pylorus feeling like a solid cylinder. This thickening spread on to the duodenum where its advancing edge could be felt and gave a sensation of cartilage to the touch. The glands along the lesser curvature were large. The pylorus and adjacent infiltrated tissues had a semi-translucent appearance as if they were cedematous. The whole mass was suspended from the liver by adhesions. The posterior wall of the stomach was drawn through the gastro-colic omentum and an anastomosis with the jejunum made by simple suture as in the other cases. The patient vomited a little mucus on returning to the ward, after which she was never sick again. The sutures were removed on the ninth day. On the twelfth day she had some fish. At the end of three weeks she got up. She then weighed six stones ten pounds. The tumour at this date was scarcely perceptible on palpation. I last saw her on March 24th, 1902. She weighed eight stones one pound and was perfectly well.

My first impression in this case was that the tumour was malignant. The very short history, without any previous illness suggestive of ulcer, and the large size of the tumour, which was apparently only three months old, certainly favoured this view. Against it was the early age of the patient and the presence of free hydrochloric acid. When the abdomen was opened, however, the appearances were much more characteristic of inflammatory rather than malignant disease. The infiltration of tissue was evidently spreading from left to right, showing no tendency to invade the stomach itself. The old scar in the stomach wall was apparently the result of an ulcer long since healed. In the pylorus itself a recent ulcer probably existed, but without actually opening it I could not absolutely prove its presence. I was unable to introduce my finger into the pylorus from the stomach cavity. The rapid disappearance of the tumour was the most remarkable instance of this phenomenon which I remember to have seen, occurring as it did within a month of operation.²

CASE 4. *Duodenal ulcer (?) ; gastric dilatation ; posterior gastro-jejunostomy through the gastro-colic omentum ; recovery.*—A man, aged 43 years, was admitted into the Bradford Royal Infirmary under the care of Dr. Campbell on Sept. 24th, 1901. He gave a history of "stomach trouble" of four years' standing. He said that he had "more or less constant" epigastric pain varying in severity. Pain came on about one and a half hours after food and was for a time very severe. It gradually abated but was renewed at the next meal. He only vomited occasionally and the material ejected was not very large in quantity. There had never been any hæmatemesis. He had lost three stones in weight in the past three years. On admission his condition was as follows. He was very thin and his expression was pinched and careworn. The tongue was very dirty and the breath was offensive. There was no abdominal tumour but the stomach was dilated, showing peristalsis on palpation. There was a well-marked succussion splash. The stomach held four pints easily. The contents obtained by the stomach tube were fermenting. Free hydrochloric acid was present. There was some rigidity and resistance of the abdominal wall over the pyloric region. Six weeks' medical treatment combined with lavage produced only slight improvement and on Nov. 11th Dr. Campbell transferred him to my care and three days later I opened the abdomen under ether.

The stomach was reached through the usual incision. It was considerably dilated but there was no pyloric tumour. Beyond the pylorus, however, there were some dense adhesions round the duodenum very suggestive of the presence of duodenal ulcer. As the patient was very feeble I refrained from further exploration and at once performed a posterior gastro-jejunostomy in the same manner as in the preceding cases. He recovered rather slowly from the operation. There was no vomiting after the first two hours. His pulse, however, remained small and rapid for a day or two and he seemed to lack the power of recuperation. After the first week he improved and a fortnight later got out of bed. He then weighed eight stones seven pounds. Before leaving hospital a fortnight later he weighed nine stones five pounds. He was very well and had lost his grey ashy complexion and dirty tongue.

