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No. 4.

REPORT ON THE PANDEMIC OF INFLUENZA, 1918–19.

MINISTRY OF HEALTH.

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REPORT ON THE PANDEMIC OF INFLUENZA, 1918–19.

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REPORT ON THE PANDEMIC OF INFLUENZA, 1918–19.

CHIEF MEDICAL OFFICER'S INTRODUCTION.

To the Right Hon.
Christopher Addison, M.D., M.P.,
Minister of Health.

SIR,

1. I HAVE the honour to submit a report on the Influenza Epidemic of 1918 and 1919. This report, taken in conjunction with the *Report on the Mortality from Influenza in England and Wales during the Epidemic of* 1918–19 recently issued by the Registrar-General (Cmd. 700), constitutes the official record which it is necessary to make concerning the outbreak. The report has been reduced to the narrowest compass compatible with an adequate presentation of the facts and the inclusion of records from various parts of the world. There can be no doubt that as an historical survey it will prove invaluable for future reference in the event of subsequent epidemics.

2. This document deals with one of the great historic scourges of our time, a pestilence which affected the well-being of millions of men and women and destroyed more human lives in a few months than did the European war in five years, carrying off upwards of 150,000 persons in England and Wales alone. The report is divided into three parts. Part I. has been prepared by Dr. Major Greenwood, with the assistance of Dr. Thomas Carnwath, both of them Medical Officers of the Ministry, and with the collaboration of Dr. Herbert French (Physician to His Majesty's Household and to Guy's Hospital) and Sir Frederick Andrewes (Professor of Pathology and Bacteriology at St. Bartholomew's Hospital, University of London). It deals with Influenza in Great Britain and Ireland, and will be found to be a contribution of exceptional interest and suggestiveness. I desire to express my high appreciation of its value, and of the public service its writers have rendered in its preparation. Part II. has been compiled by Dr. Robert Bruce Low, C.B., and Dr. S. P. James, Medical Officers of the Ministry. It presents an account by the former of the incidence of Influenza in Europe and the Western Hemisphere, and by the latter of its incidence in Australasia and parts of Africa and Asia. The section contains a large amount of information not hitherto collected respecting the epidemic in many parts of the world.

Part III. contains 12 special papers reporting inquiries into different aspects of Influenza as it occurred in this country. Several of them are by Medical Officers of the Ministry, but we are fortunate in being able to include papers from Dr. James Niven, the Medical Officer of Health of Manchester, and some of his colleagues in the Public Health Service, and a paper by Dr. Brownlee.

3. This Report does not contain a detailed history of the previous outbreaks of Influenza in this or other countries. In 1852 Dr. Theophilus Thompson described, in his *Annals of Influenza in Great Britain*, the record as it concerns this country from 1510 to 1837; the epidemic of 1847 was described by Dr. T. B. Peacock; Hirsch published in his Geographical and Historical Pathology a list of outbreaks in different countries from 1173 to 1875; in 1890 Dr. Clemow ventured even further back and discussed the probable influenza epidemics from 877 to 1481; in the following year (1891) was issued an official report to the Local Government Board by Dr. Franklin Parsons on the epidemics of 1889–92 (also recorded by Dr. Dixey); and in 1891 and 1894 there appeared the two volumes of Dr. Charles Creighton's book on the *History of Epidemics in Great Britain*, containing his review of the occurrences of the disease in this country. Speaking generally it is evident that since early times there have been outbreaks of epidemic sickness not distinguishable from what we now think of as influenza, though it is certain that the generic term has been used to cover a wide variety of catarrhal conditions. Creighton begins his list of influenza epidemics with 1173, and his examples include the epidemics of 1510, 1540, 1557, 1580, and that of 70 years later, the outbreak of 1657, which was described by Willis in what Creighton characterises as "the first systematic piece of epidemiology written in England." Then we come to the period of Thomas Sydenham and his description of the "epidemic coughs" and subsequent *febris comatosa* of 1675 and 1679. He argued that one epidemic constitution passed gradually into another. There followed at irregular intervals a series of outbreaks throughout the 18th century; in 1833 a severe epidemic of influenza occurred, succeeded by less conspicuous prevalences until the pandemic of 1847. This was reported to have caused an excess of 5,000 deaths during the six weeks that it lasted. In the three worst weeks (December) it raised the deaths at the age of childhood 83 per cent., among adults 104 per cent., and in old age 247 per cent.

The subsequent course of the disease may be traced on the accompanying chart. Except for a small epidemic in 1855, the trend of prevalence was steadily downward until 1870. During the two following decades influenza almost disappeared from the death records of this country. Then came the great pandemic of 1889–1892, which initiated a new phase in the

evolution of the disease—the phase of complete victory in which "infective power is maintained, even enhanced, and "to this is added a toxicity surpassed by few epidemiological "competitors." Since 1890 influenza has maintained its position as a factor of great importance in the causation of mortality. Outbreaks of considerable magnitude occurred in 1895, 1900, 1908, and 1915, but that in 1918–19, which is the subject of the present report, far surpassed anything previously experienced. In 1918 there were 3,129 deaths from influenza per million of population in England and Wales. The corresponding figure for 1919 was 1,170.

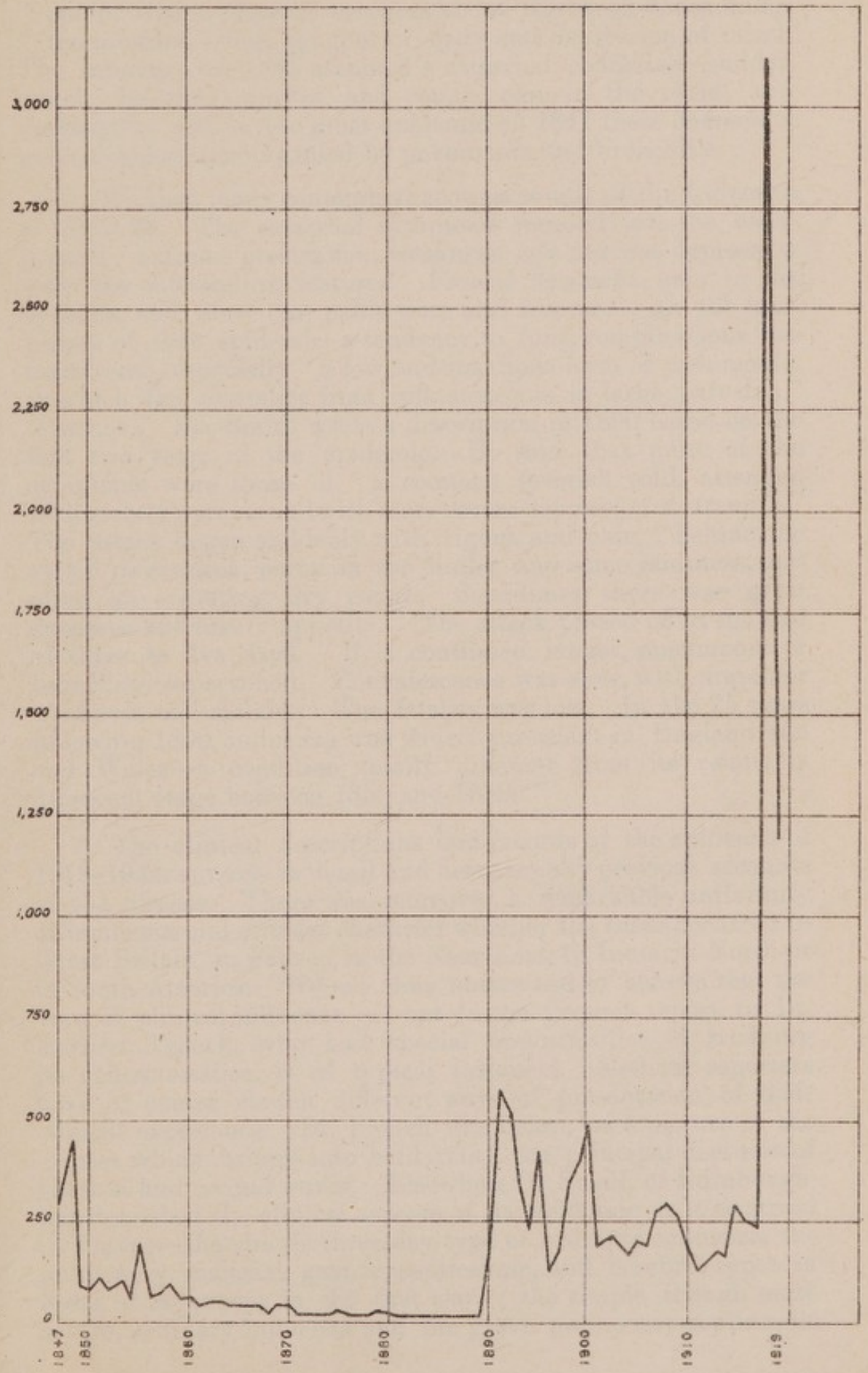
The pandemic of 1889–92 revealed for the first time the disease in successive waves separated by short intervals. Dr. Franklin Parsons furnished a lengthy report on the epidemic of 1889–92, treating of its occurrence both in this and other countries. He also discussed the etiology (Pfeiffer's bacillus had not then been isolated), incidence, and local behaviour of the disease, giving brief notes on some of its clinical features. Dr. Klein added an appendix on the pathological aspects. Dr. Parsons concluded that the disease was imported (probably from Russia), that it followed the lines of human intercourse, that it was spread from person to person, that it prevailed independently of season and climate, that the first cases were mild and the subsequent cases more severe. Whilst the report did not greatly add to our knowledge of clinical influenza, it dealt fully with incidence, and it effectually dispelled the then prevalent belief that the disease was miasmatic. Parsons showed, by his detailed records of the behaviour of the disease in areas and in institutions, that it spread, independently of the weather, from person to person.

The Clinical Character of Influenza.

4. The influenza of the 17th century had, as its most constant symptoms, cough, vomiting, hæmoptysis, aches and weariness in the head, back, and limbs, and some fever. In addition there was frequently thrush and epistaxis. According to Sydenham the disease was of the nature of "epidemic ague" up to 1685, but afterwards changed its character to that of "pestilential fever." In 1729 Huxham, of Plymouth, described the disease as "a catarrhal febricula, racking pain in the head, "delirium with incessant cough, slight dyspepsia, anorexia, "langour, and rheumatic pains"; a few years later he characterised it as having also rigors, "flying pains" in the back, and violent headaches; and once more, after ten years, he adds pains in the joints and universal lassitude as the leading features. In 1767 Heberden described the attack seen by him as beginning with severe chills, then a troublesome cough, acute pains in the head, back, and abdomen, with fever, and prostration. Fifteen years later the College of Physicians

To face p. VI.

ENGLAND & WALES. DEATH-RATES PER MILLION POPULATION. FROM INFLUENZA 1847-1919.



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said that "the universal and almost pathognomonic symptom " was a distressing pain and sense of constriction in the " forehead, temples, and sometimes in the whole face, accom- " panied with a sense of soreness about the cheek-bones under " the muscles," with langour of body and depression of mind. The influenza of 1833 assumed a catarrhal character—sudden attack, headache, coryza, and cough, pain in the chest, and depression; and in the great epidemic of 1847 these characters predominated, accompanied by pneumonia and bronchitis.

5. We have many contemporaneous accounts of the Influenza of 1889–92. The catarrhal symptoms receded into the background; extreme prostration, weakness, and nervous depression were the outstanding features. Frontal headache, pain in the eyeballs, and muscular pains were also common. In the later phases of that epidemic a tendency to lung complications was manifested, especially "a low and insidious form of pneumonia, to which the mortality from influenza was in large part due" (Parsons). Pye-Smith wrote a description in 1890 based on the first two years of the epidemic. He said that most of the symptoms were those of "a common feverish cold, attended with greater pyrexia and with more severe depression of strength." The attack began suddenly with rigors, and pain "behind the eyes," prostration, pains in the limbs, and some faintness, and often an irritating dry cough. Sometimes there was great dyspnœa and loss of appetite. The attack passed off at the end of three to five days. If it continued longer, pneumonia or bronchitis supervened. Convalescence was slow, with muscular weakness and malaise. The fatality was low. In the 25 years following 1890, influenza was widely prevalent in England and and Wales—a condition totally different from its relatively quiescent stage between 1850 and 1889.

6. The clinical descriptions and records of the epidemic of 1918–19 far surpass in detail and accuracy any previous accounts of the disease. There was, moreover, a remarkable uniformity of incidence and clinical character whether the cases occurred in Great Britain, in France, in the Near East, in India, in North or in South America. We are thus authorised to believe that the view of clinical influenza set out in the present report by Dr. Herbert French, who had special opportunities of studying its characteristics, is of typical instances. Medical reporters have of course chosen different ways of presentation of their clinical experience. Dr. French sets before us a picture of the disease which brings into bold relief the principal features of the first and second waves. Elsewhere Dr. Small, of Edinburgh, has described the clinical aspects of the epidemic as it occurred in France—the simple three-day type of ordinary influenza, the pulmonary, malarial, gastric, septicæmic, and cerebral types in order of frequency in the first wave; the simple, though more severe, ordinary influenza and the grave pulmonary-septicæmic

type of the second wave. Dr. Burnford, in Macedonia, found that soldiers from various parts of the world suffered clinically from (*a*) pharyngitis, with or without implication of the mucosa of the nose and sinuses, (*b*) laryngitis, (*c*) tracheitis, (*d*) bronchitis, (*e*) bronchiolitis, or (*f*) pneumonitis, with involvement of the whole lung tissue—indicating a progressive infection of the respiratory tract.

7. All observers are agreed as to the differentiating characteristics of the two waves, the first in June and July, 1918, the second in October and November, 1918 (the third presenting the features of the second, in February, 1919). The *first wave* was one of "the three-day-fever" type. There was sudden onset, lassitude and prostration, general aching, a rapid rise in temperature, a relatively slow but unstable pulse, coated tongue, loss of appetite, a sore throat, and a congested nasopharynx, headaches, a rapid convalescence, and a low mortality. Though the respiration was quickened there was no dyspnœa, gastro-intestinal symptoms were common but not severe, and, as a rule, there was no albuminuria. There were, of course, variations and complications, the most frequent involvement being the respiratory tract. Whilst coryza was rare, epistaxis and other hæmorrhages were common; substernal pain occurred, and was worse owing to cough, which was almost universal. The progressive changes in the sputum were significant. The chief nervous conditions were psychical, and sleeplessness was common.

On its clinical side the outbreak was remarkable, not for its virulence or mortality, both of which were low, but for its complete change of age incidence. It attacked youth. On its pathological side a damaged condition of the capillary vascular system was distinctive, leading to hæmorrhages in the mucous and serous membranes, the respiratory tract, and the lungs. In some ways it seemed to be a "bacteriæmia" localised in particular in the pulmonary blood vessels, and hæmorrhage paved the way for secondary infections.

8. The clinical features of the *second wave* ushered in much more severe forms of the disease. As Dr. French points out, of a thousand individuals attacked in the autumn, about 800 suffered from the three-day-fever type, though of somewhat unusual severity; the remaining 200 displayed pulmonary complications, and of these 80 may be thought of as moderately severe and 120 as desperately ill. Of the 120, between 60 and 80 would prove fatal (Abrahams). Many cases began as in the first wave, though epistaxis appeared to be particularly common. Some cases became pulmonary at the outset, others in a few hours or days. The pulmonary involvement was an acute infective inflammation, sometimes progressing as a broncho-pneumonia, at other times it swept through the body like a virulent toxæmia or septicæmia. In some cases there were

few or no physical signs, in others dulness, bronchial breathing, crackling râles, bronchophony, and pectoriloquy; in others signs of coagulative œdema, hæmorrhage, abscess, and collapse. The normal progress of a pneumonia case was rare. There was pain, headache "behind the eyes," ear-ache, cough, a high respiration rate ("polypnœa"), variable fever, followed by a toxaemic or pulmonary complication on the third or fourth day. The toxæmia was heralded by early cyanosis, delirium, a rapid pulse and epistaxis. The heliotrope cyanosis indicated a bad prognosis; it was evidently not cardiac in origin, but an intoxication, recalling the "purulent bronchitis" experienced in 1916–17 at Aldershot (expectoration of enormous quantities of purulent sputum, heliotrope cyanosis, and high fatality), itself an infection due in part to *B. Influenzæ* and in part to the *Micrococcus catarrhalis*, streptococcus, or associated organisms. There are grounds for believing that both forms of cyanosis were caused by an albuminous exudate in the alveoli and interstitial tissue of the lung. The pulmonary complications which arose were various, and included œdema, broncho-pneumonia, hæmorrhage, effusions, and sometimes abscess. The onset of the lung changes was indicated by alteration in the character of the sputum, increased respiration rate, pain, and diminished respiratory movements and air entry (Sundell). The mild type of the first wave and the contrasting type of the second and third waves seem to have been very much the same everywhere, except that different stages of the epidemic did not occur at quite the same periods or with uniform sequence in different countries.

9. It should be added that in February 1920 there was, after nine months' quiescence, a further rise in influenza in this country and a sharp outbreak in some of the chief cities of the United States (beginning in January) and in Switzerland and Denmark. The disease was mild and caused relatively few deaths, the age incidence reverted, with some exceptions, to that of pre-epidemic years (a high incidence in childhood and old age, and low in early adult life).

The Causation of Influenza.

10. The problem of the cause of influenza is not yet solved. Clinicians, bacteriologists, and epidemiologists are all agreed that Pfeiffer's bacillus is associated with the disease, but whether it is the cause remains unproven. Dr. French thinks it has not yet been deposed from its place as the causal organism, but Sir Frederick Andrewes considers that its position as the primary cause has been in no way strengthened by the experience of the recent pandemic. Many observers would be found to agree with Dr. French that in all probability this organism was the primary one responsible for the three-day-fever type of influenza common in the summer and autumn of 1918; but

that the graver cases were due to a combination between the Pfeiffer bacillus and an additional organism or organisms (streptococcus, diplococcus, or diplo-streptococcus) virulently or symbiotically associated with it. In a word they would suggest that the "influenza" cases were caused by Pfeiffer's bacillus, whilst the septicæmia or pneumonia types were due to an associated group of organisms. The significance of this association, however, requires further investigation.

11. As Sir Frederick Andrewes points out in his lucid and able review of the bacteriology of influenza, there are three means at hand enabling us to form a useful opinion on the part played by a specific organism. First, there is its presence in cases of the disease—its presence, isolation, separate culture, and subsequent inoculation and reproduction of the disease. The fourth of these postulates has not been even approximately satisfied, but the presence of the organism in clinical cases of the disease, its isolation from the body, before or after death, and its cultivation have all been verified in an absolutely and relatively considerable number of instances. Even here, however, we are met with the substantial difficulty that in the summer of 1918 the presence of the bacillus was not uniformly or universally demonstrated. During the earlier outbreaks of the disease in 1918 Pfeiffer's bacillus was found in some countries in a large proportion of cases; in other countries it was sometimes found and sometimes not; in Germany only quite exceptionally was it found. Speaking generally, the *B. Influenzæ* was absent in post-mortem examination and diplo-streptococcus was present. It is difficult to believe that differences of media or method fully explain this discrepancy, for though the bacillus is not easily demonstrated, it had been frequently isolated in previous outbreaks of the disease. However that may be, we are in fact faced with (*a*) the absence of a universal and uniform demonstration of it, which we get for instance of the tubercle bacillus in tuberculosis, (*b*) an absence of specific result invariably following its inoculation in animals, and (*c*) even the relative failure to convey the disease to man by spraying with cultures of Pfeiffer's bacillus.

The second means at our disposal is a study of the specific immunity reactions. We know that particular antibodies arise in the blood of the patient suffering from influenza, as a result of the activity of the causal organism in the body; in a word, an expression of the body's attempt at defence. Now it is known that, both by agglutination and by the complement fixation test, the serum of the patient suffering from influenza shows specific reaction to Pfeiffer's bacillus (between the second and sixth days of the disease), a reaction which blood from the normal person fails to produce. Whilst such a reaction demonstrates the frequent intervention of Pfeiffer's bacillus it does not establish that organism to be the primary cause of the disease. A third line

of evidence is sometimes obtainable in the effect which a pure vaccine has upon the disease. If it could be shown that a vaccine prepared from pure cultures of Pfeiffer's organism cured or prevented the disease in a large number of trials, this fact would be strong evidence in favour of its etiological importance. In truth, however, such pure vaccines have been too rarely used to furnish us with a basis of general reasoning. It is true that our experience of mixed vaccines is wider ; this experience, however, is hard to interpret, and even had such vaccines been proved to be of great value in influenza, their polyvalence would make it impossible to assign to each constituent its correct rôle in the etiology of the disease. We are, therefore, left at the end of the pandemic with our previous knowledge of Pfeiffer's bacillus confirmed but not much extended. Sir F. Andrewes summarises our present knowledge of this bacillus as follows :—

(1) It is not infrequently present in the pharynx of normal persons, living there as a temporary and apparently harmless saprophyte.

(2) It is still more commonly found in catarrhal conditions of the respiratory tract, and its presence under such circumstances may, or may not, be associated with fever and constitutional symptoms.

(3) It is the apparent cause of certain chronic inflammatory conditions of the throat, nose, and accessory sinuses.

(4) Certain cases of meningitis have been associated with the presence of this bacillus or of one closely related to it.

(5) In rare instances it has been found to be the exciting cause of malignant vegetative endocarditis.

(6) It is closely and profusely associated with lesions of the respiratory tract in human influenza and is toxic for animals, though specific influenza has not been truly reproduced in them. It is frequently present in the respiratory complications of the disease in man. "Its "case remains unproven, and the crucial tests to "which it has been submitted seem to indicate it "rather as a secondary infection of the highest "importance and significance than as the primary "'materies morbi.' At the same time it cannot be "asserted that as a primary cause it is wholly out of "court." On the other hand "the complications to "which the epidemic has owed its abnormal fatality "have been due to secondary infections, in which "Pfeiffer's bacillus and the hæmolytic streptococcus "have played a predominant part."

Sir F. Andrewes explains in his chapter why the evidence in favour of a "filter-passing" virus as the primary cause of the disease, suggestive though it may be, is not at present of a conclusive nature.

12. It is 28 years since Pfeiffer announced, at the end of the last pandemic, his discovery of the bacillus known by his name. It may appear at first sight to be somewhat disappointing that we have not been able within a generation to decide finally as to its relation to the disease. This is due in the main to our limited knowledge of the natural history of the disease, its lack of definition, its protean manifestations, and its liability to numerous complications which tend to confuse the issue for the bacteriologist. But his technique and methods are improving and his scope is rapidly widening. Protozoology, bacteriology, parasitology, immunology, agglutinins, filter-passing viruses, the carriage by healthy persons of pathogenic and saprophytic organisms, the assumption of virulence by saprophytes, secondary infections—all these now come within the ambit of the bacteriologist as well as the study of the nature of bacteria themselves. Nor must we forget to avail ourselves of the aid of epidemiological, statistical, and clinical study. Nothing is more certain than that the growth of knowledge in pathology in any direction advances growth in all directions; and the investigation of the cause of diseases allied influenza, to common cold, all the catarrhal conditions, all the diseases which accompany it as predecessors or successors is of first rate importance in solving the problem of the etiology of influenza itself.

The Epidemiology of Influenza.

13. The epidemiological features of the pandemic are even more complex and puzzling than the clinical characteristics, and the present report does little more than present a large body of data which await interpretation. Some facts concerning the incidence and behaviour of the disease we certainly possess, though a satisfying explanation of them may be lacking; other data are of insular or local interest and do not throw much light on the pandemic; a further group of observations remains as yet unverified. Almost all our data—and this report presents but a selection of them—require careful and prolonged study, a judicious co-ordination and much further research. We have passed, it is true, from the former theories regarding origin—the miasmatic theory, the telluric and climatic theories; the importation theory; or the idea that influenza is a specific disease like anthrax, arising and progressing within narrow and definitely circumscribed limits. These are hypotheses no longer confidently held. They do not answer the fundamental questions raised by the pandemic through which we have passed.

14. With the object of obtaining a bird's-eye view of the problem we may briefly summarise some of the principal features of the epidemiology of the recent outbreak, without discussing at length their causation or significance.

15. And in the first place we have to do with *an epidemic wave-form* in influenza. The outbreak of 1918–19 expressed

itself in a *three-wave type*, of which the second wave was much the most severe. The first wave, as measured by mortality, began in this country at the end of June, reached its zenith in the first half of July, and fell by the end of July; the second wave commenced in October and reached its summit early in November, remaining high for two to three weeks, and falling slowly to the end of December; the third wave began at the beginning of February 1919, rose to its summit (higher than that of the first wave but not so high as that of the second) in the third week of February, and fell slowly to the end of March. As regards period of wave the first occupied approximately six weeks (with eight weeks' quiescence), the second twelve weeks (followed by four weeks' quiescence), and the third eight weeks. As regards mortality the Registrar-General states that during the 46 weeks from June 23, 1918, to May 10, 1919, the total deaths attributed to the disease in England and Wales were 151,446, approximately three-quarters of which occurred in the second wave. It will be understood that whilst this is the record as a whole for this country, and particularly for its urban communities, there were considerable local variations in periodicity, incidence, and case mortality. For example, at Liverpool the first wave, as measured by mortality, was less and the third wave greater than in the country as a whole; at Birmingham and at Manchester the third wave reached its highest in March; at Cardiff and at Portsmouth there were practically only two waves, in October and March; at Coventry there was one substantial wave reaching its summit in November; and at Sheffield there were three waves—July, November, and March, but the November wave was incomparably the most severe. So also in Stockholm there was an August wave and an October one; at Chicago a very high incidence (with a relatively low mortality) in October only; in other countries also variations occurred. The broad facts remain that the epidemic presented three waves and that they differed in form. The first short, sharp and high; the second slower in formulation, flatter, higher and more destructive of life; the third also slow in formulation, lower, prolonged in decline, and partially reverting to the older influenzal form —a condition of things which suggests a biological factor modified by environment, but not, unhappily, directly controllable by human agency.

16. In the second place we must recognise that in many parts of the world influenza broke out in the summer of 1918 with *apparent suddenness and showing an undoubted rapidity of evolution*. It is true that in 1915, in England and Wales, an excess of deaths was attributed to influenza, but the excess disappeared in 1916 and 1917, to be followed in the summer of 1918 by a sudden explosion at the end of June. As in 1890–92 the primary wave thus generated after some years of relative quiescence spent itself after a few weeks, though it attained a

great elevation. The secondary wave passed much more slowly through its phases, and whether anticipated or not it came with much less suddenness. In Germany and in Austria there were outbreaks of influenza in 1915–1916 and in 1917, but in both countries the outbreak of 1918 exploded in July. In America, too, influenza had been occurring in greater or less degree in 1915, 1916, and 1917, but it became epidemic in mild form in April and May, 1918, and in September developed more virulent characters and swept over the country, mainly from east to west. In Paris the epidemic broke out in July; in Spain at the end of May; in Italy in April; in Greece in May; in Persia and Mesopotamia in June; in India in June; in Australia and New Zealand in July. Such was the universal experience of emergence and the rapid evolution was similar to it. The disease simply had its way. It came like a thief in the night and stole treasure.

17. Thirdly, the epidemic was remarkable in respect of the *mortality*. It should be borne in mind that the fatality of influenza is low, but its incidence is so vast that the number of deaths create an excessive mortality. Thus, it comes about that the epidemic destroyed more lives in the whole world than did the European war in five years. The peculiar character of this pandemic was that the type of age distribution which had consistently characterised influenza mortality for many years suddenly and completely changed with the onset of the summer epidemic of 1918. Deaths at 0–15 years of age increased from 7 to 11 per cent. (of the epidemic of 1889–92) to 25 per cent.; at 15–35 years of age, from 8 to 10 per cent. to 45 per cent.; at 35–55 years of age there was little or no change; but deaths at 55–75 years of age and upwards, which formerly provided 60 to 70 per cent. of the total registered, contributed in this epidemic only 10 per cent. up to 75 and 2 per cent. over 75 years. In a word, this epidemic presented a sudden and very remarkable change in the behaviour of influenza. It destroyed not the very young or the old, but the adolescent and the adult. What is the explanation of this complete change of age incidence? The customary explanation is that the older persons in the population may have enjoyed an immunity, owing to attacks in a previous epidemic (in this case 28 years before) or in intervening prevalence. But the obtainable evidence is to the effect that the degree and extent of such acquired immunity is slight, transient, variable, and incomplete. A second explanatory suggestion attributes the change in age incidence to alterations in the circumstances of the population. Soldiers have been aggregated for war purposes, young men and women in munition works, large sections of adult populations have moved in bulk owing to trade or transport exigencies, and thus the disease had greater opportunity of fastening upon these aggregated populations under exceptional surroundings. But this epidemic was a pandemic, and all classes came within its ambit, the

change of age incidence occurred in non-belligerent countries not directly affected by the privations of war, and in all parts of the world; lastly, it occurred suddenly at the outset (and not as a sequela), differentiating itself clearly from the influenzas of 1917. A third explanation is that the adolescent and adult population were suffering from the debilitating influences of war, from strain, and from exposure. No doubt this factor played a part, but we cannot escape the knowledge that the epidemic ravaged populations of this age-period who were not subjected to these unfavourable circumstances. A fourth suggestion is of a more narrowly biological character. It propounds the view that the epidemic was the pathological expression of a new strain of infecting virus, or one to which adolescent and adult tissues were particularly susceptible. This seems to require for its support evidence of importation or of origin of such a virus, and we have few or no facts in that behalf; moreover, the characters of the disease which proved most fatal and which we must assume would be attributable to such a new or enhanced virus had already manifested themselves sporadically and even epidemically in England, France, and America in the years immediately preceding the pandemic; and, still further, it must not be forgotten that the changed age incidence obtained at the beginning of the first wave of the pandemic and in the mild three-day type of influenza, as well as during the second and more severe wave of fatal septicæmia. The age incidence, in short, was peculiar to the whole emergence of the pandemic, mild or severe, and it was almost universal, under diverse conditions of economic, social, and racial environment.

18. A fourth characteristic of the recent epidemic was its relation to the *social conditions of the people* affected. In 1847 Farr recorded the fact that the influenza of that periodic epidemic was more deadly to people living in insanitary conditions and attacked in particular "persons labouring under zymotic diseases," such as whooping cough, measles, and typhus fever. It is interesting therefore to find that in the recent epidemic neither of these propositions can be proved to hold good. The mortality fell alike upon "the sanitarily just and unjust;" the Registrar-General's report makes this clear in a set of valuable tables showing the incidence on the towns and in the London boroughs; it was not associated with, nor apparently did it increase the severity of, zymotic sicknesses or tuberculosis. The facts did not even demonstrate, as will be seen in the discussions in the present report, that domestic overcrowding was a principal factor in the spread of the disease. It must not be supposed, however, that social conditions played no part in the matter. The great probability is that the social evolution proceeding in the later years of the nineteenth century raised the sum of the etiological factors to near their critical values, and maintained them closer to those critical values than in any

earlier age. Even in a world-wide pandemic the problem of epidemic influenza may be largely an internal problem of each nation, a problem of social relationship, of social factors, of domestic habit and life. The materials of the conflagration come from within. As the compilers warn us, the wolf is in the fold all the time. "His ravages depend as much upon the disposition of the sheep within the fold as upon his appetite."

19. There is a fifth point of great interest and importance which has led to much discussion respecting the epidemic, and that is the *relation of influenza to other contemporary diseases.* Is there an "epidemic constitution" of influenza? Is it an epidemiological entity by and of itself, or is it a manifestation of an infection or group of infections also represented in other diseases? To answer these questions we must consider our growing experience of the "setting" of an influenza epidemic. Attention is drawn in the report to various epidemiological records, which go far to establish—

(*a*) that the epidemiological features of the cycle of years within which influenza explodes are different from those of the influenza-free cycles;

(*b*) that preceding epidemic influenza there is often a rise in general morbidity of the population, an "epidemic constitution" develops favourable to influenza, there are early though often mild and atypical clinical forerunners of the disease, and parallel or allied clinical maladies are seen;

(*c*) that there are concurrences, similarities, and inter-relationships between outbreaks of cerebro-spinal fever, poliomyelitis, and outbreaks of influenza, bronchitis, and pneumonia.

Many facts lead us in the direction suggested in these three propositions. For example, the epidemiological history of England and Wales for several years previous to 1918, as previous to 1847, was different from the normal. In his special report on Influenza to the London County Council in June 1919, Dr. W. H. Hamer furnished evidence of such differences in London; the present report discusses the subject still more widely; and we have ample indications of the incidence of disease elsewhere which preceded the influenza outbreak. There were outbreaks of infectious pneumonia in German prisons and camps in 1915; cerebro-spinal fever and poliomyelitis had been increasing in Great Britain in 1915–1917; there was exceptional prevalence of "influenza" or "pseudo-influenza" in the United States of America in 1915 and 1916; obscure but extensive febrile "pneumonic" outbreaks occurred on both eastern and western war fronts in 1916 and 1917; in the same years there were the epidemics of purulent bronchitis, bronchiolitis, and pneumonia at Aldershot (Hallows, Eyre, Abrahams, and French), apparently a primary influenzal infection followed by strepto-

coccus; in the winter of 1917–18 there was much purulent bronchitis among young soldiers in France, as there had been at Aldershot, with pneumonia, pleurisy, empyema, and toxæmia (Abercrombie, Hammond, Rolland, and Shore), and showing cyanosis, nummular sputum, pyrexia, and high mortality; and there was, in England and Wales at least, a continuous smouldering of influenza itself which in 1915 caused more deaths than in any previous year of this century. It seems impossible to escape the conclusion that these various conditions bore a fundamental relationship to each other and to the pandemic of influenza. Or we may consider another group of facts. There is an essential similarity between the epidemic incidence curves of influenza and bronchitis; an increased incidence of bronchitis and pneumonia not infrequently precede an increased incidence of influenza; the true curve of influenza mortality can apparently only be constructed by combining with it the simultaneous waves of pneumonia and broncho-pneumonia. A similar relationship seems to obtain, though it cannot at present be measured, between the incidence of "common colds" of feverish type and of influenza itself. Again, we have also to bear in mind that not only do historic records associate chronologically outbreaks of influenza with the sporadic or even epidemic occurrence of obscure nervous illness, but that the more exact clinical methods of modern times reveal close analogies between the general and nervous symptoms of acute poliomyelitis, of lethargic encephalitis, and of cerebro-spinal fever and those of the more severe forms of influenza. The convergence of these paths of investigation leads to the surmise that the admittedly vague concept of an "epidemic constitution" does indeed correspond to something in nature; that there really is a development over a series of years of various clinical forms of infective disease which tend to assume a generic type and to prevail before and after an epidemic of influenza.

