deteriorate and might prove a great source of infestation to the surrounding villages.

2. Infestation and disease rates show no predilection for either sex.

3. Earliest incidence of lymphangitis was found to be at the age of 7. The highest incidence of disease is in the age group (46 to 50) and the lowest in the (6 to 10) age group.

4. In agreement with the findings of Iyengar (*loc. cit.*) in Travancore and Brown (*loc. cit.*) in U.S.A. the filarial infestation rate in apparently healthy persons is very much higher than in persons showing clinical signs of disease in whom it is almost *nil.*

5. The importance of blood examination in determining the incidence of the disease in a given area has been stressed.

6. The predominant filarial infestation in the area surveyed is *Mf. bancrofti* and the vector is *C. fatigans.*

7. Malayi infestation has been detected and *Mansonioides uniformis* and *indiana* were found breeding in ponds and irrigation canals in which Pistia Marsillia grow.

REFERENCES

Brown, H. W. (1945) .. *Amer. J. Pub. Health,* **35,** 607.

Iyengar, M. O. T. (1938). *Indian Med. Res. Mem.,* No. 30. Thacker, Spink and Co. (1933), Ltd., Calcutta.

BIBLIOGRAPHY

Barraud, P. J. (1934) .. *The Fauna of British India, Diptera,* Vol. V, *Culicidæ.* Taylor and Francis, London.

Iyengar, M. O. T. (1932). *Indian J. Med. Res.,* **20,** 671.

PLAGUE-MENINGITIS

By E. G. H. KOENIGSFELD

CAPTAIN, R.A.M.C.

and

K. P. S. NAMBIAR, L.M.P.

(*Cantonment Hospital, Wellington*)

This is a report on 2 cases of plague-meningitis observed by us among the comparatively small material of 41 instances of plague treated at Wellington, Nilgiris, in 1945-46.

We consider our cases worthy of record as the cerebro-spinal type of plague has been described in very few instances only, the disease being believed to be of extreme rarity.

Lewillon, Devignat and Schoetter (1940) reported in 1940 on plague causing primary meningitis. Unfortunately we have not been able to obtain their paper. Meyer of California, according to Manson-Bahr (1940), described a chronic relapsing meningeal form of plague, in which the patients exhibited meningeal symptoms and high fever. *P. pestis* was isolated in these cases from the blood. Wu Lien-Teh, Chun, Politzer and Wu (1936) in a detailed review of plague comment with regard to the meningeal form that only rare instances of this kind have been observed. According to these authors one case was seen by Paso in 1925 in a Spaniard and another one by Lafort *et al.* in a native of Daker. The latter case showed *P. pestis* in the C.S.F.

As far as we can see no such cases have hitherto been described from this country.

Case Records

1. A woman, aged 47, from Coonoor, Nilgiris, fell suddenly ill with high pyrexia, malaise and subsequent delirium on 16th April, 1945. She was admitted to hospital where she developed a moderate enlargement of the axillary glands. A gland smear was taken, which on examination at the Pasteur Institute, Coonoor, was negative for *P. pestis.* She was treated with sulphonamides and iodine injections with the result that her condition showed considerable improvement within a couple of days. At the request of her family she was allowed to be taken home after a stay at hospital of less than one week. While at her home she continued to run an irregular temperature. On 6th May, 1945, one of us (Nambiar) was called out to see her as she had again felt very ill for the past 24 hours. At that stage she was found to be delirious and very restless. She vomited and retched incessantly. There was definite nuchal rigidity, and Kernig's sign was positive. The axillary gland had remained stationary without sign of impending suppuration. She was re-admitted to hospital where lumbar puncture was done. Culture of C.S.F. at the Pasteur Institute, Coonoor, showed *P. pestis.* The patient died the following day. She is reported to have been protected with anti-plague vaccine 2 months previously.

2. A girl, aged 7 years, was admitted to hospital on 7th April, 1946, for fever of 2 days' duration. On admission the child was apathetic, listless and markedly dehydrated. Eyes were sunken, not congested; the tongue was dry and coated. Spleen and liver were not palpable. Respiratory and circulatory system showed nothing abnormal. Left axillary glands were moderately enlarged and tender. Temperature 101°F. in axilla, pulse 128; gland smear examined at the Pasteur Institute, Coonoor, was positive for *P. pestis.* The patient was treated with sulphadiazine in full doses and intravenous glucose (25 per cent) 20 c.c. daily and fluids orally in abundance. On the 5th day of her stay at hospital she developed an inguinal bubo. The axillary glands suppurated and had to be incised. After this the patient appeared to be improving and the temperature returned to normal by 16th April. However, on the 21st, her condition became worse with a relapse of

fever and pains in all the joints. The left knee joint by then was swollen and inflamed. She was put again on sulphadiazine and the temperature settled again within 4 days with complete subsidence of the arthritis.

