of the blood may not appear to be those of malaria. In this case the 12 per cent. mononuclear count may be apparently absent, though not really so. Thus a total white blood corpuscle count of 12,000 may give but 8 per cent. of large mononuclears. Still, this would represent a very high percentage for 12,000 white cells, and probably equals about 16 per cent. large mononuclears for a white corpuscle count of 6,000 per c.mm. A number of my cases gave the mononuclear count in spite of leucocytosis, such as Nos. 39 and 79, Table I; and 44, 125, 146, and 161, Table II. On the other hand, considering the high total white corpuscle count in Nos. 160 and 165, the 8 per cent. large mononuclear counts given in these cases is more than sufficient to justify one in calling the cases malarial.

3. If the temperature is up to 103° or more at the time the blood is taken the malaria count may be absent. As it is not always possible to be present at the period of remission in the fever, and should the temperature be above 103°, it is advisable not to take the blood until remission occurs.

Is a Leucocytosis ever found in Uncomplicated Malaria?

Cases 160, 161, 165, and 166, Table II, were examples of very severe malaria with leucocytosis. In all of them parasites were very numerous in the peripheral blood. In the diagnosis of liver abscess from malaria by the blood count, leucocytosis in the former is so constant as to be almost diagnostic. To this I would add that these four cases furnish the exception "that proves the rule." These cases would seem to indicate, however, that when this exceptional leucocytosis does occur parasites are so numerous in the blood that diagnosis from a fresh specimen is an easy process.

Leucocytosis in malaria seems to be comparatively rare, as it has not been much noted before. Dr. Manson, in his book on *Tropical Diseases*, pp. 98 and 99, edition 1900, mentions that an increase to as high as 30,000 may occur in the white cells in certain pernicious malarials, and Captain Rogers³ mentions the possibility of it at certain stages of the malaria paroxysm.

These were all severe cerebral cases, and were either very delirious or comatose. They were admitted into the contagious ward at first as suspected cases of plague, and clinically there was nothing to distinguish them from septicaemic plague. They occurred during the month of September, when our most severe pernicious malarials occur in India. Immediately on admission I examined the bloods, and, finding parasites in the fresh specimens, at once transferred them to the general ward.

If I might be permitted to theorize on the point, I would say that leucocytosis in those severe malarials indicates a reaction of the blood to the intense poisoning, and that in these same severe cases the leucocytosis varies directly with the intensity of the poison. The converse to this latter rule no doubt holds good—namely, that the more chronic the malaria the more marked the leucopenia, as in Case 63, Table I, and Cases 85 and 112, Table II, which were markedly cachectic from malaria, and all died.

Case 143, Table II, deserves special mention, as it very well exemplifies the diagnosis from blood count alone of malaria. The patient, a European policeman of good physique, was admitted to the plague ward as a suspected case of mild plague. He had a well-marked right femoral bubo, temperature 105°, with severe constitutional symptoms, though not delirious. There was no wound on the leg or foot and no sign or history of venereal disease. Fresh specimens carefully examined showed no malaria parasites, but the blood count was so typical that I transformed him to the general ward. His temperature fell in two days under quinine, with occasional intermissions every third day afterwards. The bubo disappeared.

Value of Blood Counts in the Diagnosis of Post-operative and Post-puerperal Fevers in the Tropics.

Cases Nos. 83 and 146, Table II, were examples of what a blood count can effect in the diagnosis of post-puerperal fevers. The nature of those cases was in doubt, and with this of course the proper line of treatment uncertain, up to the time the blood counts were done. On subsequently giving quinine the temperatures came down almost at once. No. 83 had had fever for four days, and 146 for five days after miscarriage and confinement respectively.

Cases 128 and 185, Table III, were markedly in contrast with the foregoing. Case 128 was thought to be possibly malarial and quinine given, but she died of puerperal fever. Here, again, the blood count foretold the condition at an early date.

