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## Epidemiology of meningococcus meningitis

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THE  
EPIDEMIOLOGY of MENINGOCOCCUS  
MENINGITIS

JOHN H. WATERMAN

Senior Thesis

1933

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## The Epidemiology of Meningococcus Meningitis.

### INTRODUCTION

"The most vivid, and certainly the most distressing, pictures covering the walls of the memory chamber of my brain were painted by the invisible hand of epidemic disease."

Victor C. Vaughan.

Among all the important diseases occurring in epidemics none is less satisfying to the epidemiologist than meningococcus meningitis. It presents features which are not often seen in other epidemic diseases. In the extensive literature and ancient history of the disease it has received a great number of names such as spotted fever, spotted typhus, black fever, exanthematic typhus, brain fever, petechial fever, phrenitis, epidemic cephalalgia and cerebrospinal fever. At times its features have been so strange and baffling and so different from the characteristics usually associated with contagious diseases that its contagiousness has even been questioned by many writers. In spite of the vast amount of work done and the thoroughness of technique followed, it has usually been impossible, especially in civil life, to trace the progress of the contagion from one locality to another, even in periods of widespread epidemics. There seems to be no regular progression or extension of the disease. It moves by leaps and bounds and seems to strike at haphazard, one country suffering a severe epidemic, another country entirely free. Simultaneously affected localities are often separated by those that

almost entirely escape the infection. Contrary to other epidemic diseases the evolution of an epidemic of meningococcus meningitis is usually slow and gradual and there is no regular cycle. The seasonal incidence though more stable than some of its other features, is often variable. A survey of its age incidence varies greatly with different observers and the cases are scattered and seem to be grouped around several small foci rather than a single and definite focus. Physicians rarely contract the disease and often a multiplicity of cases in a family or crowded dwelling is unusual or even absent entirely in some of the more severe epidemics. Only a small percentage of the population contracts the disease as compared to other epidemic diseases. The usual rate during epidemics is that of 1 to 2 cases per 10,000 population in the large cities, which is considerably smaller a morbidity rate than that of measles, diphtheria, pneumonia, typhoid or influenza during periods free from epidemics from these diseases.

Despite all this, however, the disease retains its prominence and often becomes the bogey-man of the public health officer because of its severity of attack and high mortality rate, usually fifty per cent. Added to this, and undoubtedly partly because of the peculiarities of the disease which we have already mentioned, there is not in the possession of the medical profession definite measures for its control. A few sporadic cases often cause the greatest of alarm and demands are immediately placed upon the public health officer that "something be done" forcing him to apply measures of doubtful

value or to rely upon routine carrier surveys to allay the public apprehension.

In spite of these difficulties our knowledge of meningococcus meningitis and measures for its control has been greatly increased through careful observations of a number of epidemics, especially those in military life. That further epidemiological studies are clearly indicated is brought out by J. F. Norton and I. E. Bailey in reporting observations on epidemic meningitis and its relation to carriers in Detroit during the period from February 6, 1929, to February 6, 1931, when they say, "Most of the data on epidemic meningitis is of conditions found under military life except for the research of Bruhns and Hohn. Civilian material is far from complete."

#### HISTORY

The history of epidemic disease we may trace back to the early Greeks. In speaking of it Greenwood says,

"The epidemiology of classical and Hellenistic Greek science was logical and self-consistent. According to Galen there were three factors- two innate or acquired aptitudes of the body, the temperamental and procatarctic; and one external, the atmospheric katastasis. The latter determined the quality and the two former the severity of an epidemic.

'So often as the katastasis of the atmosphere departs from its proper nature into the hot and humid, pestitential diseases must needs arise, yet will those chiefly be affected who were

beforehand saturated with excrementous moisture while those who labor moderately and are temperate in diet remain refractory to such diseases.' (De Februm Differentiis.)

Translating from an obsolete notation into one more congenial to our habits of thought, Galen's view is that it is rather the condition of those exposed to infection which determines the difference between one epidemic and another. He would explain the difference between the influenza epidemic of 1918-19 and that of 1931, not in terms of the *katastasis* or as we would say specific differences of the infecting agent, but in terms of contrasting resistances.

Sydenham on the other hand, enormously extended the sphere of the *katastasis*. He freed it from the naive implications of such terms as hot, cold, moist and dry in modern speech and popularized, if he did not actually invent, the doctrine of the epidemic constitution. He would have held that the influenza of 1918-19 differed from that of 1931 'essentially' just as the port vintage of 1887 differed essentially from the vintage of 1897, because the complex of essential biological and cosmic factors had changed."

The situation is but little changed today. We are still concerned with the problems of Galen and Sydenham though our terminology may be different.

The history of cerebrospinal meningitis begins in the Middle Ages though at that time it was still obscured among the group of diseases known as typhus, fever, or synochus. There is

no definite assurance that the epidemic in Germany in 1581 designated as spotted fever, or other of the many epidemics described as spotted typhus, exanthematic typhus, black fever, brain fever, phrenitis or epidemic cephalalgia were actually meningococcus meningitis. There were few if any autopsies performed so there is no confirmation, but the symptoms described and attributed to these diseases were very suggestive of those found in meningitis.

The recognized history of cerebrospinal meningitis as a disease entity and entirely separated from typhus fever begins in Geneva in 1805. On the fourteenth of February of that year a practitioner named Vieusseux saw in the poorer district of Geneva a case which, because of its striking symptoms, attracted his attention. At first he attributed it to the unsanitary conditions among the poor but within a short time the disease had spread to the best parts of the city and he felt convinced his first explanation was not correct. He did not, however, consider the disease contagious which is not surprising because there were no cases of contagion at the hospital and the commission appointed by the Government did not consider it worthy of being called an epidemic, although 26 people died of the disease. Vieusseux observed that the disease was confined to children and adults under thirty years of age. His description of the symptomatology is as follows:

"It began suddenly with extreme prostration; the face was drawn; the pulse feeble, small, and frequent, sometimes it could hardly be felt; hard and bounding in a number of cases. There

was violent headache, especially frontal. Then there appeared precordial pain or vomiting of bilious matter, rigidity of the spine and convulsions in infants. The body presented livid spots, especially after death, sometimes even during life."

His assistant, a man by the name of Mathey, reports the results of their autopsies as follows:

"The meningeal vessels were markedly congested. A gelatinous blood-stained fluid covered the whole surface of the brain. There was fluid in the ventricles. The choroid plexus was a deep red. A yellow puriform exudate was seen on the posterior aspect of the cerebral lobes and in the interior. There was no manifest change in the cerebral tissue. The same exudate was found along the optic nerves, the base of the cerebellum, and the vertebral canal."

Although, as we have described, the Government commission, of which Vieusseux was a member, reported the disease need scarcely be called an epidemic, the people of Geneva were quite alarmed, as has been mentioned is the case today even with the appearance of only a few sporadic cases. Vieusseux attributed this fear among the citizens to the same characteristics which give the disease its fear spreading power today, the lightning-like rapidity with which apparently well persons are stricken and the high mortality rate among those developing the disease.

In the spring of the following year, 1806, the disease was first seen as an epidemic in the United States at Medfield, Massachusetts. The physicians who reported it, Danielson and Mann, described it under the title, "A Singular and Very Fatal Disease which lately made its appearance in Medfield, Massachusetts." The description given by these authors is so typical,



both as to symptomatology and to pathology, that there can be no doubt but that they were dealing with cerebrospinal meningitis.

The "Historisch-Geographische Pathologie" of Hirsch, published at Stuttgart in 1886 and summarized by Heiman and Feldstein in their book "Meningococcus Meningitis", divides the history of the disease into four periods to which we may add an additional four to bring it up to the present time.

