presented by the fact that a considerable number of strains are relatively inagglutinable have not been surmounted. A serum prepared by immunization with one strain may agglutinate only a small proportion of other strains. For example, Elser and Huntton in America tested a mono-valent serum against 65 strains, with the result that 25, or 40 per cent. of them, were not appreciably affected. Similar results have been got by Arkwright and other workers.

On the other hand, some of these inagglutinable strains may have slight but definite absorptive powers, and may, when injected into animals, produce serums which affect agglutinable strains, though remaining without action or with only slight action on the homologous organisms.

It is possible, however, that these difficulties may yield to further research, which the present outbreak will doubtless stimulate, but it has to be remembered that in the coccal group generally these serological difficulties due to apparent high specificity of the individual strain are constantly met with—for example, in the case of the *pneumococcus*. The phenomenon is not confined to this group, however, as very occasionally strains of *B. typhosus* are met with which are relatively inagglutinable, but which are able to produce agglutinins for other strains.

With regard to the distribution of the *meningococcus*, it has never been isolated from dust, air, or fomites generally, and, indeed, its short-lived character renders very remote the possibility of its transmission by such media.

Apart from the actual case of the disease, the *meningococcus* is found in the naso-pharynx of a certain proportion of persons living in more or less contiguity with cases. I will refer only to its occasional presence in the naso-pharynx of healthy persons at epidemic-free periods. In the book on the *carrier problem* by Arkwright and myself¹ a list of observations on this point is found, and Sophian in his book² publishes a similar list. Some observers have found a small percentage of carriers in such populations, while others have found none, but the total number of cases examined has not been large. The only large series is that by Mayer and Waldmann (1910), who examined 9,111 men in the Munich garrison at an epidemic-free period and found roughly 2 per cent. of carriers amongst them. In a recent paper in the *BRITISH MEDICAL JOURNAL*, Sir William Osler quoted these figures and added a point of exclamation, the inference to be drawn probably being that he (Sir William) was inclined to subscribe to Mayer's sweeping generalization from his figures—namely, that the *meningococcus* was an ubiquitous organism occurring in 2 per cent. of normal people.

This question is, of course, a most important one. If one were to accept a generalization of this kind, it would amount to placing cerebro-spinal meningitis on the same plane as pneumonia, where epidemiology does not come in—that is, if we accept the current view that a person gets pneumonia by infection from a strain which has been growing saprophytically in his respiratory tract.

Now, it must be said that at the time some doubt was cast on these observations of Mayer by his own compatriots, and in a footnote to his paper Mayer refers to this feeling of scepticism; but probably the feeling was one rather of disagreement with his generalization above referred to. In my view there is no reason to doubt the figures, but in appraising them one has to remember that the population examined was one in which cases had been occurring yearly. It is therefore not at all improbable that some of his carriers revealed at the meningitis-free period had been chronic carriers for some months, while others had been infected by such carriers. As a matter of fact, Mayer admits that 4 out of 35 persons who were carriers during the meningitis season proved to be still harbouring the *meningococcus* at the meningitis-free period. The way in which one carrier gets surrounded by a zone of carriers has been very clearly demonstrated by Selter, whose observations are to be found in the book on the carrier problem.

The presence of such carriers at an epidemic-free period is indeed necessary to explain the peculiar manner in which cerebro-spinal meningitis springs up in areas between which and previously infected areas no apparent connexion can be traced. The chronic carrier, with probably a zone of carriers round him, acts as the bridge between one epidemic and the next. A generalization, therefore, like that propounded by Mayer is entirely

unjustified at the present time, and is calculated to give a false sense of security to the general and medical public.

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CEREBRO-SPINAL MENINGITIS: DIAGNOSIS AND PROPHYLAXIS.

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SUCCESSFUL prophylaxis depends upon a clear conception of what the disease actually is. The name is unfortunate. It recognizes only one set of symptoms that are practically terminal.

The disease is more prevalent than is commonly known. It has three stages:

1. Catarrhal.
2. Septicaemic.
3. Meningeal.

The catarrhal stage is probably very prevalent. It causes no symptoms that occasion any alarm, and is looked upon as a pharyngitis, or that and a laryngitis combined.