20. Generalisations are valuable though often unreliable—particularly unreliable when founded upon a slender basis of established facts. But in the present situation we must not decline to attempt the formulation of an outline of the possible or probable cause of the pandemic. Taking into consideration the data furnished in this report we may hazard the view that the recent visitation—its character and degree as well as its age incidence—was due to the operation of an exalted virus (possibly an infecting agent, parasite or saprophyte, the virulence of which had been increased by human passage) or an unusual infection of associated or symbiotic organisms, the potential virulence of which had for many months, perhaps years, been rising to a critical and stable level. This specialised agent acted upon the adolescent and adult human tissues, which had an inherent susceptibility of response to its toxic effects engendered by a complex of factors—lowered vitality or resistance owing to strain, exposure, deprivation, or physical disability; or a virginity

of tissue soil to the particular infecting agency; or the unsuspected carriage by sick or healthy persons of saprophytic or benign organisms awakened to pathogenic virulence by the infecting virus; or tissues rendered susceptible or vulnerable by the influence of other organisms. Social factors no doubt played a part in preparing the infecting seed or the soil, and in bringing to the critical and dispersive point the conditions which had hitherto been sporadic and restricted. Among these we must in particular include the extra-domestic aggregations of persons. I cannot do better than quote the general theory of the epidemiology of influenza which the compilers of the report suggest as a "working hypothesis." They say:—

> "The story, then, of the germ of influenza is divided into three phases. The first, which lasted for many centuries, was, if we may be permitted to use teleological language, a series of attempts to maintain a high level of infectivity or dispersiveness, which attempts were unsuccessful. The second phase, ushered in by the year 1889, is marked by a partial victory of the germ, a fairly constant infective power has been secured, and much infection is produced throughout the world at frequent intervals, but the toxicity relatively to the infectivity is still slight. The final phase is of complete victory, infective power is maintained, even enhanced, and to this is added a toxicity surpassed by few epidemiological competitors. Viewed as a contest between man and 'germ,' it would seem that in the congestion of public transport and the multiplication of public assemblies and entertainments, features which increasingly characterise the development of the European type of civilisation, a strategical advantage was given to the enemy. Finally, in the provision of countless incubators, whether in garrisons, war-time factories, or abnormally overcrowded and ill-ventilated means of transport and places of entertainment, the opportunity was afforded for the development of destructive powers which secured to the enemy a decisive and overwhelming victory.
>
> "The first inference from our hypothesis is that in the seeming conflict between man and his microscopic competitors, there can never be a time when man is securely master of the universe. Intoxicated by the victories achieved over the plague (in Europe), over the enteric group, over typhus (in western Europe), and over small-pox, we are too apt to suppose that the campaign has ended in our favour, that we have little more to fear from the typically epidemic diseases and may concentrate against the endemic group. That we have just passed through one of the great sicknesses of history, a plague which within a few months has destroyed more lives than were directly sacrificed in four years of a destructive war, is an experience which should dispel any easy optimism of the kind. No instructed epidemiologist can say that the world may not have to endure during the next half century other plagues of the first order of severity.
>
> "The second inference is the essential solidarity of all mankind in the matter of epidemic sickness. In a narrow sense this solidarity has been realised since the beginnings of western civilisation. The conception of a sanitary cordon, the barring out or sealing up of an infected territory, is, indeed, an old notion. But our hypothesis extends this conception greatly, and enables us to see that the sanitary cordon is but a very small part indeed of a supra-national system of preventive medicine. The dangers to the world from epidemic sickness in this matter of influenza are

enhanced in two ways. The inevitable trend of the movement of population will keep the infectivity of the organism at a high level. This we may face with equanimity. But if anywhere in the world there be large collections of men, whether through war or economic strife, or through that dissolution of civil society which a certain degree of collective misery and disorganisation entails, herded together *en masse*, there will be opportunities for the other modification of the *materies morbi* which renders it apt to conquer the world. No sanitary cordon, no quarantine, will shield us from this danger. The porters of the infection may not be sick; to exclude even the sick has often been found a task beyond the powers of a quarantine authority; land quarantine has, in fact, never yet succeeded. To realise that the material well-being of the inhabitants of a foreign—perhaps even a hostile—country is a pressing concern of ours is very hard. Yet the teaching of this pandemic is that it is a hard truth. Any supra-national organisation for the control of epidemics will need to face it."

Means of Investigation and Prevention.

21. At the beginning of 1919, immediately on your appointment as President of the Local Government Board, you will remember, Sir, that you called for vigorous official action in regard to influenza, and it is desirable that a brief summary of the administration of the Board at that time, and of the Ministry subsequently, should be recorded. In the first place the *means of information* were revised. Primary pneumonia and acute influenza pneumonia were both made notifiable by the issue in January, 1919, of the Public Health (Pneumonia, Malaria, Dysentery, &c.) Regulations, which had been prepared by the Board in 1918. A system was instituted of cables from consular and other agents in various parts of the world, and of weekly returns in the army, navy, air force, and civil communities (including schools, institutions, factories, &c.), to overcome, as far as might be, the absence of compulsory notification of influenza, which was deemed impracticable owing to difficulties of definition. The returns received were systematically charted, studied, and published, and in this way the foundations of an Intelligence Department were prepared. Secondly, we appointed a *Standing Committee* of the medical heads of the various Departments in Whitehall concerned in the matter, including the Army, Navy, and Air Force Medical Services and the Medical Research Committee, which met weekly for "intelligence," consultation and co-ordination. It was also thus possible jointly to provide and supervise numerous local inquiries in different parts of the country, which had been commenced by the Local Government Board in 1918. Thirdly, the Ministry *advised local authorities* throughout the country to organise treatment, nursing, and "home-help" for influenza-stricken houses, and issued a revised circular of advice (with popular leaflets), pressing on the public and on local authorities the precautions which preventive medicine suggested as advisable in regard both to protection and treatment. Numerous

examples of such advisory publications will be found in the present report. Fourthly, the Ministry, in conjunction with the Medical Research Council, made arrangements for the first time for *linking up medical officers of health with bacteriological experts* in different parts of the country for the systematic clinical, pathological, and epidemiological study of the disease. In this way was instituted an organisation for co-ordinated research and investigation ready to deal with the epidemic in its *local* foci of prevalence and in its varied forms. Lastly, the Ministry supplied *an anti-influenzal vaccine.* A large quantity of the vaccine (*circa* 1,850 litres) was prepared for the Ministry, partly at the Lister Institute and partly by arrangement with the War Office, at the Royal Army Medical College, Millbank, in accordance with the following formula: —

400 million Bacilli Influenzæ (Pfeiffer),
200 „ Pneumococci,
60 „ Streptococci,
per cubic centimetre of vaccine.

Several strains of the influenza bacillus were employed, and Types I. and II. of the pneumococcus. The vaccine was intended primarily for use as a prophylactic, and was recommended to be given in two doses separated by an interval of 10 days. For adults the first dose was ½ c.c. and the second dose 1 c.c. The vaccine was distributed through the Government Lymph Establishment to medical officers of health, free of cost, for issue to medical practitioners within their districts. It was also supplied direct to schools and hospitals, and made available to medical officers of passenger ships bound for infected ports. Up to the end of October, 1920, 23,559 bottles —sufficient for the preventive inoculation of 377,000 adults— were distributed in response to 1,248 applications. Each bottle of vaccine was accompanied by a form, on which the medical practitioner was asked to record, in respect of each person inoculated, certain particulars, including dates of previous attacks of influenza, date of inoculation, and subsequent history.

22. It was, of course, recognised that such a vaccine had but limited possibilities; for, first, we do not know the cause of the disease, and thus we are ignorant of the basic element for such a vaccine; secondly, the presence of the disease in the individual is not followed by a stable and complete natural immunity, and we are therefore without substantial grounds of hope, in any artificially prepared vaccine, for the production of an acquired immunity; and thirdly, it must not be forgotten that to introduce a prophylactic vaccine in the midst, and, indeed, at a late stage, of an epidemic is to court failure and imperil the credit of the vaccine. On a balance of considerations, however, it was thought desirable to attempt to meet the public demand by providing a polyvalent vaccine of the nature described above.

As was anticipated, the results have not been sufficiently substantial or uniform to lead us to any definite conclusions, though there are indications that the use of the vaccine did reduce in some cases the risk of fatality. Various forms of vaccine have been tried in different countries, hitherto without much success, for the reasons already given.

The Prospect of the Future.

23. "The problem of influenza is still unsolved," write the compilers of this report; "its solution will be one of the great events in the history of medicine." And they go on to ask the inevitable question which arises in men's minds, What is the world's outlook upon future pestilences or dangers of pestilence? The answer is that it is gloomy. "The conclusion to which we "are led is that the generation of a great pestilence such as "influenza or pneumonic plague is dependent upon disturbance "of social order involving for absolutely large numbers of "human beings the endurance of conditions of insalubrity which "afford for invading parasites a suitable field of modification. "So soon as the new properties have been stabilised no barrier "against the pandemic or epidemic extension will avail, nor "will those individuals or nations who have not suffered the "primary evils be more resistant to the disease than their fellows. "No impartial spectator can doubt that at the present time, and "almost certainly for a generation to come, there will exist in "many nations and over wide tracts of country precisely the "type of misery which we suspect to be the appropriate forcing "house of a virulent and dispersive germ."

24. The prospect is not cheerful, but we must face it with equanimity and all the resourcefulness of the spirit of adventure and quest. One thing is certain, that the fundamental requirement to make us masters of our fate is a universal improvement in the standard of health and the conditions of life. No technical device, no narrow or specific remedy for pestilence, can ultimately triumph apart from a sanitary environment for the community and the sound nutrition of the individual. They are the bed-rock. Out of them spring the sources of national vitality. Hardly less certain is it that we require, and must seek till we find, more knowledge. We have in substantial degree the means of controlling tuberculosis and syphilis, malaria and plague; we fail to control these four pestilences largely because we do not use the means; and education is perhaps the answer to that. But in the case of influenza and its allies we are not yet in possession of the means, and whilst we press forward with the improvement of sanitation, of nutrition, and of the conditions of life, we must apply ourselves anew to search and research into the causes of primary and secondary infections, into epidemic catarrh and the common cold, into carriers, and

into immunity. That is perhaps the principal lesson which is taught us by our experience of the great pandemic.

25. Two other practical steps remain. First, we must fortify our administrative methods for dealing with such scourges as influenza, and secondly, we must instruct the whole population, child and adult, in the laws of preventive medicine. The administrative arrangements introduced in 1919 by the Ministry require to be consolidated and perfected, particularly in relation to (*a*) the development of a system of "intelligence," (*b*) the effective co-ordination of central and local agencies for the synthetic study and prevention of the disease, and (*c*) the provision of further means of adequate treatment and nursing. Lastly, there is public instruction in the practice of preventive methods. The infection of influenza and its allies appears to be conveyed by the secretions of the respiratory surfaces. In coughing, sneezing, and even in loud talking these are transmitted through the air for considerable distances in the form of fine spray. The channels of reception are also normally the respiratory surfaces of mouth, nose, and throat—though dust, unclean hands, and infected materials may be the means of conveying infection. It is manifest, therefore, that the closer the bodily contact the more readily will transmissions occur; hence the paramount importance of persons abstaining thus directly to infect each other, and also of avoiding overcrowding and thronging of every sort, whether in places of public resort, public conveynces, or factories. Well-ventilated rooms, nourishing food, and an open-air life afford some defence; and in times of prevalence of respiratory catarrh or sore throat the frequent use of an appropriate gargle (for example, 20 drops of liquor sodæ chlorinatæ in a tumbler of warm water) and nasal wash may be recommended. In regard to individual prophylaxis, both the clinical experience of Dr. French and the epidemiological inferences drawn by the staff of the Ministry concur in supporting the belief that a simple hygiene of the mouth and nose is of more value than any specific medication. At the first feeling of illness, or rise of temperature, it is the private and public duty of the patient to go to bed at once, to remain at rest and in warmth, and to place himself under medical supervision, for it cannot be too clearly understood that it is the complications of influenza which disable or destroy life. Thus it seems probable that, though our knowledge of influenza is strictly limited, we are not wholly without means of reducing its incidence and mitigating its consequences.

26. So far as concerns the provision of some simple and infallible prophylactic against this terrible disease—the use of the adjective is, unhappily, fully justified—the outcome of this report must fairly be described as meagre. But in one particular, and that not the least important, this intensive study of a great and historic epidemic is well worthy of public attention. In the general annual digest which I had the honour

of submitting to you a few months since, a document founded upon a review of the whole volume of mortality and morbidity in this country, I emphasised the unitary character of the problems of health and disease—the fact that there are no short cuts to a sanitary millennium. The story of influenza as unfolded in the present report teaches the same lesson. A drama which is acted in a few weeks seems to be the product of years of preparation. There can be no question as to the deduction to be drawn. The only sure defence against sudden and devastating epidemics is to raise the whole standard of life of this nation, indeed of the whole comity of nations. That conclusion from the present evidence will, I believe, still stand long after the specific hypotheses of etiology and epidemiology here suggested have become merged in the wider synthesis which future research must render possible.

I have the honour to be,
Sir,
Your obedient Servant,

GEORGE NEWMAN.

Ministry of Health,
Whitehall,
October, 1920.

REPORT ON THE PANDEMIC OF INFLUENZA, 1918–19.

PART I.

Influenza in Great Britain and Ireland.

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CHAPTER I.

The History of Influenza, 1658–1911.

That to understand the ætiology of a disease we must study both its historical and contemporary manifestations is as much a truism to the epidemiologist as the parallel proposition in the science of social and economic institutions. Acquaintance with the historical evolution of, for instance, typhus fever, prepared the discerning for events of recent years; hardly less valuable have been the lessons drawn from the history of continued fevers or of bubonic plague. It is natural, therefore, that great expectations should be founded on the results of the historical method applied to epidemic influenza. The most cursory perusal of the annals of disease makes it plain that epidemic and even pandemic influenzas have frequently recurred in the past; the salient features are so impressive, the onslaughts so dramatic, that we hope to derive a wealth of information from the literature of earlier times. These expectations are not altogether unfounded; the search for historical information is repaying, but the reward has not been so great as the enthusiast perhaps proposed to himself.

All diseases present, as Sydenham wrote long ago, a very evident diversity in their clinical manifestations; but the range of variation is not equally wide for all. Sporadic cases of plague may deviate from the normal description of a text-book widely, but in an epidemic of this disease the proportion of victims conforming to the text-book type is large; hence it is that the history of plague can be followed into the earliest ages, and its prevalence recognised there with certainty. There is no more doubt that the disease described by the authors of the book of Samuel (i. 5) was bubonic plague than that the same disease ravaged London in 1665, and destroyed several thousand lives in India in 1919. No such certainty of identification attaches to typhoid fever; expert clinicians and epidemiologists may disagree as to whether epidemics portrayed by so great an observer as Sydenham were or were not typhoid fever. A similar doubt envelops the nature of 18th century epidemics, which may have been of malignant scarlet fever or of diphtheria. Such difficulties of clinical identification arise in the study of influenza, and no *purely clinical* criterion of it has been established; the nearest approximation to such is the definition that outbreaks of epidemic disease in which a majority of the affected exhibit initially or early in the course catarrhal respiratory symptoms are outbreaks of influenza.

The impossibility of establishing a clinical recognition mark as satisfactory in influenza as the bubo in plague has induced a majority of thinkers to base their identifications upon demographic or epidemiological grounds, even to establish a unity

in diversity, to make the very diversity of its clinical forms a mark of influenza, provided this clinical diversity be concomitant with the epidemiological characteristics of (*a*) extremely rapid evolution in time and place, (*b*) a fatality slight in comparison with the case incidence. For these and other reasons the remarkable 16th century epidemics known as the English sweats have been regarded by some as instances of epidemic influenza.

The formal objections to this view of the subject are, no doubt, grave. If we *define* epidemic influenza as a disease explosive in its manifestations, spreading rapidly, and relatively non-fatal, there can be no logical objection to regarding the English sweats as influenzas, as well as other earlier and later outbreaks of epidemic sickness, but we cannot by this method establish criteria ; it is circular reasoning to say that such and such an outbreak of " undoubted " influenza *proves* that rapidity of dissemination and low fatality characterise the disease ; this is only inferential if the definition of influenza be based upon other considerations. Some who have been impressed by the force of this objection would seek to define influenza, in terms of pathology, as a disease produced by infection with a specific germ, the bacillus of Pfeiffer or another. This definition is theoretically as inadequate as, and practically less useful than, the foregoing.

It is logically inadequate, because an epidemiological unity may not be a bacteriological unity ; diseases produced by the infections of different distinct organisms of the coli-typhoid group may present both clinical and epidemiological unity ; on the other hand, the epidemiological problems raised by infections with the same organism, the bacillus of Eberth, through different channels may be disparate. The definition is practically useless so far as the employment of historical records is concerned, since direct evidence of the nature of bacterial factors is necessarily lacking before the 19th century.

When thus contrasted, the defects of both methods are glaring, but it may appear that an amplification of the former process deprives it of patent weaknesses. If into the definition of influenza, while maintaining the characteristics of *apparent* suddenness, undoubted rapidity of evolution, and usually low fatality, we import the conception of peculiar attendant circumstances, such, for instance, as a break in the uniformity of epidemiological history prior to the outburst, and also keep in mind the catarrhal element of the explosive disease as a frequent clinical experience, we shall reach a plan of identification which is not logically assailable. To make this proposal intelligible we shall first examine some events in the 17th century which came under the notice of Sydenham.

In the autumns of 1675 and 1679 epidemic coughs, followed by pleurisies and pneumonias, were prevalent in London. In the

former year the deaths "within the bills" were 275 in the week ending 2nd November, 420 in the following week, then successively 625, 413, 349, 308, 266. These are deaths from all causes; in the worst week the deaths attributed to consumption rose from 68 to 99, of "aged" from 40 to 67, of "Tissick" from 10 to 35. In 1679 the return for the week ending 11th November was 328; 541 in the next week, then 764, 457, 388. The trend of these figures is one which modern vital statistics of what we agree to term influenzal periods exhibit; there is no doubt that what happened in 1675 and 1679 happened also in 1889–90. But these are not the only epidemics occurring about the same time which Sydenham considered noteworthy. He has left a special account of a new fever which began to rage in London in February 1685, "atque Febri quae per "octennium ultimo praegressum invaluit, esse prorsus dis- "similem." In his detailed account Sydenham puts great stress upon the symptoms of involvement of the nervous system, observing that "haec Febris species prae ceteris quas mihi "unquam videre contigit, cerebrum petere quasi apta nata "sit," and although he asserts an equal liability to it in infants and adults, he doubts whether the clinically similar diseases in young and old be really identical ("serio advertendum est, an, "quae hoc modo tractatur Febris, pro certo hujusce Constitu- "tionis fuerit, non vero alterius speciei: quod diligenter "perquiri debet in Febribus iis quae infantes corripiunt, in "omnibus quibuscunque annorum Constitutionibus"). Finally an examination of the bills of mortality shows that although the gross total of deaths was almost the same as in the previous year, their allocation was different, fever (with "spotted fever") and smallpox being both increased.

The epidemic of 1685, therefore, was characterised by (*a*) a great severity of involvement of the brain and nervous system, (*b*) a possible predilection for the adult, (*c*) no serious effect upon the gross mortality of the year. Each of these characters has been seen in the epidemics of cerebrospinal fever of England and Wales since 1914; we shall meet some at least of them in the 18th century. With respect to the forms of illness environing these outbreaks, Sydenham speaks at length of the comatose type of the continued fevers of 1673–75 (inter hujusce Febris symptomata eminebat affectus quidam comati haud dissimilis), while he characterises the fevers in and around 1678 as favourable to intermittents, "quae jam ab anno 1664, ad tredecim scilicet annos, Urbe fere exulaverant." It is significant that in 1657–59 epidemics occurred in London which have an indisputable title to be classed with those of 1675 and 1679, giving point to Creighton's remark that "Sydenham does not appear "upon the scene until 1661; but when his epidemic constitu- "tions do begin, it is with intermittents or agues, which lasted, "according to him, until 1664. Perhaps, if Sydenham's experi- "ence had extended back to 1657, he would have made his

" aguish constitution to begin with that year, and to go on " continuously until 1664." *

It is to be added that within a few years of Sydenham's retirement another epidemic catarrh prevailed in London—in the summer of 1688. " Never," says a contemporary writer, " were so many people sick together; never did so few of them die," while the bills of mortality exhibit the same rapid fluctuations as in 1675 and 1679. For the five weeks beginning with the last of May, the deaths run, 368, 518, 539, 435, 437.

The interpretation put upon such a sequence as this by Sydenham is expressed in his doctrine of epidemic constitutions, for a discussion of which reference may be made to the works cited in the footnote†; in the view of those epidemiologists who define influenza in the fashion explained above we are to look upon epidemic or pandemic outbursts of influenza, not as isolated events, not even as terms of a simply periodic series, but as focal points in a system of related phenomena; the nexus, which we may term, if we will, an influenzal constitution, includes many *apparently* disconnected outbreaks of disease, very different in clinical form from explosive influenza, but usually comprising signs and symptoms of nervous origin, this or that segment of the nervous system being specially signalled out. Such forerunners and *sequelæ* also have this in common, that they puzzle both the public and the medical practitioner, and are given new names.

It will be apparent that the need of justifying or condemning this definition, while increasing the abstract importance of historical study, reduces the practical value of many historical data. The criticisms to which it is subject are evident. The capital danger is the basing of an induction upon too scanty collections of particular instances. That in chronological association with the influenza of 1918–19 we have seen cerebrospinal fever, and " encephalitis lethargica," that after the influenza of 1679 Sydenham saw something which we shall hardly distinguish from epidemic cerebrospinal fever, and before it noted nervous forms of " fever " which reminded him of what occurred 15 years before, also in association with an influenza period, are particular instances insufficient for an induction. We are not indeed called upon to prove that

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* *History of Epidemics in Britain*, Vol. 2, p. 314.

† Sydenham's own account is to be found in the six books of Medical Observations, the first Epistle and the first Schedula monitoria (specially concerned with the new fever of 1685). A general account of the theory is contained in Greenwood's paper in *Proc. Roy. Soc. Med.* (Section of Epidem.), July 1919. Its detailed application to the case of Influenza is given in Dr. Hamer's report on Influenza to the London County Council, June 1919, while this author's *Milroy Lectures* (1906) and paper in the *Proc. Roy. Soc. Med.* (Section of Epidem.), November 1916, sustain a similar or identical thesis.

obscure nervous outbreaks were *always* followed by explosive influenza, but we should be able to show that such influenza does always cast a shadow before or behind. Power to apply the crucial test implies the existence of an objective and systematic record of what actually happened, not in one but in many series, covering more than one cycle of years within which influenza has been epidemic. But the intellectual qualities needed to guarantee the accuracy and perspicuity of such a record are rare endowments; Sydenhams are not found in every generation. That some chronology of disease is possible from the 17th century to our own time is true, and that it has sufficed to authorise many conclusions drawn by such profound epidemiologists as Creighton, Hirsch, and Haeser is also true; but, for our particular inquiry, miscellaneous annals, the products of many minds and therefore subject to the unconscious bias of many "personal equations" and fashions of nomenclature, are of doubtful value. There does, however, exist one other chronological record, extending over a considerable space of time, which is the work of a single highly competent recorder, and we think it desirable to compare the impression conveyed by it with that derived from Sydenham's observations; we refer to John Huxham's annals of disease in Plymouth and the neighbourhood, which cover the years from 1728 to 1752 inclusive. Within these termini four outbreaks of epidemic influenza ravaged this country. In the autumn of 1729 the London deaths in the worst week were 160 per cent. of the average, in the January outbreak of 1733 they were nearly thrice the normal; in the late summer of 1737 the excess was nearly 50 per cent., and in the spring epidemic of 1743 the deaths in the week of maximum mortality were 2½ times the average. These epidemics in London exhibited the other features (rapid evolution, low fatality, prevalence of catarrhal symptoms) which we still associate with epidemic influenza, and each has been noticed by Huxham at Plymouth. It will be worth while to inquire whether the influenza years in Huxham's experience can be objectively characterised as differing in some material way from the run of other years *apart from* the explosion of epidemic influenza.

The reader may be reminded of the way in which Huxham compiled his annals.* Each month of the year for 25 years is separately dealt with; the meteorology of the month is tabulated and then a longer or shorter account of the reigning diseases is given. Interspersed through the diary are essays usually on the symptomatology and treatment of different diseases. The language is Latin, but it is not the Latin of a classical imitator (as is that of Sydenham's works), and consequently more intelligible to the modern reader.

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* *Observationes de aere et morbis epidemicis.* J. Huxham. First two volumes, London, 1752. Third volume, London, 1770.

Let us now inquire whether the epidemiological events grouped around the explosions of influenza exhibit any objective contrast to those of other sequences of years. We will begin with the epoch centered in April–May 1743, under the former of which months Huxham describes the epidemic influenza in words frequently quoted and well known to all. Passing backwards from the end of April, when the explosion occurred, we find that in April intermittent fevers were common, especially tertians; in March "Variolae frequentes, Febres intermittentes plures et quotidianae et tertianae." In February there were some tertians and a catarrhal fever; nothing of importance appears until we get back to September 1742, when measles were epidemic; in August intermittents are recorded and "perpauci aliter morbi." In April, one year before the explosion,gaol typhus was epidemic at Launceston. Passing forwards in time from April 1743, for each succeeding month until September, stress is put upon the intermittents. In May, "Febres quotidianae et tertianae creberrimae longe lateque." In June, "intermittentes vero, tertianae potissimum epidemicae." In July hardly anything but intermittents; in August, "multae adhuc Febres intermittentes ruri." In the following year there were some intermittents in March; they were epidemic in May, scattered in June, and are mentioned in August and November. In December we find reference to a malignant catarrhal fever affecting French and Spanish prisoners of war, very fatal, attended often by *petechiæ* and sometimes by parotid abscess, which, with its extension to the civilian population, is the chief matter dealt with until June, when a change to frank typhus is recorded and a few intermittents are noted. Waiving the discussion of this malignant catarrhal fever, which can hardly have been other than typhus, the most striking thing in the chronology is the record of epidemic intermittents in temporal proximity to the influenza; they were very common both before and after the explosive outburst. This might perhaps be thought either a normal course of events or an idiosyncracy of the writer in his choice of material to record. But, as a matter of fact, from July 1745 to December 1752, when the record ends, during which period *no* explosion of influenza occurred in England, intermittent fevers are only *twice* again recorded, viz., under April 1748 ("nec non intermittentes sed Rure potissimum") and under April 1751, when we read of "Febres intermittentes aliquae."

We shall now examine the sequence of years from 1728 to 1742 with particular reference to intermittents.

From March 1728 (when the intermittents were "frequentiores longe quam assolent in hac regione") to October, intermittents hold the field as reigning epidemics; in the last months of the year little sickness was recorded. The first five months of 1729 hold nothing of interest. In June erysipelas and smallpox were epidemic, "Febres intermittentes remit-

" tentes praecipue, lentae, nervosae frequentes. Cephalalgiae " et Mania passim." From July to November, first a variolous fever and then frank smallpox claim attention, until we have epidemic influenza in November, soon swallowed by smallpox, which is the reigning malady until we reach April 1730, when intermittent and remittent fevers, often with light delirium, are more frequent; these again ravage in May, and "ex " levi errore in continuas malignas, nervosas, transibant haud " infrequenter." The last six months of the year are uneventful. In 1731, coughs and pneumonias prevailed in the spring, there were intermittents in July, more or less smallpox from June to September.

In 1732 there was much whooping-cough, smallpox (in April), a few (*passim*) intermittents in August, then epidemic measles until December. 1733 opens with measles still epidemic, then in February we have the explosion of influenza, extending into March, still present in the villages in April, when pneumonia and pleurisy were fatal, while in May "Febres pulmonariæ ac intermittentes, invadunt plures," and still occur in June and July. August was healthy, and there is little to remark in the remainder of the year.

In 1734 there was much spring pneumonia and a special epidemic of anginous fever in April, which extended into June; in May and June there were intermittents. In July also there were intermittents; also " furit etiamnum Febris epidemica " inexplicabili quodam angore quosdam, vel intra sex saepe dies " perimens. Hos leve primo corripuit Delirium, quod utique " pejoris fuit ominis, quam si, more Bacchantium, aperto " exarserant Furore." This strange disease, often associated with diarrhœa and vomiting, was fatal in the next month; in the last three months of the year smallpox was epidemic. At the beginning of 1735 smallpox was still reigning, followed in February by what was probably ship typhus. From March to June typhus and smallpox prevailed. In June some intermittents (*haud infrequentes*). Until November typhus and smallpox continued to be noteworthy, and slow nervous and putrid nervous fevers are entered for the last two months. In a general summary Huxham remarks on the prevalence of intermittents and remittents in the wet years of 1734 and 1735. In 1736 there is little noteworthy before June, when there were some (*passim*) intermittents, also in July, when mention is made of miliary fevers, which continue in the diary until November. The notes for November and December suggest a local explosion of influenza, for we read of "Tusses, Destillationes, Coryzae, " Raucedines, levi non raro comitante Febricula, per omnes " undique. Plures vel in iisdem Aedibus simul ac semel " corripiuntur; evanuere vero haec omnia facilime, modo se in " lectulo paululum continerent et multa tepida potione sudores " elicerent." This can hardly have been anything but an explosive influenza, for in January 1737 we read that the

" Febricula catarrhalis innumeros etiamnum infestat prout Mense Decembri," while there was a coincident horse sickness and not a little pleurisy and pneumonia amongst men. From May to August there is frequent mention of miliary fevers; in September there is reference to anomalous intermittents, and in November we reach the explosion of influenza, which is historical, like that of 1743. At the beginning of 1738 we have the dregs of the influenza; pneumonia and pleurisies occupy attention until July; a few intermittents occur in May. From August to November, smallpox and measles are important, a catarrhal fever of children is recorded in September; in the last two months of the year we again read much of pneumonia and pleurisy. In the first four months of 1739 measles are very prominent; there were some spring and autumn intermittents, but the situation is dominated by smallpox, first mild, then malignant, down to December.

In 1740 we read, under February, of " Febris quaedam " catarrhalis cum acute capitis dolore et vertigine accedente " aliquando phrenitide, multos detinet." In May " Febres pulmonariae " with dark *maculæ* prevailed, and smallpox increased; this continued into June, when ship typhus, introduced earlier from the Fleet, claims attention; there was smallpox in July and August, dysentery in September, and smallpox raged more or less in the remaining months. No reference is made to intermittents this year. 1741 was a year of smallpox, which was the reigning disease until September, and is mentioned every month; there were some intermittents in May and some anomalous intermittents in September. 1742 is under the sway of smallpox and measles until April, when we reach the gaol typhus, to which point in time we carried our inquiry backwards, starting from the 1743 influenza.

From this necessarily condensed summary of a long story, certain conclusions may be drawn. We think Huxham's narrative establishes the proposition that the epidemiological features of the cycle of years within which influenza exploded once or more were, *apart from such explosion*, different from the characteristics of the influenza-free epochs. Huxham's crowded references to intermittents around the explosive manifestation of influenza in 1743, his silence between 1745 and 1752, the paucity of his remarks on the subject during the quiet years 1739–42 are paralleled in the story told by Sydenham. We are also justified in making a comparison between the strange nervous fevers incidental to Huxham's narrative and Sydenham's new fever of 1685. With these long-sustained chronologies by skilful observers to guide us, it is not fantastic to suppose that the inter-relation of " agues " and influenzas to which Dr. Creighton called attention in the section of his history dealing with the 16th and 17th centuries, is more than casual and not a mere reflection of some theoretical pathology.

As an annalist Huxham had no successor of equal professional ability continuously recording observations through so long a term of years; but one of the witnesses of the 1762 pandemic has left some notes which recall Huxham's findings. This was Dr. John Rutty, who described the 1762 explosion in Dublin.* The influenza came in May 1762, and was, thought Rutty, "plainly a disease of foreign extraction, having visited "Copenhagen a month before it reached London, and London "about a month before it arrived here; and it gradually "extended itself over every province of the kingdom." Rutty did not therefore himself connect the explosion with anything which went before in his experience. This is his record of the previous months:—

> "Some mild intermittents appeared in April; but in May intermitting fevers were very frequent, particularly the tertian, and pleurisies and chin-coughs still continued to be epidemical. In the beginning of May a fever was common which resembled the scarlet fever in every circumstance except the eruption. In the beginning, after the shivering, the face was much flushed, with a severe headache, dry, burning skin, quick pulse, and soreness in the throat; sometimes attended with superficial ulcerations in the tonsils, and often a crick in the neck. In some it disappeared in three or four days; in many, after running out to the fourteenth day or longer, the skin peeled off in branny scales, as in the scarlet fever, though no eruption had appeared."

The explosion of influenza began in the middle of May.

Creighton has cited three witnesses—Baker and Reynolds in London and Barker of Coleshill—to the prevalence of "agues" between 1780 and 1785 (the influenza explosion was during the summer of 1782), while Willan remarked on the absence of intermittents in London during the last 10 years of the 18th century, a period free from pandemic influenza.

No chronological records apt to bring out these points have been published by the observers of the 1803 or of the 1831–37 explosions, but Dr. Peacock, in his treatise on the 1847 influenza,† took occasion to remark that, "throughout the "spring and early summer, intermittent fevers were common, "and, in March, April, and May, purpura was frequently met "with, either as a primary or secondary disease." The remark quoted from Peacock is the latest of its kind. The chronological method was already obsolete in 1847; the introduction

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* "*A Chronological History of the Weather and Seasons and of the Prevailing Diseases of Dublin*," by John Rutty, M.D.: London, 1770. (The portion dealing with the influenza of 1762 is reprinted in Dr. Theophilus Thompson's "*Annals of Influenza*": London, 1852.)

† "*On the Influenza or Epidemic Catarrhal Fever of* 1847–48." T. B. Peacock, M.D.: London, 1848.

and subsequent development of vital statistics dissuaded physicians of the calibre of Sydenham and Huxham from giving to the compilation of chronicles an attention better rewarded in other scientific pursuits. All reform involves some loss; while conferring upon us great benefits, the vital statistician has deprived us of testimonies which would have been valuable in the present connection. Henceforward we must rely almost wholly upon statistical records and nearly always upon bills of mortality. If the clinical sequences noted by our ancestors still occurred, we should hardly expect to find their traces in the records of mortality. The nosology of the "agues," "intermittents," and "remittents," of the obscure nervous fevers, and of other concomitants of the explosive influenzas may be as obscure as ever, but their marshalling under less ambiguous statistical banners is easy; intermittent and remittent fevers or agues have no longer a significant share in the recorded causes of death; whether, as Welsh* put it, the revolution has been in the cycle of disease or in the mind of the practitioner, whether what Sydenham and Huxham claimed to have seen might have been seen by them in our time, are mere matters of conjecture. We shall find that vital statistics reveal an unusual state of the public health before the explosion of influenza in 1847, and do so again before the great catastrophe of 1918; but we shall gather little intelligence from the years ending in 1890. The real significance of the observations just described is therefore likely to remain a matter of dispute, and to be differently assessed in accordance with the habit of the inquirer. The most important practical inference is that an objective chronicling of disease, the keeping of a diary upon the model of Huxham, is not the task of supererogation some have deemed it to be; such records would be valuable additions to the material of a health intelligence service.

With these necessarily incomplete observations we must leave the pre-statistical history of influenza and pass to the events of the mid-nineteenth century.

The Influenza of 1847–48.

Between the explosions of 1837 and of 1847, influenza prevailed extensively in Central Europe, during 1841, and there are signs of it in this country (*vide infra*) in the same year; it is claimed by Hirsch to have been generally diffused over North America in 1843, and the same writer assigns epidemics to England in 1843 and 1846; but there was certainly no pandemic before 1847, the earliest date of onset assigned to that year being September for France.

Our General Register Office was established in 1837, and the first annual report deals with the year 1838, but the early

* Cited by Murchison, *Treatise on the Continued Fevers of Great Britain*, 3rd Edition, p. 44.

years were transitional and the methods of tabulation in the experimental stage out of which Dr. Farr developed a system destined to become the great exemplar of vital statistical methods.

Some hundreds of deaths attributed to influenza were registered in England and Wales in each of the years from 1838 to 1842, and in 1841 the total reached 1,659; figures are not available from 1843–46. In London the figures are recorded for all but two of these years; in 1841, *i.e.*, the year with 1,659 in England and Wales, there were as many as 220 in London, in 1843 there were 104, and in 1846 115. In no other year were there more than 100. Thus there is a suggestion of increased prevalence in 1841, and perhaps also in 1846, the year before the explosion.

In the year of the epidemic and before the explosion, the public health of the country was unsatisfactory. Dr. Farr records that the first quarter of the year was unhealthy, and that in the second quarter "common cholera was fatal; scurvy prevailed "more or less over the country, from the want of vegetable "food, the potato having failed; all food became scarce and "dear; typhus broke out, is still epidemic, and shows no signs "of decline." Purpura was unusually common in London (a fact noted by Peacock, *vid. sup.*). In the third quarter there was a slight improvement in the country generally, but none in London, where typhus never killed less than 50 in the week and claimed as many as 111 victims in the third week of September, while the weekly average for this quarter in the previous five years had been only 30. The great influenza which began in November increased the mortality in the least unhealthy districts of London at the rate of 18 per 1,000 per annum, but in the unhealthiest by 34 per 1,000 per annum.

There is then no doubt that before the epidemic the public health was unsatisfactory—a circumstance attributed by most epidemiologists to the reflux of the great Irish famine and typhus of 1846. After the influenzal wave had passed, the country was destined to be submerged by a flood of cholera in 1848–49, so that, from the sanitary point of view, influenza was intercalated between two epochs of profound unheathiness. Under the rubric of influenza the deaths of 2,215 men and 2,666 women were recorded in 1847; there were 3,810 deaths of men and 4,153 deaths of women in 1848, 738 and 873 in 1849. Judged by this imperfect criterion, the metropolis suffered much more than the crowded industrial districts of the North-Western area; these districts, with nearly equal populations, having 1,913 and 1,278 deaths respectively. To study the epidemic in detail, however, it is best to confine attention to London, a course which will be taken in our subsequent account, because for the metropolis the weekly records of deaths at ages have been regularly tabulated and published. The relevant documents for 1847–48 have been collated by Dr. F. A.

Dixey*, and Table 1 is a condensation of his results. To this is added Table 2 which shows the deaths at ages from certain

TABLE 1.

London Influenza, 1847–48. *From 46th week of* 1847 *to 12th week of* 1848.

Week.	Influenza.			Pneumonia			Bronchitis.			Phthisis.		
	0-15.	15-60.	60-.	0-15.	15-60.	60-.	0-15.	15-60.	60-.	0-15.	15-60.	60-.
1847.												
Nov.												
46th week	3	1	—	66	7	6	29	8	21	12	101	8
47th ,,	3	—	1	76	15	4	31	11	19	13	87	8
48th ,,	3	10	23	137	20	13	80	55	61	22	119	12
49th ,,	38	48	112	219	56	31	96	96	151	21	158	19
50th ,,	80	113	181	221	44	29	85	83	131	14	168	10
Dec.												
51st week	75	67	128	114	34	11	72	54	108	19	122	7
52nd ,,	37	41	64	107	13	11	37	25	45	16	87	7
53rd ,,	47	35	45	124	15	9	63	38	37	20	132	4
1848.												
Jan.												
1st week	42	34	26	109	11	5	47	28	31	13	129	8
2nd ,,	39	32	31	133	16	9	51	28	46	15	129	10
3rd ,,	24	30	35	105	21	11	42	42	54	23	122	7
4th ,,	12	19	25	128	25	3	52	44	47	20	144	5
5th ,,	9	19	31	110	16	10	42	54	61	29	116	10
Feb.												
6th week	12	15	20	94	25	10	42	32	47	25	115	7
7th ,,	8	7	12	81	12	3	31	25	24	16	98	5
8th ,,	13	11	9	70	15	4	37	28	21	18	110	7
9th ,,	5	4	9	67	9	5	29	16	23	9	100	6
Mar.												
10th week	3	3	5	60	12	—	42	21	32	23	108	10
11th ,,	3	4	3	57	5	3	33	15	33	17	125	5
12th ,,	6	3	7	64	13	2	34	18	24	25	104	10

diseases in London for 1847, 1848, 1849; Table 3 contains the death rates at ages for influenza (England and Wales) in 1847–48, and a similar table calculated from the All-England data in the non-epidemic period of 1848–72 (the material is given in Dr. Farr's decennial review of 1861–70); taking into account other causes of death, the excess mortality attributable to the epidemic in London was nearly 7,000, and the most considerable contributor to the excess, apart from influenza, was bronchitis; in the maximum week of the epidemic the incidence of bronchitis was nearly 800 per cent. above the normal.

* *Epidemic Influenza* by F. A. Dixey, Oxford, 1892.

Table 2.

Deaths at Ages in London from Influenza, Pneumonia, Bronchitis, and Phthisis in 1847, 1848, 1849.

Age Groups.	Proportion of Population at Ages.	Four named Causes.		Influenza.		Pneumonia.		Bronchitis.		Phthisis.	
		Nos.	Per Cent.	Nos.	Per Cent.	Nos.	Per Cent.	Nos.	Per Cent.	Nos.	Per Cent.
	1847.										
0–15 -	31·5	5,634	33·3	304	24·3	3,168	73·8	1,298	29·9	864	12·3
15–60 -	62·4	7,969	47·2	334	26·7	716	16·7	1,177	27·1	5,742	81·9
60 and over	6·1	3,293	19·5	615	49·0	406	9·5	1,868	43·0	404	5·8
Totals -	100·0	16,896	100·0	1,253	100·0	4,290	100·0	4,343	100·0	7,010	100·0
	1848.										
0–15 -	31·6	4,945	35·9	202	30·6	2,793	79·6	1,138	37·5	812	12·4
15–60 -	62·3	6,867	49·9	207	31·4	499	14·2	769	25·3	5,392	82·1
60 and over	6·1	1,955	14·2	251	38·0	218	6·2	1,127	37·2	359	5·5
Totals -	100·0	13,767	100·0	660	100·0	3,510	100·0	3,034	100·0	6,563	100·0
	1849.										
0–15 -	31·7	4,999	37·6	40	31·5	2,918	81·1	1,239	38·1	802	12·7
15–60 -	62·2	6,378	47·9	31	24·4	444	12·4	753	23·1	5,150	81·4
60 and over	6·1	1,927	14·5	56	44·1	235	6·5	1,261	38·8	375	5·9
Totals -	100·0	13,304	100·0	127	100·0	3,597	100·0	3,253	100·0	6,327	100·0

Table 3.

Death Rates per Million from Influenza (Persons), England and Wales.

Ages.	1847–48.	1848–72.	1890–91.	1881–90.	1891–1900.	1901–10.
0—	713	149	306	26	288	161
5—	80	13	55	4	49	31
10—	49	7	46	3	41	24
15—	51	9	115	7	97	53
25—	79	12	197	12	151	86
35—	139	20	347	19	281	147
45—	284	41	595	27	492	264
55—	809	125	1,060	45	1,013	574
65—	2,372	439	1,985	83	2,214	1,325
75—	5,510 } 5,883	1,103 } 1,192	3,355 } 3,522	191	4,703	2,927
85—	11,243 }	1,816 }	4,821 }			

TABLE 3—*continued.*

Expressed as Percentages of the Rate at Ages 0—5.

Ages.	1847–48.	1848–72.	1890–91.	1881–90.	1891–1900.	1901–10.
0—	100	100	100	100	100	100
5—	11	9	18	15	17	19
10—	7	5	15	12	14	15
15—	7	6	38	27	34	33
25—	11	8	64	46	52	53
35—	19	13	113	73	98	91
45—	40	28	194	104	171	164
55—	113	84	346	173	352	357
65—	333	295	649	319	769	823
75—	773 } 825	740 } 800	1,096 } 1,151	735	1,633	1,818
85—	1,577 }	1,219 }	1,575 }			

It was mentioned above that 1,611 persons were recorded to have died of influenza in 1849, and the total is above 1,000 in every year until 1861,* reaching 3,568 in 1855. Thereafter it declined and in the decennium ending with 1889 was only thrice more than 100, viz., in 1880, 171; 1883, 107; 1885, 138. That the disease entered in the death registers as influenza between the years 1847 and 1890 was necessarily identical with the malady of pandemic times is, as pointed out by the late Dr. Parsons in his official report, doubtful. Amongst the public, "influenza" has almost as vague a connotation as a "touch of liver" and, for the reasons explained above, the doctor had not (and still has not) any instrument of precision which enabled him in this matter to rise superior to the temptation of a conveniently loose phraseology. But it is going too far to assert that the large fluctuations of the deaths attributed to "influenza" in some of the years are capricious or to believe that no persons died in the ten years preceding 1890 of "undoubted" influenza. One small but instructive incident suggesting the need of caution was the localised outbreak of a disease in the district of Northallerton during February 1887; it was reported on by Dr. Page of the Local Government Board; a very brief abstract of his findings is printed in the report of the Board's Medical Officer for 1887–8 and some further particulars are quoted by Dr. Parsons in his study of influenza. These particulars are sufficient to prove that had the disease affected a wider area no one would have hesitated to classify it as influenza in the sense of either 1890 or 1918. We do not therefore think it proven or probable that the pathogenic

* All the figures are tabulated in Parsons' Report to the L.G.B. on the Influenza of 1889–90 (Cd. 6387). This report and its successor by the same author (C. 7051 of 1893) are hereinafter cited as Parsons I., and Parsons II. For a more minute analysis of the statistics of the 1890–2 influenzas than given in the body of the present report, reference should be made to the work of Dr. Parsons amplified in certain directions by the above quoted monograph of Dr. Dixey.

materials of a conflagration were absent from this country between 1880 and 1889. Yet it has to be plainly stated and fully grasped by the reader that the recorded history of an epoch which, as we now know, came to a definite end in December 1889 in no way prepared us for the subsequent events.

We have seen that the influenza period of 1675–88, of 1729, of 1734, and 1743 are set in circumstances which are harmonious one with another; we have this on the faith of continuous records kept by skilful observers. One witness, Rutty, speaks to a like sequence in 1762; three or four give concordant accounts of the 1782 period; we can properly attribute our confusion respecting the 1803 and the 1833–37 influenzas to the decay of one mode of recording and the infancy of another. For 1847, the evidence, not indeed of "nervous" and "intermittent" satellites but of profound disturbance in the public health apart from the explosion of influenza is unchallengeable. Almost as strong evidence is available respecting the antecedents of 1918. But little in written history can be brought forward as foreboding an impending change in the years immediately before 1890. So far as "influenza" deaths are concerned 1889 had the lightest toll of the decennium; 55 in England and Wales, 5 only in London, three of these of young children. The general death rate of the year was very low; measles and diarrhoea were more prevalent than in 1888, but below the rate of any other year in the decennium. Such vague headings as cerebro spinal fever, remittent fevers, agues, covering morbid conditions which might be harbingers of a new "constitution," provide statistics which show mere random fluctuations. Nor is there much non-statistical evidence; no epidemic studied by the medical inspectors of the Local Government Board between 1881–90 (other than the Northallerton incident) and published in official reports throws direct light* upon the antecedents of the influenza of 1890.

In 1889 this country had been free from pandemic influenza for more years than in any previous epoch since the middle of the 17th century. The pandemic period of 1658 is separated by less than 20 years from that in Sydenham's cycle which was followed within 10 years by the events of 1688–1693. Not quite 20 years more bring us to the influenzas of 1710–12 and the succeeding intervals continue to be of about 20 years; thus the years from 1712 to 1732 bring us to the middle of another epoch which closed in or about 1743; another twenty years pass and we have the pandemic of 1762; 1782 and 1803 are again storm centres. The gap lengthens to nearly thirty years, then we have three pandemics within six years, 1831, 1833, 1837. The following interval is much shorter, only ten years, and the resulting outbreak was of the first order of severity. Statistics give no reason to suppose that

* Dr. F. G. Crookshank (*Journ. Roy. San. Inst.*, September 1920) has, however, justly called attention to the severe epidemic of pneumonia in Middlesbrough and its neighbourhood during 1888 which the late Dr. Ballard investigated (App. A., No. 18, *Report* of M.O. to L.G.B. for 1888).

influenza was truly epidemic in England in any year later than 1858, so that the interval free from explosions must be put as at least 30 years, although during this period influenza was pandemic in Europe and North America in 1874–5 and in North America in the previous year. Judging from the earlier experience we ought to have had pandemic influenza in 1875; actually the only even minor outbreaks before 1890 and after 1874 were in Norfolk (1878) and the Northallerton incident mentioned above. Hence both in respect of interval as well as of lacking portents in our general mortality experience, the events of 1889 and following years were surprising. The subsequent history reveals other novelties.

In table 4 are shown the deaths attributed to influenza in London for each year since 1889 to the middle of 1919.

TABLE 4.

London Influenza.

Date.	No. of Deaths.
1890	648
1891	2,336
1892	2,242
1893	1,526
1894	750
1895	2,156
1896	496
1897	671
1898	1,283
1899	1,817
1900	1,950
1901	664
1902	1,073
1903	644
1904	709
1905	689
1906	895
1907	967
1908	1,350
1909	1,216
1910	723
1911	496
1912	535
1913	862
1914	613
1915	1,058
1916	915
1917	751
1918	12,927
1919 (to 10th May)	3,871

We shall shortly have to examine more detailed statistics, but this table suffices to bring out some important points. Although it is practically certain that the attack rate of

influenza was higher in 1890–1 than in any subsequent year until we reach the summer of 1918, the deaths attributed to influenza in 1890 are among the fewest of the series; in the first decennium, 1896 alone returns a lower number. In the second decennium, the lowest total that of 1903 is almost equal to that of 1890, while in the decennium now drawing to a close only 1911 and 1912 provide decidedly smaller quotas. The years 1890–93 are to be counted pandemic years; 1895 was another. This remark applies to 1898–1900, and London was not exempt from the pandemic of 1907–8 which, as may be inferred from Table 5, affected most of the States making returns. Apart from the statistical

TABLE 5.

Influenza Deaths.

——	Population in Millions, 1905.	1905.	1906.	1907.	1908.	1909.	1910.
England and Wales.	34·0	6,953	6,310	9,257	10,112	8,992	6,504
Scotland - -	4·6	912	313	425	419	314	274
Ireland - -	4·4	1,219	978	1,720	1,798	1,303	1,332
Denmark (urban population).	—	268	58	244	299	146	149
Norway - -	2·3	138	54	177	150	46	186
Sweden - -	5·3	404	113	429	912	209	534
Switzerland -	3·5	1,202	486	1,018	663	766	463
Prussia - -	37·1	6,380	2,516	5,512	8,824	4,252	4,098
Bavaria - -	6·5	1.733	485	1,648	2,130	657	1,098
Saxony - -	4·5	765	277	693	998	430	630
Wurtemburg -	2·3	545	180	537	648	287	411
Baden - -	2·0	329	99	456	418	164	256
Holland - -	5·6	783	516	925	946	557	714
Belgium - -	7·2	1,364	1,136	1,379	1,316	1,171	1,221
France - -	39·2	No data	6,891	16,024	7,765	11,782	5,797
Portugal - -	5·7	1,605	1,354	2,194	1,463	1,616	—
Spain - -	19·0	14,287	10,478	18 565	9,103	10,511	9,835
Italy - - -	33·2	6,870	4,309	8,043	4,450	5,719	2,780
Connecticut -	1·0	237	207	532	396	193	264
Massachusetts -	3·0	443	203	510	337	237	250
Michegan - -	2·6	649	321	897	633	370	458
Maine - -	0·7	243	109	278	241	170	105
Japan - -	47·7	2,720	1,676	4,319	2,304	2,803	2,641
New South Wales	1·5	183	145	330	168	107	130
Victoria - -	1·2	133	243	276	166	110	100
Queensland -	0·5	53	59	222	177	71	63
South Australia	0·4	27	38	57	35	23	26
West Australia -	0·3	16	29	16	37	24	18
Tasmania - -	0·2	16	24	33	31	12	7
New Zealand -	0 9	70	132	223	64	47	141
TOTALS -		50,517	39,739	76,939	57,003	53,089	40,487

adjustments needed to make the annual figures strictly comparable, something is to be allowed to fashion of nomenclature; but when all discounts equitably due have been made it will still be found that the position lost in 1890 has never been regained, that such phrases as the return of influenza, the reimportation of influenza, etc., are mere figures of speech; we have never lost it again since 1889.

We shall now attend to some finer points. Table 6 provides the annual death-rates at ages for London from influenza, bronchitis, pneumonia, and phthisis for each year from 1891 to 1911. The age grouping used in the weekly returns has been retained, although it is statistically inferior to that of the

TABLE 7.

The Special Outbursts.

Year.	Duration.	Average Weekly Deaths in the Epidemic.				Proportion of Mortality from Influenza and Pneumonia borne by Age Group 20–40.
		Influenza.	Pneumonia.	Bronchitis.	Phthisis.	
						Percentage
1890	15 weeks from 4th January 1890.	38·5	147·3	354·4	213·2	19·9
1891	17 weeks from 18th April 1891.	124·5	145·1	216·2	166·5	15·4
1891–92	18 weeks from 19th December 1891.	120·9	180·9	447·3	184·3	11·6
1893–94	27 weeks from 4th November 1893.	44·9	162·7	263·7	165·9	12·3
1895	16 weeks from 16th February 1895.	116·6	178·9	382·0	179·6	10·0
1899–1900	28 weeks from 2nd December 1899.	75·4	194·1	287·1	180·3	13·3
1908	22 weeks from 4th January 1908.	50·7	159·5	149·8	139·6	10·1
	WEEKLY DEATHS IN 1889.					
	1st Quarter - -	—	95·9	269·9	151·0	10·2
	2nd ,, - -	—	77·5	120·2	149·2	11·5
	3rd ,, - -	—	46·5	80·6	137·5	12·6
	4th ,, - -	—	91·5	219·5	158·3	12·4
	CENSUS POPULATIONS.					Proportion of the Populations, aged 20–40.
	1891 - - - -	4,211,743				33·9
	1901 - - - -	4,536,541				35·4
	1911 - - - -	4,521,685				34·6

TABLE 6.

Death-rates per Million from Influenza, Pneumonia, Bronchitis, Phthisis, in London, 1889–1911.

Ages.	0–5.				5–20.				20–40.				40–60.				60–80.				80 and upwards.				All Ages.			
—	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.
1889	4	4,307	7,662	527	—	155	80	500	—	333	159	2,604	—	868	1,726	3,761	8	2,279	12,039	2,137	—	3,866	38,782	119	1	982	2,179	1,872
1890	114	5,743	10,033	640	24	237	110	604	115	633	292	3,025	321	1,656	2,824	4,262	611	3,411	16,087	2,489	1,051	5,782	46,960	526	156	1,489	2,977	2,170
1891	498	5,820	9,544	540	87	227	106	598	253	677	267	2,777	877	1,993	2,940	4,021	3,315	4,604	18,769	2,152	7,873	7,011	55,054	402	554	1,640	3,118	2,013
1892	395	5,684	8,409	607	62	217	85	523	216	483	203	2,636	876	1,667	2,307	3,677	3,335	3,933	15,715	2,019	9,660	5,616	47,119	337	533	1,450	2,631	1,890
1893	302	5,669	7,685	700	64	265	90	547	204	744	203	2,621	545	2,158	2,162	3,624	1,938	4,541	14,418	2,174	4,559	7,579	44,104	330	356	1,681	2,431	1,910
1894	178	5,859	6,708	594	33	174	54	493	94	390	140	2,357	277	1,081	1,422	3,438	897	2,600	9,629	1,890	1,829	4,734	31,576	538	174	1,233	1,811	1,748
1895	505	6,722	7,714	543	57	194	59	505	175	385	186	2,459	733	1,151	2,135	3,620	3,058	3,066	14,732	2,163	10,223	5,744	46,585	158	496	1,378	2,446	1,834
1896	144	6,598	6,802	564	19	181	52	466	64	336	121	2,346	173	963	1,249	3,593	530	2,460	9,289	1,949	1,239	4,957	28,710	568	113	1,264	1,726	1,776
1897	147	5,792	5,943	576	24	156	39	444	76	313	115	2,307	231	1,025	1,280	3,631	827	2,189	9,492	2,145	2,227	4,251	32,493	557	152	1,145	1,679	1,782
1898	233	6,187	5,707	601	48	159	50	426	106	357	119	2,307	431	1,035	1,434	3,696	1,837	2,446	10,552	2,052	4,565	7,939	32,551	595	289	1,224	1,750	1,792
1899	316	6,570	5,696	581	48	207	41	498	185	502	158	2,372	621	1,589	1,840	3,909	2,250	3,380	13,260	2,329	8,905	6,131	45,158	827	406	1,489	2,058	1,897
1900	206	6,586	5,648	452	56	221	47	444	174	579	133	2,185	673	1,795	608	3,760	2,564	3,907	12,368	2,336	9,833	7,064	40,330	668	432	1,594	1,928	1,780
1901	143	6,043	4,961	463	22	174	34	424	69	398	103	2,132	216	1,347	1,267	3,471	787	3,166	10,173	2,245	2,438	5,766	33,574	891	146	1,349	1,613	1,705
1902	187	7,373	4,934	571	34	195	37	437	97	452	112	2,052	325	1,369	1,258	3,409	1,413	3,160	11,363	2,179	4,712	6,052	42,920	508	237	1,497	1,742	1,681
1903	147	6,344	3,599	523	19	171	24	415	65	344	76	1,950	201	1,113	804	3,303	731	2,910	6,994	2,196	2,778	7,468	27,778	638	142	1,285	1,156	1,621
1904	127	6,940	3,616	569	28	211	18	483	50	393	95	2,054	203	1,274	1,043	3,435	975	3,666	9,291	2,112	3,143	7,587	36,186	943	156	1,458	1,406	1,708
																	Ages 60 and upwards.											
1905	116	6,817	3,426	484	14	227	30	391	60	443	76	1,808	195	1,428	942	2,976	1,096	4,381	10,628	2,003	—	—	—	—	133	1,530	1,335	1,503
1906	135	6,235	2,835	547	33	233	23	414	68	426	69	1,856	274	1,386	835	3,000	1,354	4,310	9,583	1,835	—	—	—	—	198	1,453	1,182	1,530
1907	146	7,820	3,446	544	31	209	20	446	91	424	84	1,816	287	1,488	940	2,897	1,400	4,701	10,230	1,908	—	—	—	—	214	1,663	1,328	1,515
1908	112	6,480	2,408	499	36	214	21	376	99	424	71	1,806	381	1,345	868	2,742	2,265	4,562	9,829	2,011	—	—	—	—	298	1,487	1,180	1,469
1909	114	6,540	2,303	409	28	246	24	387	97	499	87	1,724	330	1,699	1,070	2,744	2,000	5,438	11,514	2,059	—	—	—	—	268	1,667	1,352	1,443
1910	85	6,460	2,275	358	18	225	22	352	55	422	61	1,564	208	1,365	784	2,333	1,160	4,450	9,156	1,768	—	—	—	—	162	1,488	1,115	1,273
									Ages 20–45.				Ages 45–65.				Ages 65 and upwards.											
1911	77	6,042	2,370	526	20	198	26	409	46	416	110	1,780	215	2,690	1,528	2,306	864	3,476	12,351	1,543	—	—	—	—	110	1,238	1,151	1,349

annual reports, and variations of classification, one consequence of which is to raise the influenzal rate in later years, necessitate caution in making comparisons. In Table 7 summaries of the features of the special exacerbations or explosions are set out. In Table 8 are shown the proportional mortality from notified

TABLE 8.

Influenza. Proportion borne by Age Group 20–40.

Year.	Influenza.	Pneumonia and Influenza.	Pneumonia.	Bronchitis.	Season of Year.	Proportion of Population at Age 20–40.
1890	24·9	19·9	18·6	4·6	Winter	33·8
1891	15·4	15·4	15·5	3·2	Summer	33·9
1891–92	12·9	11·6	10·7	2·7	Winter	34·0
1893–94	17·3	12·3	10·9	2·7	,,	34·3
1895	11·2	10·0	9·2	2·5	Winter and spring.	34·5
1899–1900	13·3	13·3	13·3	2·5	,,	35·3
1908	10·3	10·1	10·1	1·9	,,	34·8

influenza, pneumonia, and bronchitis borne by the age-group 20–40, allowance being made for changes of age constitution; but chance fluctuations of undefined magnitude are to be expected. Age corrections are not, of course, applicable to the data of the recent prevalence.

It has already been seen (Table 3) that the participation of different age-groups in the mortality was different in 1890 from the experience of 1847; the very much greater share taken by the young adults in 1918–19 makes it pertinent to inquire whether a uniform trend in this direction can be found in the interval between 1890 and 1915. Although it is permissible to conclude that during the past 30 years the share of mortality borne by young adults has been greater than in the 1847 experience, there is no sign of a uniform progression. These figures relate to death-rates; we shall discuss incidence rates in a later section.

Before passing to the detailed study of the events of 1915 and subsequent years it will be proper to make some general reflections upon the epidemiological history of the quarter of a century which succeeded the pandemic explosion of 1889–90. Whatever view may be taken of the incidents, or lack of incident, which ushered in the new cycle, the general truth of Dr. Creighton's remark (uttered in 1893) that the new age differed profoundly from anything that had gone before will by now be plain to the reader. Since 1890 the endemic prevalence of what has at least been called influenza main-

tained itself at a height entitling it to be regarded as an important cause of recorded mortality. In no single year have less than 450 of the inhabitants of London come by their deaths from "influenza"; in each of 11 years more than a thousand fatalities have been allotted to this cause group. Five times the tide has risen sufficiently to justify the word "explosion" or "epidemic" and mention of the fact in the press. We may not be able to explain these events, but we must take serious note of them.

To explain outbursts of epidemic disease an appeal has always been made to one of two conflicting principles. As Dr. Singer has expressed it: "The history of the doctrine of "epidemics may be summed up in one sentence, as a struggle "between the ideas of *miasma* and *contagion*." Down to a quite recent time the doctrine of miasmatic influenza probably commanded a majority of supporters, and it was incumbent upon Dr. Parsons to devote much space in his report on the influenza of 1890 to a demonstration that the principal argument in favour of a miasmatic or air borne *materies morbi*, viz., the spread of the disease with a velocity exceeding that of human travel and its alleged appearance in localities exempt from human importation was fallacious. Perhaps no medical man now adheres to the doctrine of miasma, but the dramatic character of the epidemics, the patent discontinuity in respect of prevalence between epidemic and non-epidemic years, has led to almost universal acquiescence in a theory of re-importation. It is argued that the disease has been reintroduced at intervals from some other country in which the *materies morbi* had acquired a peculiar virulence or infectivity, that not otherwise can we account for the catastrophic increase of cases and deaths.

Not to pause upon the fact that this explanation proceeds *ab ignoto ad ignotius*, since nothing that has been ascertained of the immediately exciting causes of influenza gives any warrant for believing that the conditions of those remoter countries into which the origin of influenza has been exiled —the favourite location is Russia—are peculiarly favourable to an exaltation of infectivity, we may note that the basal assumption is unsound. It is not true that a sudden transition from a low endemic to a dramatically epidemic level of prevalence needs the postulation of a new factor. Sir Ronald Ross first showed mathematically that a disease, the continued existence of which depends upon the conjoined action of several factors, might as a consequence of a small and undetectable variation in one of the factors suddenly assume epidemic proportions. So long as one or more of the factors remain below a critical value the disease smoulders and may pass almost unregarded; let the critical point be passed and the disease will increase with great rapidity. A numerical example will make the theory more vivid. Under certain

conditions,* when the number of anophelines per head of population in a district is 40 the malarial case rate will be 5 per 1,000; an increase of anophelines to 44 will be associated with a threefold increase of malaria; when the mosquitoes reach 46 the malaria rate will be 50 per 1,000, and when the anophelines have reached 49, 10 per cent. of the human population will be infected. Thus an increase of anophelines so slight that it "could scarcely be detected by our present methods" might be attended by an increase of the malaria rate amounting to a severe epidemic.

The application of this reasoning to our present problem is obvious. It does not, of course, exclude the possibility of re-importation being a factor of the situation, but, just as the evidence collected by Dr. Parsons convinced most epidemiologists that contagion could not be excluded as a vehicle of dissemination, that no instances of outbursts without the possibility of contagion were known, the analysis of Sir Ronald Ross shows that we might have explosions of influenza even if Russia or Spain did not exist and the British frontiers were hermetically sealed. We should blunt Occam's razor if we failed to inquire whether any factors might exist, the variation of which above or below a critical value would explain the observed sequences. In the case before us we must start from the recognition of the fact that whatever the nature of the aetiological elements they are such that social evolution in the later years of the 19th century raised them more nearly to the critical value, and has maintained them ever since closer to that value than in any earlier age. We must observe that this remark applies not only to Great Britain, but to all Western Europe and to the North American Continent. European experience since 1889 is, indeed, closely paralleled by that of the United States. In Massachusetts, for instance, the death rate from "influenza" never reached 100 per million per annum between 1868 and 1890, and never again fell below that level until 1902, exceeded 200 per million five times between 1890 and 1901, and in 1918 attained the same calamitous dimensions as seen in Europe.

The historical student recalling the substitution principle of Robert Watt, approved by Farr, and, perhaps, extravagantly eulogised by Creighton, will inquire whether in the extinction or decline of some national disease for which influenza is substituted we are not to look for the needed variation. At the first blush we seem to open up here a promising path. The most noteworthy feature of our vital statistics, so far at least as adults are concerned, in the last quarter of the 19th century was the decline of the tuberculosis death rate. The effect of this has been presumably to leave in the population an appreciable percentage of persons who would otherwise have died of tubercular disease. But, as all records of influenza explosions

* "*Prevention of Malaria*," by Sir Ronald Ross, 2nd edition, London 1911.

show, and as so many early writers have observed, the infection of influenza, if not specially attracted by the phthisical, is at least particularly deadly to them. Might it not be that the addition to the population of a steadily increasing percentage of those susceptible naturally to infection through the respiratory tract is precisely the slight change of a factor of prevalence which is required? Can it be that the slighter intensity and frequency of influenza explosions between 1910 and 1918 is due to the fact that the decline of tuberculosis was less active in the decennium 1901–10 than in the previous decennium? Does not this hypothesis cast a ray of light upon the especial severity of the disease upon young adults in the last great pandemic, immediately prior to which the conditions unfavourable to the now considerable population of young susceptibles to phthisis had been accentuated all over the world through the direct and indirect consequences of war?

A superficial examination of European vital statistics would indeed seem to lend support to this hypothesis. In 1901–5 the death rate per million from pulmonary tuberculosis in England and Wales was 1,218, and the influenzal death rate 192; in 1905–10 the phthisis rate fell to 1,107, and the influenza rate rose to 235. In France and Italy on the other hand the tuberculosis (pulmonary) rates hardly changed.

For the great towns of France the successive quinquennial rates were 2,673 and 2,675 for Italy, 1,050 in each quinquennium. The influenza rates of these two countries showed much less increase than in England; that of the French cities only rose from 205 to 212; that of Italy actually fell from 156 to 150. But the very figures we have quoted are enough to show how rash it would be to build anything upon them. No allowance for differences of age constitution would make it probable that the tuberculosis rates in reality differ so much from country to country as the figures assert. Even if they could be accepted at the face values a wider survey destroys the illusion. In Massachusetts the tuberculosis rate fell more sharply than in England and Wales from 1,630 to 1,438, but the influenza rate instead of increasing declined (125 to 97). In Spain, which seems always to have an enormous *statistical* prevalence of influenza,* both pulmonary tuberculosis and influenza declined together.

The obvious inference is that a comparison of international vital statistics cannot lead us to any reliable conclusions on this point. There remains the suggestion that the steady increase of movement and intermingling of population associated with the improvement of communications may be the factor we are in search of. We do not mean that owing to improvements in methods of transit it is now easier for the *materies morbi* to be brought from its supposed endemic centre into Western Europe, although this is a possibility, and Dr. Parsons (I. 53) wrote in

* *See* p. 236.

1891 that "the progress of the epidemic over the globe, taking "Russia as its starting point, has been more rapid than that of "previous epidemics." We are now examining the facts, which Dr. Parsons (II. 39) in his later study said, pointed "not "to dissemination of the disease over the globe from a single "centre as appeared to be the case in 1889–90, but to the "revival of epidemic activity at or about the same time in "several different centres in different parts of the world." The centralisation of industries, the tendency not merely in England, but in all advanced industrial nations, for the workers in cities to seek habitations in the outer suburbs, was already making itself felt in the eighties of the last century, and has proceeded with an accelerated velocity in the past 30 years. This evolution has been favourable to the public health in the relief of housing congestion in the centres of the urban districts which it effected, but it has had consequences which may be less advantageous from the present point of view. The larger unit of industrial production in which numerous human beings are brought into contact one with another, even if under conditions definitely more favourable to life than those obtaining in the smaller industrial units of an earlier age, must render frequent the opportunities of transfer from one to another of *materies morbi* formerly confined to separate groups.

Suppose we had a chess board over the squares of which the leaves of some plant were scattered and a number of caterpillars were then thrown at random upon the board; suppose also that the leaves were capable of nourishing the caterpillars, but not especially attractive to them. Then it is to be expected that the insects will sample the leaves on the squares upon which they alight, but wander further afield until all the squares have been visited. If partitions had been erected around each square, the caterpillars must have stayed where they fell and been forced by hunger to consume the not too attractive nourishment provided. In the one case we shall find slight effects of the invasion over the whole board; in the other some squares will have been swept clean, others will remain untouched. This apologue illustrates the possible effects of concentration in great industries. When the nature of the disease and normal powers of resistance to it are such that successful invasion needs repeated attacks, massive dosage, the distribution of workers into a number of small badly constructed work places will produce the maximum of evil. When the condition of spreading is less exacting, the infectivity high and normal resistance low, aggregation of workers, however favourable the conditions, so long as mixture occurs at all, will achieve the maximum result. It is probable that tuberculosis is in the former category hence the apparently paramount importance of home conditions, and the deplorable results of

home industries; it is possible that influenza is in the latter class. When we recollect that a corollary of centralised main industries and centrifugal housing is the great tide of railway, tram and tube traffic which has arisen so steadily during the past thirty years, it is *possible* that we have come upon the varying factor which now always oscillates so close to the critical point of epidemic prevalence. It is probable that all epidemic diseases owe their epidemicity to the conjoined working of numerous factors, that the failure of any one of these factors to pass a critical point destroys the whole sequence. The history of bubonic plague, of smallpox, and of typhus has afforded examples.

We have in the plague history of England an epoch of approximately 350 years during which the conditions in this country were such that some slight and inappreciable variation of a hidden factor was sufficient to precipitate an epidemic; in our own time, on the other hand, the introduction of infection, even the establishment of an epizootic through a wide area not only did not result in a general epidemic, but did not even give rise to a local outbreak; the disease flickered, killed half a dozen persons, and went out. What link was wanting in the chain we do not know and, in view of our aetiological ignorance respecting so clear cut and assiduously studied a disease as plague, we need not attach great importance to the speculative identification of the required factor of epidemic influenza with the modern tendency to peripheral housing and centralised industries. Yet the speculation has at least one character of a true hypothesis in that it will co-ordinate the seemingly contradictory experience of influenza through the past two centuries.

If it is a fact that promiscuous intermingling of a random population will raise the disease from an endemic to an epidemic level or rather make the transition from one to the other but a step, we should expect that under social conditions not securing such intense mixing, epidemics of influenza will only occur under special conditions.