1. 1805-1830, during which the disease was general in the United States. In Europe it occurred in isolated epidemics.
2. 1837-1850. During this period there were widespread epidemics in France, Italy, Algiers, the United States and Denmark.
3. 1854-1875. In this period the disease was widely diffused throughout most of Europe, the adjoining countries of Western Asia, the United States and parts of Africa and South America.
4. 1876-1882. During this period there were isolated epidemic outbreaks.
5. 1893-1903. During this period there were epidemics in France, Germany, Austria, Norway, Scotland, Ireland, Bosnia, Italy, Algeria and the United States, especially New York.
6. 1904-1911. A severe epidemic prevailed in New York, and another in Prussia. There were also severe epidemics in Great Britain, France and the west coast of the United States. During the period from 1904-1907 the disease was more widely spread throughout the world and killed more people than in any previous period of its history.
7. 1915-1919. During the period just previous the morbidity rate had been steadily increasing and with mobilization of troops for the Great War severe epidemics occurred in England in 1915-1919, and in the United States in 1917-18. Hamer describes these epidemics as "The Precursors of the Great Influenzas of 1918-19. For enlisted men as a cause of death meningitis stood fifth in the United States and fourth in Europe.

8. 1928-1931. During this period there have been moderately severe epidemics reported in the United States, especially in the Central States and Great Lakes Region. The data for this period is not yet complete.

During the first period (1805-1830) three important contributions to the American literature of the disease were made: the paper by Danielson and Mann reporting the epidemic in Medfield, Massachusetts; the communication by a committee of the Massachusetts Medical Society; and the classical book by Elisha North entitled, "A Treatise on a Malignant Epidemic Commonly Called Spotted Fever". A frequency of eruptions and respiratory complications was noted during this period.

During the second period (1837-1850) chiefly the clinical features of the disease were studied and described, especially by the French clinicians, notably Lespes and Tourdes.

In the third period (1854-1875) a very valuable paper was written by Webber of Boston in 1866, although he did not distinguish definitely cerebrospinal meningitis from typhus fever, inferring that it might be a complication of the latter. An important contribution regarding the contagiousness of the disease was made by Smith in 1873 reporting an epidemic he had studied in New York City. He says, "My statistics, therefore, harmonize with the doctrine of noncontagiousness, but it is obviously very difficult to determine from clinical experience whether an epidemic constitutional disease is absolutely noncontagious, or contagious in a very low degree. Cerebrospinal fever is one or the other, but if contagious it is apparently less so than either typhoid fever or Asiatic cholera."

Stille also wrote a monograph during this period which was a report of 98 cases seen in the Philadelphia Hospital during 1866-67. He does not say so definitely but leaves the reader with the impression that he believes the disease to be non-contagious.

In the fourth period (1876-1882) there are no records of epidemics in the United States and there were no very extensive epidemics anywhere so that little was added to the literature during that time. Councilman, Mallory & Wright, however, in a report of the State Board of Health of Massachusetts in 1898, show that there was an average of 150 deaths per year in Massachusetts from 1878 to 1896. The minimum was 78 in 1878 and the maximum 171 in 1888.

In the fifth period (1893-1903) there was an extensive report by Berg of an epidemic in New York in 1893. Also an epidemic in Maryland in 1893 was reported by Flexner and Barker. A severe epidemic in Portugal in 1901-1903 was reported by Bettencourt and França. The bacteriology of cerebrospinal fever was firmly established during this period.

The sixth period (1904-1911) was one characterized by extremely severe epidemics and in 1905 the morbidity reached eighty per 100,000 population in New York. In Glasgow it was 84.7 in 1907 and in Paris 10 in 1909. There were extensive researches made emphasizing the importance of germ carriers in the spread of the disease.

During the seventh period (1915-19) there were especially severe epidemics in England and later in America. Valuable contributions were made in the preventive control, bacteriology and

treatment of the disease in the publications of the Medical Research Council in London in reporting the epidemic among the military forces. Vaughan and Palmer gave us valuable additions to the statistics in a report on communicable diseases in National Guard and National Army of the United States during the period from September 1917 to March 1918. Although not published until 1929, Sir William Hamer in his book "Epidemiology, Old and New" makes a valuable contribution concerning the meningitis epidemic of 1915-1919 and its relation to the influenza epidemic of 1918-1919.