The septicaemic stage sets in when invasion of the blood stream has taken place. It is apt to be mistaken for influenza, and often it ends there and nothing occurs to cause any suspicion that there has been a mistake in diagnosis. Occasionally it happens that the true nature of the disease is recognized, and it is notified, but before time has been found to perform lumbar puncture the whole train of serious symptoms has disappeared. The result is a reversal of the diagnosis, and some degree of discredit to the original examiner of the case.

The diagnosis depends upon the recognition of the existence of these three stages.

Fever, headache, and other constitutional symptoms need not be expected in the mild catarrhal stage. An examination of the pharynx shows redness—catarrhal as opposed to granular pharyngitis is seen. But it must be remembered that the former may be superimposed upon the latter, and thereby confusion may arise. The question of the nature of the infection can only be settled bacteriologically. A pure culture is practically never to be got directly, and the usual methods must be observed to separate different organisms. Certain streptococci and pneumococci are very frequently found, but the important point is to find out the presence or absence of meningococci. This is done by the excellent method devised by Gordon.

The question arises whether if meningococci are found and there is no other symptom but pharyngitis, is the disease to be attributed to the meningococcus? It certainly ought to be, for the following reasons:

1. The meningococcus resembles the gonococcus in having great vitality in a favourable nidus, and in being extremely susceptible to adverse influences when removed to situations where other organisms could still survive. Implanted successfully upon the pharyngeal wall it tends to hold its own, just as does the gonococcus when it reaches the site of its predilection. (The utility of gargles and other forms of local treatment has yet to be proved in dislodging it, but the attempt to do something in the way of thorough local treatment should not be neglected.)

2. In all probability the tendency is for the organism to make headway and to enter wandering cells and travel eventually into the blood stream. Even in the blood it may do little mischief beyond causing a toxic headache and other symptoms usually attributed to influenza. Cases of this sort may have typical or more usually atypical and protean nervous symptoms, and may suddenly recover or become quite easily diagnosable clinically.

3. Instead, however, of the disease progressing so far it may become chronic, and spread over a greater area and cause laryngitis. Sufficient resistance is raised by the leucocytes to prevent living cells entering the blood stream, and a state of perimucosal induration arises that is very painful and tender, and causes trouble in swallowing and moving the neck. This is not the later neck rigidity pathognomonic of meningeal irritation. These symptoms are all aggravated by the association of other organisms, such as the *Streptococcus marinus*, and may be successfully treated by a vaccine chiefly of that organism. The symbiotic association of these other organisms probably aids rather than hinders the successful survival of the meningococcus, but they alone without it may cause similar symptoms.

4. The gonococcus and the meningococcus are intra-leucocytic organisms. Phagocytes seem to give them a shelter that defends them from the action of the antibodies that ought normally to be formed in organismal invasions of the body. No doubt they are carried in this way to the meninges. Gonococci have the same properties, and in suitable shelter, such as joint cavities, thrive and cause trouble. The meningococcus occasionally enters a joint, and, vice versa, the gonococcus occasionally enters the cerebro-spinal canal.

The usual gonococcal disease is confined to mucous surfaces. Similarly the usual meningococcal disease is one of mucosa. It is already known that carriers are more numerous than so-called cases, but really all carriers and cases alike are, no doubt, genuine cases. Therefore the prophylaxis of meningococcal disease is the treatment of these early cases, preventing them from infecting non-immunes, and from themselves developing more advanced stages of the disease. It amounts to the examination bacteriologically of all cases of sore throat during epidemics, the elimination of meningococcal cases, and the special segregation and treatment of these. The chief burden of this work would fall upon the bacteriologist; but there could be no question of the benefit that would result, and the ultimate saving of time to men in training, and the saving of life too.

It has just been observed that on suitable media, and exposed for several days to temperatures generally recognized as unsuitable, the meningococcus is *still* viable, and can be subcultivated. It is inferred that this is perhaps possible in Nature.

As a measure of prophylaxis it is suggested that carriers ought to be treated by autogenous vaccines.