We may put it in this way. The epidemic is generated by the correlated action of several factors *a*, *b*, *c*, *d*, *e*, &c., if the intensity of one of these, *a*, say, exceeds a certain minimum, then very small variations of *a* and also of the remainder are needed to generate an epidemic. When *a* is varying upon a lower level great changes in all are necessary. If *a* be the measure of commingling which we suppose to be more intense than ever before, small variations of it and concomitant small variations of the others, which must include general resisting powers, are enough to lead to an outbreak. When *a* is on a lower level this is not so; there must be big changes. Therefore as precursors of epidemics at a time when *a* swings about a lower level we shall have evidence

of general or special unhealthiness in the community; when the status of *a* has been raised we shall not. *En revanche* after the other factors have fallen to their common average no more will be heard of influenza as an epidemic for many years under the old system; under the new, breaking out in an apparently healthy community the disease will continue at quite short intervals to pass above the flash point, and be responsible for epidemics. This seems to be what has happened in influenza. We can also, upon this supposition, make light of the difficulties and conflicts of testimony as to the direction along which influenza extended to this country. Somewhere in the decennium 1880–90, the *a* factor, which we identify with a certain frequency of commingling in a population, members of which carry the infective principle, reached a critical intensity and the epidemic of 1890 was generated; it appeared in many different countries at about the same time, not because some volcano outside their borders awoke to a new and sinister activity, but because the parallel developments of civilisation in all reached approximately the same level at nearly the same time; the earlier or later development of the particular intra local epidemic depended upon local and minor variations of the several factors. The materials of the conflagration came from within in each case, but were not brought to red heat at the same moment.

An unstable equilibrium has existed throughout Europe and America the disturbance of which has had for consequence the frequent epidemics of the past quarter of a century. The impossibility of making any accurate prediction as to when such will occur depends upon the present difficulty of accurately measuring the variations of the postulated factor. In one sentence, the problem of epidemic influenza is largely an internal problem of each nation; there is no question of shutting the wolf out of the sheep fold, he has been regularly lying down with the lamb for years; his ravages depend as much upon the disposition of the sheep within the fold as upon his appetite. Thus broadly stated, the hypothesis neglects some portion of the truth; however combustible the material, there can be no fire without a spark, and when the fire has been started, it may spread to material not naturally combustible. We must not ignore this consideration, but it has, perhaps, been over emphasised in current discussion.

We have felt authorised to devote so much space to the display of what is after all an unproven hypothesis, a mere speculation, because of the important inferences to which it leads.

The wisdom of insuring against fire depends at least as much upon the consequences of a fire if it break out as upon the probability of a conflagration; the value of a hypothesis depends both on its intrinsic credibility and its practical

consequences. Were this hypothesis to be true, it would follow that the problem of influenza is a graver problem now than in the past. When the postulated factor normally fluctuated well below the flash point, influenza could only become pandemic if some grave antecedent disturbance of the public health occurred. Signs would be vouchsafed which the instructed observer could recognise as portents; we have seen them in the chronologies of Sydenham and Huxham, also perhaps in the unsavoury conditions of 1846–7. But when the pendulum swings always close to the danger limit, we shall not receive the warning, we shall always live in the shadow of a possible disaster. Our position in face of influenza will be that of the 17th century physician in regard to plague, and our remedy will not be specific but general, not found in the exclusion of infection from our shores, not even in specific immunisation, but in the more laborious and less dramatic task of attending to the general principles of hygiene. This will, of course, still leave a possibly wide field of usefulness for *ad hoc* or specific measures when the conflagration rages or is immediately in front of us, and it is by no means a counsel of despair. But it removes influenza from the class of capricious or alien foes to be dealt with on simple military lines.

With these tentative reflections we must leave the general historical problem of influenza. We can no longer regard the miasmatic or cataclysmal theory as adequate; the researches of Parsons and others in the early years of the present cycle effectively destroyed that theory. Neither, we think, can influenza be reduced to the category of imported infections, the consequences of which might be obviated by attention to a foreign endemic focus. We believe that the general treatment of the problem will ultimately be found to be parallel with that appropriate to the now obsolete plague of typhus in Great Britain, and we have little reason to expect that the years 1918–9 are to play that part in the history of influenza assigned by fate to the years 1665–6 in English experience of plague.

TABLE 9.

MALES.—*Deaths from Influenza, all forms of Pneumonia and Bronchitis, in England and Wales from* 1889–1917.

Ages.	0–5.			5–10.			10–15.			15–20.			20–25.			25–35.			35–45.			45–55.			55–65.			65 and over.		
—	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.
1889	24	7,567	14,001	—	493	234	—	156	51	1	330	44	—	379	62	—	1,088	258	—	1,511	680	—	1,678	1,626	1	1,575	3,203	3	1,866	7,691
1890	291	8,471	15,083	45	602	276	38	236	66	86	455	74	107	752	96	250	2,035	394	345	2,827	1,042	357	3,175	2,521	382	2,822	4,351	514	2,763	9,783
1891	957	9,671	16,899	127	576	271	108	184	54	246	410	70	252	653	92	668	1,847	368	1,000	2,730	1,019	1,321	3,235	2,551	1,471	2,911	4,998	2,471	3,115	11,480
1892	698	8,958	15,038	97	501	248	88	195	48	175	431	50	194	428	77	489	1,350	331	745	2,077	811	996	2,421	2,000	1,312	2,253	4,078	2,879	2,617	10,390
1893	460	8,559	12,316	101	608	231	76	201	59	162	447	57	163	585	60	428	1,604	266	577	2,471	706	668	2,621	1,759	727	2,381	3,499	1,446	2,626	8,782
1894	365	9,275	12,792	46	435	193	43	159	37	98	325	37	117	377	44	277	1,082	220	335	1,595	532	418	1,748	1,309	519	1,639	2,519	1,015	1,970	6,973
1895	802	9,987	13,165	79	521	185	69	178	40	157	378	49	185	455	78	411	1,162	263	557	1,645	659	774	1,903	1,796	1,043	1,886	3,587	2,096	2,212	9,452
1896	297	9,917	11,899	39	558	160	24	161	33	62	341	38	77	478	56	179	1,146	197	220	1,812	548	266	1,979	1,266	272	1,904	2,668	476	2,034	6,704
1897	405	9,754	11,157	54	432	136	31	162	35	91	352	36	108	463	49	263	1,169	189	353	1,772	539	412	2,056	1,298	497	1,844	2,754	871	2,159	7,113
1898	595	10,115	10,386	72	449	106	59	172	35	132	332	40	154	431	47	345	1,144	153	478	1,751	507	581	1,883	1,339	761	1,822	2,834	1,833	2,035	7,523
1899	619	10,378	10,562	90	531	141	68	201	35	158	417	39	189	598	50	440	1,495	207	704	2,187	616	797	2,429	1,610	983	2,235	3,347	2,037	2,389	8,672
1900	620	11,370	10,761	95	583	154	67	194	34	177	483	38	238	678	52	528	1,707	244	730	2,686	612	976	2,898	1,742	1,310	2,667	3,691	2,942	2,881	9,389
1901	288	9,950	9,088	41	505	139	44	185	29	108	377	38	97	534	45	256	1,327	152	329	1,979	489	433	2,289	1,383	486	2,116	2,943	830	2,361	7,506
1902	418	12,187	9,034	59	593	114	52	217	28	83	458	36	105	631	47	246	1,633	160	392	2,403	443	503	2,682	1,201	620	2,709	2,796	1,164	3,013	6,982
1903	353	11,183	8,132	39	483	77	31	222	19	86	388	26	84	475	39	240	1,244	159	314	1,822	331	398	2,174	964	501	2,196	2,312	1,021	2,808	6,422
1904	350	12,137	8,237	50	586	97	28	215	21	67	418	32	71	533	37	200	1,243	142	282	1,830	461	360	2,081	1,136	471	2,275	2,720	903	2,901	7,695
1905	351	11,510	7,248	51	587	84	34	203	23	83	435	29	94	607	43	251	1,377	133	322	2,178	365	438	2,446	1,095	599	2,584	2,533	1,164	3,141	7,408
1906	296	10,118	6,110	51	536	84	40	210	17	85	434	21	84	581	37	237	1,487	136	300	2,057	347	401	2,392	1,044	579	2,629	2,496	1,051	3,183	7,164
1907	396	12,368	7,563	68	618	97	41	224	28	106	411	41	114	561	30	299	1,565	189	443	2,132	435	568	2,529	1,218	778	2,648	2,752	1,618	3,357	8,070
1908	396	11,154	6,555	58	530	89	51	207	25	102	399	23	127	504	43	340	1,345	143	490	1,909	415	634	2,178	1,089	883	2,350	2,646	1,761	3,096	7,835
1909	301	10,870	5,942	63	655	81	36	233	32	107	476	28	129	570	34	302	1,554	161	419	2,206	413	563	2,580	1,165	749	2,773	2,792	1,631	3,817	8,756
1910	259	9,972	5,430	52	558	66	34	198	19	77	340	24	86	476	38	222	1,274	124	315	1,843	359	419	2,207	955	568	2,424	2,414	1,221	3,249	7,643
1911	233	9,937	5,498	37	576	97	31	178	25	52	350	32	75	516	31	174	1,364	172	241	1,856	414	309	2,057	1,126	360	2,142	2,607	719	2,606	7,980
1912	223	9,618	5,210	31	574	93	29	164	25	64	345	33	67	426	46	182	1,313	148	275	1,823	489	359	2,107	1,248	458	2,209	2,766	991	2,808	9,236
1913	261	10,154	5,390	43	541	93	35	182	36	72	340	27	102	455	49	287	1,188	182	354	1,813	504	505	2,047	1,208	627	2,123	2,887	1,076	2,662	9,136
1914	242	10,334	5,101	46	657	117	37	206	23	88	372	22	88	440	39	231	1,389	189	372	2,082	535	450	2,327	1,254	549	2,274	2,864	1,003	2,935	9,473
1915	454	12,833	6,334	61	741	128	59	291	40	120	457	43	134	591	70	354	1,557	249	512	2,293	722	704	2,718	1,692	908	2,882	3,779	1,922	3,551	12,011
1916	231	8,786	4,593	55	592	104	33	257	34	86	423	47	79	558	57	235	1,287	205	368	1,835	627	598	2,293	1,510	792	2,466	3,262	1,806	3,308	11,099
1917	260	9,279	4,612	57	668	110	30	287	36	88	555	59	74	495	81	175	1,331	293	342	2,216	678	515	2,614	1,518	702	2,742	3,354	1,481	3,460	10,840
CENSUS POPULATIONS AT AGES (MALES).																														
1891	1,767,562			1,693,372			1,610,858			1,465,175			1,247,346			2,089,010			1,611,077			1,191,789			770,124			606,588		
1901	1,855,361			1,738,993			1,670,970			1,607,522			1,472,644			2,485,954			1,931,943			1,396,209			907,945			661,072		
1911	1,936,113			1,847,295			1,747,631			1,654,895			1,502,652			2,831,655			2,336,508			1,694,333			1,085,156			809,370		

TABLE 9A.

FEMALES.—*Deaths from Influenza, all forms of Pneumonia and Bronchitis, in England and Wales from* 1889–1917.

Ages.	0–5.			5–10.			10–15.			15–20.			20–25.			25–35.			35–45.			45–55.			55–65.			65 and over.		
—	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.	Influenza.	Pneumonia.	Bronchitis.
1889	15	6,335	12,110	1	468	232	—	194	46	—	205	53	1	271	76	—	714	268	—	809	705	1	783	1,508	2	1,023	3,123	6	1,626	9,703
1890	240	7,170	12,817	50	575	296	51	208	71	62	313	72	79	417	104	203	1,034	371	227	1,352	969	246	1,340	2,177	282	1,527	4,362	668	2,299	12,179
1891	688	8,009	14,026	150	541	301	101	183	63	203	287	77	242	389	87	597	935	402	729	1,251	966	1,035	1,431	2,367	1,373	1,756	4,874	2,947	2,685	14,460
1892	556	7,415	12,455	118	568	231	84	208	55	184	250	62	181	290	85	467	819	331	654	1,066	867	915	1,150	1,937	1,392	1,505	4,224	3,513	2,389	13,450
1893	400	7,222	10,256	92	542	250	78	222	62	131	293	58	166	342	93	380	979	294	484	1,285	759	507	1,248	1,728	765	1,519	3,678	1,858	2,492	11,773
1894	377	7,634	10,845	58	430	183	46	169	34	86	209	40	87	244	71	234	649	238	264	845	526	336	914	1,223	521	1,084	2,723	1,383	1,792	8,817
1895	634	8,137	10,943	93	498	197	75	174	58	143	216	57	138	282	73	375	652	285	491	915	753	634	988	1,747	1,076	1,253	3,956	3,048	2,167	12,558
1896	217	8,426	10,114	49	499	200	47	166	44	52	223	39	57	250	53	135	664	221	201	838	528	216	972	1,231	252	1,147	2,707	615	1,890	8,711
1897	296	8,033	9,354	65	491	156	50	173	37	87	241	46	91	235	40	193	709	220	282	893	529	289	926	1,223	495	1,073	2,701	1,155	1,907	9,227
1898	491	8,559	8,777	78	451	127	58	158	40	105	218	43	118	232	54	306	655	193	405	913	525	508	917	1,254	847	1,123	2,787	2,479	2,102	9,851
1899	499	8,716	8,503	78	487	145	80	186	48	115	244	45	149	320	52	363	794	225	479	1,072	630	703	1,114	1,613	1,023	1,368	3,454	2,843	2,500	11,396
1900	491	9,277	8,956	93	506	160	87	213	33	124	265	39	164	324	70	409	886	215	548	1,196	718	856	1,299	1,607	1,495	1,575	3,814	4,295	2,612	12,251
1901	246	8,329	7,643	54	482	125	38	158	39	65	259	40	75	274	45	195	747	178	260	963	545	315	1,039	1,294	421	1,232	2,929	1,085	2,307	9,885
1902	298	9,909	7,603	40	627	139	49	227	33	83	325	31	98	405	41	237	1,021	166	311	1,278	448	381	1,405	1,127	589	1,698	2,745	1,638	2,748	9,971
1903	267	9,398	6,830	65	512	91	41	178	25	59	253	26	76	312	37	229	778	138	238	1,024	414	329	1,172	898	507	1,426	2,179	1,444	2,677	8,012
1904	278	8,963	6,995	47	553	113	32	204	25	62	280	29	71	320	34	167	867	178	216	1,146	426	284	1,082	1,058	482	1,617	2,597	1,273	3,094	10,155
1905	271	9,365	5,990	42	588	106	48	201	29	83	279	26	87	353	28	196	923	125	270	1,191	404	346	1,278	1,013	547	1,703	2,518	1,676	3,418	9,715
1906	219	8,467	5,011	60	566	111	42	264	25	54	242	27	63	321	33	204	927	123	255	1,182	357	338	1,233	938	484	1,696	2,454	1,467	3,301	9,206
1907	346	10,388	6,035	70	607	85	55	232	21	75	275	21	90	353	37	247	881	143	355	1,225	419	448	1,344	1,066	755	1,750	2,785	2,385	3,499	11,169
1908	305	8,963	5,441	85	501	80	51	206	22	96	253	35	114	318	26	273	847	117	368	1,056	374	521	1,224	969	823	1,507	2,504	2,634	3,233	10,275
1909	240	9,015	4,787	61	610	98	46	227	28	71	278	33	91	381	44	277	1,031	126	355	1,260	419	432	1,464	1,100	751	1,973	2,801	2,368	4,135	11,978
1910	191	8,229	4,350	52	510	74	32	203	20	57	222	32	72	270	35	163	833	107	213	1,062	329	308	1,144	849	508	1,496	2,113	1,655	3,250	9,482
1911	148	8,134	4,604	33	550	95	30	193	24	33	226	33	35	271	43	134	692	129	183	990	377	236	1,015	949	366	1,325	2,398	914	2,664	10,021
1912	185	8,129	4,308	42	503	88	38	186	29	45	195	44	55	233	44	140	666	158	202	962	407	239	987	1,054	402	1,263	2,489	1,327	2,879	11,818
1913	176	8,338	4,330	42	536	90	28	169	21	56	207	29	81	241	39	166	687	156	243	945	403	299	952	1,069	478	1,262	2,407	1,455	2,688	11,171
1914	194	8,513	4,563	25	610	109	45	219	37	48	222	41	61	259	43	149	662	139	225	971	440	276	1,077	1,102	458	1,376	2,475	1,377	3,145	11,823
1915	363	10,256	4,931	63	759	118	59	275	38	96	294	44	98	331	54	255	842	192	318	1,172	485	459	1,402	1,296	715	1,731	3,069	2,830	3,898	15,679
1916	190	7,148	3,774	45	538	102	46	246	32	68	259	41	83	252	54	180	731	164	276	952	426	380	1,142	1,054	677	1,466	2,501	2,563	3,377	13,726
1917	191	7 308	3,624	53	566	118	39	265	31	70	249	48	76	280	38	143	653	168	205	1,019	412	311	1,425	987	526	1,475	2,446	1,949	3,245	12,954
CENSUS POPULATIONS AT AGES (FEMALES).																														
1891	1,785,928			1,701,806			1,612,709			1,485,690			1,399,066			2,288,718			1,717,729			1,305,085			886,879			766,014		
1901	1,861,347			1,748,298			1,670,770			1,638,621			1,648,278			2,769,886			2,064,062			1,505,982			1,035,305			856,681		
1911	1,918,270			1,849,501			1,752,057			1,681,726			1,673,066			3,124,580			2,509,373			1,833,936			1,213,229			1,069,146		

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CHAPTER II.

A General Account of Influenza in the United Kingdom during 1918–19.

In the first chapter we recapitulated the modern history of influenza, bringing the story down to the year 1911; we are now to enter upon a closer examination of recent events* in the course of which we may find it needful to re-consider the conclusions of our predecessors or the inferences provisionally drawn by us from their experience.

The public health of England and Wales immediately prior to and during the first years of the war exhibited features of interest. So far as influenza itself is concerned, it will have been noticed in tables 9 and 9A of chapter I. that 1915 returned from the whole of England and Wales (but not from London) more deaths attributed to influenza than any other year of the 20th century, not excepting 1908, a fact briefly alluded to by Dr. Stevenson in his annual letter. But this fact did not arouse much general interest, more attention was directed to the increase of deaths from poliomyelitis and from cerebro-spinal fever. The former disease which had been responsible for 177 deaths in 1914, was credited with 178 in 1915, 200 in 1916 and 174 in 1917. Cerebro-spinal fever, the records of which amounted to but 161 deaths in 1907, almost the same number (163) in the year before the war, killed 194 persons in 1914, ten times as many, 1,974, in 1915 (this being the highest statistical prevalence since 1876), 1,214 in 1916 and 1,531 in 1917. As a contributory factor of the total mortality even the largest of these items is not daunting (it is worth noticing that in 1915, the number of deaths of persons at ages 15–45 attributed to influenza was 1,887, almost as many as the *total* of deaths from cerebro-spinal fever, but little attention was devoted to the subject of influenza by the general public who were greatly alarmed by the epidemic of "spotted fever") and seems trifling in comparison with the harvest reaped by pandemic influenza three years later. We are also aware that the uncertainties of statistical comparison, already great in connection with "influenza," reach a climax in the group of quasi epidemic nervous diseases. None the less, we think that during these years there was an unusual prevalence of illnesses which the 17th and 18th century epidemiologists would have described in terms similar to those quoted from Sydenham and Huxham in our first chapter.

Hence we think that the opinion of some epidemiologists, amongst whom we may mention Dr. Hamer and Dr. Crookshank,

* A complete analysis of the mortality statistics is not attempted in this report. Such an analysis has been issued by the General Register Office (Cmd. 700), and to it those needing full details are referred.

that the association or juxta-position of cerebro-spinal fever, polio-encephalo myelitis, the Heine-Medin complex and epidemic influenza is not fortuitous, may be well founded. But a careful examination of the facts, has not warranted a more decided affirmation. We pointed out in the last chapter that no indications of the kind are furnished by the records of 1888–90, an omission to be set off against the discoveries of 1914–18. We can of course explain this discrepancy away. Thus we might argue, firstly, that the increased number of medical men in a position to make and record epidemiological observations and, secondly, some distrust of statistical averages rightly founded upon the disturbance of vital statistics consequent upon war, have both combined to revive the ancient habit of recording medical observations, so that, during the war, we were again *in pari materia* with the 18th century annalists. But such explanations are *ad captandum*, we may be unconsciously influenced by a desire to establish a striking generalisation, to emancipate epidemiology as an independent scientific method from its thraldom to bacteriology, it is prudent not to advance beyond the point reached above, viz., adhesion to the belief that before the influenzas of 1918–19 the quality of diseases affecting the population of this country was abnormal. Any further observations upon the general epidemiological question had better be reserved for a later chapter.

The mass of observations upon the influenzas of 1918 and 1919 is so great, that the compilation of a record intelligible to the reader and not a mere palimpsest, blurred by the superposition of many narratives, is a task of difficulty; it necessitates the exclusion of much detail not perhaps less worthy of discussion than what is retained. Such selection is also liable to unconscious bias, in that observations favourable to the opinions adopted by the compilers may be given greater weight than others of more dubious interpretation.

We have felt, however, that the importance of furnishing the student with a study of the subject which shall be intelligible is such that the risks indicated ought to be taken; it is believed that sufficient indications of sources and critical annotations have been provided to guide the reader in need of more detailed information and criticism.

In the present section some of the broad statistical facts respecting the later epidemics in the United Kingdom are set out. Owing to improvements in the methods of registration and tabulation at the General Register Office, especially such as took effect in the year 1911, it is now possible to provide better coefficients of mortality than were available in earlier times.

The mortality at ages in the metropolis expressed in the form set out in Chapter I. of this Report is no longer the only or

Table 1.

Deaths from Influenza, Pneumonia, Bronchitis, Phthisis in London. Death Rate per Million 1912–14. *Total Deaths* 1915–19.

Ages -	0–5.				5–20.				20–45.				45–65.				65 and upwards.				All ages.			
Year.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.
DEATH RATE PER MILLION.																								
1912	86	5,265	1,929	497	8	140	25	401	51	409	135	1,698	245	1,508	1,664	2,510	942	3,758	13,868	1,535	118	1,181	1,212	1,343
1913	122	6,081	2,111	533	25	195	30	422	83	447	136	1,678	388	1,428	1,647	2,412	1,517	4,171	14,945	1,440	191	1,303	1,282	1,324
1914	116	5,598	1,760	460	22	202	22	505	61	414	126	1,769	237	1,471	1,613	2,589	1,169	4,014	13,824	1,409	139	1,240	1,179	1,403
TOTAL DEATHS.																								
1915	62	3,372	1,091	249	39	315	32	710	167	869	334	3,482	321	1,370	1,581	2,011	469	1,192	3,984	334	1,058	7,118	7,022	6,786
1916	38	2,318	735	179	24	224	28	698	98	573	197	3,183	286	1,065	1,219	1,905	454	1,008	3,395	331	900	5,188	5,574	6,296
1917	41	2,591	798	228	25	232	34	833	88	619	219	3,294	248	1,098	1,187	1,947	337	968	3,268	365	739	5,508	5,506	6,667
1918	1,493	3,307	791	189	2,067	699	43	865	5,890	1,500	264	3,403	2,220	1,247	1,340	2,004	1,216	1,048	3,151	343	12,886	7,864	5,589	6,804
1919 (Jan. to Sept. 3th.)	319	1,380	451	110	344	213	28	465	1,660	601	174	1,890	1,024	722	955	1,148	642	618	2,643	196	3,989	3,543	4,251	3,809

Table 2.

Weekly Deaths from Influenza, Pneumonia, Bronchitis, Phthisis in London during the Epidemics of 1918–19.

Ages		0–5.				5–20.				20–45.				45–65.				65 and upwards.				Totals.				Temperature, Mean.	
	Week.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Influenza.	Pneumonia.	Bronchitis.	Phthisis.	Max.	Min.
	1918.																										
May.	18th week	—	41	9	2	1	4	—	12	2	9	2	45	—	18	18	30	—	20	55	5	3	92	84	94	55·7	42·0
	19th ,,	—	53	17	5	—	1	1	9	2	9	3	51	—	17	16	32	5	12	51	6	7	92	88	103	62·7	44·4
	20th ,,	—	37	17	3	—	8	—	11	1	13	3	48	2	16	14	28	3	21	31	7	6	95	65	97	69·7	47·9
	21st ,,	—	44	2	—	1	7	—	15	1	17	—	56	3	20	9	29	1	7	36	6	6	95	47	106	72·4	50·0
June.	22nd ,,	—	36	5	3	—	5	—	15	1	12	3	53	2	12	9	35	2	10	30	10	5	75	47	116	70·0	46·0
	23rd ,,	—	28	2	7	—	5	1	19	—	11	1	57	3	17	7	31	2	14	25	9	5	75	36	123	70·0	45·3
	24th ,,	1	27	4	3	1	1	1	19	2	6	3	52	1	14	15	34	—	10	25	6	5	58	48	114	70·0	49·4
	25th ,,	—	21	6	3	—	5	—	12	3	21	6	54	3	15	10	31	4	10	26	5	10	72	48	105	67·3	47·6
	26th ,,	5	22	4	4	13	12	—	20	29	22	2	72	18	17	13	27	2	11	28	5	67	84	47	128	68·6	46·4
July.	27th ,,	5	32	6	5	50	30	—	17	104	49	7	76	37	28	12	36	22	7	30	5	218	146	55	139	75·4	51·6
	28th ,,	10	56	1	2	51	27	2	27	144	64	6	74	60	32	22	27	22	21	43	10	287	197	74	140	71·6	51·6
	29th ,,	11	43	4	6	25	6	4	23	89	37	2	67	45	22	12	39	22	14	27	6	192	132	49	141	74·1	55·6
	30th ,,	11	23	4	1	12	8	—	20	34	33	5	58	19	16	14	31	10	9	26	14	86	89	49	124	70·0	54·4
	31st ,,	3	16	4	3	3	6	1	8	13	10	3	66	11	14	6	40	8	26	20	3	38	72	34	120	—	52·6
Aug.	32nd ,,	1	15	2	2	2	2	—	12	7	13	1	65	10	5	9	35	1	3	19	3	21	38	31	117	71·6	53·0
	33rd ,,	2	12	2	4	—	4	1	8	5	6	—	47	6	13	9	35	7	7	13	6	20	42	25	100	75·6	52·4
	34th ,,	—	11	5	5	2	1	—	15	2	6	3	59	4	6	4	31	4	5	15	4	12	29	27	114	77·7	57·4
	35th ,,	—	8	3	4	3	3	—	13	5	6	1	38	4	6	7	31	—	12	23	3	12	35	34	89	67·1	51·6
	36th ,,	—	20	2	3	3	1	—	12	1	5	2	61	4	9	5	30	4	10	14	8	12	45	23	114	69·0	51·9
	37th ,,	—	15	6	—	3	—	1	12	2	6	1	47	6	8	8	31	3	9	18	4	14	38	34	94	64·4	50·4
Sept.	38th ,,	1	10	7	4	1	3	—	16	2	9	1	61	3	7	8	22	2	9	24	8	9	38	40	111	67·7	52·4
	39th ,,	2	23	5	4	3	5	—	11	6	13	—	59	3	4	11	47	3	10	32	6	17	55	48	127	61·3	47·9
	40th ,,	—	18	4	—	3	8	1	16	9	22	2	55	1	18	12	43	4	16	36	4	17	82	55	118	56·0	41·4

41st	,,	4	23	6	3	18	23	—	16	45	49	2	40	8	23	18	30	5	14	36	3	80	132	62	92	69·7	46·9
42nd	,,	27	40	5	3	89	28	1	19	137	80	7	71	58	32	34	28	19	16	49	7	330	196	96	128	—	39·9
43rd	,,	144	87	14	2	260	53	2	17	621	93	15	100	172	28	39	49	59	17	73	6	1,256	278	143	174	Not	given
44th	,,	338	198	18	4	461	77	4	20	1,163	143	9	128	351	54	44	48	145	50	105	9	2,458	522	180	209	,,	,,
45th	,,	308	213	34	4	404	69	5	37	1,215	154	18	146	355	68	76	40	151	35	107	5	2,433	539	240	232	,,	,,
46th	,,	288	200	33	4	260	58	4	22	761	115	14	107	277	51	63	55	119	40	122	12	1,665	464	236	200	,,	,,
47th	,,	152	160	21	1	158	37	1	330	509	67	13	90	218	53	74	53	141	24	142	7	1,178	341	251	181	—	33·3
48th	,,	86	94	14	3	104	30	1	21	434	76	16	95	197	36	55	43	121	30	141	17	942	266	227	179	50·0	39·4
49th	,,	53	64	11	3	64	22	1	17	266	42	14	80	144	21	49	37	133	27	135	8	660	176	210	145	53·9	45·6
50th	,,	32	81	17	—	30	13	1	15	129	19	6	51	74	27	24	35	57	19	71	7	322	159	119	108	—	44·1
51st	,,	23	54	18	3	19	10	1	11	74	25	5	52	45	15	29	48	25	18	75	1	186	122	128	115	46·3	39·3
52nd	,,	11	47	19	4	14	4	—	12	39	14	3	68	16	21	28	29	15	17	57	4	95	103	107	117	48·3	33·7
1919.																											
1st	week	6	51	27	3	8	15	1	17	20	16	7	62	20	20	27	39	11	13	74	9	65	115	136	130	46·4	38·9
2nd	,,	3	56	29	3	7	3	—	20	22	17	2	59	17	15	26	34	19	21	79	6	68	112	136	122	—	36·0
3rd	,,	4	51	21	4	5	3	—	14	12	9	6	63	15	18	15	33	7	15	96	8	43	96	138	122	47·0	34·6
4th	,,	3	53	33	3	—	7	1	9	9	5	6	51	15	15	28	31	6	14	79	7	33	94	147	101	39·1	29·4
5th	,,	6	62	26	4	3	7	2	19	26	21	4	62	18	21	39	40	5	29	86	4	58	140	157	129	35·1	30·0
6th	,,	11	71	26	3	5	8	1	13	44	32	6	76	26	41	54	44	14	30	139	10	100	182	226	146	34·0	29·0
7th	,,	15	98	36	5	22	13	1	22	131	70	18	91	78	42	86	49	27	41	199	5	273	264	340	172	40·3	23·4
8th	,,	45	121	33	2	56	27	1	13	289	74	20	85	158	72	113	45	105	64	254	10	653	358	421	155	48·3	37·9
9th	,,	53	123	37	2	78	16	2	17	380	63	14	68	188	68	76	50	109	37	207	7	808	307	336	144	43·9	34·6
10th	,,	58	89	22	2	54	10	6	12	235	50	9	68	151	38	60	47	99	31	164	8	597	218	261	137	50·9	39·0
11th	,,	33	70	16	4	39	12	2	14	188	26	5	64	106	25	54	29	69	25	137	5	435	158	214	116	50·4	38·3
12th	,,	22	55	18	5	18	8	—	11	93	14	11	59	52	23	30	40	45	24	113	3	230	124	172	118	42·6	33·4
13th	,,	15	47	16	6	6	8	—	17	46	15	10	52	37	23	49	37	21	25	97	7	125	118	172	119	44·1	31·1
14th	,,	12	42	15	6	7	6	1	10	39	19	5	59	19	15	42	37	14	21	103	9	91	103	166	121	49·1	30·4
15th	,,	8	34	10	1	7	7	—	14	45	10	10	52	27	23	33	44	18	13	106	7	105	87	159	118	57·9	41·9
16th	,,	9	37	7	1	5	2	—	12	26	25	4	60	18	24	22	26	13	24	96	4	71	112	129	103	58·6	43·1
17th	,,	3	29	7	4	6	4	1	17	11	18	3	52	12	13	17	45	11	19	66	5	43	83	94	123	53·0	37·9
18th	,,	4	23	9	3	4	6	—	15	12	13	4	62	15	22	20	28	9	18	69	5	44	82	102	113	52·3	38·7

even the best criterion. It is, however, so desirable for epidemiological purposes to preserve comparability that we tabulate the metropolitan experience (Table 1, p. 37) in precisely the form utilised above, save that after 1914 the war-time conditions render estimates of population at ages impossible. The figures for England and Wales are included in Table 9, p. 31. Without considering any other statistics than these, some broad generalities are at once admissible. These are :—

(1) That the mortality in England and Wales, as a whole, attributable directly or indirectly, to influenza, is without any precedent in magnitude ;

(2) That that of the metropolis affords no parallel more recent than 1847 ;

(3) That the toll taken at the young adult ages of life is without *any* know West European or North American precedent.

The first of these conclusions needs no further support than that provided by the figures. With respect to the second, it appears from Dr. Dixey's tabulation (*vide supra*) that the excess mortality debitable to the London epidemic of 1847 amounted to close upon 7,000 deaths. Dr. Farr's contemporary estimate, furnished before the returns were completely analysed, put the figure at 5,000, while Dr. Hamer computes the 1918–19 toll at approximately 18,000 lives. By 1911, the population of London was double that of 1847 ; in 1918–19, the considerable loss of males at working ages *may* have been largely compensated by the influx of operatives and of temporary clerks employed in the Government offices. Assuming a mere equality of populations in 1911 and 1918, an epidemic on the scale of 1847 would have destroyed 14,000 lives, a loss below the truth, but not so greatly below it that we can regard 1918–19 as out of all proportion more fatal to life in London than 1847.

The third point, viz., the greater toll of young lives, is brought out in the tables already mentioned, and shown still more dramatically in Diagram I., taken from Dr. Stevenson's paper read to the Royal Society of Medicine in November 1918.