In this case I omitted to excise the redundant gastric mucous membrane when making the anastomosis and the sequel shows the importance of this step. About a month

after he left the hospital he complained of vomiting occasionally after meals and I readmitted him so as to have him under observation. The following facts disclosed themselves. He was perfectly well for a time and was quite conscious of food passing through the artificial opening. Then, without any apparent reason he found that the stomach did not empty itself after a meal. This usually occurred after an ordinary breakfast. He then had a good deal of pain and ultimately vomited, when he was at once relieved. During an attack there was a well-marked succussion splash. Sometimes he would go a week without one of these attacks, or, on the other hand, he might have them two days running. In the intervals washing out the stomach showed it to be empty, the fluid returning quite clear and not increased in quantity. He had attacks much more frequently when he took only liquid diet. He was distinctly better on solid food and when he did not drink at meals. On two occasions the vomited matter contained some blood. These observations show clearly, I think, that there was some occasional accidental blocking of the artificial opening and I think there can be little doubt that this was due to a fold of mucous membrane. Since his second stay in the hospital these attacks have become less and less frequent and are apparently ceasing altogether. He is putting on weight and looks very well.

In conclusion, I would urge a trial of this method of performing the posterior operation. It is an easy and rapid method and combines the advantages of both the anterior and posterior routes. It certainly provides most efficient drainage of the stomach even when the patient is lying on his back. On two occasions I have noted fluid passing from the stomach into the intestine immediately the clamps have been removed. I am not aware that this route has been previously used by other surgeons, but in any case I would urge a trial of it as I feel confident that it possesses great advantages. On the other hand, so far as my limited experience of it goes, I have found no objection to the method.

Bradford.

NOTE ON THE DIAGNOSTIC AND PROGNOSTIC VALUE OF THE LEUCOCYTE VARIATIONS IN ASIATIC CHOLERA.

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DURING the last six months I have made some examinations of the blood in cases of cholera in order to determine if any guidance in prognosis and treatment could be derived from the conditions met with, more especially as regards the use of transfusion. Although the cases so far examined are much too few in number to allow of any final conclusions being drawn on this large question, yet in the course of the inquiry certain variations in the leucocytes have occurred so constantly as to appear to have a diagnostic value of their own which may be worth drawing attention to.

In Table I. blood estimations in 23 cases of cholera are given and in Table II. will be found six cases of non-choleraic diarrhoea of various kinds and of such severity that the patients were admitted into the cholera observation ward as suspected cases of true cholera. These last all subsequently proved not to be cases of cholera, but they so closely resembled the specific disease clinically as to be just the class which is most difficult to diagnose from cholera in practice, and hence they serve as good controls for purposes of comparing the blood changes. In 15 of the cholera cases stools were submitted to a bacteriological examination, and in 10 of them pure cultures of comma bacilli were obtained. In the others similar bacilli were seen under the microscope, but pure cultures were not obtained owing to their being mixed with so many other varieties, the stools being faecal in character in these cases, having been taken in a late stage of the disease. When a typical rice-water stool was obtained there was usually no difficulty in isolating comma bacilli. The cultural characters of those isolated varied greatly, as in Cunningham's well-known observations in Calcutta, but on preparing a serum

² This patient was seen in June, 1902. There was then a large malignant mass in the abdomen with all the accompanying signs of carcinoma. There was absence of free hydrochloric acid.

by subcutaneous inoculation of one of them into a rabbit everyone of the other varieties clumped typically with it, although a comma bacillus obtained from water failed to do so. This points to all the different varieties being specific cholera organisms, an interesting point which forms part of my larger inquiry and cannot be further dealt with here. In the first two cases of Table II. no comma bacilli could be isolated, but material for examination was not received from the other cases.