The patient, Case 185, Table III, was delivered ten days before the blood examinations, and had had fever since her confinement. She looked very ill, and might have been clinically either a severe malaria, typhoid, or puerperal case. It will be seen that the leucocytosis is exceeding high (the highest, in fact, of any of my blood counts). I gave the opinion from the blood count that she was suffering from septicaemia (there were no local signs whatever), and I recommended antistreptococcus serum. In two days after the injections of five phials of the serum the temperature fell to normal, and remained so for seven days more. On the next day (eighteenth day since confinement) the temperature ran up again to 103°, with rigor; and on examining the blood I got the following results: Red cells, 2,528,000; white, 18,000; ratio, 1—140. Polynuclears, 70 per cent.; lymphocytes, 19.3 per cent.; mononuclears, 10.7 per cent. In spite of some leucocytosis still present it will be seen that the large mononuclears are very high. Fresh specimens showed no parasites. I gave it as my opinion that the patient was now suffering from malaria. She got no quinine, but her temperature fell to normal. Two days later the fever was repeated, with rigor, hot stage, and sweating. Quinine was now given, and the patient got well. In my opinion this patient's life was probably saved by the blood count.

Case 148, Table III, also deserves mention. She was operated on by Caesarean section seven days before the blood examination, and had had fever for four days. I concluded that the blood condition was indicative of some local inflammatory process, and suggested "stitch abscess." This was carefully sought for and found, and on removing the offending stitch the temperature fell, and the patient completely recovered.

Diagnosis of Liver Abscess from Malaria.

It is no unusual occurrence in India to get cases of fever, with pain, tenderness, and enlargement of the liver, in which large doses of quinine is the proper line of treatment, though exploration of the liver seems clinically to be indicated. Cases 22, 28, 45, and 132, Table II, are examples. The livers were carefully explored without success in Nos. 28 and 132. No. 28 recovered under quinine. No. 132 was taken out of hospital four weeks later than the blood count, in a dying condition. Nos. 22 and 45 recovered under quinine. Contrast the blood counts of those cases with Nos. 26, 32, and 34, Table III, which were clinically not unlike malaria remittents, without local signs, at the time of the blood counts. All were subsequently successfully operated on for liver abscess.

I cannot conclude this paper without owning my great indebtedness to Captain Leonard Rogers, I.M.S. Without the help he gave on all occasions when I sought it, I should probably never have had the patience to persevere until I possessed the skill necessary to examine the blood properly and interpret the results.

Finally, I would repeat, for the benefit of those who wish to test the value of blood counts, especially in malaria, that the skill to examine the blood and to interpret the results will only be obtained by repeated examinations of normal bloods, and that the greatest difficulty to surmount is in differentiating large mononuclears.

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BERI-BERI AMONG LASCAR CREWS ON BOARD SHIP.

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BERI-BERI among Lascar crews is a common occurrence, and it may therefore be interesting, in view of the controversy as to its etiology, to look at what is likely to be the cause of the disease on board ship.

Passing over the predisposing causes, such as occupation, age, sex, climate, and overcrowding—and the latter does not occur on board ship (P. and O.), as the Lascars have the same superficial and cubic space as is allowed to European seamen—we come to the specific causes.

The anaemic theory is exploded, the cockroach theory has also little ground, as although they are numerous, yet they have practically no chance of getting infected from faeces, as the latrines are too well looked after. This also may be said of the metallic theory, since the only way of infection would be through cooking utensils. One is therefore drawn by a

process of exclusion to fall back on the rice and germ theories.

I am inclined to favour the view that rice is a prime factor in the disease, and that probably the germ, whatever it is, is intimately connected with the rice. I am certainly of the opinion that rice alone will not give rise to beri-beri, for beri-beri is unknown among the rice-eating millions of Bengal. Still we must remember that rice is the staple diet of the Lascars, and when this diet is changed and they are taken from the infected area, Dr. Manson admits himself they will recover (if not dead within a fortnight).¹

The following is the scale of provisions for Lascars or other native seamen as per Board of Trade regulations:

	lb.	oz.	drms.
Rice	...	8	0
Dhal	...	4	0
Ghee	...	2	0
Salt	...	0	8
Salt fish at sea	...	4	0
Curry stuffs	...	0	10
Fresh meat in harbour	...	4	0
Vegetables	...	8	0
Tea	...	0	4
Sugar	...	0	8
Lime juice after being ten days at sea	...	1	0

I think the burden of evidence is distinctly in favour of the rice theory, and many papers have been written to support this view. In the Japanese army and navy in 1884-1885 beri-beri was a serious complaint, but it was almost entirely eliminated by the improvement in diet, the conclusion being reached that it and rice were intimately connected, and that lack of nutritive substance was the cause, and consequently the introduction of barley into the diet along with rice caused a speedy diminution of the disease, and that no other hygienic condition had anything to do with the result.