On 4th May, after one week of definite improvement, she complained again of headache and lost her appetite. Temperature rose again up to 102°F. that evening. From now onward her general condition, which was poor enough from the very beginning, deteriorated rapidly. On the following day she exhibited all signs of gross meningeal irritation. She vomited all the food given to her, showed marked head retraction and opisthotonos, and became delirious and restless. The pulse at that stage was thready and irregular.

In consideration of the fact that her condition after the long and debilitating illness was obviously hopeless we decided not to perform a lumbar puncture. During the following days, while she was gradually sinking, she became more and more apathetic, but the nuchal rigidity persisted and Kernig's sign remained positive. She died on 7th May.

Commentary

In the first case the diagnosis 'plague-meningitis' was confirmed by the findings of *P. pestis* in the C.S.F.

In our second case no lumbar puncture was performed and the diagnosis had to be made on clinical grounds. Even so there can hardly be any doubt that the symptoms were due to a genuine meningitis and not to mere toxæmic meningism. Head retraction and opisthotonos indicate severe meningeal irritation, which, in our opinion, can only be explained by an acute break-down of the blood-C.S.F. barrier, if not for the *Pasteurella* itself so at least for the toxins. We attribute equal significance to the vomiting which synchronized with the delirium and nuchal rigidity.

It was a striking feature of both cases that the meningeal signs occurred in the course of a prolonged illness after some initial improvement. Both patients seemed to respond favourably to sulpha drugs and succumbed only at a comparatively late stage of the disease. Sulphadiazine probably lessened the virulence of the germ to a certain degree, without rendering it entirely harmless, with the effect that the disease took a more chronic course and meningitis had time enough to develop. If this conclusion is correct more cases of plague-meningitis may be observed in the near future as sulpha drugs are now in general use in the treatment of plague.

There is still one question likely to be asked in connection with our cases: 'What is the appearance of the C.S.F. in simple, uncomplicated bubonic plague?'

We are not aware that systematic examinations of the fluid have been carried out so far, and have, therefore, performed bacteriological tests and cell counts of the C.S.F. in our latest series of 5 cases of bubonic plague.

The results of all our tests have been entirely negative. Neither did the bacteriological examination of the clear C.S.F. show any growth of organisms on culture, nor was there any increase of the number of leucocytes. The intracranial pressure was normal.

Summary

Two cases of plague-meningitis have been described. One case has been confirmed bacteriologically by the finding of *P. pestis* in the C.S.F. The other case was diagnosed on clinical grounds.

A tentative explanation has been given for the occurrence of the disease and the possibility that more cases may be reported in future is pointed out.

No evidence could be obtained as to any change of the C.S.F. in uncomplicated cases of bubonic plague.

We wish to convey our thanks to Lieut.-Colonel G. S. Caithness, Wellington, for permission to publish this paper, to Lieut.-Colonel M. L. Ahuja, Director, Pasteur Institute, Coonoor, for kind permission to use the laboratory reports, and to Dr. D. I. G. K. Menon, for his advice and suggestions in writing this paper.

REFERENCES

LEWILLON, R., DEVIGNAT, R., and SCHOETTER, M. (1940). *Ann. Soc. Belge de Méd. Trop.*, **20**, 79. (Abstract—*Trop. Dis. Bull.*, **37**, 828.)

MANSON-BAHR, P. H. (1940). *Manson's Tropical Diseases.* Cassell and Co., Ltd., London.

WU LIEN-TEH, CHUN, J. W. H., POLITZER, R., and WU, C. Y. (1936). *Plague: A Manual for Medical and Public Health Workers.* National Quarantine Service, Shanghai Station, China.

SEROLOGICAL TECHNIQUE (contd.)

By S. D. S. GREVAL

LIEUTENANT-COLONEL, I.M.S.

and

A. B. ROY CHOWDHURY, M.B. (Cal.)

(From the Laboratory of the Imperial Serologist and Chemical Examiner to the Government of India, School of Tropical Medicine, Calcutta)

DETERMINATION OF BLOOD GROUPS FROM STAINS

THE basis is the absorption of the known isonins by the unknown isogens of the stains proved to be caused by human blood only. The details are:—

1. A potent serum ab in which titre of a equals that of b is selected and its Minimal Dose of Equal and Simultaneous Agglutination, MDESA, determined, thus: