of disease. He had noticed the eagerness with which the audience listened to any suggestion of a new physical sign. Further knowledge of diseases must follow on the more exact study of early subjective symptoms. Dr. Buzzard gave as an instance the pains of tabes dorsalis, and pointed out the characters by which they could be distinguished long before there were any physical signs of the disease. The developments of modern psychology constituted an attempt to apply to mental disease the close study of its history on the lines which had been followed with such good results in organic neurology.

Dr. J. P. LOWSON (London) insisted upon the need for a word to distinguish between disturbances of function whose origin was mental from disturbances of function whose origin was a structural change. Hysterical amnesia corresponded to the patient's notion of "memory."

Mr. ARCHIBALD STANLEY PERCIVAL (Newcastle-on-Tyne) had seen one case of retrobulbar neuritis which he afterwards heard suffered from a temporary paresis of the sixth and fourth nerves. He would like to ask whether retrobulbar neuritis had not been observed in such cases as those to which allusion was made by Dr. Collier. In early retrobulbar neuritis there were no objective signs; one had to rely on the presence of a central scotoma (partial or complete) and on light-sense tests. Further, retrobulbar neuritis might have just the same significance as choked disc; if there were an adhesion in the nerve sheath at the site of the optic foramen so that there was a block in the perineural (potential) canal at this site, signs of choked disc could not occur, but they were replaced by signs of retrobulbar neuritis. It was essential that examination for the symptoms of this condition should be made early, before the patient's mental processes became dull. He had seen a case of retrobulbar neuritis in which the diagnosis of a cerebral tumour was controverted on the ground that there was no ophthalmoscopic evidence of an optic neuritis. The patient died, and a cerebral tumour was found. In a small proportion of cases (about 1 per cent. or so) an anatomical peculiarity existed in which retrobulbar neuritis would replace the ordinary choked disc of cerebral tumour.

Dr. BEDFORD PIERCE (York) wished to ascertain the significance of certain common symptoms. Many patients said that their illness began with the feeling of "something snapping in the head," or of "the brain moving or turning over," or of "water running inside the head." What, he asked, was the physiological meaning of these abnormal sensations?

Dr. R. N. GOODMAN (Kingston) said that, as far as the outlook of the general practitioner was concerned, the use of the term hysteria should be given up or severely limited. The distinction between organic and functional conditions was that the former persisted and the latter cleared up. He instanced the occurrence of angio-neurotic conditions of headache with prolonged course and ultimate recovery which were only saved from being called hysterical because the tender points and swellings could be felt by the finger. Hysteria might safely be used in cases where the exaggeration of symptoms took new forms from time to time, and so were indications of a mental disturbance. In all other cases the use of the term hysteria was a confession of ignorance.

BLACKLOCK and CARTER, of the Liverpool School of Tropical Medicine, showed last year that *Anopheles plumbeus* may become infected with the malarial parasite, if kept at 82° F. after its infecting meal. It is possible, therefore, that the species may act as a carrier of malaria in this country. It frequents woods, and breeds in rot-holes in branches of trees or in hollows in their roots. At the request of the Ministry of Health the Mosquito Investigation Committee of the South-Eastern Union of Scientific Societies is inquiring into the distribution of the species, and as to the stages and situations in which it survives the winter. It hopes to present an interim report on the larvae before the end of this year. *A. plumbeus* has borne several other names, including *A. nigripes*.

It is reported that magnesium sulphate and calcium carbonate are used extensively in Italy for the adulteration of cocaine. Some specimens do not contain even a trace of cocaine.

D

NOTES ON BLACKWATER FEVER.*

BY

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DURING my service as officer in charge of a medical division in Macedonia I saw a large number of cases of blackwater fever. Some of the chief points in 14 of these cases are tabulated:

Case.	Previous Malaria.	Malaria Parasites.	Quinine.	Result.
1	+	+	V	Recovered.
2	+	+	V	"
3	+	+	V	"
4	+	+	M	Died.
5	?	+	V	"
6	+	0	V	Recovered.
7	+	0	V	"
8	+	+	M	"
9	0	+	V	"
10	?	+	V	"
11	+	+	O M	"
12	0	+	V	"
13	0	+	O	"
14	+	+	M	"

O=oral; M=intramuscular; V=intravenous.

All the patients were Serb except Cases 5 (Greek) and 11, 12, and 13 (British); all recovered except Cases 4 and 5.