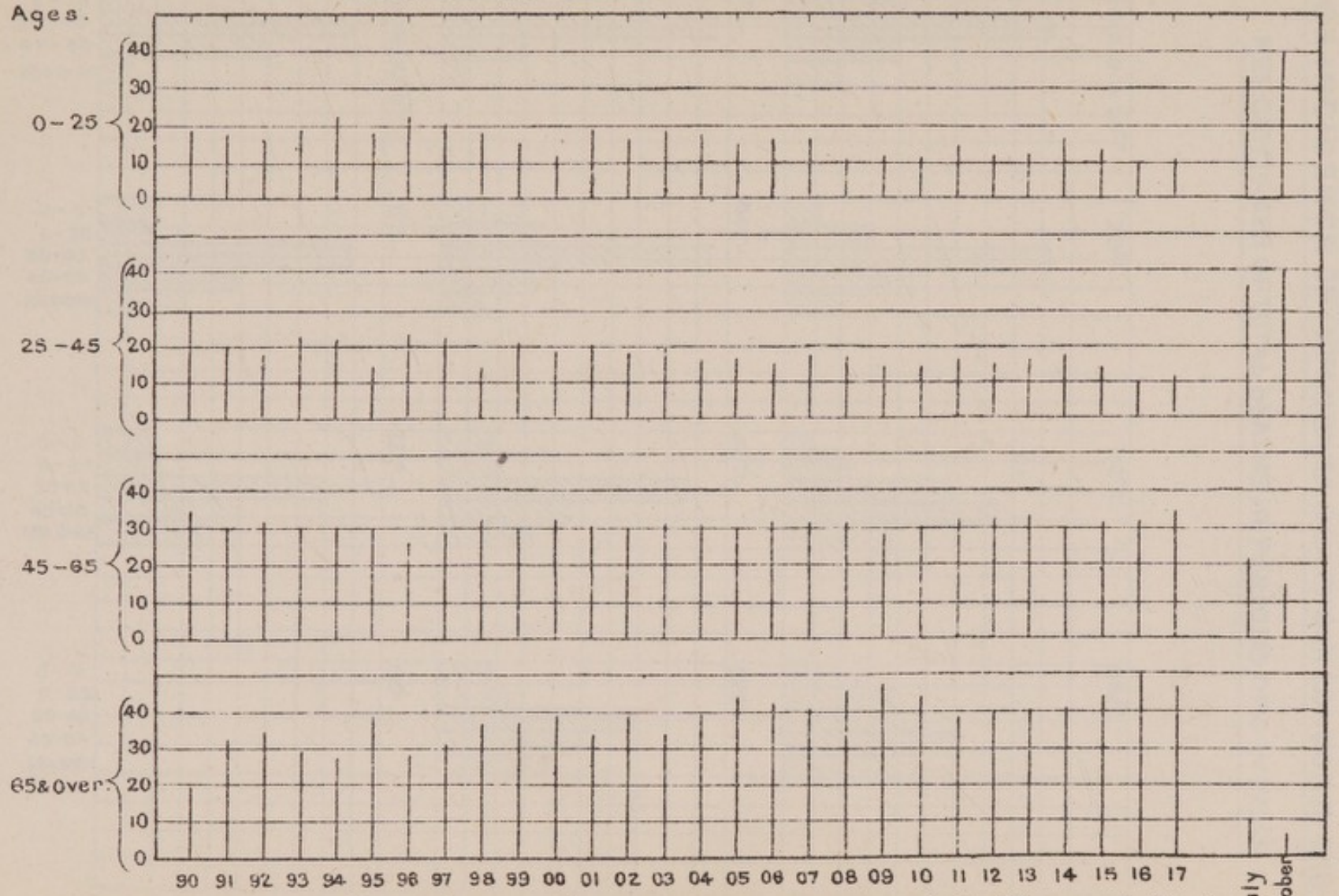
Diagrams 2 and 3 refer to London experience (Table 2, p. 38).

It will be noted that the change is almost abrupt, and that the relatively heavier incidence of death upon the young ages increased up to the crest of the second wave ; thereafter it receded, although the approximation to a pre-1918 proportionality in the third wave never became close.

These statistics refer to deaths only and for the population as a whole, we have no incidence data. We, have, however, such figures for particular samples. Table 3 and Diagram 4 relate to block census enumerations made in various towns.

FROM THE PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE JAN: 1919. DR T.H.C. STEVENSON C.B.E.

DEATHS FROM INFLUENZA IN LONDON 1890 – 1918.



The Sum of the four columns relating to any one year amounts to 100 per cent. The two sets of columns relating to the year 1918 refer to the July & October outbreaks. the latter being covered up to November 2nd only.

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To Face p. 40

DIAGRAM 2.

AGE INCIDENCE OF INFLUENZA DEATHS IN LONDON IN 1918-19.

PERCENTAGES OF TOTAL DEATHS IN PERIODS OF 4 WEEKS.

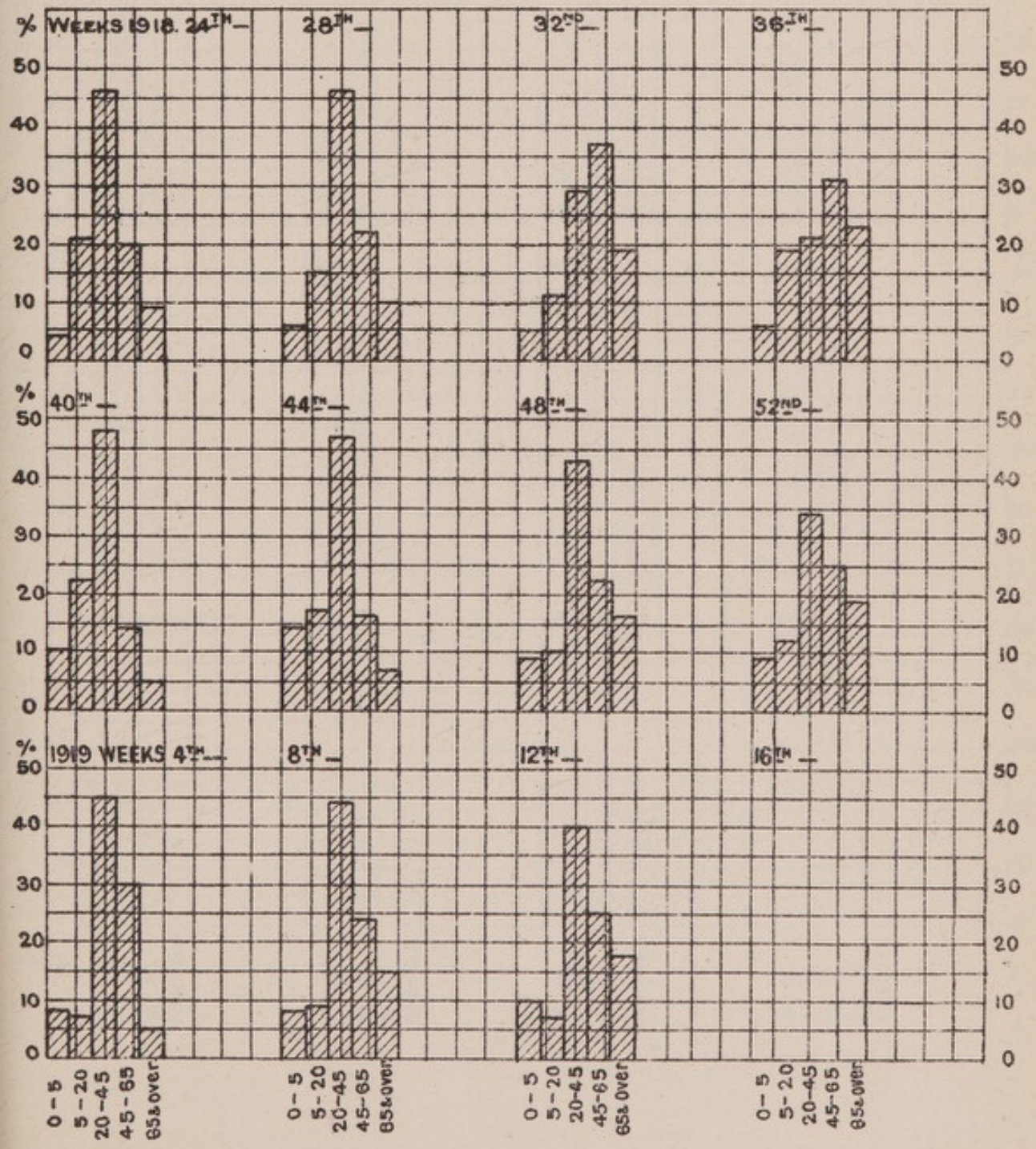

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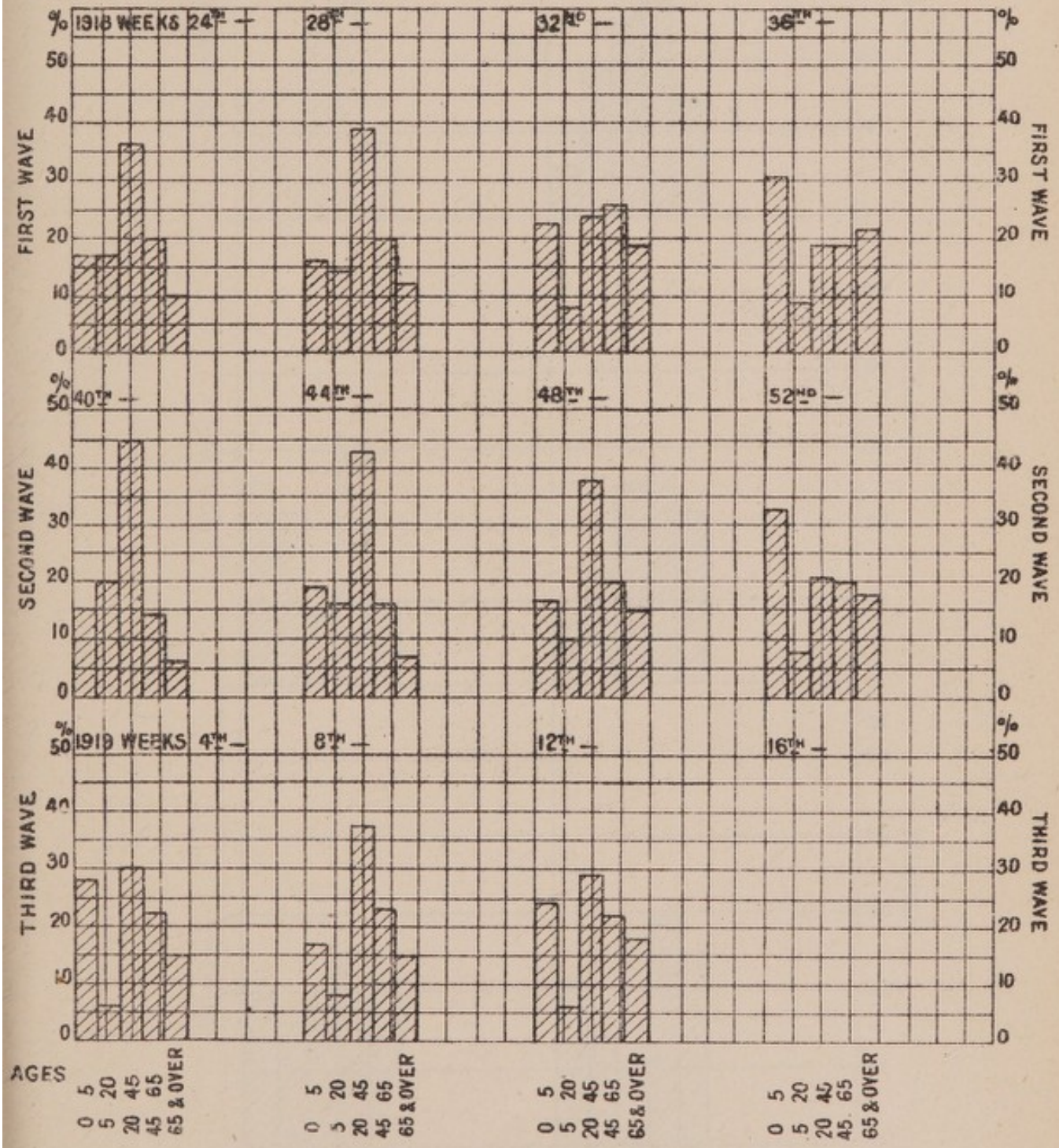
DIAGRAM 3.

AGE INCIDENCE OF INLUENZA & PNEUMONIA DEATHS IN LONDON (JUNE 1918-APRIL 1919)

PERCENTAGES OF TOTAL DEATHS IN PERIODS OF 4 WEEKS.



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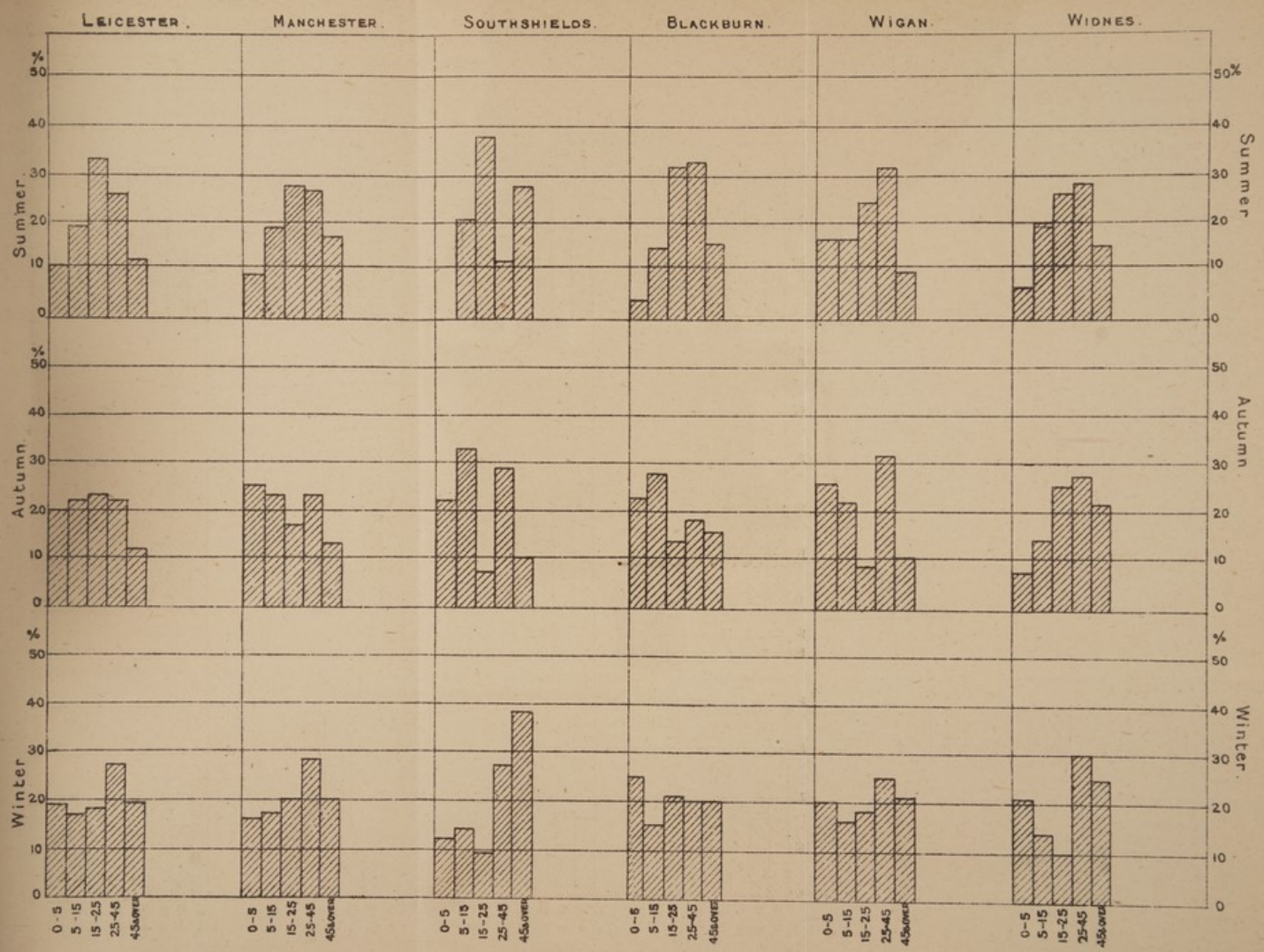
(Chapter 2).

DIAGRAM 4.
AGE INCIDENCE OF INFLUENZA CASES.



The Diagrams show the proportional distribution of cases which would have occurred in each town had equal numbers of persons been exposed to the same risk in each age group.

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TABLE 3.

Age Incidence. Cases per cent. of Population.

Ages.	Leicester.		Manchester.		South Shields.		Blackburn.		Widnes.		Wigan.	
FIRST WAVE.												
	Per cent.		Per cent.		Per cent.		Per cent.		Per cent.		Per cent.	
0–5	3·6	10·56	6·7	9·38	0	0	1·5	3·82	4·1	7·24	3·4	17·00
5–15	6·6	19·35	13·6	19·05	2·9	21·01	5·9	15·00	11·5	20·32	3·3	16·50
15–25	11·2	32·84	19·8	27·73	5·3	38·41	12·7	32·32	15·5	27·39	5·0	25·00
25–45	8·7	25·52	19·1	26·75	1·7	12·32	13·0	33·08	16·2	28·62	6·3	31·50
45 and over	4·0	11·73	12·2	17·09	3·9	28·26	6·2	15·78	9·3	16·43	2·0	10·00
Total	34·1	100·00	71·4	100·00	13·8	100·00	39·3	100·00	56·6	100·00	20·0	100·00
SECOND WAVE.												
0–5	14·7	19·86	12·2	24·60	6·5	21·81	8·3	23·32	3·2	8·20	9·3	25·62
5–15	16·6	22·44	11·3	22·77	9·7	32·55	9·8	27·53	5·9	15·13	8·1	22·38
15–25	17·2	23·24	8·4	16·94	2·1	7·05	5·0	14·04	10·3	26·41	3·3	9·11
25–45	16·4	22·16	11·5	23·19	8·6	28·86	6·9	19·38	11·0	28·21	11·5	31·77
45 and over	9·1	12·30	6·2	12·50	2·9	9·73	5·6	15·73	8·6	22·05	4·0	11·05
Total	74·0	100·00	49·6	100·00	29·8	100·00	35·6	100·00	39·0	100·00	36·2	100·00
THIRD WAVE.												
0–5	7·6	18·67	1·8	15·52	4·3	12·08	9·8	24·56	10·6	20·51	11·0	20·07
5–15	7·0	17·21	2·0	17·23	4·9	13·76	5·9	14·79	7·0	13·54	8·8	16·06
15–25	7·2	17·69	2·3	19·83	3·2	8·99	8·3	20·80	5·4	10·44	10·0	18·25
25–45	11·1	27·27	3·2	27·59	9·5	26·69	8·1	20·30	15·6	30·17	13·5	24·64
45 and over	7·8	19·16	2·3	19·83	13·7	38·48	7·8	19·55	13·1	25·34	11·5	20·98
Total	40·7	100·00	11·6	100·00	35·6	100·00	39·9	100·00	51·7	100·00	54·8	100·00

NOTE.—The first column under each locality gives the observed percentage attack rate. The sum of these entries gives the number of attacks which would have occurred had 100 persons in each age-group been exposed to risk. The second column expresses the rates at ages as percentages of the sum of the first column.

Table 4.

Case Rate per Million of Notified Cases of Influenza, Pneumonia, Bronchitis in Copenhagen.

Week.	Ages.			
	0–5.	5–15.	15–65.	65 and over
1918:				
10th week	1,671	576	604	866
Mar. 11th „	1 892	587	526	1,206
12th „	1,874	670	454	1,082
13th „	1,542	418	282	743
14th „	1,727	418	387	804
Apr. 15th „	1,415	586	381	464
16th „	1,561	575	418	557
17th „	1,746	492	232	959
18th „	2,076	482	264	557
May. 19th „	1,764	545	247	681
20th „	1,525	450	280	619
21st „	2.003	471	288	434
22nd „	1,139	419	238	619
June. 23rd „	1.083	429	230	805
24th „	1,175	273	215	588
25th „	883	209	274	619
26th „	661	220	215	526
27th „	496	219	336	217
July. 28th „	826	796	2,379	433
29th „	2,646	3,081	10,456	1,268
30th „	3,122	6,436	23,348	1,392

Week	Ages.			
	0–5.	5–15.	15–65.	65 and over.
1918:				
31st week	5,566	7,275	15,714	1,052
Aug. 32nd „	2,534	4,695	7,330	866
33rd „	1,561	2,799	3,589	495
34th „	1,046	2,148	1,940	402
35th „	478	912	1,007	248
Sept. 36th „	551	618	554	372
37th „	642	1,205	657	248
38th „	1,322	2,442	1,262	680
39th „	1,616	3,732	1,763	836
Oct. 40th „	3,490	11,047	3,257	804
41st „	13,409	38,573	10,445	1,300
42nd „	21,492	51,781	24,396	2,072
43rd „	22,153	36,110	30,333	1,794
Nov. 44th „	14,989	19,025	21,303	1,947
45th „	8,450	10,072	13,483	1,670
46th „	4,500	5,356	6,837	1,051
47th „	2,682	3,658	4,649	805
Dec. 48th „	2,498	3,741	4,400	804
49th „	3,858	6,855	6,202	959
50th „	4,886	7,756	5,681	1,083
51st „	4,555	7,798	4,777	1,206

Week.	Ages.			
	0–5.	5–15.	15–65.	65 and over.
1918:				
Dec. 52nd wk.	4,409	5,839	5,020	928
1919:				
1st week	5,933	8,563	8,171	1,856
Jan. 2nd „	7,716	10,314	8,511	1,237
3rd „	5,198	7,096	6,659	1,763
4th „	4,592	5,283	4,418	1,052
Feb. 5th „	4,520	4,024	3,682	1,422
6th „	5,034	4,014	3,183	1,607
7th „	6,356	4,560	4,546	1,485
8th „	7,678	5,324	5,745	1,762
Mar. 9th „	6,704	5,682	5,234	2,319
10th „	5,346	3,930	4,816	1,423
11th „	5,015	4,497	4,445	1,855
12th „	3,987	2,725	3,322	1,268
Apr. 13th „	3,674	2,736	3,130	1,670
14th „	3,251	2,715	3,153	1,391
15th „	3,820	2,600	2,972	1,052
May. 16th „	3,232	1,529	1,674	1,392
17th „	3,656	1,583	1,751	1,855
18th „	2,976	1,562	1,524	1,082

(Chapter 2)

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DIAGRAM 5.

NOTIFIED CASES OF INFLUENZA, PNEUMONIA, BRONCHITIS.

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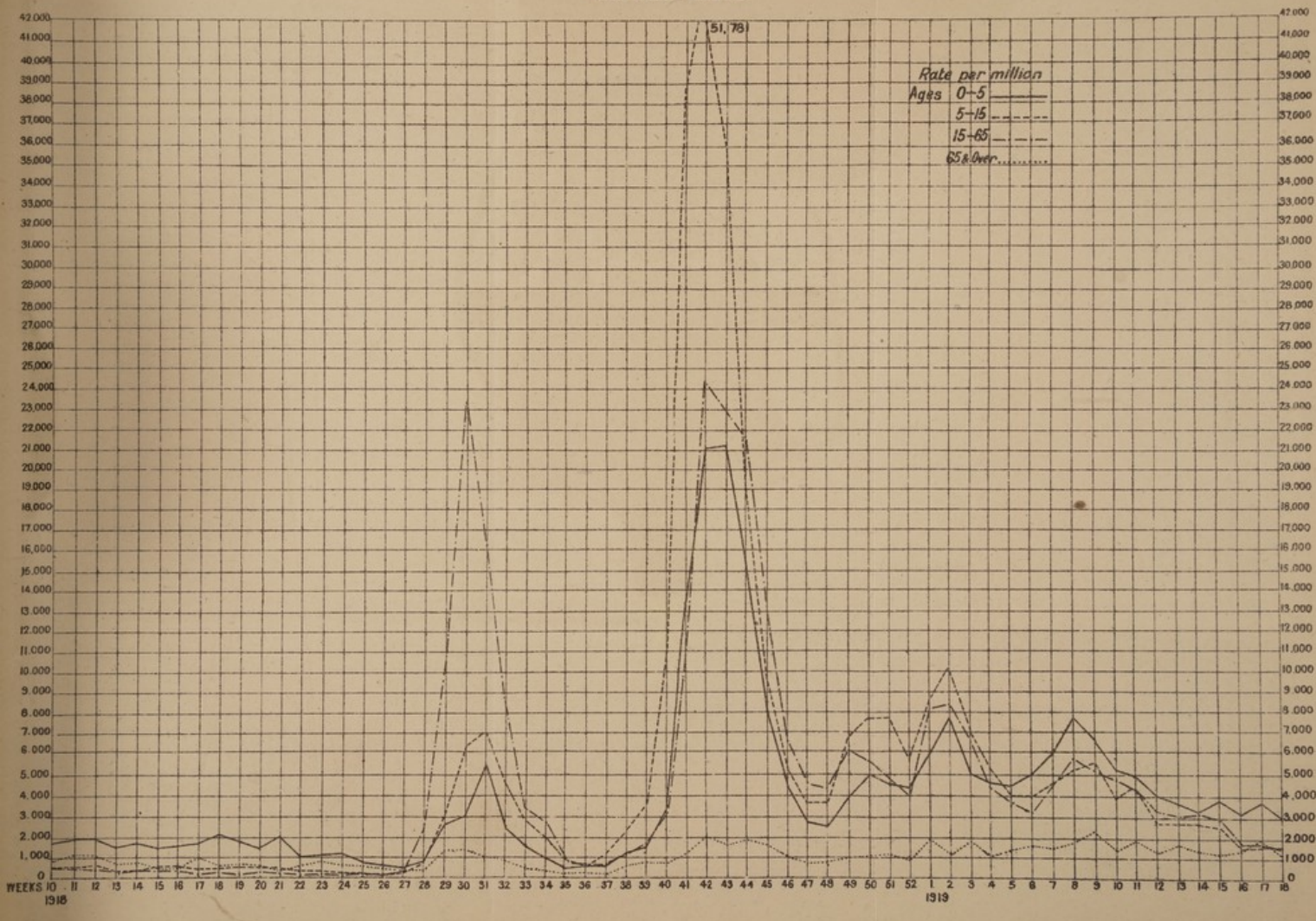

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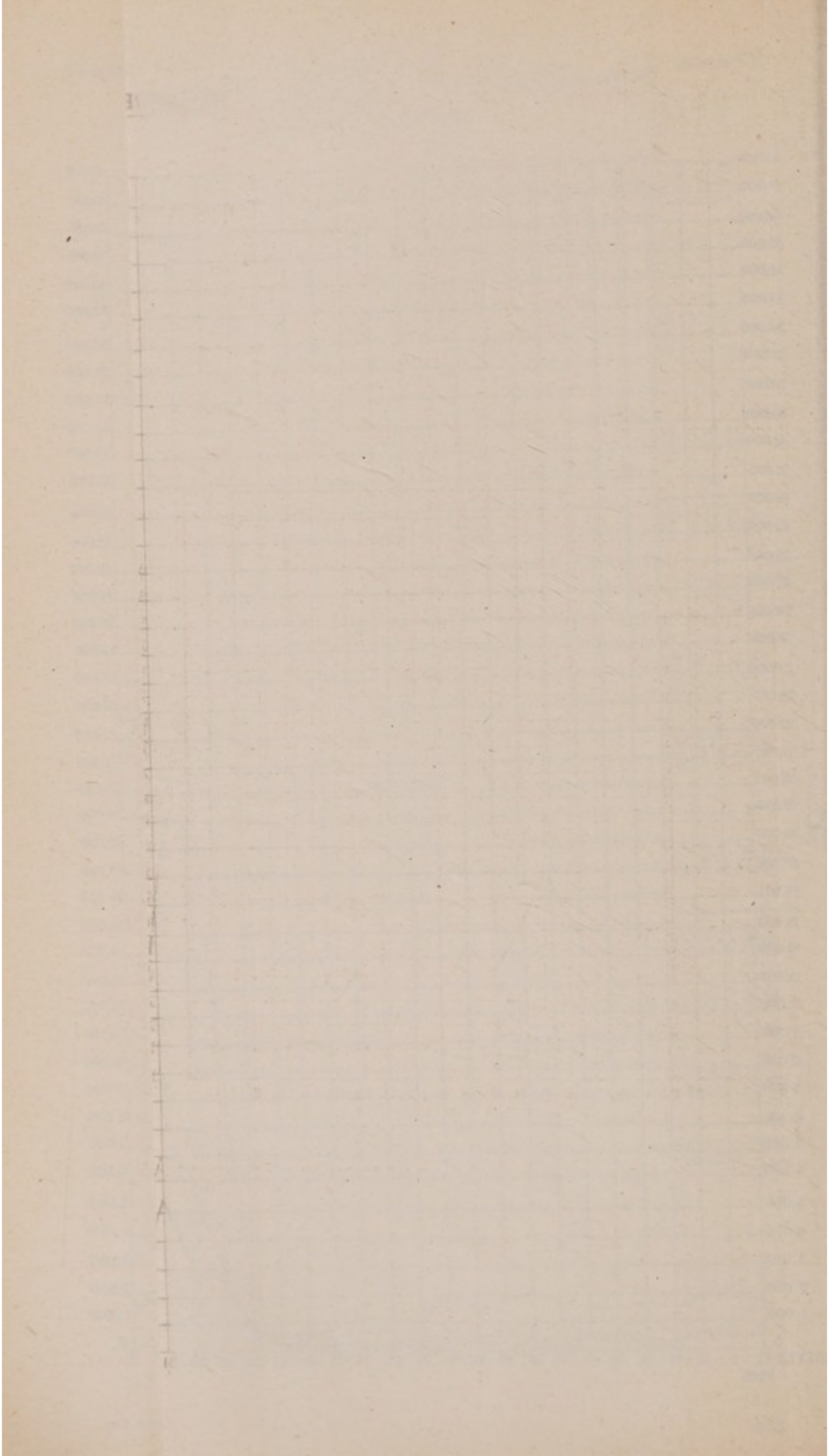

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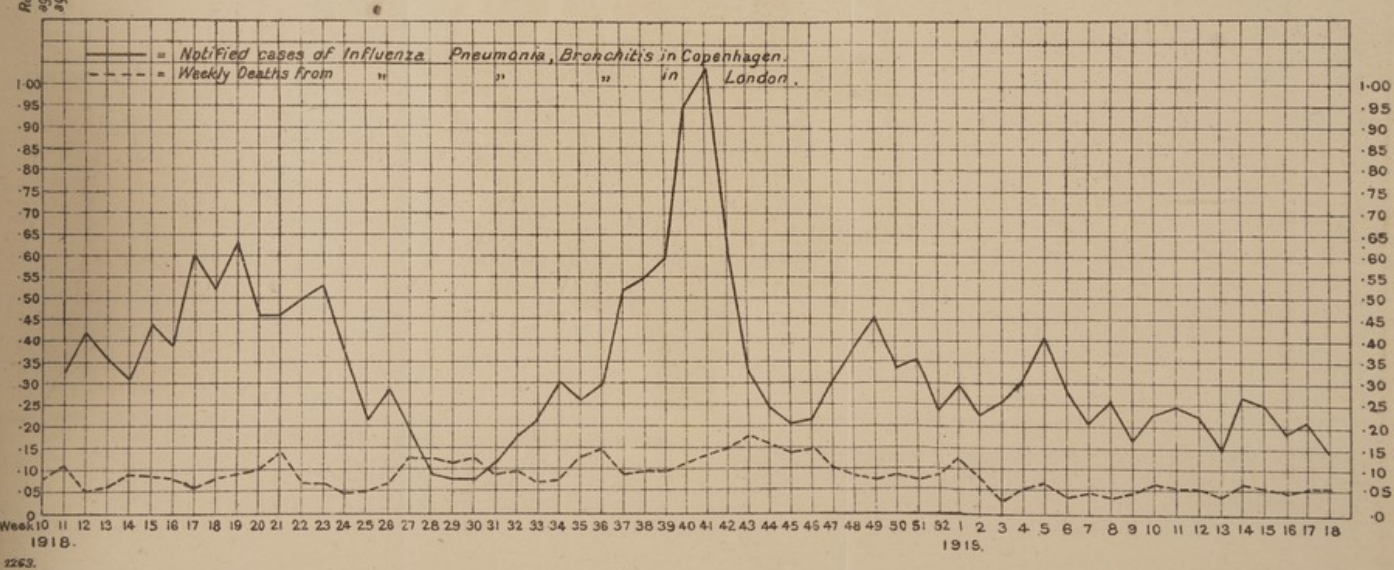
DIAGRAM 6.

RATIO OF AGE GROUPS 5-15 AND 15-65.



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These results are remarkable. They exhibit minor irregularities, dependent upon the fluctuations characteristic of sampling, but, as will be demonstrated in a later chapter, there is effective concordance between the individual returns. In all cases, the autumnal wave of influenza was less felt by those in the prime of life, and more by adolescents than the summer visitation. Since, as the mortality returns show, no such change in the incidence of death occurred; on the contrary, the proportionate toll borne by the adults continued to increase, it may be inferred that the relative fatality of the disease amongst those in the prime of life considerably increased in the autumn epidemic—an inference confirmed by clinical observation.

The change of age incidence between summer and autumn which the block censuses indicate is exhibited in an extreme form by the notification statistics of the city of Copenhagen. A full account of the epidemics as witnessed in foreign countries will be found in other chapters of the report, so that we need not here discuss points of detail. Table 4, p. 42, shows that at Copenhagen the change of age incidence characterising the onset of the summer influenza was as great as here, while Diagrams 5 and 6 bring out the difference between summer and autumn waves. It will be noticed that the goal at which

TABLE 5.

Proportional Distribution of Influenza Notifications in Copenhagen.

Year.	0–5.		5–15.		15–65.		65 and over.		Total.
	Notified Cases.	Per Cent. of Total.	Notified Cases.	Per Cent. of Total.	Notified Cases.	Per Cent. of Total.	Notified Cases.	Per Cent. of Total.	
1889-1900	876	9·04	1,415	14·60	6,892	71·12	508	5·24	9,691
1901–5	388	6·06	716	11·18	4,917	76·80	381	5·95	6,402
1906	88	4·65	182	9·62	1,483	78·42	138	7·30	1,891
1907	486	5·44	820	9.17	7,082	79·23	550	6·15	8,938
1908	544	5·07	937	8·73	8,611	80·19	645	6·01	10,737
1909	230	4·96	465	10·02	3,667	79·01	279	6·01	4,641
1910	319	6·71	463	9·74	3,678	77·38	293	6·16	4,753
1911	255	4·93	672	13·00	3,926	76·15	306	5·92	5,169
1912	407	7·06	706	12·24	4,353	75·49	302	5·24	5,768
1913	196	7·19	256	9·39	2,111	77·47	162	5·94	2,725
1914	235	6·14	430	11·24	2,964	77·15	198	5·17	3,827
1915	420	5·57	894	11·86	5,798	76·95	423	5·61	7,535
1916	565	6·02	1,087	11·58	7,201	76·70	535	5·70	9,388
1917	368	5·40	707	10·38	5,331	78·29	403	5·92	6,809
1918, July to August.	824	3·28	2,603	10·35	21,561	85·75	157	0·62	25,145
Sept. to Dec.	5,076	7·94	16,316	25·52	42,218	66·03	330	0·52	64,940
1919, Jan. to April.	2,821	8·90	6,239	19·68	22,329	70·45	308	0·97	31,697
July 1918 to April 1919.	8,721	7·22	25,158	20·83	86,108	71·29	795	0·66	120,782

the English town samples seem to have been aiming in this matter of age incidence was reached and passed in Copenhagen. The incidence of influenza upon adolescents in the autumn was not only relatively, but, for a time, absolutely heavier than upon the adults. Table 5 compares 1918, with earlier Danish experience. We are authorised to regard this variation as an important epidemiological fact. Qualitatively similar variations of age incidence in successive epidemics have often been recorded in history; a well-known historical instance is the *pestis puerorum* of 1361 coming after the Great Death of 1349 and destroying many of the young children spared by the greater plague. The customary explanation is that the survivors of the first in series of a set of epidemics are immune, so that if the first selectively attacked a particular age group, the successor must spare survivors of that group. This explanation is not wholly satisfactory. It will be proved in a later section that the degree of immunity conferred by passage through an attack of influenza has never been complete and has varied from locality to locality in a way which may be significant. In particular it will appear that in towns, such as Leicester and Manchester, each of which exhibits the change of age incidence, the amount of protection conferred by a first attack of disease has been ostensibly different. Immunisation of age groupings is not then a completely satisfactory explanation. This finding compels us also to reconsider the hypothesis naturally tendered to describe the almost abrupt change of age incidence which characterised the first wave of influenza in contrast with previous experience. That change would be naturally supposed to mark the importation into the country of some new strain of organism, or of a germ not before domiciled here. Indeed no other evidence in support of introduction *ab extra* can be found. A study of the distribution of deaths in England and Wales attributed to influenza during the first weeks of the summer pandemic brings out the existence of cases in the north eastern ports but none in the western ports (the natural entrance of infection from the new world) or in the southern ports (excepting London) receiving travellers from Europe. The distribution in fact corresponds to that of population, the greatest number of deaths being returned from London and the North Midland manufacturing districts. Of course this is a merely negative result, not excluding importation—since the rapidity of inland transit even in war time is such that, with a mild disease of short incubation period, the records of deaths in early weeks may give an incorrect picture of the lines of spread—but it *is* negative and the abrupt change of age incidence in July remains our best evidence in favour of a theory of importation. That both here and in Europe this age incidence again changed, and that the change is not fully explicable as an immunising phenomenon, appreciably weakens the case for importation, since what has demonstrably happened

within a country during an epidemic period might well have taken place before. Recognition of this fact while not lifting the veil of mystery since we are still to learn why, if the disease were *not* imported, it changed when it did change, and also why an identical change occurred in many different countries, emphasises the need of caution in framing hypotheses.