The eighth period (1928-1931) is marked by the publication of the report of the meningitis epidemic in Detroit (1928-1930) and the relation of carriers to the waves of the epidemic by Norton and Bailey. Norton also makes a valuable report of the occurrence of secondary cases directly attributable to contacts. The report of a recent attack of meningitis in Missouri with epidemiological and administrative considerations is made by Laybourne. There are also numerous Public Health and Military studies as well as the report of the League of Nations on the epidemiology of meningitis from 1929-1931. The general summary of this period is that we are still without efficient control measures.

In general, from the history of the disease and a study of the literature of the various periods we can say that: Cerebrospinal meningitis has existed from early times. It is widely scattered throughout the world. Only a small portion of any community usually contracts the disease. Its

epidemics are small, limited and sporadic. The epidemic characteristics are essentially the same today as in its early history.

#### ETIOLOGY, THE MENINGOCOCCUS

No epidemiological study would be complete without a brief account of the etiological factors of the disease, although in the case of the meningococcus the material is vast and forms a subject in itself with many of its problems yet unsolved. We shall endeavor to confine ourselves, however, only to its more salient features.

Although Leichtenstern in 1885, and at about the same time, Schwabach found intracellular diplococci in conjunction with cases of cerebrospinal fever, it remained for Weichselbaum, in 1887, to definitely associate the diplococcus with meningitis. He gave the organism the name of *diplococcus intracellularis meningitidis*. It is a small gram-negative diplococcus about one micron in diameter and appears with adjacent sections flattened, not unlike the gonococcus. The meningococcus belongs to a group of five gram-negative diplococci, any or all of which may be found in the nasopharynx. For this reason it is extremely important that the organism be positively identified in the making of carrier surveys where a swab of the nasopharynx is depended upon. The finding of a gram-negative coccus in the nasopharynx is not sufficient to declare the individual a carrier. The members of the group may be distinguished by obtaining their reaction with various sugars. Vaughan

quotes Elser and Huntoon as being responsible for this method of differentiation. In a report of M. H. Gordon to the Medical Research Council he classifies four strains of meningococci and identifies them by means of a polyvalent serum. His serum will agglutinate all strains of the meningococci in dilutions of from 1-200 to 1-2000. After determining that the diplococcus is a meningococcus the special strain can be determined by the use of specific sera.

Concerning the pathogenicity of the meningococcus Dr. Gordon and his colleagues say: "It is now generally agreed that during an outbreak of cerebrospinal fever, for every case in which the meningococcus succeeds in setting up meningitis there is a plurality of persons in whom it does not get further than the nasopharynx. But, as a rule, if the human defence is unsuccessful, there is no infection that on occasion is capable of producing death more swiftly than cerebrospinal fever." They bring out also the question as to the variance of the pathogenicity of meningococcus itself, apart from the variations in body defence and have attempted in their experiments to discover the special attributes upon which its pathogenicity depends. They found, experimenting with mice, that the major portion of the pathogenicity of the very virulent meningococci is labile and would appear to be closely associated with the ability of the coccus to multiply actively in the tissues of its host. They also found that even when dead the meningococcus still possesses considerable pathogenic

power, definitely proved to be due to a haemolytic substance, a reducing agent and a powerful endotoxin.

Outside the body the meningococcus is quite delicate. It is mentioned by Hoyne that any chilling of a spinal fluid specimen may make it useless for cultural purposes. He recommends the making of cultures on special medium directly from the patient if possible. It is undoubtedly for this reason that many of the earlier efforts at finding the meningococcus in the nasopharynx of those suffering with the disease and in contacts met with negative results.