It may be noted that 9 of the 14 cases had had malaria previously, 3 had not, while in two cases the facts could not be ascertained. Case 12 arrived from England at the end of August. She had not had malaria, and had not taken quinine. She began to suffer from blackwater fever on October 10th. In 12 of the 14 cases malaria parasites were found. They were all of the malignant tertian type. Case 13 is interesting in this respect. Very numerous young malignant rings were present in the blood at 11.30 a.m., and blackwater began at 3 p.m. the same day.

The cases were of all degrees of severity. In the majority there was only one attack, which lasted for two days, and then the urine became clear. A few of the cases had a second attack. Case 11 had three attacks, and had a desperate struggle for life each time. The temperature was only slightly raised in all the cases. They all showed jaundice, and albuminuria usually persisted for a few days after the haematuria.

NOTES ON THE TWO FATAL CASES.

Case 4.

A Serb, aged 46, admitted to hospital on September 5th, 1918, complaining of headache, pains and cough. He had been ill for four days. The temperature was 97°. He had suffered from malaria in 1917. He was jaundiced, anaemic, and appeared to suffer from profound toxæmia. He passed urine in bed, and it was not till September 10th that a sample was obtained. This contained haemoglobin, broken-down red corpuscles, epithelial cells, and blood and epithelial casts. He was treated with intramuscular injections of quinine, but signs of pneumonia supervened. The spleen became palpable, vomiting was severe, and death occurred on September 12th. The temperature never reached 99°.

A post-mortem examination showed a flabby, dilated and fatty heart, with blood-stained endocardium and some recent vegetations on the mitral cusps. The lungs were oedematous. The right lower lobe showed red and grey hepatization. The liver was enlarged and fatty. The gall bladder was distended. The spleen was three times its normal size. There was an increase of fibrous tissue with soft pulp and obscure Malpighian bodies. The kidneys were enlarged, with cortex and papillae swollen and congested. The pancreas was hard and fibrous. The alimentary canal presented nothing noteworthy.

On microscopic examination the liver showed a moderate degree of fatty degeneration. There was a golden coloured pigment in many of the liver cells, and part of this gave the

* A communication to the Pathological Society of Great Britain and Ireland.

free iron reaction. The spleen contained pigment in leucocytes and endothelial cells and also free. The kidneys showed some pigment, probably malarial, in some of the glomerular capillaries. The tubules were catarrhal and contained much broken-down material, evidently consisting of partially disintegrated red corpuscles. There were also numerous casts. The broken-down material was most abundant in the collecting tubules. The bone marrow was myelocytic but showed no great change. The heart muscle was atrophic; there were a few haemorrhages between the fibres. The right lung showed croupous pneumonia.

Case 5.

A Greek, admitted on September 25th, 1918, after four days' journey. He was anaemic and yellow, and passed blood-stained urine in bed. The spleen was enlarged, the lungs seemed healthy, but the heart was dilated and the pulse was almost imperceptible. He received an intramuscular injection of quinine bilydrochloride, 20 grains, at 2 p.m., and as the pulse had improved he received an intravenous injection of 20 grains at 6 p.m. This was repeated the next day in a pint of saline solution. Malignant tertian parasites were found in the blood. He died at 2.50 p.m. The temperature had never exceeded 99° F.

Post-mortem examination showed a dilated heart with flabby muscle. The lungs were congested, the liver pale and fatty, and the gall bladder distended. The spleen was six times the normal size. The kidneys were dark in colour.

Microscopic examination of the liver showed extreme fatty degeneration. Every here and there were necrotic areas. The cells contained much brown pigment, and a small portion of this gave the free iron reaction. The spleen showed fibrosis and great masses of brown pigment in endothelial cells and large leucocytes. The kidneys showed catarrh of the convoluted tubules. There were blood corpuscles and detritus in the collecting tubes and ascending limbs of Henle's loops—most in the former. The bone marrow was myelocytic. The giant cells were numerous and appeared healthy. The heart muscle was extremely atrophic with exudation between the fibres.

I wish to submit that these cases strongly support the view that blackwater fever is a manifestation of malaria, and that the onset of the haematuria or haemoglobinuria may be determined by exposure to cold. The majority of my Serb patients had taken part in the attack that led to the Bulgar capitulation, and all of them had suffered cold and privation.

Tropical haemoglobinuria or haematuria is commonly recognized in three forms—(1) malarial haematuria, (2) quinine haemoglobinuria, (3) blackwater fever.

Malarial haematuria is characterized by the presence of malarial parasites and the absence of jaundice. In an experience of some 20,000 cases of malaria¹ I have only seen one example, and I regard the condition as due to a localization of parasites in the urinary passages or a local lesion resulting from the parasite or its toxins, and comparable to malarial appendicitis, pneumonia, or keratitis.

