eye is larger but not drawn up. No adhesions of iris in either eye. Media clear. Fundi normal.

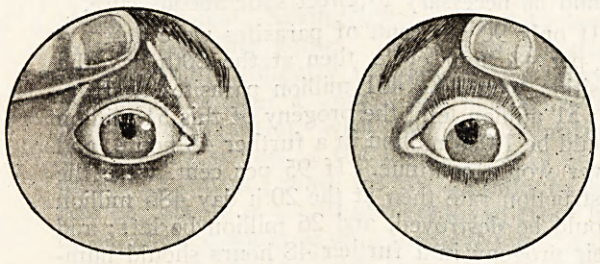
(14) *P.*—Both eyes operated on 23rd December 1921. Vision with + 10 D. in each eye = 6|6. Both pupils keyhole in shape. No adhesions of iris either in front or behind. This is one of the best results I have seen after the Smith operation.

(15) *M.*—Both eyes operated on 22nd December 1921. Age 60. Vision = 6|9 in each eye with + 11 D. Colobomata small. No adhesions of iris. Media clear. Fundi normal.

(16) *H.*—European aged 70. Right eye operated on 3rd January 1922. No complications at the time of operation or afterwards. Vision = 6|9 with + 10 D. and can read J ii with + 13 D. Coloboma large. No adhesions of iris. Media clear. Fundus normal. Still doing his work on the staff of a newspaper. Left eye cataractous.

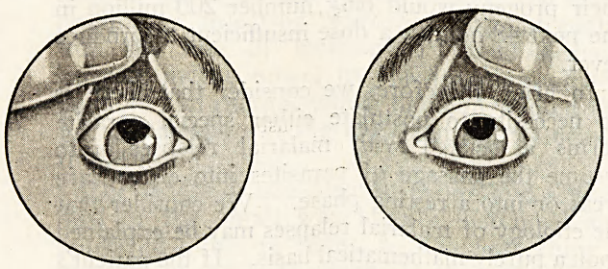
(17) *B.*—Right eye operated on 3rd March 1922. Vision = 6|6 with + 10 D. Coloboma medium sized. No adhesions of iris. All media clear. Fundus normal.

PLATE I.



A B

PLATE II.



C D

The illustrations A. B. C. D.

A. and B. are drawings of the eyes of a patient operated on four months ago. *A* shows the type of pupil I am in the habit of obtaining by doing a small iridectomy.

B. is a somewhat larger pupil. It might be thought that the iris was adherent at the nasal corner of the wound but this is not so. The vision in both these eyes = 6|6.

C. and *D.* are eyes of No. 6. Ayah.

In the right eye there was a slight escape of vitreous but it will be noted that she has still good vision. Left eye, no escape of vitreous.

I have given the latter drawings in order to show the type of coloboma in a case of escape of vitreous (C); and in (D) where the iris could not be properly replaced and is adherent to both corners of the wound.

A NOTE UPON SPLEEN PUNCTURE FINDINGS IN MALARIA.

By R. KNOWLES, and HUGH W. ACTON,
MAJORS, I.M.S.,
and
S. A. S. BIRAJ MOHAN DAS GUPTA,
Calcutta School of Tropical Medicine and Hygiene.

A paper read at the Medical Research Section of the Indian Science Congress, Lucknow, January 1923.

Now that the earlier views of Schaudinn (1902) on malarial relapse as being due to a production of merozoites by parthenogenesis on the part of the macrogametocytes,—(a process which, even if it existed, would be strangely anomalous from the protozoological point of view),—have been shewn to be entirely erroneous by J. D. Thomson, (1917), the problem of the true etiology of relapse in malaria remains to be solved.

With a view partly to obtaining light upon this problem; partly to test its value as a diagnostic procedure; and partly also to study the process of destruction of malarial parasites; we commenced in October 1922 to spleen puncture all cases of malaria admitted to the Carmichael Hospital for Tropical Diseases with enlarged and hard malarial spleens. In all 15 such cases were spleen punctured during the subsequent two months; including cases of benign tertian, quartan, and malignant tertian infections. The slides were stained by Leishman's stain, and were most carefully studied.

Results.

It was soon evident that spleen puncture is a method of no diagnostic value in chronic malaria. Occasionally in splenic films one may come across a crescent of *P. falciparum,* where examination of peripheral blood films has been negative; more frequently the presence of abundant hæmozoin pigment may give evidence that the case is one of malaria; yet few if any viable parasites are ever encountered in such films in afebrile and relapsing cases. Whereas spleen puncture is the readiest method of diagnosis in kala-azar, it is of little diagnostic value in malaria.

Where viable forms are encountered they are occasional trophozoites within the erythrocytes; and, with the exception of gametocytes, which seem to be less numerous in splenic films than in films of the peripheral blood, no other viable forms are encountered. What the films do show, in abundance, however, are parasite forms lying free in the plasma and undergoing degeneration.