The red corpuscles and hæmoglobin.—All the patients in Table I. were Hindus or Mahomedans except the patient in Case 15 who was a Chinaman. In the first line of the table the normal figures for healthy natives of India belonging to the poorer and ill-fed classes are given, the hæmoglobin in particular being much lower than the European standard, as I pointed out several years ago, being only on the average 70 per cent. of Gowers's standard. On the other hand, the number of red corpuscles is about 5,000,000, so that the hæmoglobin value is only 0.7. In such a severe form of diarrhoea as occurs in cholera the number of the red corpuscles and the percentage of hæmoglobin both rise with the concentration of the blood and should serve as a guide to the amount of fluid lost. The estimation of the specific gravity of the blood would be a more rapid way of arriving at the same result, but it was only carried out in a few cases. In two of the cases the number of red corpuscles rose to over 8,000,000 per cubic millimetre and both patients died, but one patient out of three cases in which they reached between 7,000,000 and 8,000,000 recovered, as did one of the patients (Case 1) in Table II. On the other hand, four patients out of five cases in which they numbered 5,000,000 and under also died, while of 11 cases in which they were between 5,000,000 and 7,000,000 seven patients recovered and only five died. There does not, therefore, appear to be any constant relationship between the degree of concentration of the blood as indicated by the number of the red corpuscles and the death-rate. The hæmoglobin varied closely with the red corpuscles, the hæmoglobin value being about normal in the majority of the cases, although somewhat high in a few of them. In only one was it below normal—namely, in Case 16—and this patient had hæmorrhagic stools.

Leucocytosis.—It has long been known that leucocytosis is a well-marked condition in Asiatic cholera, while it is said to be most marked in the severest and most fatal cases. These statements are borne out by my observations, leucocytosis having been present in every case which was examined in the earlier stages—that is, within the first two days of the disease. In nine cases it was present in comparatively slight degree, less than 20,000 leucocytes per cubic millimetre, and five of these patients recovered.¹ On the other hand, out of 14 cases in which this number was exceeded 11 patients died and only three recovered, these three, however, including two out of the three cases in which over 40,000 leucocytes were found per cubic millimetre. A high degree of leucocytosis is, then, a bad prognostic sign and a slight degree a good one, yet a very high degree is not incompatible with recovery. The constancy of the occurrence of leucocytosis in cholera is of importance in distinguishing it from some forms of acute diarrhoea in which it is absent, as in most of the cases in Table II. It is not, however, of absolute value, as leucocytosis occurs in ptomaine poisoning and sometimes in cases of acute dysentery beginning with severe diarrhoea, Cases 3 and 1 in Table II. being cases in point, and it is just these which for a time present the greatest difficulty in diagnosis.

The differential leucocyte count in cholera.—While working recently at the blood changes in plague I found in some of them a peculiar form of leucocytosis characterised by an unusually large proportion of lymphocytes in proportion to the degree of total increase of the white corpuscles. Thus, with a total count of from 20,000 to 60,000 white corpuscles, instead of finding from 80 to 90 per cent. of polynuclear cells and only about 10 per cent. of lymphocytes, the polynuclear cells showed little or no increase above the normal percentage, but the lymphocytes formed from 20 to 30 per cent. of the whole, a condition which I have not met with in the leucocytosis of inflammatory conditions or in such diseases as pneumonia or cerebro-spinal fever. Unfortunately,

neither a marked increase in the total leucocyte count nor this peculiar proportion of the different kinds occurs at all constantly in plague, so that I have not found these changes of much practical value in differentiating plague from other diseases, as a negative result is of no value in excluding plague, although I think the finding of the condition mentioned may be of value in confirming such a diagnosis. These observations led me to make similar differential counts in cholera cases, 500 corpuscles always being counted, with the result that much more constant changes of a different nature were met with, which I have found to have great diagnostic value in the limited series of cases which have so far been examined. In cholera the percentage of polynuclear cells is increased to somewhere about 80 per cent., but it seldom rises much above that point. The lymphocytes are very markedly decreased, having been under 10 per cent. in 13 out of 17 cases examined on the first or second day of the disease. On the other hand, the large mononuclear cells are not only decreased proportionally with the decrease in the lymphocytes, but are usually increased above the normal percentage as well as absolutely. The result of these two coincident changes is that the large mononuclear cells usually outnumber the lymphocytes often to the extent of two to one, instead of being in the normal proportion of one to five. This peculiar change occurring in a leucocytosis I have not met with in any other disease than cholera. Moreover, the increased percentage of the large mononuclear cells becomes more marked as the disease progresses, especially in fatal cases. The marked character of this change is still better seen by taking the total number of the different forms of leucocytes, as shown in columns 15 to 17 of Table I. The polynuclear cells show a great increase in proportion to the degree of leucocytosis. The lymphocytes show no proportional increase, being actually below the normal in many cases and only showing a comparatively slight increase in those cases which in the leucocytosis is of a very marked degree. On the contrary, the large mononuclear cells show an extraordinary increase, being not infrequently more than 10 times the normal number. Case 12, in which no leucocytosis was present when examined on the fourth day, was one of convalescence from cholera in a malarial subject with marked anæmia, but even here the percentage of large mononuclear cells is very high and the total number is double the normal. The constancy of this decrease of the lymphocytes and great increase of the large mononuclear cells in this series of cholera cases is very remarkable and is in marked contrast with the cases of non-choleraic diarrhoea in Table II., for in not one of these last is either the percentage or the total number of large mononuclear cells increased above the normal limits in spite of leucocytosis being present in two of the cases.

