Indian Medical Review'.

223. MAJOR KILOH: I would augment Lt.-Col. Bose's remarks, by saying that there is no such thing as a weak positive formalin test. It is either negative or positive, and, in a positive test, the mixture becomes solid, with a white of egg appearance, so that, on turning the test-tube upside down, no fluid emerges. Furthermore, there are two other conditions in which the test is positive, i.e. Schistosomiasis and Trypanosomiasis, but these are not likely to confuse the issue in India.

224. LT.-COL. ROGAN: It has been stated that the diseases, other than kala azar, which give a positive aldehyde test, namely trypanosomiasis and schistosmiasis, are non-existent in India. It is worth noting that trypanosomiasis may be found among African troops in India.

225. DR. WIG: I agree with Lt.-Col. Bose that we in the Punjab should now know more about kala azar. I want to ask him and the previous speaker a few questions. Have they studied the X-ray appearances in the lungs of kala azar patients? I am asking this question, because out of the five cases of kala azar which I have so far met in this province, (all imported cases), three were referred to me because they showed some opacities in the lungs. My suspicion was aroused because these opacities did not resemble those of Tuberculosis, either in situation or appearance, sputum was persistently negative and spleen was enlarged. Later enquiries showed that the cases had been living in endemic areas. These cases were definitely proved to be kala azar by later investigations, and yielded to the antimony treatment. While I was not able to follow two of them, in one the opacities cleared up completely. I remember that the subject was mentioned at one of the Tuberculosis Workers Conferences, and some workers from Bengal mentioned that the opacities in the lung can appear in certain cases of kala azar. One of the cases mentioned above, had never been to the east of Mussoorie in India. He was an Anglo-Indian boy, studying in some school at Mussoorie. I want to know from Lt.-Col. Bose, what is the western-most limit of kala azar in India.

226. COL. CAMERON: I had the pleasure of seeing Mediterranean and Sudanese Kala Azar in the Middle East, and of discussing them with a world authority, Prof. Adler. On arrival in India, I felt I knew Kala Azar, but association with Lt.-Col. Bose showed me the blanks in my knowledge. I offer grateful acknowledgment to Bose, for his help in guiding my footsteps in this respect. We disagreed over the relative merits of urea stibamine and diamidino-stilbene in treatment. The Indian variety of Kala Azar is more susceptible to the latter, than the Mediterranean and Sudan varieties.

I find that splenic puncture gives positive information, more often than sternal. I have also seen good results from hepatic puncture.

227. LT.-COL. BOSE, in reply to Dr. Wig, said that kala azar cases had been reported as far as Delhi and Jhansi, but not further west, as far as he knew; he was on the look-out for such cases among the soldiers and the non-combatents who had been recruited from the Punjab and N. W. F. P. He further stated that the presence of radio-opaque areas in the lungs were consistent with this infection, in view of the fact that Prof. De of Calcutta Medical College, had conclusively proved the presence of L. D. bodies in every organ of the body, after post-mortem examination.

In reply to Col. Cameron, he stated that he had been misunderstood, in that he had never tried Stilbamidine (diamidino-stilbene), which was a new M & B product, and reputed to be effective in Sudan Kala Azar, and therefore could not express an opinion on the comparative merits of the drug.

228. MAJOR-GENERAL CURSETJEE—"*Closing Address*"

I came to this conference to learn. I have learned quite a lot. I hope I shall remember it all. I trust that you have all learned something too. I shall have the advantage over you all; I shall not have to remember it so long. When you all go back to your hospitals, do not be jealous of your knowledge, but pass on what you have learned to your brother medical officers and subordinates, even to your sweepers. You may "walk with princes" but do not lose "the common touch". In the Army, we must all aim at the highest standard. If we do not, the enemy may have a higher and we cannot be expected to win. The side that makes the least mistakes, wins. We always win in the end, but, the fewer mistakes we make, the sooner will we win.

If this Conference has been a success, it is largely due to the I. G. of Civil Hospitals, Punjab, Principal and Staff of the Medical College, Lahore, who placed every facility at our disposal for the week, and the members of the medical profession of this city, who gave us the value of their experience and knowledge. We have met them on cordial terms and we hope we shall have opportunities to repeat these occasions in the future. I am sure that all the Army members will join me, in according them our most generous and sincere thanks.

W. B. C. ENUMERATOR

229. LT.-COL. DAUKES, while deploring the levity shown by certain members of the audience, demonstrated an apparatus for helping in the enumeration of a differential white cell count. It consisted of a box, with a sloping curved floor and a block of wood in the right side, at the far end of which there were six holes, conveniently arranged for location blind, simply by feel. Into these, marbles were dropped, representing the different kinds of leucocytes, each marble being dropped into the appropriate hole for the kind of leucocyte seen. The marbles then fell through the block, out of the side of the box, into a specially partitioned tray. 100 marbles were put into the box and, when these were exhausted, the totals in the different partitions of the tray were counted quickly and easily. (Subsequent improvements had been made on this, and time tests carried out, which showed that, while a little time might be saved on one person writing down his own results, the main benefit was that it was a little more convenient than writing down the results. Lt.-Col. Daukes' opinion was, that while it was a refinement which might have been of some use, it was no very great improvement on the present methods).

230. PROF. GOYLE: In closing this session, I have to thank all the speakers who have read papers and have taken part in the discussions, which have been very interesting, and instructive indeed. I thank the organisers of the conference, especially Col. Schlesinger, on behalf of the Civil Medical Men, for inviting us to be present at today's meeting.

(The Conference finally closed at 17.30 hours).

N.B.—Reference Paragraphs 209 and 210, a sample of the plant was sent to Lt.-Col. D. R. Thomas, O.B.E., I.M.S., Chemical Examiner, Punjab, for identification and analysis. It is called 'OYA' in Punjabi and 'RANDI' by Hazara District people; it grows in the hills or in semi-hill areas.

Report by Lt.-Col. Thomas :—Ranunculaceae—Travellers' Joy (Cleematis Vitalba L.) The extent to which this species is poisonous is not clear, though all parts are stated to be poisonous, acrid and narcotic, while the juice tends to blister the skin. It is remarked by Cornevin that it is less poisonous in spring, when the ass and goat browse on it to a considerable extent without serious trouble, than later, when it cannot be eaten without danger.

Toxic Principle: Travellers' Joy appears to contain strongly poisonous substances which have not been closely investigated. Greshoff found a Saponin in the leaves. The poison is dissipated by heat.

Symptoms : When eaten in quantity, the young shoots are diuretic, violently purgative, causing dysentery and, in rare cases, death. Applied to the exterior, it is irritating and even vesicatory, (Cornevin).

(*Ed.*)

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