It has usually been held that malarial parasites are destroyed in the body by a process of phagocytosis on the part of the macrophages, large hyaline mononuclear leucocytes and endothelial cells. We have been quite unable to confirm this view. Examination of the spleen films shews that only such parasites as are extracellular, and have been swept off the surface of the erythrocytes and caught in the meshes of the splenic pulp are destroyed. The stages seen are:—(1) Early destruction of the parasite chromatin by chromorrhexis and chromatolysis. (2) After

this the parasite cytoplasm becomes vacuolated and is destroyed by cytolysis; so that parasite forms are seen which are devoid of chromatin, but which can be identified as degenerated malarial parasites by the presence of hæmozoin pigment. (3) At this stage the degenerating masses of parasite cytoplasm appear to tend to adhere together in the intracellular spaces, where we meet with sheets of degenerated parasite cytoplasm studded with hæmozoin pigment. (4) Finally the cytoplasm also goes to pieces; free hæmozoin pigment is left; and this is phagocytosed by the macrophages and endothelial cells. Spleen puncture films in malaria are often very pretty studies of such "pigmented mononuclears."

The Etiology of Malarial Relapse.

Many different views as to the true etiology of malarial relapse have been put forward: that there may be special and as yet unrecognised parasite forms especially associated with relapse; that the parasites may survive by finding their way out of the blood stream into endothelial cells and therein assuming a resting phase; that the parasites may retire into special visceral areas where they are relatively safe from destruction.

We cannot see the necessity for the assumption of any such theories; and our spleen puncture findings fail to shew any such special and as yet unrecognised forms; or any evidence of any process other than destruction of parasites going on in the spleen. Liver puncture was carried out on a case of relapsing quartan malaria; and here the findings were again similar to those in the spleen. The spleen appears to function as the great site of destruction of parasites, and in no way as their reservoir; multiplication by schizogony appears to be restricted to the interior of the erythrocytes, either in the peripheral or in the internal circulation.

It has been estimated by Ross and David Thomson (1910) that the "dose" of trophozoites necessary to produce the clinical febrile symptoms of malaria is approximately 250 million parasites. It is also held in connection with *P. vivax* that the mature schizont produces about 20 merozoites; although we have found experimentally that this figure is too high, and that a mean of 14 merozoites is nearer the mark. Taking the above figures at 250 million and at 20 merozoites per schizont, however, then the progeny of a single injected malarial sporozoite of *P. vivax*, if its schizogony were to proceed uninterruptedly, should be 512 millions on the 18th day after infection; by the 22nd day 204,800 million malarial parasites should be present in the patient's circulation; and the patient should be dead. As, however, the tendency in even untreated cases of malaria is towards spontaneous—even though temporary—cure, it is obvious that there must be a tremendous destruction of parasites in the body; and this takes place in the spleen, possibly also in the other internal viscera. Parasites which have entered into the erythrocytes are safe from destruction, and with them schizogony proceeds undisturbed. Parasites which, for any reason, fail to penetrate into the erythrocytes are swept off into the plasma, are caught in the meshes of the spleen pulp and there destroyed by chromorrhexis, chromatolysis and cytolysis. Also, as the patient becomes resistant to the infection, gametocyte formation sets in; every trophozoite which becomes a gametocyte represents a non-multiplying form introduced into the parasite population; and the formation of gametocytes exerts a brake-like action upon the schizogony cycles.

The rate of destruction of parasites, even in untreated cases of malaria, must—indeed—be tremendous. If we take the above figures for a benign tertian infection of production of 20 merozoites per 48 hours by each schizont; and an approximate number of 512 millions in the circulation at the 18th day after infection; and disregard the brake-like effect of gametocyte production; then, for *P. vivax* infection, a destruction rate of not less than 98 per cent. per 48 hours would be necessary to effect spontaneous cure.

If only 90 per cent. of parasites were destroyed per 48 hour cycle, then at the 20th day the position would be 461 million parasites destroyed; 51 million left; the progeny of this 51 million would be 1,020 million in a further 48 hours, and fever would continue. If 95 per cent. were the destruction rate then at the 20th day 486 million should be destroyed, and 26 million be left; and their progeny in a further 48 hours should number 520 million; a dose still sufficient to cause fever. If 98 per cent. are destroyed, however, then of 512 million parasites present, 502 million would be destroyed, and only 10 million left; and their progeny would only number 200 million in the next 48 hours; a dose insufficient to produce fever.

In brief, therefore, we consider that there is no necessity to postulate either special parasite forms associated with malarial relapses or to assume the passage of parasites into either safe areas or into a resting phase. We consider that the etiology of malarial relapses may be explained upon a purely mathematical basis. If the patient's powers of resistance are such that 98 per cent. of *P. vivax* parasites, for instance, are destroyed per 48 hour cycle, then cure, and probably permanent cure without relapse, is assured. If the destruction rate is lower, and at a figure of only 90 to 94 per cent., the disease is still in its progressive phase. At a figure between 95 and 96 per cent. a condition of balanced equilibrium is reached at which schizogony is still proceeding at the normal rate; but the patient's powers of resistance are sufficient to keep the total number of trophozoites below the febrile threshold; whilst the brake-like factor introduced by gametocyte formation is also important in reducing the infection to afebrile limits. Should the patient's powers of destruction of merozoites become reduced from any adverse or extraneous cause, however, the destruction rate will fall;

the schizogony success rate will be proportionately greater; a febrile dose of merozoites result; and fever recur.