It will be advisable to mention here the nature of the cases in Table II. in which all of the patients were admitted into the cholera observation ward as possibly suffering from cholera. The first two patients were sent by the port medical officer as being suspected cases of cholera and were examined by me very shortly after admission, when the subject of Case 1 in particular presented the general aspect of a patient suffering from cholera. The higher number of red corpuscles and the percentage of hæmoglobin in Case 1 were equal to those met with in severe cases of cholera and a slight degree of leucocytosis was present. The differential leucocyte count, however, of both cases failed to show the increase of the large mononuclear cells, which at that time had been found in 17 consecutive cases of cholera, and I thought that I had met with exceptions to the general rule. On the next day it appeared that the patients were suffering from very acute attacks of dysentery and they were removed from the cholera observation ward. In Case 3 the patient was taken ill 10 hours after a meal at a Bengali hotel and was admitted soon after as a case of cholera. He had profuse diarrhoea and sickness, but all the stools were coloured brown and were never typical of cholera and he made a good recovery, the physician in charge subsequently coming to the conclusion (without knowing the result of the blood examination) that the case was one of ptomaine poisoning and not cholera. Case 4 was similar to the last, only it proved fatal, but no history could be obtained and unfortunately the stools of these last two patients were not sent for bacteriological examination. It is therefore just possible that they may have been cholera cases which did not show the blood changes described in this paper, but in the absence of exact proof I can only accept

¹ In one of the fatal cases granular kidneys were found. This is of interest in connexion with the fact that in 70 post-mortem examinations on cholera cases which I analysed recently I found granular kidneys to be much more frequent than they were in the cases of those dying from other commoner diseases, showing that if a patient had granular kidneys there was very little chance of his surviving an attack of cholera.

TABLE I.—CASES OF ASIATIC CHOLERA.