Quinine haemoglobinuria is widely recognized, but I have never met with it in spite of the fact that all my malaria cases never received less than 30 grains of quinine daily for many successive days, and I have given as much as 60 grains intravenously within an hour.

The arguments adduced against the malarial origin of blackwater fever are the following:

1. The rarity of blackwater fever in places where severe malaria is prevalent. This seems to me a doubtful proposition. The incidence of blackwater is often transient, and I agree with Parsons and Forbes² that it is often overlooked.

2. The seasonal incidence of blackwater fever differed from that of malaria in Macedonia, blackwater being most common in the winter months. I think that this may be accounted for by the factor of exposure to cold.

3. It is frequently stated that the administration of quinine often causes blackwater fever, and that blackwater fever is not benefited by quinine. I am prepared to controvert both statements. Of course blackwater fever may follow the administration of quinine if the dosage is inadequate or if a sufficient number of corpuscles has been affected before it is given. I have administered quinine intravenously in large doses in blackwater fever at every stage and have never seen harm follow, and latterly intravenous quinine in saline was my routine treatment. In that practice I was by no means alone.³

4. It is frequently stated that malaria parasites are rarely found in blackwater fever. This is contrary to my experience.

Noël Paton⁴ and I pointed out in 1903 that haemolytic agents fall into two groups:

1. True haemolytics, such as glycerin and distilled

water, dissolve out haemoglobin, which is set free in the plasma and appears in the Bowman's capsules in the kidneys.

2. Toxic agents, such as toluylene-diamine and phenylhydrazin, poison the corpuscles but do not liberate haemoglobin. Very large doses of phenylhydrazin kill animals with the formation of a brown pigment in the blood resembling but not identical with methaemoglobin and with haematuria. Haemoglobin does not appear in the Bowman's capsules, but blood pigment appears in the urinary tubules and in their epithelium. In smaller doses there is no haematuria, but pigmentary changes occur in the liver, spleen, and kidneys.

The distribution of damaged blood corpuscles and pigment appears to be identical in blackwater fever with that found in animals poisoned with the second class of haemolytics. Blackwater fever is therefore regarded as due to destruction of blood corpuscles by the malarial parasite. The parasites associated with blackwater fever are the malignant type which does most damage to the corpuscles, has the greatest tendency to cause local symptoms, and sporulates and liberates toxins mainly in the internal organs. A moderate amount of damage may lead to jaundice only. A greater destruction of red corpuscles may be more than the usual haemolytic organs can deal with, and blackwater fever results. The influence of cold is, I think, an important causal factor. W. Hunter⁵ showed that normal animals became more deeply jaundiced than spleenless animals after poisoning with the second class of haemolytics. Paton⁴ and I confirmed this observation, and concluded that the greater jaundice in the normal animals was simply due to a more rapid fixing of the poisoned corpuscles in the portal system.

One does not overlook the important influence that cold has been shown to exert in determining an attack of paroxysmal haemoglobinuria, but it is unnecessary, I think, to invoke immunity reactions to explain its influence in blackwater fever. Cold may be regarded as acting much in the same way as the spleen does in the experimental animals, by retaining a larger proportion of corpuscles in the internal organs, and thereby exposing them to a larger contact with the haemolytic organs. Such a view might afford a simple explanation of apparent anomalies in the incidence of blackwater fever both as regards time and place.

Summary.

Fourteen cases are tabulated. In twelve of these malaria parasites were found. All the cases were treated with large doses of quinine and twelve recovered. It is contended that malaria is the essential cause of blackwater fever, and that exposure to cold may be an important factor in determining its onset.

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RESUSCITATION IN DEATH UNDER ANAESTHESIA.

BY

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THERE appears to be a need for the greater advocacy of cardiac massage as a measure of resuscitation in death and impending death under anaesthesia. Records of fatalities still appear without mention of cardiac massage having been practised, and without any reason for its non-employment being advanced. Such a case, for instance, was described last year, and not one of the twenty-six contributors to the correspondence which followed made any comment upon the statement that "artificial respiration was carried on for nearly two hours, but there was never the slightest response." It is true that the means employed for resuscitation of this case—death occurring during an adenoid operation—was not the issue particularly raised for discussion; but the absence of comment could not but have implied acquiescence in the correctness and adequacy of the treatment. To those, however, who have proved the merit of cardiac massage the case is but one of many which clearly indicates that this operation has