It is believed that routine examination of contacts will ultimately be more valuable and less laborious than that either of alleged contacts under present conditions or of whole regiments, which is a counsel of perfection and too large a problem to tackle.

but capable of transferring them to others more susceptible. The latter hypothesis was supported by many observations conducted by many observers in many different countries. Carriers might or might not show obvious signs of inflammation of the naso-pharyngeal mucous membrane, and might carry the organism for some months. Adults more frequently acted as carriers than children. Of thirty carriers, in twenty-six the organism was obtained from the naso-pharynx on one occasion only. A carrier could transport the organisms for long distances, and the readiness with which they could be distributed by sneezing, coughing, etc., was well known. Kissing and the use of utensils afforded other means. The organism was present in the naso-pharynx in the early stages of the disease, and exceptionally persisted for a long time. In one it was found on the thirty-fifth day from the onset. The detection of the organism in the blood afforded a reasonable explanation of its route from the naso-pharynx to the meninges. Its usual site was the upper part of the pharynx and about the posterior nares, and it could readily be reached by a small swab on a bent wire introduced through the mouth. A widespread distribution of carriers had been demonstrated. Among 30,000 men 40 cases of meningitis had occurred. These were widely distributed, no two cases arising in the same hut or tent. The greater part of one battalion was examined for carriers, and of 887 men 3.3 per cent. carriers were found. The number of carriers might amount to twice, ten times, or forty times the number of cases. The condition which made a search for carriers desirable was the possibility of their isolation during the early stages of an epidemic. The total number of persons harbouring the micrococcus should be looked upon as constituting the epidemic. The carriers formed the bulk of an epidemic, the cases forming only a small minority. Epidemics of meningitis occurred when and where carriers became numerous.

Dr. W. H. HAMER said that epidemics of the disease had been described in the fifteenth, sixteenth, and seventeenth centuries, and he did not accept the statement that the disease began in 1805. Sydenham's description of a new fever in 1685 corresponded closely. During the last eight or ten weeks there had been a large number of deaths from diseases variously called pneumonia, bronchopneumonia, and influenza. He thought that there was only a small increase of deaths from cerebro-spinal fever. He questioned whether the various organisms described played any more important part than that of secondary invaders.

Professor W. J. R. SIMPSON said that the disease had been met with frequently in the tropics during the last few years. There had been recrudescences, but the disease had not yet arrived at its climax. One epidemic had occurred in the northern part of the Gold Coast, another in Nairobi. In the latter the cerebro-spinal fluid had been examined in a large number of cases, the organism being found in 80 per cent. If not found in the cerebro-spinal fluid it was isolated from the blood. Meningococcal serum had not proved of use for the natives, but good effects had followed the employment of soamin injected into the buttocks once a day for five days. No equivalent results had been obtained in the epidemic in Belfast, where the mortality was 38 per cent. In the Nairobi epidemic, of eight Europeans affected only one died. In West Africa there was a notion among the natives that the cattle and fowls were also affected. This had not been confirmed, but the point was worthy of being kept in mind.

Dr. F. E. BATTEN spoke of the endemic form which was constantly present in London. It had a seasonal incidence, beginning in February and reaching its maximum in April, but varying somewhat in different years. The meningococcus was of a different strain from that of the Belfast epidemic, as the blood of one did not agglutinate the organisms of the other. The endemic cases were not as a rule so acute, but at times were very severe. Fulminating cases occurred, accompanied by purpura and rapid death. These cases were nursed in the general ward, and he had never known the disease to spread, nor, indeed, had he ever known a case start in hospital. An interesting comparison was with poliomyelitis, which also showed a seasonal variation, reaching its maximum in August. In it there was the same absence of transference from case to case. He had never been able to obtain any good evidence

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The Interpretation of the Features of Cerebro-spinal Meningitis Epidemics and Preventive Measures.

At a meeting of this section held on Friday, March 5th, the President, Dr. C. J. MARTIN, being in the chair, the discussion was reopened by Dr. J. C. G. LEDINGHAM, whose contribution is published in full at p. 465.

Dr. J. A. ARKWRIGHT said that a striking feature concerning epidemics of cerebro-spinal meningitis was the apparent isolation and want of ascertainable contact between the patients. The majority of houses afforded only one example, but the proportion differed in different epidemics. In one epidemic, of 1,957 houses implicated, 6.9 per cent. furnished more than one case. Cases often occurred simultaneously in the same house. If not simultaneous, a second case might arise after an interval considerably longer than that of the accepted incubation period. Such observations were against the view that the infection was spread from one patient to another. To overcome this difficulty two hypotheses were possible: (1) the virus might be spread abroad, and remain capable of infecting for long periods. This was, however, impossible in the case of the *Diplococcus intracellularis*. (2) The organisms might be transported by carriers—persons themselves insusceptible,