#### MODE OF DISSEMINATION

In the earlier history of cerebrospinal fever, those who suspected the contagious nature of the disease were baffled in their attempts to explain the mode of transmission of the organism. But in 1896 Keiffer found the meningococcus in the nasopharynx and two years later Councilman, Mallory and Wright did the same thing with a large number of patients. The important discovery, however, came in 1901 when Albrecht and Ghon found the organism first in the throat of a man whose child had died of meningitis and later in the nasopharynx of perfectly healthy individuals who so far as was known had had no contact with the disease. Since that time there have been extensive surveys made to determine the number of healthy carriers, both among contacts and non-contacts. Most of this work has been under military conditions. The two most notable works in civilian life are probably those of Bruns and Hohn in 1908, and Norton and Bailey in 1931. Both sets of observers found that the number

of healthy carriers, both in the immediate vicinity of the patient and in the general community, runs approximately parallel with the number of cases of the disease. The percentage of healthy carriers, however, decreases with the increase of the epidemic, the increase in carriers having come just previous to the increase in the number of cases. Thus the peak in the number of healthy carriers is reached just before the peak of the epidemic and when the epidemic has reached its peak the number of carriers is on the decline.

The following tables, the first by Bruns & Hohn and the second from Norton and Bailey, illustrate this point clearly.

(See Page 15 for tables)



TABLE I

Bruns &amp; Hohn

	No. of cases meningitis	No. of well persons exam'd	No. of carriers found	Pctg of carriers among well persons examined	No. of families examined	No. of families with carriers	Total no. persons in these families	No. carriers among these persons	Pctg carriers among these persons
March	148	56	34	60.7	7	7	23	14	61.7
April	278	360	116	32.2	39	37	135	67	50.0
May	327	408	97	23.8	43	40	172	81	47.0
June	188	352	84	23.9	23	18	93	34	36.6
July	146	323	49	15.1	21	11	67	18	27.0
Aug.	68	287	21	7.3	22	7	119	10	8.5

TABLE II

Norton &amp; Bailey

Meningococcus carriers in Detroit by 2 mo. periods-  
February 6, 1929- February 6, 1931

Date 1929-1931	Contacts				Non-Contacts			
	No. cases occurring	No. persons examined	No. positive	Percent positive	No. persons examined	No. positive	Percent positive	
2-6 to 3-31	131	709	332	46.8				
4-1 to 6-1	310	1,406	207	14.7	117	5	4.3	
6-2 to 7-31	178	777	23	3.0	169	0	0.0	
8-1 to 10-2	70	359	12	3.3	393	2	0.5	
10-3 to 12-4	51	260	9	3.5	81	0	0.0	
12-5 to 2-4	110	668	39	5.7	113	2	1.8	
2-5 to 3-31	164	891	31	3.5	138	2	1.45	
4-1 to 5-31	140	757	23	3.4	207	1	0.5	
6-1 to 7-31	49	210	2	0.95	188	2	1.1	
8-1 to 10-2	26	147	2	1.36	162	0	0.0	
10-2 to 12-1	18	90	4	4.5	164	3	1.8	
12-2 to 2-6	25	142	1	0.7	259	2	0.8	
Total	1,272	6,416	685	10.7	1,991	19	0.95	

It can be seen from the table showing the percentage of meningococcus carriers in Detroit that the average percentage of positive cultures for contacts was ten times that for non-contacts. The percentages found by Bruns and Hohn for contacts and non-contacts do not show quite as marked a difference. In one survey they found 36.7% carriers among close contacts, 22.5% among less intimate contacts and 7.9% among non-contacts. The actual number of carriers in proportion to the number of cases has been known to vary for different epidemics and is said by Vaughan to be as much as from two to forty times the number of cases. It is interesting to note that the percentage of non-contact carriers in the Detroit epidemic varied but not in the same proportion as the percentage of contact carriers. At no time was the non-contact carrier rate over 4.3% and that at the height of an epidemic.

The question of the duration of the carrier state has been carefully studied and it has been found that as a rule the meningococci disappear from the throats of healthy persons within three weeks. Of 685 carriers found in Detroit during the recent work of Norton and Bailey 551 had frequent cultures made until they were released at the end of the two week quarantine period. Almost 70% were found to be negative within 6 days. Of the 551 carriers, 152 or 27.6% were still positive at the end of the 14 day quarantine. They note it is of interest that the percentage of relatively persistent carriers showed a rapid decrease as the epidemic continued.