REFERENCES.

Schaudinn, 1902, "Plasmodium vivax, (Grassi und Felliti), der erreger des Tertianfiebers beim Menschen." Arb. aus Kaiser Gesundheitsamte. Bd. 18, p. 169.

Ross, Sir Ronald, F.R.S., and Thomson, David (1910). "Some Enumerative Studies in Malarial Fever." *Annals Trop. Med. and Parasitology*, Vol. IV, p. 267.

Thomson, John D. (1917) "Notes on Malaria." *Journal Royal Army Medical Corps.* Vol. 29, No. 4, p. 379.

A Mirror of Hospital Practice.

A CASE OF INTESTINAL OBSTRUCTION COMPLICATED BY TETANUS.

By F. J. W. PORTER, D.S.O.,

MAJOR, R.A.M.C. (Retired).

Mrs. Y, aged 32, sent for me on July 18th about 1-45 p.m., having been advised to come into my nursing home. There was a history of increasing difficulty in getting her bowels to move for over a year. She had acute intestinal obstruction with frequent vomiting, and the distension of the abdomen was enormous. I did not wait for an ambulance but had her placed in a taxi and removed from her hotel at once, picking up a medical man as my assistant en route. On arrival and before taking her out of the car, I gave one tabloid of hyoscin compound hypodermically.

At 2-15 p.m. I gave spinal analgesia and opened the abdomen in the middle line after previously injecting novocain and adrenalin into the line of incision. I found a hard growth in the bowel at the junction of the sigmoid and rectum which had constricted the gut as though a string had been tightly drawn round it.

The whole of the large intestine and a good deal of the small was hugely distended and appeared likely to burst, so the first thing I did was to incise the sigmoid through a band and give exit to gas and fæcal matter. After emptying as much as possible, the small incision was closed and I adopted a similar procedure in the transverse colon and cæcum. The appendix being diseased was removed. The bowels were replaced and the upper part of the wound closed. The sigmoid just above the growth was sutured to the parietal peritoneum in the lowest part of the wound and a purse string suture placed in the bowel. I had hoped that I had emptied the whole of the large bowel and did not therefore clamp it above the intended site of incision. As soon however as I had incised the gut inside the purse string, an enormous amount of fluid fæces escaped and it was fully 15 minutes before I was able to insert a large glass Paul's tube and tie the suture.

I had made no attempt of course to excise the growth and unite the bowel for, on account of the tremendous thickening and œdema of the wall of the gut, any suture would inevitably have become loose within 12 hours, with a fatal result from leakage into the peritoneal cavity. The patient slept throughout the operation and her condition at the end of it was extraordinarily good.

Great quantities of fluid fæces escaped into a kidney tray and it was at least 7 days before the bowels had fully emptied themselves. There was no difficulty in keeping the patient clean so long as the glass tube remained in the bowel, but after it had come out incessant labour was involved by the constant escape of fluid fæcal matter into the cotton wool dressings. The diarrhœa which always ensues in these cases added to the difficulty.

Pituitrin in ½ c.c. doses was given intramuscularly every three hours for the first three days and an injection was invariably followed by a large escape of fluid within half an hour.

Next morning I started continuous drip saline and glucose with brandy, but although my apparatus is a perfect one I could not succeed in getting the patient to retain it.

She now began to vomit in an effortless manner and obviously had post-operative hæmatemesis as well as some dilatation of the stomach. I therefore propped her up and passed a stomach tube, giving exit to a lot of gas and a great deal of dark fluid. The stomach was washed out with hot bicarbonate of soda solution until the washing became clear. The vomiting ceased at once and she became quite comfortable. After 12 hours it returned and I repeated the washing. Next morning she was vomiting again, but as she dreaded the passage of the tube, I allowed her to drink large quantities of hot soda solution and wash out her own stomach in this way. Although the pulse remained of good volume and tension I thought it advisable to get fluid into her intravenously and gave her 2 pints of saline-glucose-brandy and adrenalin in this way. She was already so uncomfortable that I did not want to add to her troubles by giving it subcutaneously.

This was repeated three times in all and to the last lot I added a preparation named "New-Hormonal" which had been supplied to me by Merck & Co., in answer to my request for an intravenous purgative. I have been for many years seeking something which could be given in this way and every surgeon who does much abdominal work must long at times for something which can be relied upon to act on the bowels, in cases of post-operative distension accompanied by vomiting, in which it is impossible to give anything by the mouth and where it is of vital importance to get an evacuation.