Then, again, there are Captain Rost's investigations,² in which he examined rice liquors from different sources and of different kinds, and he found organisms in these liquors which proved identical.

Then, again,³ example is given of outbreak of beri-beri on board a ship, where the disease resisted all treatment until the rice was thrown overboard and another fresh lot taken in, and then it stopped. As against this theory there is quoted the outbreak of beri-beri in the Dublin Asylum,⁴ but it is questionable whether this was beri-beri; also an instance of beri-beri breaking out among European officers.⁵

The two following cases are of interest. The first, as illustrative of the danger which young and inexperienced surgeons in tropical diseases are apt to fall into in giving a favourable prognosis in a case of beri-beri on board ship; the second, because it looked as if the patient had developed beri-beri by direct contagion, a view which is not upheld by any of the authorities on beri-beri.

CASE I.

A Lascar reported sick on the morning of October 1st, 1900, on voyage home from Calcutta.

History.—He complained of pain in the calves of his legs, and said he felt tired and unable to work. Serang said he was a good worker, and was not lazy.

Present State.—Pulse 84, small, compressible, regular. Respirations 16; temperature 98°. Bodily conformation only fair; he was rather thin, medium height. Visible mucous membranes normal. There was slight oedema over the flat of both tibiae, but none anywhere else. There was a certain amount of anaesthesia over the front of the tibiae, but it was difficult to make out. The muscles of the calves were flabby, and on pressing against the bones the patient called out. No other tenderness could be elicited. The circulatory system was normal, except that the second pulmonary sound was accentuated. The respiratory system was normal. The tongue was clean and moist; the bowels slightly constipated; the liver dullness was normal. The knee-jerks were present and equal; ankle clonus was not obtained; the superficial reflexes were present. There was slight ataxia. I asked him to button his coat, and I noticed there was slight difficulty. His gait was not ataxic, but he walked as if tired. He passed his urine without any trouble, took his food, and slept well. The urine was of specific gravity 1020, acid, and straw coloured. No albumen.

Treatment.—Isolated him, putting him in an airy cabin in the other end of the ship, changed his diet and put him on the usual treatment.

Progress.—His symptoms remained much the same for the next six days, food being taken well. On the eighth day I visited him before breakfast and found him smoking a cigar. This and his general appearance put me off my guard, and I told him that I thought he would be ready for duty next morning. He complained, however, of some pain in his abdomen. That same night (9 p.m.) he commenced vomiting, and though at the time I did not recognize the significance of this symptom I saw, however, that there was something seriously wrong with him. He was pale and complained of post-sternal pain and dyspnoea. The pulse was quick and of low tension, intermittent, the extremities cold. There was epigastric pulsation; the cardiac impulse was diffuse. The deep cardiac dullness extended from 1 in. outside nipple line to the right of the sternum. The first sound a

the apex was loud and followed by a loud blowing murmur conducted into the axilla and to the angle of the left scapula and could be heard there. The second pulmonary sound was both accentuated and reduplicated. The first sound at the base was followed by a murmur which was conducted into the carotids. He died in great distress in the early morning conscious up to the last.

Manson⁶ in his book on *Tropical Diseases* gives a striking picture of such a case. "He was struggling for breath, his eyes were starting out of his head, and his whole attitude was expressive of the utmost distress." No necropsy was held, and he was buried at sea. A lesson which seems to be taught by this is that when beri-beri is diagnosed one must, at least on board ship, always be prepared for serious symptoms suddenly developing themselves. The case just described was one which I did not consider sufficiently ill to prevent his working and certainly his symptoms were such as to make one take such a view, yet he died within a few hours. The vomiting was probably due to the pneumogastric nerve becoming affected and death due to subsequent implication of the cardiac plexus.