The following table shows the carrier state lasting less than fourteen days:

TABLE III

Norton &amp; Bailey

Days to 1st negative culture	No. carriers	Percent of Total
1-3	146	36.6
4-6	129	32.3
7-9	81	21.1
10-13	40	10.0
Total	399	100.0

During a recent outbreak of cerebrospinal fever in the Royal Air Force, which was carefully studied and reported by Whittingham, Kirkpatrick and Griffiths, 75 per cent of the meningococcus carriers undergoing nasopharyngeal disinfection daily were negative within four weeks and the remainder 25 per cent were negative at the end of six weeks. The accompanying table shows the length of time the carriers remained positive during the Royal Air Force outbreak.

TABLE IV

Nasopharynx clear of meningococcus	No. of Carriers	Percentage of Carriers
In 2 weeks	18	15)
" 3 "	48	40) 75%
" 4 "	24	20)
" 5 "	15	12½) 25%
" 6 "	15	12½)
Total	120	100.0

Although the observations in the Royal Air Force outbreak in Uxbridge are with too small a number of carriers to be of great value the question of the value of nasopharyngeal disinfection is brought out by a comparison with the carriers in the Detroit observations. In the Detroit quarantine no nasopharyngeal disinfection was used and yet almost 70% of the carriers were negative within six days and 72.4% were negative within two weeks. In the Uxbridge outbreak, where there was nasopharyngeal disinfection among quarantined carriers by means of a spray twice daily, only 15% were negative at the end of two weeks. Only 55% were negative at the end of three weeks and four weeks were required to reach a percentage of 75. Since we have no data on how quickly the remaining 27.6% of positives released from the Detroit quarantine at the end of two weeks cleared up, we cannot make the entire comparison but the percentage of negatives among carriers without treatment is so large, over four times that of those with daily nasopharyngeal disinfection, that it places a stigma of doubt as to the value of the nasopharyngeal spray in clearing up meningococcus carriers. Further observations along this line will aid in our knowledge of control measures.

Apparently the meningococcus has little effect on the health of carriers although there may be in some cases an inflammation of the nasopharynx of such mild degree that there is seldom any complaint. More rarely there may be a severe coryza and in a very few instances a carrier may have slight malaise and a mild headache. The figures of the report of the Royal Air Force outbreak may be considered as fairly representative. The

percentage of the carriers in normal health may be questioned as being a little low but it must be remembered that in civilian life it is much easier to pass over a case of mild pharyngitis than under the strict regime of military regulation. Of the 132 carriers observed 19 had a pyrexia of an influenzal type, 26 had acute nasopharyngitis, 75 remained apparently in perfect health. If the 12 cases of cerebrospinal fever are included, then 43% of the carriers showed signs of some acute infection, 57% remained in normal health and only 10% of those who carried the meningococcus in the nasopharynx contracted the specific meningitis.

It will be noticed in this report that 19 carriers are reported as having a pyrexia of an influenzal type which indicates a possibility of a relationship between influenza and meningococcus meningitis. This possibility was thoroughly investigated by Mr. G. H. Day in London in 1914-1915, and Sir William Hamer reports him as having established the fact that the closeness of the relationship between influenza and cerebrospinal fever is beyond all question. Unfortunately this work was done only on a basis of the association of definite cases of meningococcus meningitis to influenza so that we have no figures as to the proportion of carriers of the meningococcus who suffered from influenza. We may assume that the number was large, however, since the number of cerebrospinal fever cases giving a history of influenza seven days or less previous to the attack of meningitis was ten times that of the "expected." The "expected" number of cases having association with influenza is interpreted as the probable number based on the influenzal rate at that particular time.