Some further consideration of the epidemiological possibilities will be reserved for a later section of the report.

Returning to the general statistics of England and Wales it is pertinent to inquire whether any general distinction of mortality between the successive waves pointing to some interconnection, whether due to immunity or not, is discernable.

TABLE 6.

Deaths per 1,000 *from Influenza in* 111 *London and County Boroughs in Summer, Autumn, and Winter*, 1918–19.

	Death Rate per 1,000 from Influenza Epidemics in—				Mean Standard Death Rate per 1,000 in 1911–12–13–14.
	London and County Boroughs.	Summer.	Autumn.	Winter.	
29 London Boroughs.	City of London	0·76	10·08	2·27	15·05
	Battersea	0·96	15·11	4·07	13·65
	Bermondsey	1·04	14·80	3·66	17·78
	Bethnal Green	1·63	14·55	2·91	16·93
	Camberwell	1·17	13·31	3·10	13·60
	Chelsea	0·78	14·13	5·87	13·90
	Deptford	0·59	13·29	2·40	14·70
	Finsbury	1·27	14·54	3·08	18·88
	Fulham	1·20	12·53	3·33	13·90
	Greenwich	1·47	13·78	3·17	13·93
	Hackney	0·94	13·58	2·85	13·65
	Hammersmith	0·66	9·45	3·22	14·13
	Hampstead	0·22	8·26	4·36	11·05
	Holborn	1·09	13·64	4·69	15·83
	Islington	0·90	11·95	4·23	14·63
	Kensington	0·54	7·90	3·21	13·60
	Lambeth	1·06	11·62	3·64	14·15
	Lewisham	0·37	10·65	3·01	10·80
	Paddington	0·82	11·09	4·71	13·20
	Poplar	0·92	14·34	3·22	17·13
	St. Marylebone	0·98	10·79	4·64	14·78
	St. Pancras	1·35	15·00	4·98	15·40
	Shoreditch	0·92	12·40	4·35	19·68
	Southwark	1·09	13·15	3·39	17·88
	Stepney	1·23	11·27	2·66	16·78
	Stoke Newington	1·18	10·16	3·72	12·53
	Wandsworth	0·60	9·52	3·76	11·20
	Westminster	0·92	10·80	5·97	13·53
	Woolwich	0·81	12·86	3·16	13·15

	London and County Boroughs.	Death Rate per 1,000 from Influenza Epidemics in— Summer.	Autumn.	Winter.	Mean Standard Death Rate per 1,000 in 1911-12-13-14.
25 Midland County Boroughs.	Birmingham	1·46	8·47	3·94	15·88
	Bristol	0·95	11·32	4·40	13·53
	Burton-on-Trent	0·95	19·51	2·76	13·20
	Coventry	0·77	13·02	0·74	12·98
	Derby	1·22	11·54	3·94	13·08
	Dudley	1·78	8·60	1·94	16·10
	East Ham	1·16	14·20	2·28	11·48
	Gloucester	1·36	12·73	4·41	13·18
	Great Yarmouth	1·06	13·31	5·86	13·35
	Grimsby	1·47	11·09	2·60	14·88
	Ipswich	0·50	10·38	3·81	13·10
	Leicester	1·52	16·10	5·73	14·03
	Lincoln	0·53	11·47	3·47	12·85
	Northampton	0·40	10·09	2·32	13·23
	Norwich	0·22	11·71	2·92	12·80
	Nottingham	1·26	18·55	3·47	15·28
	Oxford	0·89	17·34	1·94	11·40
	Smethwick	1·83	10·87	5·14	14·45
	Southend-on-Sea	1·02	8·74	4·03	10·93
	Stoke-on-Trent	2·50	12·42	7·31	19·33
	Walsall	2·93	14·00	2·88	16·50
	West Bromwich	2·98	17·94	6·02	16·30
	West Ham	1·46	14·56	3·19	15·35
	Worcester	0·90	13·22	2·97	13·30
	Wolverhampton	1·22	15·26	7·32	15·53
41 Northern County Boroughs.	Barnsley	2·88	16·24	9·39	18·1
	Barrow-in-Furness	1·30	9·95	4·80	15·1
	Birkenhead	1·28	12·51	4·84	16·0
	Blackburn	2·30	9·68	6·40	16·7
	Blackpool	1·11	6·70	4·86	14·4
	Bolton	2·52	6·87	6·94	16·5
	Bootle	1·36	12·28	6·59	18·4
	Bradford	1·58	9·51	8·71	16·0
	Burnley	2·78	8·31	3·93	18·4
	Bury	2·52	8·33	9·12	15·6
	Carlisle	2·09	6·12	2·39	16·4
	Chester	2·08	8·10	3·11	14·9
	Darlington	2·66	8·38	6·19	14·0
	Dewsbury	2·47	13·12	6·47	17·5
	Gateshead	1·81	7·02	5·13	17·6
	Halifax	1·33	9·91	4·12	15·6
	Huddersfield	2·78	4·38	6·28	15·4
	Kingston-upon-Hull	1·46	14·89	4·78	15·5
	Leeds	1·53	11·42	5·23	16·3
	Liverpool	0·90	7·42	5·52	19·8
	Manchester	1·96	10·11	5·63	17·9
	Middlesbrough	2·53	9·98	4·85	20·3
	Newcastle-on-Tyne	1·83	8·44	8·75	16·7
	Oldham	3·01	10·84	4·00	18·7

	London and County Boroughs.	Death Rate per 1,000 from Influenza Epidemics in— Summer.	Autumn.	Winter.	Mean Standard Death Rate per 1,000 in 1911-12-13-14.
Northern County Boroughs—*cont.*	Preston	2·92	11·56	5·46	17·7
	Rochdale	4·32	10·90	3·73	16·6
	Rotherham	0·93	17·37	4·73	16·4
	St. Helens	1·86	11·64	7·92	18·9
	Salford	2·55	8·01	7·80	18·3
	Sheffield	1·69	16·69	3·39	16·5
	Southport	0·94	9·26	7·62	12·6
	South Shields	1·86	16·14	6·75	17·9
	Stockport	1·84	10·56	4·00	16·3
	Sunderland	2·37	16·00	5·51	17·5
	Tynemouth	1·22	17·55	7·41	16·4
	Wakefield	2·39	7·98	10·28	15·3
	Wallasey	0·64	9·46	6·74	12·9
	Warrington	3·10	12·44	7·59	17·1
	West Hartlepool	1·83	14·36	6·20	16·4
	Wigan	2·35	9·08	7·63	19·8
	York	1·04	10·77	2·75	13·3
12 Southern County Boroughs.	Bath	0·51	8·77	2·48	11·3
	Bournemouth	0·54	5·13	3·89	9·9
	Brighton	0·66	11·34	5·11	12·6
	Canterbury	0·36	12·90	5·19	11·6
	Croydon	0·74	10·62	2·67	11·1
	Eastbourne	0·65	6·86	5·00	10·2
	Exeter	0·40	10·29	4·79	13·5
	Hastings	0·23	10·73	3·55	11·4
	Plymouth	1·18	9·54	2·48	15·1
	Portsmouth	0·73	12·32	4·10	13·2
	Reading	0·83	10·52	3·70	11·3
	Southampton	0·70	11·15	3·85	13·4
4 Welsh County Boroughs.	Newport	1·48	5·22	5·32	14·1
	Cardiff	1·07	10·32	3·95	14·9
	Merthyr Tydfil	2·00	6·88	3·16	16·2
	Swansea	0·80	10·54	4·60	15·9

This is a subject which can be less fallaciously appraised when incidence rates are available but the wider area covered by mortality returns merits some survey. In Table 6 are contained averages obtained from the data of county and metropolitan boroughs. The gross correlation of rates in successive waves is not sufficient, it is necessary to take account of the normal variations of public health in the various places and four variables have therefore been handled. (1) Standardised death rate for all causes in 1911–14 used as a criterion of normal mortality, (2) Influenza death rate in third quarter

of 1918, (3) Influenza death rate in fourth quarter of 1918, (4) Influenza death rate in first quarter of 1919.

TABLE 7.

Correlation between the Mean Standard Death Rates for 1911–14, *and the Death Rates in the Summer, Autumn, and Winter Epidemics of Influenza.* (*Deaths per* 1,000.) 29 *London Boroughs and* 82 *County Boroughs.*

Variables (the other two Variables remaining Constant).	London and County Boroughs. (111.)	County Boroughs alone. (82.)	London Boroughs alone. (29.)
	Correlations and Probable Errors.		
Summer Death Rate and Mean Standard Death Rate.	+·5700±·043	+·6102±·047	+·2407±·120
Summer Death Rate and Autumn Death Rate.	−·1872±·062	−·1798±·072	+·2479±·118
Summer Death Rate and Winter Death Rate.	+·2328±·061	+·1489±·073	−·1705±·122
Autumn Death Rate and Mean Standard Death Rate.	+·2325±·061	+·1836±·072	+·3927±·106
Autumn Death Rate and Winter Death Rate.	−·0926±·063	−·0625±·074	+·0980±·124
Winter Death Rate and Mean Standard Death Rate.	+·1689±·062	+·2452±·070	−·0996±·124

In Table 7 are entered the partial correlations of the variables, both for the whole collection of county and metropolitan boroughs and for the county and metropolitan boroughs separately. From the point of view of geographical study the second method is better, since with the former the weight attaching to the 29 metropolitan boroughs exaggerates the influence of London upon the deduced averages. The coefficients of correlation are those determined by the ordinary method of multiple correlation from grouped data. Thus the first correlation in the table is that between the summer death rate (crude) from influenza and an average pre-war standardised death rate, the autumnal and winter influenzal death rates being made constant. Having regard to the various probable errors, the coefficients determined from the whole 111 observations do not differ appreciably from those derived after exclusion of the metropolitan boroughs.

The first point to notice is that the pre-war standardised death rate is substantially correlated with the influenzal death rate of the summer quarter, less closely associated with the influenzal death rates of the subsequent quarters. Professor

Raymond Pearl, of Baltimore, has found a substantial correlation between the influenzal death rate of 1918 in American cities and the general death rates in those cities for 1916;* the actual figures, owing to differences of procedure, are not comparable; but Professor Pearl's remark, that "an essential "factor in determining the degree of explosiveness of the "outbreak of epidemic influenza in a particular city were the "normal mortality conditions prevailing in that city," is compatible with our result.

The next point is the negative correlation between the summer and autumn influenzal death rates, the positive correlation between the summer and winter death rates from influenza, and the absence of any correlation between the autumn and winter influenzal death rates. A facile and possibly correct explanation is that the summer epidemic conferred immunity against the autumn wave but not against the winter form. We shall hereafter show that there is evidence of personal immunity acquired by passage through a summer attack against attack in the autumn and less evidence that the autumn or summer disease conferred protection against the winter epidemic wave. But this is rather too facile We may find nothing shocking in the idea that the summer disease produced some immunity, but it is going far to postulate that it actually lowered resistance to the winter influenza, and, before accepting this, we must consider whether there is any simpler arithmetical interpretation. The first suggestion is that our correlations only reflect differences of age incidence. The influenza death rates are merely crude rates, and the age distribution of deaths was different in the winter from that of summer or autumn. Hence, a population favourably constituted by age for summer attack was unfavourably constituted for the winter disease. But this should lead to a negative correlation, and the correlation is positive. Another interpretation is that the correlations only partly depend upon any phenomenon of immunity, and merely express, so far as the winter and summer correlation is concerned, that local circumstances, other than those measured by the pre-war standard death rate, which favoured one outburst of influenza, also favoured the other. If this be the explanation, we must suppose that, in the autumn, some other factor was involved of sufficient potency to swing the correlation over from positive to negative. That factor may well have been acquired immunity. We can then read our riddle as follows:—Ordinarily there is a decided correlation between the factors, other than such general hygienic conditions as are measured by a

* "*Influenza Studies* 1.—On certain general Statistical Aspects of the 1918 Epidemic in American Cities." Raymond Pearl. (Reprint No. 548 U.S.A. Public Health Reports, August 1919.)

standardised death rate, which favour the explosion of epidemic influenza; hence there is usually a positive correlation between the death rates of influenza in successive epidemics. But this correlation may be modified by the acquirement of immunity. Between the autumn and summer waves there was sufficient biological kinship for a definite degree of immunity to be attained. Hence, in this combination, and in this alone, the correlation is negative, but, not being significantly positive as between autumn and winter, there, too, some immunity existed. Such appears to be the simplest interpretation of our results. But, although we have thought proper to analyse the statistics by this method, we do not attach, and the reader should not attach, great importance to the analytical results.* In an ideal statistical universe, the method of multiple correlation provides an almost perfect instrument for the appraisement of the relative importance of various factors of a complex result. *Our* universe is not ideal. Not to speak of irregular errors in the influenzal rates due to faulty nomenclature and the technical difficulty mentioned in the footnote, the pre-war standard death rate is an imperfect measure of the sanitary status of a county borough in 1918, while the change of age incidence to which we have adverted seriously impairs the value of the crude influenza death rate as a measure of epidemicity. As an instrument of prediction (by the formation of a regression equation) we have not found the arithmetical results of sufficient value for it to be worth reproducing them in this place.

Course of the Epidemic in Scotland.

The statistics of the epidemic in Scotland have been reported by Dr. Dunlop. Diagram 7 and Table 8 exhibit some of the main results. The broad features of the pandemic in North Britain are identical with those of England and Wales; the relatively light death roll of the first waves, the heavy mortality

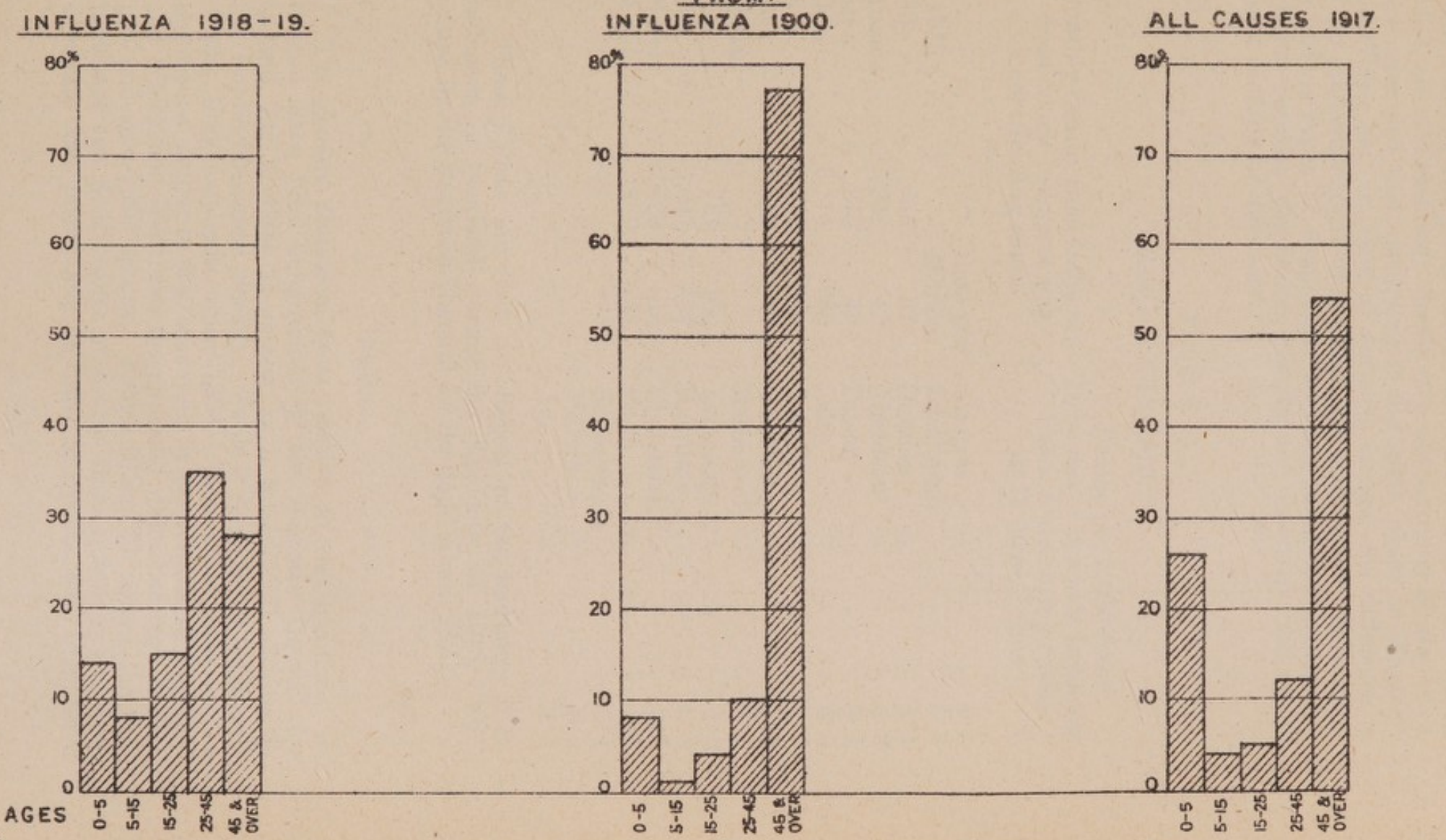
* As an additional warning, we may note that an analysis of the 29 metropolitan boroughs leads to values of the coefficients of partial correlation different from those obtained either for the whole of the data or for the county boroughs alone. In one case—that of the correlation between summer and autumn influenzal death rates for standard death rate and winter influenzal rate constant—the difference is significant, the London value being *positive*, viz., $\cdot 428 \pm \cdot 138$. Such a result, while, perhaps, evidence of some geographical peculiarity or, again, of a failure to allocate institutional deaths satisfactorily, necessarily raises in the mind of a statistician suspicion as to the meaning of any of the coefficients. It is well known that if we mix two records in each of which the variables are quite independent one of another, but their mean values are not the same in the two records, then *the mixture* will display an arithmetical correlation which has no physical significance; is, in fact, spurious. The operation of this principle might seriously vitiate the interpretation of any results deduced.

DIAGRAM. 7

AGE DISTRIBUTION OF DEATHS IN SCOTLAND

(PERCENTAGE OF THE TOTALS)

FROM:-



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of the autumn, and the excessive toll upon adults in the prime of life, are all exhibited. Relatively, the mortality of the third wave exceeded that attributed to the same phase in England and Wales, but, in view of seasonal factors, the difference is of no epidemiological importance. A scrutiny of death rates in various types of community does not bring out any sign of relation between population, density, and mortality rate which could usefully be discussed.

TABLE 8.

Scotland 1918–1919.

Monthly Deaths from Influenza or in which Influenza was a contributing cause.			Deaths per 1,000 from Influenza or in which Influenza was a contributing cause in 16 large Towns.	
		Rate per Million.		
1918			Glasgow	4·1
July	768	157	Edinburgh	5·3
August	232	47	Dundee	4·1
Sept.	240	49	Aberdeen	3·2
Oct.	3,714	760	Paisley	3·4
Nov.	4,272	874	Leith	4·9
Dec.	1,569	321	Greenock	3·0
1919			Coatbridge	5·3
Jan.	781	160	Motherwell	3·1
Feb.	3,041	622	Kirkcaldy	4·2
March	2,529	518	Hamilton	2·8
April	429	88	Clydebank	3·7
			Perth	4·0
			Kilmarnock	3·1
			Falkirk	5·0
			Ayr	2·7

These figures are from a "Report on the mortality from Influenza in Scotland during the Epidemic of 1918–1919" issued by the Registrar-General for Scotland [Cmd. 282.]

IRELAND.

The general statistical facts relative to the pandemic in Ireland have been summarised by the Registrar-General for Ireland, Sir William Thompson, in a presidential address to the Dublin Statistical Society. Table 9 records the registered deaths from influenza since 1864. This table produces an impression similar to that conveyed by the returns of England and Wales, and illustrates the profound difference between the pre- and post-1889 epochs. Tables 10 and 11 record the proportional age distribution of deaths from influenza and from influenza and pneumonia at different epochs, and Diagram 8

has been prepared. It will be seen that, just as in England and Wales, the brunt of the 1918 pandemic was borne by the young adult population.

TABLE 9.

Deaths from Influenza in Ireland.

Year.	Number.	Rate per 100,000 of Population.	Year.	Number.	Rate per 100,000 of Population.
1864	311	6	1892	3,742	81
1865	220	4	1893	1,310	28
1866	189	3	1894	2,032	44
1867	164	3	1895	1,896	42
1868	96	2	1896	729	16
1869	180	3	1897	1,535	34
1870	166	3	1898	2,277	50
1871	105	2	1899	1,716	38
1872	132	3	1900	4,677	105
1873	100	2	1901	1,381	31
1874	100	2	1902	1,258	28
1875	124	2	1903	1,553	35
1876	121	2	1904	1,184	27
1877	93	2	1905	1,219	28
1878	54	1	1906	978	22
1879	74	1	1907	1,720	39
1880	69	1	1908	1,798	41
1881	54	1	1909	1,303	30
1882	39	1	1910	1,332	30
1883	87	2	1911	992	23
1884	43	1	1912	1,025	23
1885	36	1	1913	1,174	27
1886	34	1	1914	696	16
1887	26	1	1915	1,568	36
1888	44	1	1916	1,014	23
1889	21	0	1917	1,438	33
1890	1,712	36	1918	10,651	243
1891	891	19			

By the courtesy of Dr. E. C. Bigger, we have been supplied with a précis of the reports of the medical inspectors of the Irish Local Government Board, and the following points emerge :—

In Dublin and the south-east, the second wave was the most fatal : 1,814 deaths were registered in the Dublin district during the three months ending 31st December 1918, the maximum of any week being 383, in the week ending 26th October. The total for the quarter ending 31st March 1919 was 665 ; the maximum, 153, in the week ending 15th March. The immediate cause of death was usually septic pneumonia.

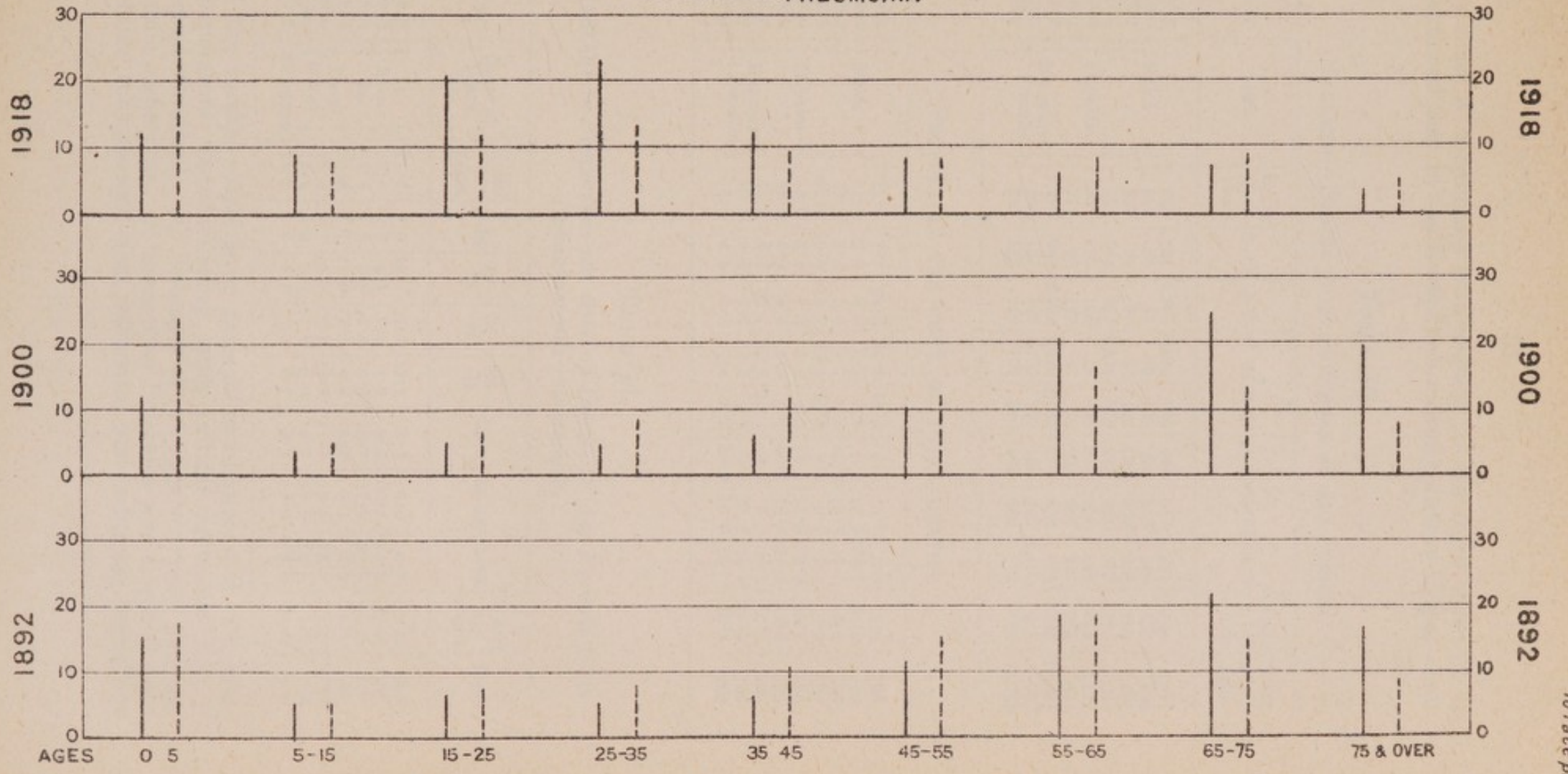
In Belfast and the north-east a similar condition prevailed. Points of special interest noted in this division are : (1) The

(Chapter 2)

DIAGRAM 8.

—IRELAND—

PROPORTION OF DEATHS AT AGES FROM INFLUENZA AND PNEUMONIA IN 1918, 1900, 1892.



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frequency of meningitis as a sequela in the third wave (it is mentioned that in the North-Irish military district 70 per cent.

Table 10.

Ireland—*Deaths from Influenza at Age Periods.*

Year.	Sex.	Under Five Years.	5–15	15–25	25–35	35–45	45–55	55–65	65–75	75 and Upwards.	Total all Ages.
1918	Males	625	431	1,205	1,390	706	448	317	321	148	5,591
1900	Males	257	64	86	92	122	202	418	528	437	2,206
1892	Males	306	75	97	86	112	198	305	358	294	1,831
1918	Females	621	533	997	1,032	579	427	321	373	177	5,060
1900	Females	238	69	106	78	103	257	534	622	464	2,471
1892	Females	241	93	122	85	98	204	357	417	294	1,911
1918	Males and Females.	1,246	964	2,202	2,422	1,285	875	638	694	325	10,651
1900	Males and Females.	495	133	192	170	225	459	952	1,150	901	4,677
1892	Males and Females.	547	168	219	171	210	402	662	775	588	3,742

PROPORTION PER CENT. OF TOTAL DEATHS.

1918	Males	11·2	7·7	21·6	24·8	12·6	8·0	5·7	5·8	2·6	100
1900	Males	11·8	2·8	3·9	4·2	5·5	9·2	18·9	23·9	19·8	100
1892	Males	16·7	4·1	5·3	4·7	6·1	10·8	16·7	19·6	16·0	100
1918	Females	12·3	10·5	19·7	20·4	11·4	8·4	6·4	7·4	3·5	100
1900	Females	9·6	2·8	4·3	3·2	4·2	10·4	21·6	25·2	18·7	100
1892	Females	12·5	4·9	6·4	4·5	5·1	10·7	18·7	21·8	15·4	100
1918	Males and Females.	11·8	9·0	20·7	22·7	12·1	8·2	6·0	6·5	3·0	100
1900	Males and Females.	10·6	2·8	4·1	3·6	4·8	9·8	20·4	24·6	19·3	100
1892	Males and Females.	14·6	4·5	5·8	4·6	5·6	10·7	17·7	20·7	15·8	100

Table 11.

Ireland.—*Proportion per cent. of Deaths at Age Periods from Influenza and Pneumonia.*

Year.	Cause of Death.	Under 5 Years.	5–15.	15–25.	25–35.	35–45.	45–55.	55–65.	65–75.	75 and upwards.	Total.
1918	Influenza.	11·8	9·0	20·7	22·7	12·1	8·2	6·0	6·5	3·0	100
1900	Influenza.	10·6	2·8	4·1	3·6	4·8	9·8	20·4	24·6	19·3	100
1892	Influenza.	14·6	4·5	5·8	4·6	5·6	10·7	17·7	20·7	15·8	100
1918	Pneumonia.	28·8	7·1	11·6	13·0	8·7	8·4	8·1	9·2	5·1	100
1900	Pneumonia.	23·5	4·1	5·7	7·8	10·8	11·7	16·4	12·8	7·2	100
1892	Pneumonia.	17·4	5·3	6·7	7·0	9·9	14·2	17·9	13·8	7·8	100
1917	All causes	15·8	3·4	5·5	5·6	6·7	8·2	11·0	19·6	24·2	100

of the cerebro spinal fever cases followed attacks of influenza, temperature charts showing, after critical influenzal fall, a second elevation). (2) Instances of infection conveyed through public gatherings. A dance near Coleraine was attended by a soldier convalescent from influenza, and influenza broke out

among the 60 guests, several of whom died. (3) It is stated that a first attack conferred immunity, but no statistical evidence is afforded.

In the Cork and South Ireland district, while septic pneumonia, as elsewhere, was the usual complication, enteritis was frequently observed, and led to confusion with enteric. A widespread outbreak of this type was studied in the Ardfert dispensary district of the Tralee union. The cases all presented many features common to enteric—high temperature, foul tongue, sordes, abdominal distention, diarrhœa with stools of the characteristic "pea soup" appearance. The fever usually lasted from one to three weeks, but the Widal reaction was invariably negative.

In the Derry and north-western district, the disease followed the customary course.

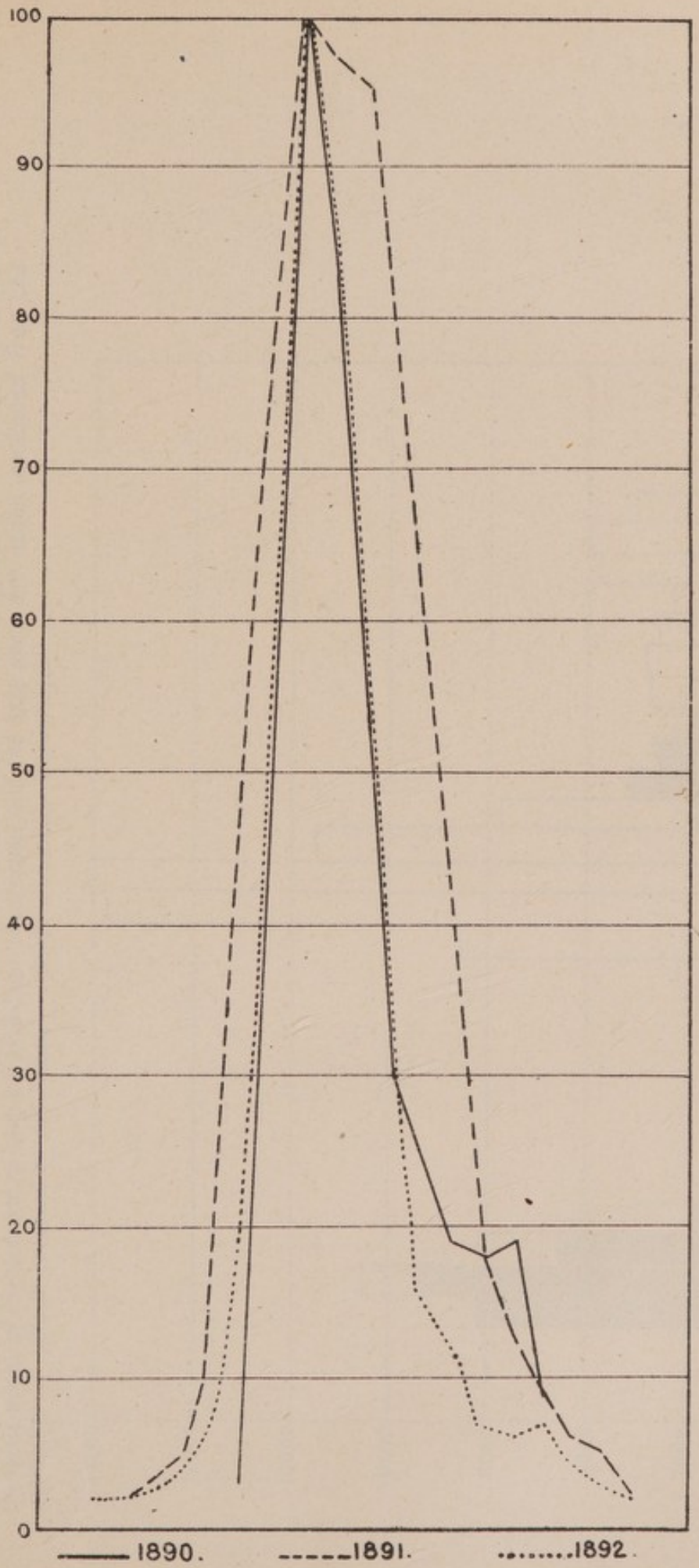
It will be seen that the Irish influenza did not present, either in respect of prevalence or severity, any uniform variation from the type dominant in England and Wales; this is a matter of some epidemiological importance, since, from the point of view of material well being, the reaction of the war upon the Irish population was less prejudicial than in other divisions of the United Kingdom, and both the age and sex constitution and housing aggregation were not forced so far from the normal equilibrium. Naturally this does not exclude the war factor as perhaps a principal one in the development of the disease, but it may be held to show that, given the needed preliminaries, the subsequent evolution of a pandemic will occur under a very wide range of environments.

The Form of the Epidemic Waves.

It has been remarked before that the course of subsequent epidemics after the re-appearance of pandemic influenza in explosive form has varied from the type manifested in the first phase. The accompanying Diagram 9 illustrates this in the instance of the three first waves of the 1889–92 period (deaths), the second being somewhat flatter and remaining above the endemic level for a longer period. The same character has been manifested in the late prevalences, both in the Air Force experience and that of the British Army; the October prevalence was longer drawn out than that of the summer. When the data are plotted as ordinates, the tops being joined by straight lines, as in Diagram 9, the impression is produced of almost perfect symmetry in the primary explosive influenzas of the summer and of a decided asymmetry in the autumn attack. This impression of symmetry in the first wave is not confirmed by arithmetical calculation, from which it appears that the primary outbreaks were not in any strict sense symmetrical, the decline not being really at the same rate as the ascent. This point is, however, hardly of more than

DIAGRAM. 9.