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)
Number of case.	Caste.	Sex.	Age.	Result.	Day of disease.	Hæmoglobin.	Red corpuscles.	White corpuscles.	Ratio of white to red corpuscles.	Percentages.				Total number per cubic millimetre.		
										Polynuclear cells.	Lymphocytes.	Large mono-nuclear cells.	Eosinophiles.	Polynuclear cells.	Lymphocytes.	Large mono-nuclear cells.
Normal for natives						70	5,000,000	7,500	1-666	68·0	25·0	6·0	1·0	5,100	1,875	450
1	H.	M.	30	D.	3	130	7,910,000	26,000	1-304	79·6	6·8	12·2	1·4	20,696	1,768	3,172
							8,200,000	24,000	1-335	82·2	4·2	12·8	0·4	20,287	1,029	3,086
2	H.	F.	50	D.	1	107	6,180,000	33,000	1-187	83·0	5·2	11·4	0·4	26,456	1,716	4,752
3	H.	M.	18	D.	3	107	4,500,000	19,000	1-237	80·2	6·8	12·8	0·2	15,638	892	2,432
4	H.	M.	45	R.	2	114	6,200,000	15,000	1-413	87·0	4·8	8·2	—	13 050	720	1,230
5	H.	F.	28	D.	2	—	3,160,000	26,000	1-122	85·2	6·4	7·8	0·6	22,152	1,664	2,028
6	H.	M.	36	R.	1	109	6,790,000	24,000	1-283	69·0	11·8	17·4	1·8	16,560	2,832	4,176
7	M	M.	28	R.	2	91	5,100,000	18,750	1-273	72·8	15·2	11·2	0·8	13,650	2,848	2 099
8	H.	M.	32	D.	1	85	4,890,000	15,250	1-321	79·2	7·4	13·4	—	13,278	1,248	2,043
9	H.	M.	30	D.	3	74	5,200,000	30,750	1-161	63·8	16·8	19·0	0·4	19,618	5,116	5,842
10	H.	M.	16	D.	5	81	5,000,000	15,750	1-318	80·8	8·6	10·2	0·4	12 726	1,354	1,606
11	H.	F.	30	R.	4	97	6,810,000	12,250	1-555	79·4	10·2	9·8	0·6	9,726	1,249	1,200
12	H.	M.	33	R.	4	60	2,520,000	2,250	1-1120	38·8	23·6	37·6	—	873	531	840
13	H.	M.	30	D.	3	120	7,690,000	30,000	1-256	76·8	10·4	12·8	—	23,040	3,120	3,840
14	M.	M.	35	R.	2	114	6,070,000	10,000	1-597	85·4	7·4	7·0	0·2	8,540	740	700
					7	102	6,070,000	4,000	1-1517	49·6	26·0	22·8	1·6	1 984	1,040	912
15	C.	M.	35	D.	2	102	6,430,000	16,500	1-390	76·2	8·8	15·0	—	12,573	1,452	2,475
					6	99	6,390,000	13,500	1-473	82·4	8·0	9·6	—	11,124	1 080	1,296
16	H.	M.	30	D.	—	73	5,790,000	30,250	1-191	78·8	7·0	14·0	0·2	23,837	2,117	4 235
17	M.	M.	35	D.	—	112	6,190,000	25,750	1-210	64·0	15·0	21·0	—	16,480	3,862	5 407
18	H.	M.	40	D.	2	103	5,120,000	28,500	1-180	76·4	10·0	13·6	—	21,774	2,850	3,376
19	M.	M.	35	R.	2	111	7,270,000	53,250	1-136	88·0	4·0	8·0	—	46,860	2,130	4,260
					4	107	6,560,000	27,250	1-240	87·4	3·8	8·8	—	23,816	1,035	2,398
					9	103	6,990,000	14,250	1-490	79·6	8·8	11·2	0·4	11,343	1,254	1,596
20	H.	M.	33	D.	3	109	6,840,000	33,000	1-205	70·0	9·2	10·2	0·6	23,100	3,036	3,366
21	H.	M.	30	R.	1	111	6,330,000	43,000	1-148	84·8	5·2	10·0	—	36,464	2,236	4,300
					9	102	5,640,000	18,000	1-313	77·0	9·6	13·0	0·4	13,860	1,728	2,340
22	H.	M.	25	D.	1	116	8,420,000	55,000	1-153	78·2	8·2	13·4	0·2	43,010	4,510	7,370
23	H.	M.	22	D.	2	94	6,550,000	37,000	1-177	83·4	6·8	9·6	0·2	30,858	2,516	3,552

TABLE II.—CASES OF ACUTE DIARRHŒA OTHER THAN CHOLERA.