PATH of INVASION

There is little doubt but what the meningococcus is carried into the nasopharynx by way of the inspired air. There may be other avenues of invasion but because of the fragility of the meningococcus, according to Vaughan it dies in a few minutes when expelled from the body at temperatures less than 22°C, it is unlikely that inanimate objects such as common eating or drinking utensils, soiled handkerchiefs or towels, aid in bringing the meningococcus to the nasopharynx. According to Hoyne there is still some discussion as to the path of the meningococcus from the nasopharynx to the meninges as to whether it passes through the cribiform plate or enters the lymphatics going on into the blood stream. The weight of the evidence seems to favor infection of the meninges through the blood stream. Herrick, in a recent paper on meningococcus infections, divides the disease into three stages which are practically the same as those given to it by Dopter in 1909. The first stage he considers the carrier stage, which he says seems to be borne out by the observed frequency of mild infections of the upper respiratory tract in communities in which meningococcus infection is rife. The second stage he considers a bacteremia, the clinical picture of which he says is quite definite and may be readily recognized by the alert and experienced clinician. The third or metastatic stage of the disease is the infection of the meninges. Herrick holds that the disease proceeds only through the first stage in many instances, and in other more rare cases gets no further than a meningococcemia which is often the cause of arthritis. As a rule, however, there is involvement of the meninges (90%) if the

disease once leaves the first stage. More often than not the first stage passes unrecognized and in many instances there are but few symptoms of the second stage. Herrick has found positive blood cultures in from fifty to eighty percent of his cases, however, and is undoubtedly more skilled at recognizing the early stages of the disease than most clinicians.

The important question which as yet has not been satisfactorily answered is why the meningococcus causes meningitis in only such a small minority of those in whom it reaches the nasopharynx. In Gordon's report to the Medical Research Council it is suggested that the meningococcus in the nasopharynx of carriers is a saprophyte with parasitic potentialities. There is little doubt, however, that the same strain of meningococcus in the nasopharynx of one person will have apparently no effect whatever on the health of the carrier while in another individual it will cause the most virulent and fulminating form of meningitis. With this in mind, it is significant that the disease is most frequently manifested with the highest mortality rate among those with vigorous health and tends to place the possibility of infection more upon the resistance of the individual rather than variance in pathogenicity of the germ. It is thought by some that a process of autovaccination occurs, immune bodies being developed in the blood stream as small numbers of the meningococci escape into it from the nasopharynx. As far as the membranes of the nasopharynx itself are concerned, the meningococcus is only weakly or entirely non-pathogenic. It is highly pathogenic to the meninges.

PERIOD OF INCUBATION AND INFLUENCE OF CROWDING

It is generally accepted that individuals susceptible to the meningococcus can develop the disease one to five days after coming in contact with a carrier or person suffering from the disease. It is also possible, and often the case that the disease will develop in recognized carriers as late as six weeks after having been found positive. According to Norton the difficulty in tracing the disease from case to case, together with the frequent occurrence of contact carriers, has led to the belief that the disease is most commonly conveyed by healthy carriers. In Detroit 46 cases which could definitely be classed as secondary cases, where there was already one or more cases in the house, or intimately connected, were investigated. In 20 cases the time of onset from the development of the primary case to the development of the secondary case varied from 1-4 days. In 14 cases the time was 5-9 days. In 6 cases over 15 days. It is mentioned by Norton that it is doubtful whether the six cases occurring after fifteen days from the primary case are of much value in determining the incubation period as far as infection from the primary case is concerned.

There is no factor apparently having as much influence on the development of epidemics of meningitis as crowding. By crowding it is necessary to make clear that it is the bringing together of individuals into fairly close contact under conditions favorable for the spread of the meningococcus by droplet infection, especially in moist overheated quarters. The recent outbreak in the Royal Air Force which we have already mentioned



was found directly attributable to the crowding of men around the stove in the center of the barracks room and in the canteen. With the closing of the canteen and substitution of central heating the epidemic promptly ended. It is explained by Vaughan that the epidemic in the winter of 1917-1918 was due not to the temperature which was very low, but to the fact that the cold weather drove people indoors where they congregated and came into close contact. Coughing, sneezing, and spitting are more prevalent during the winter and spring months and this associated with the fact of crowding indoors in abnormally high temperatures makes conditions ideal for the transfer of meningococci from one individual to another.