LONDON DEATHS.



This graph shows the courses of the several epidemics: the mortality of the worst week in each is taken as 100 and the mortalities of other weeks expressed as percentages thereof. The maxima are superposed.

theoretical importance, the great contrast, viz., of duration, being unaffected and revealing itself whatever method of graphical representations be chosen, Table 12, Diagram 10.

TABLE 12.

Data utilised for the Construction of Diagram 10.

Copenhagen.*			Munich.†	British Army, Home Stations.‡	§Royal Air Force, Home Stations, making Return to the Medico Statistical Department.					
					First Wave.		Second Wave.		Third Wave.	
14	273	2,746	65	8	140	(13)	68	(4)	93	(5)
68	618	2,751	160	206	217	(19)	105	(5)	102	(7)
846	893	2,442	1,430	2,706	1,045	(87)	290	(15)	200	(14)
3,868	2,171	2,330	5,480	1,749	6,083	(434)	555	(30)	323	(25)
8,514	7,564	3,684	8,650	743	7,261	(567)	635	(33)	242	(22)
6,141	13,786	3,986	4,630	459	4,404	(313)	1,682	(85)		
2,961	14,185	2,916	1,550	248	1,707	(121)	3,051	(158)		
1,491	9,412	1,961	440	162	964	(70)	2,661	(130)		
863	5,672	1.595	240	89	478	(31)	1,670	(81)		
395	2,924	1,397	120	58	344	(21)	1,237	(63)		
213	1,932	1,922	110	36	170	(10)	935	(49)		
	1,855		80		114	(6)	926	(49)		
			55		94	(5)	601	(26)		
					77	(4)	303	(16)		
							141	(8)		
							85	(4)		
							132	(7)		
							84	(5)		

* Notifications from week ending June 29th 1918, to week ending February 15th 1919.

† Notifications for 13 weeks from December 1st 1889, to March 2nd 1890 (approximate only, deduced from Leichtenstern's chart of daily notifications).

‡ Non-commissioned officers and men, from week ending December 27th, 1889, to week ending March 7th 1890.

§ The figures in brackets are the rates per 10,000 exposed. The first wave extended from the week ending June 8th to that ending September 7th 1918; the second from the week ending September 14th 1918, to that ending January 11th 1919; and the third from the week ending January 18th 1919, to that ending February 15th.

Great improvements in the art of curve fitting and the graphical representation of mathematical formulæ expressing biological laws or ways of happening due to the researches of such mathematicians as Charlier, Edgeworth, and Pearson, and of mathematical epidemiologists such as Brownlee and Sir Ronald Ross, have permitted the hope that by analysis of epidemic curves, light may be thrown upon the genesis of the epidemic.

The subject has not, however, yet been sufficiently developed to permit the realisation of these hopes in the present connection. The contrast between the explosive rise and fall of the primary and the long drawn-out agony of the second onset,

together with the latter's larger proportion of complicated cases and the greater ease with which pathogenic germs which are to be regarded as secondary invaders were isolated (*vide infra*) naturally and properly suggest that the change of form was due to a variation in, or rather a complication of, the invading organisms. There is, again, the possibility to which Dr. Topley called attention in his Gulstonian lectures,* that a slow increase of susceptibles after the first wave, combined with the exploitation of strains of high virulence and infectivity, are responsible. It is not, however, possible to adjudicate on the claims of the competing explanations with the help of purely statistical evidence. Very different biological factors might be represented by one and the same graphical form. The fact is that the second wave in the present, like that in our earlier history, was longer drawn-out and worked more havoc than the first. It was separated from the first wave by a much shorter interval than came between the first and second outbursts in the previous pandemic, but the interval was little longer than intervened between the second and third waves of the earlier visitation.

It is probable that any explanation covering the 1918 facts must also be descriptive of those of 1890–92.

Upon the whole, bearing in mind the relative ineffectiveness of naturally acquired immunity and the great change of age of incidence to which we have drawn attention, the balance of probability inclines to the view that the *materies morbi* itself underwent an evolution similar in form to, but more rapid than, the life cycles of flux and reflux which, Dr. Brownlee has contended, are principal factors of varying epidemicity. Some light may be thrown upon this by the application of harmonic analysis directed to disclose periodicities of incidence. We summarise in the Appendix a contribution to the subject by Dr. Brownlee. It is no criticism of this interesting study to remark that the nature of the data could hardly allow, and has not allowed, the deduction of sufficiently well grounded conclusions to illuminate the problem of form which we are discussing. At present no more distinct deduction from the epidemic curve seems permissible.

As before, the primary wave generated after some years of quiescence spent itself after a few weeks, in the course of which it attained a great elevation. The secondary wave, coming shortly after the primary, passed much more slowly through its phases and was much more destructive of human life. The third wave showed a slight tendency to revert to the older form. So far as the mere form of the epidemic is concerned, the analogy with historical precedents is close. The

* *Lancet*, July 5th, 1919.

interval between successive phases has diminished; the participation of age groupings has been much altered. Epidemiologically these matters are significant, but they do not pertain to the characters discussed in this paragraph.

Large as are the numbers upon which the previous discussion is based, they are necessarily contaminated by errors, and can only reproduce a blurred impression of the events. We shall gain in clarity if we review less extensive but more accurate statistics. These are provided by the experiences of ships at sea, and a valuable collection of records has been published in Dr. J. H. L. Cumpston's Report on Influenza and Maritime Quarantine in Australia.* The unique significance of this collection resides in the facts that it relates to vessels which sailed from various infected ports at different dates, which contained large numbers of men and, in several instances, carried trained medical observers. Further, in some, particular measures were taken to arrest the course of the disease, the effects of which can, therefore, be studied.

For the purposes of our discussion, we may divide the material into three groups. The first includes ships which sailed from English, Egyptian, African, or New Zealand ports during the second or autumnal wave of influenza. The second group includes ships which left England after January 1st, 1919, *i.e.*, when the second wave was spent and the third in progress. Finally, we have the experience of Australian coastal vessels which derived their infection within the confines of the Commonwealth (as will be seen in the chapter on extra European influenza, the Australian epidemic was of a milder type than prevailed in most parts of the world). In Tables 13–15, we recapitulate the experiences of the larger vessels coming within the classes mentioned. From these tables, it appears; (1) that the incidence and fatality rates of the first group notably exceed those of the second; (2) the third group is characterised both by a low fatality and a short duration, but not by a low incidence rate (in view of the small number of instances and slender complements of the ships tabulated, this inference is, of course, subject to a considerable risk of error).

Passing to the precise form of the epidemic wave, we find that three vessels of the first group carrying in each case about 1,000 passengers and crew, provide accurate chronological records.

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* *Service Publication, No.* 18, Commonwealth of Australia Quarantine Service, 1919.

TABLE 13.

GROUP I.—*Extra-Australian Infection*, 1918.

Name of Ship.	Port of Origin.	No. of Persons on Board.	No. of Cases.	No. of Deaths.	Case Rate per Cent.	Fatality Rate.	Approximate Duration of Epidemic (Days).
Niagara	Vancouver	567	156	5	27·5	3·2	30
Atua	New Zealand	163	88	16	54·0	18·2	23
Medic	" "	989	313	22	31·6	7·0	40
Makura	Victoria, B.C.	406	94	4	23·2	4·3	36
Fantome	Fiji	124	78	0	62·9	—	30
Charon	Singapore	113	23	1	20·4	4·3	17
Nikko Maru	Hong Kong	244	30	1	12·3	3·3	11
Boonah	South Africa	1,095	470	18	42·9	3·8	40
Port Lyttleton	London	929	12	1	1·3	8·3	15
Mataram	Singapore	198	61	0	30·8	—	21
Devon	Suez	1,096	95	0	8·7	—	24
Port Darwin	Egypt	1,237	90	2	7·3	2·2	23
Sardinia	England	1,378	120	10	8·7	8·3	30
Marathon	"	1,041	89	4	8·5	4·5	12
Malton	Egypt	898	16	0	1·8	—	22
Neston	England	1,903	69	0	3·6	—	25
Saxon	"	1,603	25	1	1·6	4·0	36
	Total	13,984	1,829	85	13·1	4·7	435

Mean of the Approximate Duration of Epidemic 25·6±1·44.

TABLE 14.

GROUP II.—*Extra-Australian Infection*, 1919.

Name of Ship.	Port of Origin.	No. of Persons on Board.	No. of Cases.	No. of Deaths.	Case Rate per Cent.	Fatility Rate.	Approximate Duration of Epidemic (Days).
Derbyshire	England	1,203	24	0	2·0	—	14
Delta	"	1,530	93	3	6·1	3·2	28
Ceramic	"	2,361	131	2	5·5	1·5	43
Lancashire	"	1,643	53	0	3·2	—	26
Ascanius	"	1,369	9	0	0·7	—	10
Orea	"	1,698	48	0	2·8	—	37
Kenilworth Castle	"	831	29	0	3·5	—	18
Nevassa	"	1,643	38	0	2·3	—	38
Euripides	"	1,323	53	0	4·0	—	46
Kashmin	"	1,500	23	0	1·5	—	43
Demosthenes	"	1,668	12	0	0·7	—	14
	Total	16,769	513	5	3·1	1·0	317

Mean of Approximate Duration of Epidemic 28·8±2·58.

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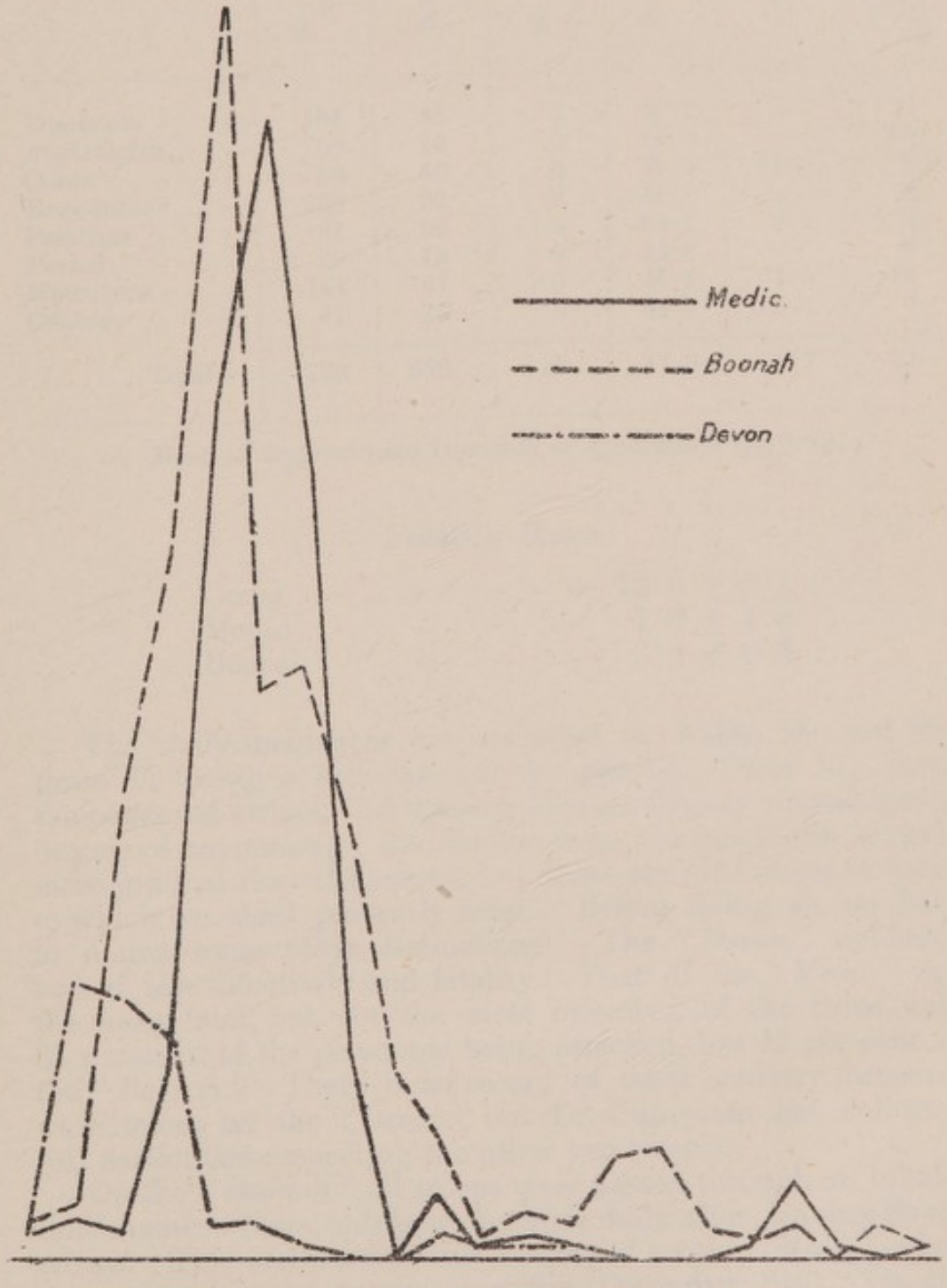
DIAGRAM II.



Graph of course of Epidemics on 3 Ships, plotted from Table 17.

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TABLE 15.

GROUP III.—*Intra-Australian Infection.*

Name of Ship.	No. of Persons on Board.	No. of Cases.	No. of Deaths.	Case Rate per Cent.	Fatility Rate.	Approximate Duration of Epidemic (Days).
Dimboola - -	101	30	0	29·7	—	8
Australglen -	50	19	0	38·0	—	13
Ooma - -	56	50	6	89·3	12·0	8
Encounter - -	220	92	0	41·8	—	8
Pacifique - -	81	52	5	64·2	9·6	8
Period - -	29	13	0	44·8	—	6
Mourilyan - -	144	67	5	46·5	7·5	18
Century - -	41	16	0	39·0	—	8
Total -	722	339	16	47·0	4·7	77

Mean of Approximate Duration of Epidemic $9 \cdot 6 \pm 0 \cdot 88$.

Fatality Rates.

Ooma - - - -	$12 \cdot 0 \pm 3 \cdot 1$
Medic - - - -	$7 \cdot 0 \pm 1 \cdot 0$
Boonah - - - -	$3 \cdot 8 \pm 0 \cdot 6$

The daily incidences are recorded in Table 16, and Diagram 11 (using a two-day unit for plotting, Table 17), shows the course of events. All three epidemics display a considerable degree of asymmetry; the decline from the maximum is much more gradual than the ascent, but there are differences of detail to which we shall presently refer. Before doing so, we have to notice some other distinctions. The "*Devon*" epidemic was of low infectivity and fatality. That of the "*Medic*" was the most fatal, but not the most infective, of the three, only 31 per cent. of the personnel being attacked, but 42 per cent. in the "*Boonah*." There is no record of what sanitary measures were taken on the "*Devon*," but Dr. Cumpston has collected full particulars respecting the other two vessels.

On the "*Boonah*," all troops were passed through an inhalation chamber (zinc sulphate method) daily after leaving South Africa. This method was only employed on the "*Medic*" when the epidemic was nearly over (on December 3rd). On the "*Medic*" troops were inoculated on 21st November, *i.e.*, when the epidemic was at its maximum; on the "*Boonah*" inoculations were done on December 12th after the maximum. It is

evident that the dates at which the inoculations were performed in each case were too far advanced to make it necessary to

TABLE 16.

Daily Case Incidence of Influenza on three Australian Ships.

"*Medic.*"				"*Boonah.*"				"*Devon.*"	
Date.	Cases.	Date.	Cases.	Date.	Cases.	Date.	Cases.	Date.	Cases.
1918.		1918.		1918.		1918.		1919.	
Nov. 11	2	Nov. 26	—	Nov. 29	3	Dec. 19	—	Oct. 14	2
„ 12	3	„ 27	—	Dec. 1	4	„ 20	2	„ 15	2
„ 13	—	„ 28	5	„ 2	13	„ 21	2	„ 16	22
„ 14	—	„ 29	1	„ 3	28	„ 22	2	„ 17	12
„ 15	2	„ 30	—	„ 4	28	„ 23	1	„ 18	11
„ 16	—	Dec. 1	1	„ 5	35	„ 24	7	„ 19	14
„ 17	17	„ 2	2	„ 6	64	„ 25	2	„ 20	6
„ 18	37	„ 3	—	„ 7	48	„ 26	8	„ 21	2
„ 19	41	„ 4	1	„ 8	23	„ 27	2	„ 22	1
„ 20	50	„ 12	1	„ 9	28	„ 28	1	„ 23	—
„ 21	52	„ 14	2	„ 10	24	„ 29	2	„ 24	3
„ 22	35	„ 15	5	„ 11	29	„ 30	1	„ 25	1
„ 23	36	„ 17	1	„ 12	25	„ 31	1	„ 30	—
„ 24	8	„ 20	1	„ 13	13	1919.		„ 31	2
„ 25	11			„ 14	9	Jan. 1	2	Nov. 2	—
				„ 15	8	„ 2	1	„ 3	1
				„ 16	7	„ 6	3	„ 4	1
				„ 17	4	„ 7	1	„ 7	1
				„ 18	2				

consider the prophylactic value of the inoculation. The main distinction between the ships is with regard to inhalations regularly carried out on the "*Boonah*"; not until the end on the "*Medic.*"

We have now to consider the forms of the epidemic waves on the ships. We will begin with the "*Medic.*"

Although at first sight the distribution might be thought to have only a single maximum, and the whole record of 313 cases to be a unitary phenomenon, a little further study makes this idea less plausible. Between December 4th and 12th 1918, no new cases occurred and there were 10 between 12th and 20th December. Can this have been a new phase? Plainly such a result might well be a mere error of sampling. But on examination of the "*Boonah*" and "*Devon*" records a similar, but more prominent sign of at least a two peaked distribution is found. It therefore, seemed to us that something might be gained by studying the course of the epidemic upon the "*Medic*" down to December 4th as if it were a complete history. To the data so limited the customary methods of curve fitting were applied. The appropriate curve proved to be Professor Pearson's "Type 4"

curve (the type of curve which has most often been found in the graduation of statistics of epidemics) and the theoretical frequencies calculated from the curve, together with the latter's constants

TABLE 17.

Case Incidence of Influenza in 2-day Grouping.

"*Medic.*"		"*Boonah.*"		"*Devon.*"	
Date.	Cases.	Date.	Cases.	Date.	Cases.
1918.		1918.		1919.	
Nov. 11 -	2	Nov. 29 -	3	Oct. 14 -	2
„ 13 -	3	Dec. 1 -	4	„ 16 -	24
„ 15 -	2	„ 3 -	41	„ 18 -	23
„ 17 -	17	„ 5 -	63	„ 20 -	20
„ 19 -	77	„ 7 -	112	„ 22 -	3
„ 21 -	102	„ 9 -	51	„ 24 -	3
„ 23 -	71	„ 11 -	53	„ 26 -	1
„ 25 -	19	„ 13 -	38	„ 28 -	0
„ 27 -	0	„ 15 -	17	„ 30 -	0
„ 29 -	6	„ 17 -	11	Nov. 1 -	2
Dec. 1 -	1	„ 19 -	2	„ 3 -	1
„ 3 -	2	„ 21 -	4	„ 5 -	1
„ 5 -	1	„ 23 -	3	„ 7 -	1
„ 7 -	0	„ 25 -	9		
„ 9 -	0	„ 27 -	10		
„ 11 -	0	„ 29 -	3		
„ 13 -	1	„ 31 -	2		
„ 15 -	7	1919.			
„ 17 -	1	Jan. 2 -	3		
„ 19 -	0	„ 4 -	0		
„ 21 -	1	„ 6 -	3		
		„ 8 -	1		

are shown in Table 18. This graduation is not a very good one as indeed, might have been expected since the decline of the epidemic as observed is very irregular, but the result is greatly superior to any graduation obtained of the data as a whole or again to any graduation by a normal curve of error, whether of the whole or of a selection of the data. Attending to the point just made, viz., the irregularity of descent, it seems possible to hold that we are dealing not with two epidemics, but even with three, the maximum of the second being not more than two units of time from the first maximum. Upon this hypothesis we should regard the complete morbid evolution as compounded of three waves, or, in geometrical language, three curves with maxima at different points have combined to produce the result. On turning to the record of the "*Boonah*" this surmise is confirmed. It is at once obvious that even the very partial success achieved in the case of the "*Medic*" by regarding the first stage of the observed sequence as a single phenomenon and graduating

it with a monomodal skew frequency curve cannot be secured; in the part of the descending limb of the "curve" so deliminated

TABLE 18.

Comparison of "Medic" *Cases with those calculated by Curve.**

	Cases in 2-day Grouping.	Cases as calculated by Curve.*
— 6	2	·7
— 4	3	1·2
— 3	2	5·8
— 2	17	25·1
— 1	77	73·1
0	102	97·0
+ 1	71	60·2
+ 2	19	24·9
+ 3	0	9·1
+ 4	6	3·4
+ 5	1	1·3
+ 6	2	·6
+ 7	1	·5

* $y = 85{\cdot}549\left(1 + \frac{x^2}{7{\cdot}349}\right)^{-3{\cdot}458} e^{1{\cdot}532 \tan -1 \frac{x}{2{\cdot}711}}$.

Origin ·785 units before 0.
Mode (101·22) at — ·185.
Skewness = ·170.

there is an evident arrest of the decline, and at a point when the observed frequencies of cases are still considerable. It is necessary to dissect the distribution into three parts, one curve having its maximum probably between the 5th and 7th of December, the second some four days later, the third 16 or 18 days from the first maximum. The resolution of a skew frequency into three components by algebraic analysis is a task not yet achieved by statistical mathematicians and would in any case depend upon constants the probable errors of which, when so small a number of observations is available, must be large. Hence we can do no more than indicate *primâ facie* appearances and suggest that in the "*Boonah*" as in the "*Medic*" a triple wave was experienced. The still scantier data of the "*Devon*" are in nowise inconsistent with the others, there is plainly heterogenity of the complete frequency and *primâ facie* a resolution into components with maxima at or about 16th, 18th, and 25th October is conceivable. In each of these three cases the second maximum must be near the first, but in the "*Devon*" and the "*Boonah*" the second component is relatively to the first, much more important than

in the "*Medic*." But in the "*Medic*" the first and dominating component is much more "explosive," it includes, and within a shorter time, a much greater proportion of the total cases. The whole epidemic was more fatal, but less infective than the "*Boonah*" outbreak. It may well be that these differences are correlated and it is *conceivable* that the prophylactic methods used on the latter vessel had something to do with the result. But since the ports of origin were different and the constitution of the personnel of the "*Medic*" (which carried numerous Italian reservists), unlike that of the other ship, it would be very rash to base any definite conclusions as to the value of the inhalations upon these differences.

The epidemiological importance of these events depends upon the apparently complex form of the epidemic curve. Upon these ships we have had performed a remarkable experiment. We have had confined within narrow bounds an absolutely large number of individuals and have maintained this confinement for a long period. The state of affairs is quite unlike that of a prison, and has little resemblance to that of an educational institution. In a prison there is cellular isolation, only limited communal mixing. In a school there is the variable factor of frequent intercourse with the general population. On the ship we do really have ordinary social life in isolation from the great world, a real microcosm. The remarkable point is that within this mircrocosm we have seen in miniature a triple wave, such as the whole world experienced, but running through its phases in weeks instead of months. But this evolution was not parallel with the world evolution, since the voyage started after the first act of the world drama. Whatever organism was "carried," it must have belonged to the autumn variety, not to the summer vintage. Hence, if this threefold wave phenomenon is not a mere illusion, it would seem to be a function of the organism as modified by passage, a modification determined by the environmental conditions. Which of the three phases shall be the most serious to humanity cannot be determined by the environment alone, otherwise we should find in all the same compound form, since the general environmental conditions were alike in all; but the milieu will determine the time relations of the successive events.* These results therefore strengthen the conclusion which we have drawn from other data, that the abnormal time relations of the phases in the world pandemic of 1918–19, the shortening of the interval between successive waves may really be dependent upon the great increase of what we have called extra-domestic aggregation so notably exaggerated by the direct and indirect consequences of the world war. We hasten to add that the

* An instance of epidemic pneumonia attacking a village community some months after influenza was prevalent, is that reported by Parsons (Rep. M.O., L.G.B., 1890, Appendix A, 12) from Scotter (Lincs.), 29 cases with 18 deaths occurred in a population of 1,070.

evidence upon which this conclusion is based is incomplete, and that the technical difficulty or, so far as the writers are concerned, impossibility of effecting a theoretically satisfactory resolution of the compound epidemic waves on the ships into monomodal components should be regarded as a serious limitation upon the value of the conclusions; but we consider that these data merit the closest scrutiny and that other epidemiologists will do well to examine them attentively. It is very unlikely that equally complete records of ship epidemics upon vessels so long at sea will become available. We turn now to the records of intra-Australian epidemics.

In our table we have only included a small number of the vessels, viz., those with relatively large outbreaks. Actually 92 vessels, carrying 7,966 souls, came under observation, and in no fewer than 34 there was but one case of influenza on board, evidence of the low dispersive power of the infection. But the tabulated results show that sometimes the disease was even more infective than the over-seas type, and there are a few instances of high mortality. Thus, the "*Ooma*," while lying at Melbourne with a crew of 11 Europeans and 45 Filipinos, but no passengers, experienced an epidemic in which the daily incidence, from February 15th, was 1, 0, 4, 10, 12, 19, 0, 3, 0, 0, 0, 1. In all there were 50 cases, and six of the patients died. As these vessels were under the continuous supervision of the Medical Quarantine Service, a good opportunity of judging whether the course of an epidemic can be modified by prophylactic methods was afforded. Of three vessels—"*Ooma*," "*Pacifique*," and "*Encounter*"—all on board the two former were vaccinated once, and all on the "*Encounter*" three times, with the Commonwealth Coryza vaccine *before* influenza broke out; two, however, had relatively more extensive outbreaks than any other ships trading between Commonwealth ports. Hence we can deduce no evidence of modification of the infectivity by prior inoculation. It is, however, noteworthy that in the "*Encounter*" and the "*Fantome*" (a vessel of group 1), where inoculation had been practised, and an extensive epidemic followed, there was no death amongst the 170 attacked persons. We are aware that the gross number is much too small to use as a basis of generalisation, but the experience is consistent with the belief, supported by other evidence, that inoculation may diminish the risk of death, although not altering the risk of infection.

In almost every case, inhalation by the zinc sulphate steam method was used daily. Hence the inference drawn from the "*Boonah*" and "*Medic*" cases is strengthened. With respect to isolation, we quote Dr. Cumpston's remarks:—

> "In every one of the vessels under consideration, a daily thermometer parade was held, and every person showing any rise of temperature was at once removed

> for observation. In addition, any person reporting sick was immediately removed. This was most thoroughly and efficiently carried out, and it can safely be asserted that the sick were removed in every case immediately on discovery. No better test of the efficacy of immediate isolation of the infectious sick could well be devised. That this method failed to control epidemics is clear from the vessels dealt with in this series. In spite of this method applied from the first day, "*Ooma*" showed 89 per cent. of personnel infected, "*Pacifique*" 64 per cent. of her personnel, "*Mourilyan*" 46 per cent., and "*Dimboola*" 30 per cent."

It may be urged that these measures were responsible for the very large proportion of instances in which the disease did not affect more than one or two persons, *i.e.*, that, on the average, it had an appreciable effect, although it failed occasionally. But when we note the greater explosiveness of the disease where it did occur, in comparison with the type witnessed in the over-seas group, it is perhaps more probable that the proportion of sporadic cases is attributable to a differentiation of the infecting germ. However this may be, it is established that when an epidemic is in course of development its wave-form cannot be modified by administrative action of the kind described. Although in point of absolute numbers the data we have just analysed are scanty in comparison with the massed figures discussed in the remainder of the chapter, they are, we think, of epidemiological importance, and strengthen the impression derived from the less detailed statistics, viz., that the epidemic wave-form in influenza is a biological phenomenon which cannot yet be controlled or varied by human agencies. Other epidemiological inferences, not, however, so securely based, are that, even when the primary focus of infection is characterised by a low virulence (as seemed to be true of Australian influenza), under special conditions—such as those of the "*Ooma*"—a very serious outbreak may occur, and that vaccination may be of importance in diminishing the rate of fatality.

CHAPTER III.

The Clinical Features of the Influenza Epidemic of 1918–19.

BY

Herbert French, C.B.E., M.D., F.R.C.P.

Introduction.

From a clinical point of view the influenza epidemic of 1918–19 resolves itself into two sharply distinguished parts, the first including the type of cases met with in June 1918; and the second, the quite different type, which abounded in the autumn (October) of 1918 and recrudesced in the spring (February) of 1919. In the June 1918 outbreak, although the cases were very numerous there was little or no mortality; there was little or no pulmonary complication; and the patients, though striken severely for the time, speedily recovered after so short an attack that it was widely spoken of as "influenza of the three-day fever type." It was in the October 1918 and in the February 1919 outbreaks that the high mortality from pulmonary and general septicæmic complications developed and gave such an entirely different clinical aspect to the disease; and it was in this part of the epidemic that the dreaded heliotrope cyanosis was so pronounced a feature of the fatal cases.

It is of importance, however, to emphasize the fact that the heliotrope cyanosed type of case, though it attracted chief notice during the influenza epidemic of 1918–19, was not a new phenomenon confined to this epidemic; it had already been met with and reported upon in connection with minor epidemics during 1916 and 1917, at which time the label given to these fatal cases was "purulent bronchitis," though I, personally, am strongly of the opinion that these smaller outbreaks in 1916 and 1917 with heliotrope cyanosis as a striking phenomenon of the worst cases, were of precisely the same nature as those of the enormously more widespread and serious "influenza" outbreaks of 1918 and 1919. (*See* below, p. 69.)

It is not possible to give exact figures as to the number of cases I myself saw. In the epidemic of June 1918 I had the opportunity to study some hundreds; but in the epidemic of the autumn of 1918 and the spring of 1919 the total number extended to many thousands; part in Guy's and other civilian

hospitals; part in military hospitals in the Aldershot Command; amongst Canadian Troops at Bramshot and Witley Camps; and amongst United States troops on board ship and in the hospitals at Liverpool and in the hospitals at Winchester and at Portsmouth. I also performed, or was present at, autopsies totalling over three figures.

A very striking point throughout was that wherever one met with the disease, and of whatever nationality the patient, the malady in each mainpart of the epidemic ran remarkably true to type; except for the rarer complications the clinical type of case, whether of the mild "three-day fever" type of June 1918, or of the much more serious type of October 1918 and February 1919, was much the same wherever one met with it. It will be convenient to deal first, and briefly, with the mild June type; and secondly and more fully with the graver autumn type.

The Clinical features of the relatively mild June 1918 *type of Case.*

In the midst of perfect health in a circumscribed community, such as a barracks, or a school, the first case of influenza would occur, and then within the next few hours or days a large proportion—and occasionally even every single individual of that community—would be striken down with the same type of febrile illness, the rate of spread from one to another being remarkable. The patient would be seized rapidly, or almost suddenly, with a sense of such prostration as to be utterly unable to carry on with what he might be doing; from sheer lassitude he would be obliged to lie down where he was, or crawl with difficulty back to bed so that barrack rooms which the day before had been full of bustle and life, would now be converted wholesale into one great sick room, the number of sick developing so rapidly that the hospitals were, within a day or two, so overfull that fresh admissions were impossible and the remainder of the sick had to be nursed and treated where they were.

The men's temperatures were raised to varying heights, generally about 103° or 104° F.; the pulse rates were less raised in proportion; the tongue was coated; the face flushed; the eyelids a little drooped as though the patient were but half awake; and in a very considerable proportion of these cases, either at the very beginning or within a few hours of the onset, there was huskiness of the voice and a tendency to hawking and throat-clearing, less often to actual cough. The throat was complained of as being sore so that it was difficult to swallow or to speak; frothy expectoration, not large in amount, was brought up rather from the mouth and pharynx and larynx than from the bronchial tubes. There was a varying degree of bright reddening of the posterior part of the palate, uvula,

fauces and pharynx without white spots or folliculi of pus; without exudation and often without any swelling of the tonsils or cervical glands. In some, on the contrary, the tonsils, besides being reddened looked swollen and enlarged and there might be tenderness on either side of the upper part of the neck below and behind the angle of the jaw, suggesting that the lympatic glands here were inflamed, too, though palpable glandular enlargement was not as a rule a feature of these cases.

Headache, especially of that type in which the head does not ache so much when it is kept still, but aches badly when there is a change of posture such as when the head is rolled from side to side, or some effort of coughing is made, was prevalent in many cases; but in the main one would summarise the symptoms as being those of lassitude and general all-over aching, with fever, a coated tongue, loss of appetite, soreness of the throat, huskiness of the voice and headache.

Most patients slept well and wished simply to be left alone.

Gastro-intestinal symptoms were not pronounced; there was no dyspnoea; water and cooling drinks were all the patient asked for, and in this state he lay in bed the next day and the day following, during both of which the temperature would as a rule remain up, though tending to fall upon the third day. The pyrexia might continue longer than this, but in many it was already coming down to normal at the end of the second day, and most of it had become normal at the end of the third day and remained so thereafter. The patient by this time was feeling almost himself again; asking for food, wishing to get up, and complaining of little more than some remains of soreness of the throat and perhaps some huskiness of voice, though the latter was better. than it had been during the preceding three days. Convalescence was rapid and the great majority of the patients were fit for their ordinary work again by the end of the week. Only in those who seemed already predisposed to bronchial catarrh as a result of being subject to emphysema, for example, or chronic bronchitis, tended to have any persistence of the bronchial infection.

Amongst those previously robust there was practically no mortality, though individuals taken by the influenza when already ill with something else may have had their end hastened by the intercurrent influenzal attack.

There was no albuminuria, no special tendency to infection of the accessory nasal sinuses; indeed no tendency to any particular complication at all. Hundreds of cases ran very much the same course simultaneously, one very much like another, and "three-day" influenza was the popular name generally given to the disease.

In regard to treatment, nothing special was indicated; rest in bed for three days and ordinary nursing without drugs led

to just as good results as active administration of medicines, such as aspirin, salicylates, quinine, or anything else.

The Clinical Features of the Cases in the severe Epidemic of the Autumn of 1918 *and of the Spring of* 1919.

Contrasted with the extensive and acute but non-fatal outbreak of June 1918, the world-wide "plague" of influenza of the following autumn and winter, with its millions of deaths, presented very different clinical characters; and in the multitude of severe cases one saw the constantly repeated picture of a dreadful malady which few physicians had seen the like before. Those who had experienced the minor epidemics of "purulent bronchitis with heliotrope cyanosis and fatal ending" that had occurred here and there in military camps in America, England and France during 1916 and 1917 had already become familiar with some of the worst features, especially the dreaded blueness, of what was probably the same malady under a different name; but now it was a question of seeing hundreds or even thousands of cases in districts in which the fatal "purulent bronchitis" had affected but a few.