(1) Number of case.	(2) Caste.	(3) Sex.	(4) Age.	(5) Result.	(6) Day of disease.	(7) Hæmoglobin.	(8) Red corpuscles.	(9) White corpuscles.	(10) Ratio of white to red corpuscles.	Percentages.				Total number per cubic millimetre.		
										Polynuclear cells.	Lymphocytes.	Large mono-nuclear cells.	Eosinophiles.	Polynuclear cells.	Lymphocytes.	Large mono-nuclear cells.
1	M.	M.	30	R.	6	105	7,420,000	12,500	1-593	87·0	9·0	4·0	—	10,875	1,125	500
2	M.	M.	28	R.	6	90	5,240,000	9,000	1-582	86·8	10·4	2·8	—	7,812	936	252
3	H.	M.	45	R.	1	92	6,190 000	20,500	1-302	93·8	4·0	2·2	—	19,229	820	451
4	H.	M.	11	D.	1	87	5,930,000	8,000	1-741	81·2	12·4	6·2	0·2	6,496	992	496
5	E.	F.	26	R.	1	—	3,280 000	3,750	1-857	58·6	32·8	14·4	1·2	2,197	1,230	540
6	E.	M.	50	R.	1	83	4,610 000	7,750	1-593	61·2	31·2	4·4	3·2	4 723	2,418	341

the conclusion of Captain Delany, M.B., I.M.S., that clinically they were not cholera cases, and I may also take this opportunity of thanking that officer for kindly allowing me to examine his cases. The patient in Case 5 was a woman who while suffering from malarial fever took two pills, after which she was violently purged 11 times and also suffered from vomiting. She was admitted into the cholera observation ward and subsequently was transferred to the general ward, where she had malignant tertian fever with ring-shaped parasites. Her blood while suffering from the diarrhoea showed the typical low total leucocyte count, with high percentage of large mononuclear cells, which is characteristic of malaria, together with a high percentage of lymphocytes, illustrating well the difference between the high large mononuclear counts of cholera and malaria respectively. Case 6 was another case of ptomaine poisoning following the consumption of prawn curry late at night, the patient having also been admitted into the cholera observation ward, but she subsequently proved not to be suffering from cholera. This series of cases of non-choleraic diarrhoea is a very small one, but they form the whole of the cases admitted as suspicious of cholera during the same period as the true cholera cases in Table I occurred, came under observation in the Medical College Hospital, and hence are just the cases which it is most difficult to differentiate from cholera. The fact that none of them show the changes met with in all the cholera cases strongly supports the diagnostic value of the peculiar leucocyte count of cholera described above, although it will be necessary to examine a much larger series of cases before the exact limitations of this new method of diagnosing cholera can be established. This leucocyte test has at least the great advantage that it can be carried out within an hour or so with only the aid of a slide, a simple stain, and an ordinary high-power microscope (a one-sixth inch lens); whereas the bacteriological diagnosis takes a day or two with the aid of a properly-equipped laboratory, while, owing to the great variability of cholera comma bacilli, both in their power of liquefying gelatin and in the presence or absence of the indol reaction, considerable experience is required for its safe application, including, in order to be quite certain, the carrying out of the serum test.

The prognostic value of the leucocyte variations in cholera.—If the leucocyte variations in cholera above described are of a specific nature then it might be expected that their degree might bear some relationship to the severity and mortality of the disease. The following data are of interest in this connexion. We have already seen that when the leucocytosis is of less degree, with a correspondingly low total polynuclear count, the prognosis is comparatively good. In the case of the leucocytes, on the contrary, we have seen that in cholera a very low percentage and a comparatively low total count is the characteristic feature. On classifying the cases according to the percentage of lymphocytes met with we find that out of 16 cases in which less than 10 per cent. were found no less than 12 ended fatally, while out of seven cases in which over 10 per cent. were present four patients recovered and only three died. On the other hand, there is no such relationship between the total number of lymphocytes per cubic millimetre and the mortality, which is what might have been expected, as the tendency of the high degree of leucocytosis in fatal cases to increase the number of lymphocytes is counteracted by the opposite tendency of the low percentage of lymphocytes in severe cases. Lastly, on turning to the large mononuclear corpuscles we find that out of six cases in which under 10 per cent. were found four patients recovered and only two died, while, on the contrary, out of 15 cases in which over 10 per cent. of large mononuclear cells were present only three patients recovered and 12 died. The total number of large mononuclear cells per cubic millimetre shows a similar relationship, as in this case the favourable low degree of leucocytosis coincides with the equally favourable low percentage in reducing the total number of these corpuscles and *vice versa*. Thus we find that recovery took place in no less than four out of five cases in which the total number of large mononuclear cells was less than 2000 per cubic millimetre, while out of 18 cases in which over 2000 were present 14 patients died and only four recovered. We therefore conclude that there is a definite relationship between the decrease of the percentage and the total number of large mononuclear cells and the severity of the disease—a relationship of the very peculiar leucocyte changes described which it would be difficult to explain on