#### AGE INCIDENCE

As mentioned in the introduction, there is some disagreement among many authorities on the influence of age in meningococcus meningitis. Primarily, however, it seems to be a disease of childhood and adolescence. In the New York epidemic of 1905 of 2,180 cases of cerebrospinal fever, 67 percent were under ten years of age. In the Prussian epidemic of the same year 80.12 per cent were below sixteen years of age. In Denver in the five year period from January 1927 to December 1931 the highest case incidence occurred in the age group from one to nine, although the highest death rate occurred between the ages of fifteen and nineteen. The age distribution in Norton's carrier study in Detroit is interesting. It was found that there is a slightly greater tendency for children between one and ten years to become carriers, which coincides with the other data we have

presented as regards the development of actual cases of meningitis. The percentage of carriers among infants is extremely small and is pointed out as worthy of note because of their known high susceptibility to meningitis. The age incidence among military groups is of little significance because of the fixed age limit of the group. Their reports, however, show 20 to 23 to be the most susceptible ages.

#### CONTROL MEASURES

Control measures for any epidemic disease must necessarily be based on the characteristics of that disease as to mode of spread, degree of contagion, relation of insanitary conditions, relation to crowding, influence of location, relation to fatigue, practicability of immunization, et cetera.

In the case of meningococcus meningitis it must be borne in mind that we have found it to be essential that in order to become a carrier the individual must come in close contact with another carrier of the meningococcus under conditions favorable to the transfer of the organism, which is most preferably a warm, moist atmosphere of a temperature over 71.6°F. Among military groups the problem of control is much more easily handled since temperatures can more easily be kept at a safe level and carriers can be quarantined until free from the infection. The report of the Committee on Standard Regulations for the Control of Communicable Diseases of the American Public Health Association recommends the following:

1. Recognition of the disease and isolation of infected persons until 14 days after the onset of the disease.

2. Increase the separation of individuals and the ventilation in living and sleeping quarters for such groups of people as are especially exposed to the infection because of their occupation or some necessity of living conditions. Bodily fatigue and strain should be minimized for those especially exposed to infection.

3. Carriers should be quarantined until the nasal and pharyngeal secretions are proved by bacteriological examination to be free from the infection.

4. Prevention of overcrowding.

In the same report immunization is mentioned as in the experimental stage. The same condition is also reported by the British Research Committee.

In a discussion of these measures McCoy states that the one measure upon which everyone agrees is that persons suffering from the disease should be isolated. It can be readily seen that other measures, especially the quarantine of carriers, are practically impossible to enforce in a large civil community.

Further, there is considerable argument as to the value of nasopharyngeal disinfection of carriers. As we have seen in a comparison of disinfected and non-disinfected carriers earlier in our discussion, the statistics indicate that carriers without nasopharyngeal disinfection become negative considerably sooner than those having a nasopharyngeal spray twice daily.

Practically the same measures as those recommended by the American Public Health Association are advocated by Laybourne in a study of epidemic meningitis in Missouri. He adds that

the control of epidemic meningitis is primarily a function of the health officer rather than the bacteriologist and the general carrier survey. In this the writer agrees to some extent, especially in the phase of education of the public to recognize the dangers of overcrowding, excessive room temperatures, and poor ventilation. There should be more instruction in personal hygiene and to this end the words of Vaughan are significant. "When man has become sufficiently well trained in personal hygiene that he is able to avoid receiving all consignments of bacterial flora from the upper air passages of his neighbors, the last of the meningococci will die." The possibility of such a situation brought about through the public health officer at the present time seems remote and since, according to Rosenau, it is not clear that any of the measures taken so far have either materially influenced the course of epidemics or prevented the spread of the disease, we must look to the immunologist for the solution of our problem of control.

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