One of the other officers told me he had seen a native suffering from beri-beri suddenly fall down dead, so that my experience is evidently not unique.

As Dr. Manson says: "In the matter of death and recovery beri-beri is a disease of surprises. It is an exceedingly risky thing to venture on a prognosis in a case of beri-beri."⁷

CASE II.

A Lascar reported sick, October 11th, 1900, on voyage home from Calcutta.

History.—He complained of weakness, pain, and swelling in his legs.

Present State.—The pulse was 100, of low tension, irregular. Temperature, 99°. Respirations, 20. Bodily conformation very good; visible mucous membranes pale; oedema was present in a marked degree. It was present on the dorsal surfaces of the feet, on the legs and thighs, and in the small of the back. Anaesthesia was well marked over the whole of both legs, and hyperaesthesia of the calf muscles was also present. The apex beat could be seen and felt in the fifth interspace, half an inch outside the nipple line; the impulse was diffuse, and there was epigastric pulsation. The deep cardiac dullness extended from the right of the sternum to the apex beat. The first sound was followed by a loud blowing murmur conducted into the axilla, but I could not trace it further; second sound sharp and accentuated. Second pulmonic both accentuated and reduplicated. At the base the first sound was followed by a loud murmur conducted into the carotids; the second sound was accentuated. A certain amount of dyspnoea was present, and there were a few fine crackles heard at the base of the left lung. The tongue was clean and moist. Bowels regular. Digestion good. No difficulty in micturating. Fluid in abdomen could not be made out. The liver dullness extended from the fourth rib to a quarter of an inch below the costal margin in the nipple line. The legs and small of the back were oedematous, but not the scrotum. The muscles were tender all up the legs. The knee-jerks were absent; ankle clonus was not obtained; all the superficial reflexes were present except the plantar. Ataxia was well marked. He could walk with difficulty, but ankle-droop was not present. Romberg's symptom was present. When I laid him down and asked him to raise his legs he had difficulty in doing so. His arms were also affected, and he had difficulty in fastening his coat. Anaesthesia was well marked in both legs, and in the small of the back, but I could not make it out in the arms. The special senses and muscles connected with them were normal. There was no difficulty in making water, but he said he did not pass so much as usual. Urine, specific gravity 1025, and high coloured, and contains lithates; no albumen. Ordinary treatment was adopted.

Progress.—On October 12th there was very much more oedema, the arms and face commencing to swell. Crepitations occurred at the bases of both lungs. Slight shifting dullness made out in the abdomen; liver dullness increased to 1 in. below costal margin in the nipple line. He was transferred to another ship at Port Said, and sent to Bombay as an invalid.

Manson⁸ says, "There is no conclusive evidence to show that beri-beri is communicable from one person to another," and I do not put forward this second case as evidence that direct communication does take place. Still the man showed no previous signs of beri-beri, and as he occupied the bunk of Case No. 1, who had died, the whole of the Lascar quarters, and his bunk in particular, having previously been thoroughly disinfected, it is a significant fact to notice that two days afterwards he developed a severe attack of beri-beri.

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¹ *Lancet*, November 23rd, 1901. ² *Indian Medical Gazette*, December, 1900, and July, 1901. ³ *Ibid.*, September, 1901. ⁴ *BRITISH MEDICAL JOURNAL*, September 24th, 1898, p. 876. ⁵ *BRITISH MEDICAL JOURNAL*, 1897, vol. II, pp. 390-392. ⁶ *Tropical Diseases*, Article on Beri-beri. ⁷ *Ibid.* ⁸ *Etiology of Beri-beri*, *Lancet*, November 23rd, 1901.

THERE are at present 186 women physicians in Philadelphia. A number of these are said to make 10,000 dollars (£2,000) a year, while a large proportion average from 2,000 (£400) to 3,000 dollars (£600) a year.