any other ground than that these changes are of a specific nature. This fact, taken with the constancy of these changes in the consecutive series of cases examined and their absence in cases of non-choleraic diarrhoea, encourage one to believe that a larger experience will confirm the conclusions derived from a study of the data recorded in the tables of this paper—namely, that the leucocyte changes described above have a considerable value as a simple and rapidly applied guide in both the diagnosis and prognosis of cholera.

Calcutta.

ENVIRONMENT AS A CAUSE OF AGUE.

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THE proceedings of the Malaria Congress recently held at Nagpur, Central India, under the presidency of Colonel A. Scott Reid, I.M.S., were full of interest and have led me once again to invite the attention of the profession to meteorological environment as a probable cause of those cases of malarial fever in which parasites cannot be discovered in the blood. In a paper read at the congress Dr. Stephens and Dr. Christophers stated that cases occurred, with fever clinically identical with malarial fever, in which parasites could not be found in the blood, after repeated search, before quinine had been given. It was also stated that in large numbers of cases of fever with enlarged spleens examined in Calcutta, the authors had seldom found the parasite in peripheral blood, although pigment was sometimes obtained by spleen puncture. Instances of failure to find the parasite in undoubted cases of malarial fever were narrated and every speaker at the congress affirmed the malarial nature of the cases of chronic fever with enlarged spleen. It would appear from this that under the term "malarial fever," as it is understood at present, are included two clinically identical, yet etiologically distinct, fevers which for convenience may be referred to here as (1) parasitic malarial fever and (2) non-parasitic malarial fever. The diagnosis of cases of the first class would, of course, rest on the recognition of the parasite in the blood by experienced observers, for, as Manson says, "negative results by amateurs are valueless, and a positive result is not much more to be relied on."¹ The second class would include all fevers clinically identical with malarial fever in which parasites cannot be discovered in the blood. It is to fevers of the latter class alone that I refer in this paper. Such fevers are, in my opinion, obviously and indisputably due to the meteorological environment under which they arise. By this environment I mean the hot, moist, stagnant atmosphere under which they become most prevalent and which was for many years considered a sufficient explanation of their cause. First, as regards this environment it is obvious that it may be of a higher degree of intensity in some places than in others, in some years than in others, and in some seasons than in others, but I hope to demonstrate that the inevitable effect of exposure to such environment is to produce an intermittent excess of water in the blood. This intermittent excess of water in the blood will be seen to afford a clear explanation of the intermittent pyrexia, the anæmia, the melanaemia, the enlargement of the spleen, and the other conditions characteristic of malaria.

I.—THE METEOROLOGICAL ENVIRONMENT UNDER WHICH INTERMITTENT FEVERS BECOME PREVALENT.

The meteorological conditions under which intermittent fevers become prevalent have long been known and are indicated in the attached chart which I have constructed from figures contained in the annual report of the Army Medical Department. It shows at a glance the difference between a malarial and a non-malarial environment. Where intermittent fever becomes prevalent, as at Singapore, both the atmospheric temperature and the atmospheric humidity are high, the mean atmospheric temperature being above 80° F., and the mean atmospheric humidity considerably above 80 per cent. of saturation throughout the year. On the other hand, where intermittent fevers are not prevalent, as in England or at Barbados, the mean atmospheric temperature or the mean atmospheric humidity, or both, will

¹ Brit. Med. Jour., May 17th, p. 1212.