Nevertheless, it is important to emphasize the fact that, although it was the "pneumonic" type of case that attracted so much attention, creating such consternation owing to its mortality, and thereby colouring the picture of the epidemic as a whole, these fatal "pneumonic" cases constituted but a minority of the whole. There were far more cases of ordinary straight-forward benign influenza than there were of "influenzal-pneumonia"; but these benign cases were overshadowed by the grave ones; and there is a little danger, if one does not emphasize the fact, that future generations might gain the impression that the whole of the 1918–19 epidemic was of "pneumonic" and grave character. Broadly speaking, I should say that out of 1,000 individuals striken by the disease fully 800 had no more than an ordinary attack of uncomplicated "influenza," a little more severe perhaps than the "three-day fever" of June 1918, but not any worse than simple influenza as it may occur at any other time. It was the remaining 200 who were so much more seriously ill, with "pneumonic" symptoms added to those of simple influenza; and of these about 80 died. The most dreaded symptom was the heliotrope cyanosis; it developed in less than half of the pulmonary cases, but once it became definite the prognosis was so bad that I should say out of every 100 "blue" cases 95 died.

It is not within my province to discuss the bacteriology of the disease, nor to enter into the question of whether the disease was really influenza at all, or something else. A separate chapter deals with these points. I have, however, been present at many discussions of them, both public and private, and my personal conclusion is that the primary nature of the whole epidemic has been "influenza"; that Pfeiffer's bacillus has

not yet been deposed from its place as the causal organism of "influenza"; that it has in all probability been the primary organism in the 1918–19 epidemics; but that whereas Pfeiffer's bacillus alone may be responsible for the symptoms in the 800 mild cases, an additional organism—sometimes the pneumococcus, sometimes the streptococcus, sometimes Friedlander's pneumobacillus, sometimes a combine of two or more of these, and sometimes, perhaps, yet other organisms—has become virulently associated with Pfeiffer's bacillus in the 200 graver cases; the pulmonary and other complications and the high mortality from septico-toxæmia being the result of the doubled or trebled infection and not of influenza solely.

Expressed in another way, too briefly no doubt to be strictly accurate, and yet helpful in interpreting the remarkable difference there was between the mild and the severe cases that were under observation simultaneously, I would describe the 800 as suffering from influenza only; the 200 as suffering from influenzo-pneumococcal or influenzo-streptococcal or influenzo-pneumo-streptococcal septicotoxæmia and not from influenza only.

It was, however, only when any given patient had absolutely recovered that one could relegate him with certainty to the "mild" category. Even the mildest case had to be regarded as potentially grave; no matter how benign the illness might appear to be at first the dreaded pulmonary complications and cyanosis might set in without any notice at all. A patient might have been ill a day or two with mild influenza and seem to be progressing well; in an hour or two the whole picture might change, and twenty-four hours later the patient might be dead. During the epidemic itself, therefore, every case had to be regarded as in grave danger; it is only when one looks back that the two big classes—800 out of every 1,000, mild and ordinary; 200 out of every 1,000 severe, pulmonary, grave—emerge clearly into view.

There is little need to enter into the clinical details of the milder cases; they were ordinary influenza cases running an ordinary influenza course. It is the "pneumonic" type of case that calls for a detailed account, and in what follows it is the "pneumonic" type that is being discussed. One point, however, calls for special mention, and that is that whether the case developed into the grave type, or remained benign, *epistaxis* was a phenomenally common early symptom. It is not possible to give statistics, for in the stress and dire overstrain of those strenuous days and nights no full records were kept; but when special inquiry was made in scores of consecutive cases, some degree of nose-bleeding had occurred in over half; in most it had been but blood enough to redden a handkerchief, but in some it had been sufficiently severe to call for treatment for the nose-bleeding itself; and all the medical men one met were commenting on the commonness of this epistaxis. It is referred to again later.

The Special Features of the "Pneumonic" Cases.

The first point to emphasize is that although the pulmonary complications were spoken of almost universally as "pneumonia," the one thing they hardly ever were was ordinary croupous lobar pneumonia in the recognised sense of the term. The word "pneumonia," therefore, has been used in this article in inverted commas, because one needs some word to use for the pulmonary complications, and "pneumonia" is the term that was applied almost universally; it would be quite erroneous, however, to suppose that these cases were ordinary influenza plus croupous lobar pneumonia. The physical signs were most variable; but the occurrence of dulness, bronchial breathing, bronchophony, pectoriloquy, and crackling rales over the greater part or whole of one lobe was frequent enough to make those who saw no autopsies believe that there was real lobar pneumonia present; and many made the diagnosis of lobar pneumonia on the physical signs, and used the term in discussion as though they thought real croupous pneumonia was present. Only in the most exceptional cases was croupous lobar pneumonia found post-mortem; the "pneumonia" of these influenza cases was almost everything else but lobar pneumonia, unless lobar "pneumonia" is held to include any variety of lung inflammation that can consolidate a whole lobe. In more cases than not there was no really "lobar" consolidation, and some died with symptoms precisely similar to the others and yet without any consolidation of the lung at all. The "pneumonia" was an acute infective pulmonary inflammation in which such consolidation as resulted was due not to croupous lobar pneumonia of the classical sort, but to a conglomeration of changes which included bronchitis and peribronchitis, coagulative œdema, hæmorrhage, collapse, broncho-pneumonia, abscess formation, and compression by pleuritic effusion, totally different to anything ordinarily seen in the post-mortem room. Hence, in speaking of these cases as "pneumonic," it must be emphasized that the pulmonary inflammations implied were those peculiar to the epidemic, and not just croupous lobar pneumonia complicating influenza.

The "pneumonic" complication—often with physical signs of but a little bronchitis when more were anticipated—would develop at any period of the influenza attack; there was no rule. In most cases the patient had been ill for a day or two with ordinary simple influenza, not necessarily severer than that of his neighbours, when there was a rapid or sudden change for the worse, and the picture changed rapidly from that of influenza to that of a chest case; and the effects of the pulmonary changes were often so fulminating that death might ensue in 24, 36, or 48 hours, in such a way as to suggest that it was not due to the lung lesions themselves but rather to a generalised and very virulent microbic-toxæmia, or actual septicæmia.

On the other hand, there was often no preliminary "influenzal" period at all, the patient being attacked from the start in such a way that ordinary lobar pneumonia of virulent or even ultra-virulent type would be the diagnosis made by most of us if one saw the case singly and not in such an epidemic; at the beginning of the outbreak it was most difficult to persuade those who had not yet seen other cases that the condition was not ordinary pneumonia; but the autopsies showed just the same conditions as did the other types of cases, and seldom, if ever, true lobar pneumonia.

Again, the pulmonary complications were often later in their development, yet equally fatal. The patient might have had no symptoms other than those of ordinary influenza for nearly a week; his temperature might be falling steadily, or might have become normal, so that danger might be regarded as past, and yet the "pneumonic" complications might set in and carry off a man who seemed almost convalescent.

Less often, and yet not infrequently, the patient might be apparently quite convalescent from "influenza," ready to be discharged from hospital, and yet go down with "pneumonic" symptoms, and die.

All types were seen in abundance—the initial, the early, the later, and the latest.

At whatever stage the pulmonary complications set in the patient generally began to complain in some way of his chest, often of pain in one side or other of the thorax, or on both sides; frequently in front, in one or other anterior axillary line below the level of the nipple; almost as frequently at one or other base; or, again, down the front of the chest behind the sternum as though he were "all raw inside there." In practically every case there was also cough, not always severe, but sometimes in itself distressing, short, dry, and hacking to begin with; looser and associated with frothy or blood-stained or purulent sputum within a few hours, or the next day. Towards the end of a severe case coughing and expectoration would be entirely absent from sheer weakness of the sufferer and his inability to cough at all. The rate of breathing became accelerated out of all proportion to the physical signs; in the worst cases the respiration rate would rise to 40, 50, or even 60 to the minute, and yet without any particular evidence of respiratory distress; orthopnea was exceptional and although the patients were breathing so rapidly they seldom, if ever, complained of actual difficulty in breathing; it was rather a frequent breathing—a polypnœa or tachypnœa—than a true dyspnœa. The condition of the skin was not constant; it might be hot, dry, and pungent as in ordinary lobar pneumonia; quite as often the whole of the patient's body and limbs would be covered with profuse perspiration, the latter often resulting in sudamina and miliaria. A definite rigor might occur at the onset of the pulmonary complication, but more often there was

nothing in the way of a definite rigor to attract notice, though the temperature, already raised, might rise higher.

The pulse rate, though raised, was seldom unduly rapid, and it was a remarkable feature of a great majority of the cases that the condition of the pulse remained good almost to the last, falling only in *articulo mortis*.

The Heliotrope Cyanosis.

The facies, at first flushed and red, with a peculiar drooping of the eyelids giving a weary look, shown in Plate 1, might remain purely red throughout, but in nearly half the cases affected by the pulmonary complications the red tint rapidly changed to one of progressive cyanosis, depicted in Plates 2 and 3. It was when this dreaded heliotrope cyanosis appeared that one knew that the prognosis had now altered so completely that the patient was almost certain to die; a small percentage of cases managed to recover, even after the cyanosis had developed, but the great majority succumbed, and it was amongst cases of this type that the great mortality of the epidemic occurred. There were, of course, cases which died without the cyanosis being pronounced, but in going round a large ward one could, without examining the patients at all beyond looking at their countenances, pick out those who were going to die with almost uniform certainty by reason of their colour alone. It was not by the temperature chart nor by the physical signs in the chest, nor by feeling the pulse that one could tell the serious cases so well as one could by their colour; the cyanotic tint might be definite in a patient who was complaining little, who was taking his liquid nourishment well, was taking an intelligent interest in his surroundings, answering questions promptly and clearly, and in no way—except by his colour—indicating that by the next day or the day after he would almost certainly be dead.

The plates reproduced here were taken from rather extreme cases and very often the degree of fatal heliotrope cyanosis fell a long way short of that depicted; but the illustrations were taken from actual cases, and there were hundreds as severe as these. Whatever the degree of cyanosis, however, it rendered the prognosis bad. It depended much upon what the original colour of the patient had been, what the amount of actual blueness of the face was; for example, a man who was naturally sallow and pale-faced would show little of the heliotrope colour in his face generally, but it would be obvious in the colour of his lips and ears. The naturally pale man tended to look rather ashen than heliotrope, but with a distinctive colour of the lips and ears that attracted at once. The plethoric man, on the other hand, would be seen to change from a frankly red countenance to a colour which, if one were to reproduce it in

painting would necessitate the admixture of more and more blue with the red until ultimately the whole face and particularly the lips and ears passed through the stages of dark red to purplish-red, to reddish-purple, to absolute purple; and then towards the very end—as shown in Plate 3—that which had been a purple face might become a pale, cyanosed, deathly countenance with purple lividity of lips and ears.

In some the cyanosis might be well marked before the patient had been ill 24 hours, and death occurred in some instances within this time from the onset.

In others, the duration might be 48 hours. In others again, the lividity came on more gradually, and the patient might remain alive for three, four, or five days, or even for a week, breathing 50 or 60 to the minute, not unconscious, not subjectively distressed, though objectively a dreadful picture; but the ending in over 90 per cent. of all the cases in which the cyanosis developed was progressively downhill towards death; the latter being preceded in many instances by delirium of a low type, associated with unconsciousness, though in some on the other hand consciousness was retained almost, if not quite, to the very last.

I have seen many cases of precisely the same type of heliotrope cyanosis as the result of what was then called "purulent bronchitis" (see *Lancet*, July 14th, 1917, p. 41), and my impression is that, though there were certain clinical differences, especially in regard to the character of the sputum, the "purulent bronchitis" cases of 1916 and 1917 were really sporadic instances of precisely the same malady as those of the epidemic of 1918–19; and it is of interest that when Dr. Eyre helped Major Abrahams and me by determining the bacteriology of these previous "purulent bronchitis" cases, he found that they also had a double infection—influenzo-pneumococcal in some places, influenzo-streptococcal in others. The only other condition in which I have seen similar facies with cyanosis has been "gassing"; but in gassed cases the patient has been in dire distress as well, whereas the influenzal "pneumonic" cases were in much less distress than were those who saw them.

For a long time the nature and causation of this peculiar heliotrope cyanosis was obscure. It was certainly not due to cardiac or circulatory failure, for the condition of the heart and pulse remained strikingly good, and although some observers have noted dilatation of the heart at autopsy in these cases, cardiac dilatation was, in my experience, quite the exception; again and again at the post-mortem examinations one was struck by the fact that the heart looked remarkably normal both in colour and in size; there may have been a little dilatation of the right side, sometimes, but certainly no more than is general in cases of death from acute conditions, and anything like extreme dilatation of even the right side of the heart was very exceptional. The

PLATE 1.—This illustrates an early case in which the facial colour is frankly red, and the patient might not appear ill were it not for the drooping of the upper eye-lids, giving a half-closed appearance to the eyes.

PLATE 2.—This illustrates a pronounced degree of the "heliotrope cyanosis." The patient is not in physical distress, but the prognosis is almost hopeless.

PLATE 3.—This illustrates another type of the cyanosis, in which the colour of the lips and ears arrests attention in contrast to the relative pallor of the face. The patient may yet live for twelve hours or more.

cyanosis was not relieved in the least by venesection, or by the administration of digitalis or other cardiac stimulants. At one time it was thought that there might be some peculiar chemical change in the blood leading to the formation of methæmoglobin, or even sulph-hæmoglobin, but repeated spectroscopic examination showed no abnormal blood pigment to be present. When, however, one had the opportunity of examining microscopical sections of the lungs, in which coagulative exudation both into the alveoli and into the interstitial tissues was often a very pronounced feature of the section, one realised that this albuminous exudate—quite different to that seen in ordinary pneumonic cases—was the probable cause of the cyanosis. The appearances in some lung sections were very similar to those of the extreme exudate that results from gassing, and layers of this albuminous fluid coming between the inspired air and the blood capillaries would necessarily interfere with the absorption of oxygen by the latter, an extreme degree of anoxhæmia being the result. The general conclusion was that the heliotrope cyanosis was due not to heart failure, nor to abnormal chemical changes in the blood, but to sheer anoxhæmia resulting from this widespread and extensive albuminous exudate into the alveoli and interstitial tissues of the lungs.

The Temperature Charts.

Short of reproducing many scores of actual temperature charts it would be impossible to show how variable the course of the pyrexia was in different cases otherwise of the same type. Facing page 76, ten charts from simultaneous cases are reproduced, five from cases which recovered and five from cases which died, and their variability speaks for itself. Sometimes the temperature dropped rapidly with speedy recovery, as in Chart 1, but Chart 6 shows how a patient may have little pyrexia, and it may seem to be falling comfortably by lysis, and yet the patient may die. Chart 2 shows termination of the illness by crisis with recovery. Chart 7, a similar sudden fall of the temperature followed by death. Chart 3, a fall by lysis, prolonged by irregular persistence for several days, ending in recovery. Chart 9, a fall by lysis in a severe case that seemed to be doing well, with subsequent rapid rise and death. Chart 10 shows a rapid fall on the third day of the disease as though the patient had terminated his illness by crisis, but the pyrexia flared up again, rising by steps to a second maximum about the 10th day, when a second apparent crisis occurred, and yet the illness continued, pyrexia recurring after the second apparent crisis, and followed by a rise after the second crisis and terminating in death. No two charts were quite alike; definite termination by crisis and recovery, as is shown in Chart 2 was very rare indeed; the chief thing which struck one on studying many hundreds of consecutive charts was that

there was nothing in the temperature chart itself to tell one whether the patient was doing well or badly.

The Respiration Rate.

The respiration rate, on the other hand, was a much more helpful guide than was the pyrexia; the rapidity of breathing in the fatal cases was even greater than that which one expects in ordinary lobar pneumonia; rates that were nearer 40 than 30 to the minute were very common, but in the worst cases the rapidity of breathing was generally over 40 and often 50 or even 60 to the minute, and this sometimes before the fatal cyanosis became evident. It was in almost all cases silent respiration without stridor and phenomenally without distress; though breathing so fast the patients themselves were seldom conscious of panting for breath; they did not complain of their breathing. They might complain of pains in their chest, or of cough, or of chest trouble, but they did not—at any rate in the great majority of cases—complain that they had difficulty in breathing or that they could not get their breath. They very seldom had orthopnea; most cases indeed lay flat down in bed and preferred not to be propped up. It was exceptional to find actual orthopnoea, and then only as a rule when there was pre-existent emphysema or myocardial degeneration or valvular disease. This absence of real *dys*pnœa was remarked on repeatedly by observers from all parts, and led to the use of the terms *poly*pnœa or *tachy*pnœa, to distinguish the condition exhibited by these patients from real *dys*pnœa.

Cough.

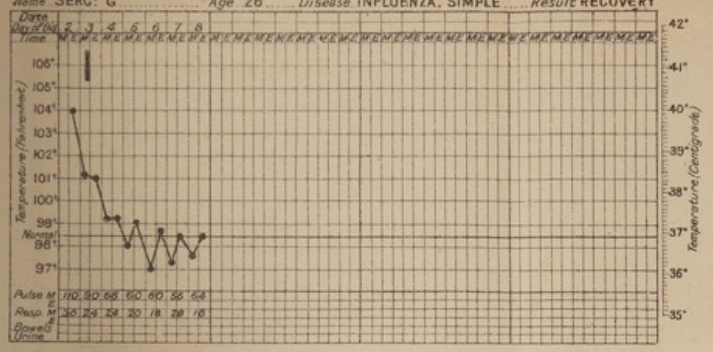
Cough was not always a prominent feature, and often it was almost absent throughout even in those most gravely affected. It bore little relation to the extent of the physical signs in the lungs, but was most troublesome when there was evidence of much bronchitis, or again in the many patients who had a pleuritic friction rub. It was apt to occur in paroxysms which were sometimes very exhausting when there was little sputum. Towards the end of the grave cases cough, which might have been very troublesome indeed in the earlier stages, generally ceased, even though the lungs were full of rhonchi and rales from apex to base; probably because the patient had too little reserve of strength for the effort of coughing to be possible.

Sputum.

The sputum was very variable indeed. Cases in which large quantities—8 or 10 ounces—of almost pure pus were coughed up daily in the way which was so remarkable in the previous cases of so-called "purulent bronchitis" were met with here and there, but these were very exceptional indeed during the main epidemic.

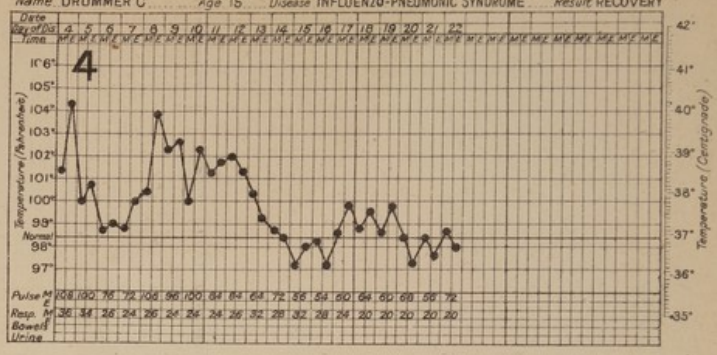
To face p. 76.

Name SERG^T G. Age 26. Disease INFLUENZA, SIMPLE. Result RECOVERY

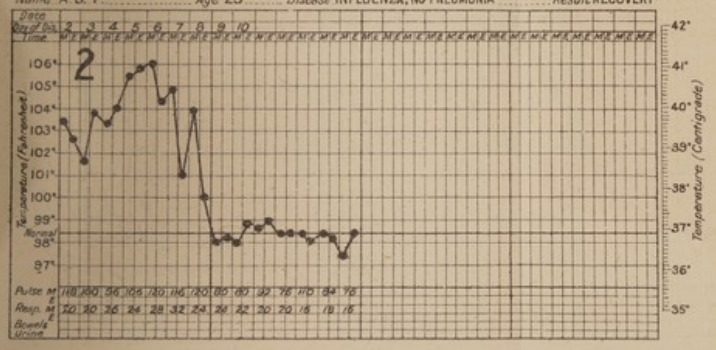


MILD CASE

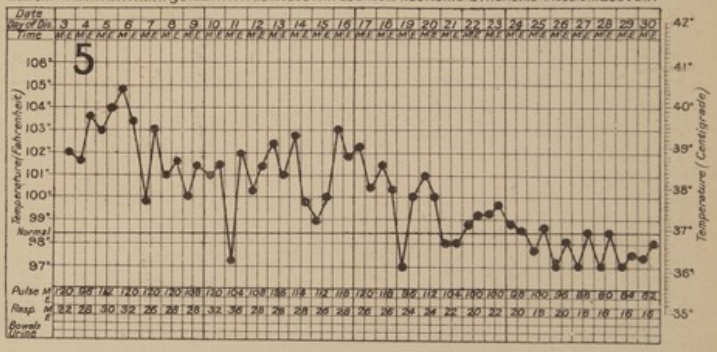
Name DRUMMER C. Age 15. Disease INFLUENZO-PNEUMONIC SYNDROME. Result RECOVERY



Name A. B. F. Age 25. Disease INFLUENZA; NO PNEUMONIA. Result RECOVERY

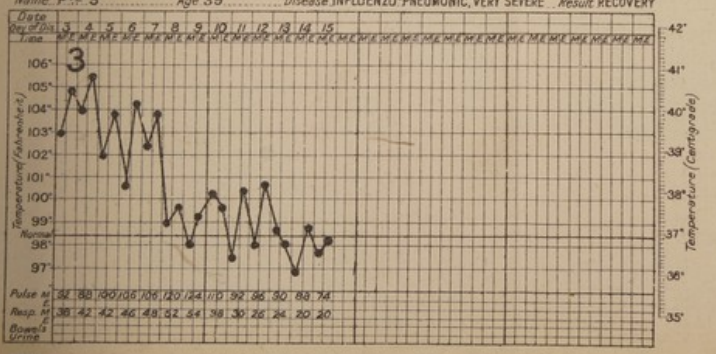


Name PTE. M. Age 22. Disease INFLUENZO-PNEUMONIC SYNDROME. Result RECOVERY



AT ONE TIME APPEARED HOPELESS

Name PTE. S. Age 39. Disease INFLUENZO-PNEUMONIC, VERY SEVERE. Result RECOVERY



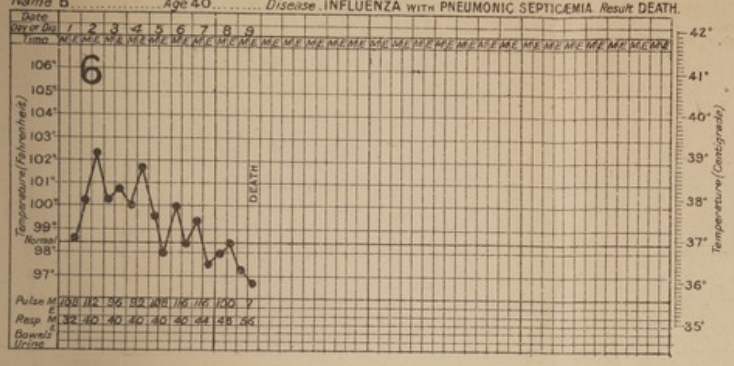
CHARTS OF 5 CASES OF RECOVERY.

2763

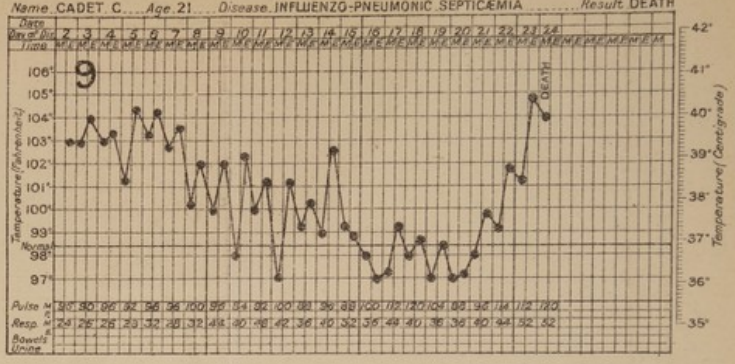
H. & C. GRAHAM LTD LITHO^RS LONDON, S.E.

To Face p. 76

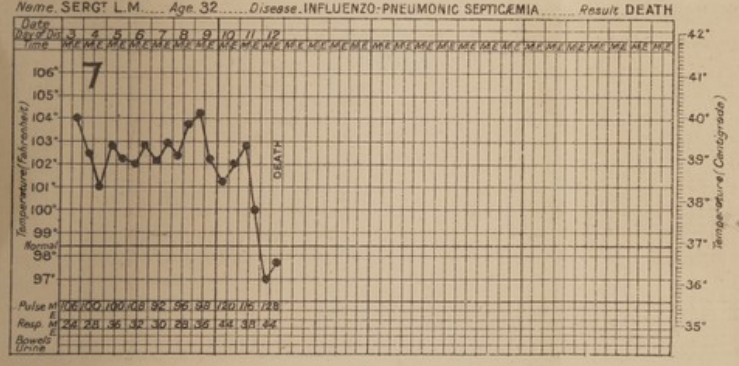
Name B. Age 40. Disease INFLUENZA WITH PNEUMONIC SEPTICÆMIA. Result DEATH.



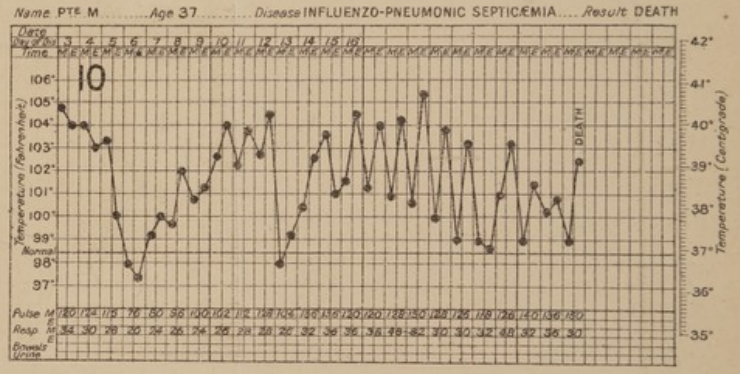
Name CADET C. Age 21. Disease INFLUENZO-PNEUMONIC SEPTICÆMIA. Result DEATH



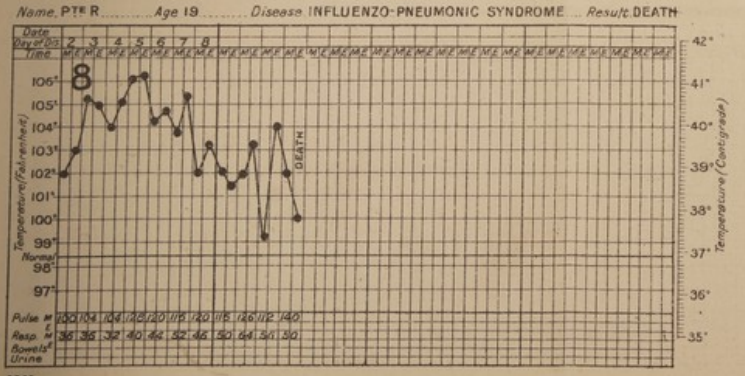
Name SERGT L.M. Age 32. Disease INFLUENZO-PNEUMONIC SEPTICÆMIA. Result DEATH



Name PTE M. Age 37. Disease INFLUENZO-PNEUMONIC SEPTICÆMIA. Result DEATH



Name PTE R. Age 19. Disease INFLUENZO-PNEUMONIC SYNDROME. Result DEATH



CHARTS OF 5 FATAL CASES.

2263

H. & C. GRAHAM LTD. LITHO'S LONDON, S.E.

Some cases had hardly any sputum at all. Frothy mucoid or muco-purulent phlegm was much more common. It had no particular odour. Sometimes there was nothing but pure froth; again there might be froth only with some bouts of coughing; pellets or dollops of muco-pus at other times in the same patient; the individual expectorations remaining separated from one another in the disinfectant in the sputum jar. Or again, the sputum might be glairy and mucoid or stringy; or it might be simply purulent, the successive expectorations running together in the receptacle like pure pus. The colour was equally variable; sometimes the sputum was all white froth; or the froth might be tinged with blood streaks; or dollops of pure clotted blood might be coughed up separately from the froth; or there might be liquid red blood expectorated in a way recalling a moderate hæmoptysis from phthisis; or the more purulent type of sputum might be tinged red or brown with altered blood, or it might be definitely glairy rusty sputum like that of ordinary lobar pneumonia; the same patient might bring up different types of sputum on successive days, and in the same ward successive patients, suffering apparently in a similar way, might be bringing up each a different type of sputum, so that no conclusion could be drawn from the appearances of the latter as to what degree of the malady the patient had.

Occasionally the colour attracted notice; instead of being white or very pale yellow, red, or brown, it was bright orange; or in other cases pale green, or greenish yellow, or bluish yellow, or greenish blue, or pale blue; apparently these peculiarities in colour were due to variations in the pigments produced by the micro-organisms present.

The Physical Signs.

The physical signs varied widely in different cases of similar type. As a general rule what struck one most was the frequent paucity of abnormal signs, when the degree of illness of the patient led one to expect many. Percussion and stethoscopic evidence of extensive consolidation was often conspicuous by its absence. Before one gained experience one thought the case was one in which this absence of the familiar signs of pneumonia was due to the consolidation being seated too deeply at the moment to be detected, and one expected dulness, bronchial breathing and pectoriloquy to manifest themselves the next day or the day after. In a small proportion of cases this proved to be so; and the unwary would regard the signs as indicating lobar pneumonia beyond doubt; but the autopsy findings would prove the lobar consolidation to be due to the conglomerate lesions mentioned previously, and not to true croupous pneumonia. In a very large number of cases, on the contrary, the classical signs of lobar consolidation never developed at all.

There might be a few scattered rhonchi over the front of the chest, and over the upper part of the lungs behind, changing from rhonchi to non-consonating rales as one passed down to the bases; but without dulness, bronchial breathing or pectoriloquy, even rales and rhonchi might be difficult to find in some cases. Again, there might be little more than deficient vesicular murmer at one or other base—the kind of condition leading one to say, "the consolidation is here and will show itself more definitely to-morrow"; and yet it did not. Again, amongst the scattered rhonchi and mucous rales one might come across a patch or two of rales that were almost or quite consonating, yet without bronchial breathing or pectoriloquy; such patches might persist, or develop further, but quite as often they became less obvious within an hour or two, or might disappear entirely. In not a few cases one might note definite bronchial breathing with crackling rales and pectoriloquy over a quite considerable area of a lower lobe at one time, and yet later on the same day the bronchial breathing and pectoriloquy might have disappeared entirely, giving place to ordinary vesicular murmur, rhonchi and non-consonating rales again; suggesting that there had been temporary collapse. On the other hand, especially in cases that survived several days, the areas, particularly in the lower lobes, in which crepitant rales could be heard, might increase until such rales would sometimes be audible all over both lower lobes, yet without bronchial breathing; the percussion note at the bases being impaired but not dull; or there might be definite dulness over one or both lower lobes with crepitant rales, bronchial breathing, bronchophony and pectoriloquy; or again basal dulness with absence of breath and voice sounds might be found on one or both sides, sometimes the result of massive collapse, sometimes of compression by pleuritic effusion. Whatever the other signs, pleural friction was to be heard in a large number of cases, though detection of the friction was often rendered difficult by the fact that patients tended to take rapid short shallow breaths, so that it was not always easy to get them to make an inspiration deep enough to render the friction audible. Such friction was most often to be found posteriorly or in the axilla, but it might occur anywhere from apex to base; the physical signs of consolidation, on the other hand, when they did develop at all, were almost always in the lower lobes, apical consolidation signs being remarkably uncommon, though met with in exceptional cases.

The extensiveness or otherwise of the physical signs, however, seemed to bear little relationship to the degree of illness of the patient; nor to his cyanosis. A man might be heliotrope blue with hardly any abnormal lung signs to be detected; or he might have signs suggesting consolidation of both lower lobes and not be blue at all. It was by the blueness and not by the physical signs that the prognosis could be measured best, and one saw quite a number of cases in which the patient went

the heliotrope colour and died without exhibiting any material departure from the normal as regards pulmonary physical signs.

Herpes of the Face and Herpes of the Ears.

Herpes facialis varied greatly in its incidence amongst different groups of cases. One might see several hundreds consecutively with none at all; and then come across a number with it. I should say that its occurrence was exceptional on the whole—less than 3 per cent.—and yet it might chance that one would occasionally find upwards of a quarter of the cases in a single ward presenting this symptom. When it did occur it was generally of just the same type as that seen in ordinary pneumonia, affecting the lip margins, chin, or alæ nasi; but in exceptional cases it was more like the type seen in spirochætosis icterohæmorrhagica, the massed vesicles and their inflamed bases extending all over the chin and lips and nostrils, out on to both cheeks, and down on to the neck, becoming hæmorrhagic and covered with bloody scabs The occurrence of herpes facialis, even of this exceptionally severe type, did not imply that the patient would not recover.

Several striking instances of bilateral herpes of the primæ were met with, both with or without herpes of the lips. The grouped vesicles developed on all parts of the prima, more often on the external surface and free margin than on the posterior surface, incommoding the patient mainly by rendering it painful for him to lie with either ear turned down to the pillow, but otherwise not related, apparently, to any other special feature of his case.

Skin Rashes and Lesions.

Apart from the herpes of lips and ears, and from the development of sudamina or miliaria in perspiring cases, pronounced skin lesions were exceptional.

General flushing, especially of the face and neck, was common; but in a very few cases did this flushing reach a degree that could be called definite generalised erythema. Now and then one met with a case, reddened sufficiently to rouse a suspicion of scarliatina; but the erythema differed from that of scarlet fever in that it was not punctate, and it involved the face and neck as well as the trunk and limbs.

Morbiliform eruptions were very rare, and when met with there was always a question as to whether the patient had not really got measles or German measles as well as influenza; so many thousands of individuals were ill together that the co-incidence of measles or almost any other illness with the influenza was almost sure to be met with; one could not say, therefore,

that the influenza itself was the cause of the few morbiliform rashes seen.

Generalised purpura was remarkably rare; in so toxic an infection one was on the look-out for it, and expected it; but it was very rare indeed, even in the worst cases; though I saw it in four, all fatal. Less uncommon was a localized purpuric eruption on the legs, below the knees, though even this was rare. Its local occurrence was not necessarily followed by death. In two cases, after a shower of purpuric spots upon the feet and ankles, hæmorrhagic bullæ developed upon the sites of the original small petechiæ, associated with extensive œdema of the toes, feet, and ankles, great pain in the feet, and tenderness; the bullæ subsequently ulcerated, and the patients, though surviving several days, ultimately died. This purpuric-vasculo-ulcerous state was bilateral and more or less symmetrical. I saw no others of this type, and I do not know the nature of this form of lesion, though I imagine it was vascular and, perhaps, the result either of multiple embolism or of multiple thrombosis of venules in the extremities.

Larger and smaller tender areas of erythema in various parts, not hæmorrhagic, were complained of by many patients; as a rule they subsided spontaneously and they did not attract great notice; but looking back at them now it occurs to me that these may have been similar lesions of lesser degree.

Epistaxis, Hæmoptysis, and other Hæmorrhages.

Epistaxis has been referred to already; it was strikingly common at the onset, usually slight, sometimes so severe as to last 24 hours or more. The fact that it was so common suggests that the naso-pharynx was an important focus from which the germs of the generalised malady were absorbed—a point debated later.

Hæmoptysis was also common, though not so common as epistaxis. The latter occurred in the simple mild cases as much as in the severe; hæmoptysis was a phenomenon of the "pneumonic" cases mainly. Occasionally, no doubt, it was due to the influenzal infection lighting up a latent tuberculous lesion; but quite apart from this, true hæmoptysis, with the expectoration of bright red liquid blood in amounts varying from half a teaspoonful to several ounces was met with so often that it could not have been accidental, over and above which it occurred in cases in which subsequent autopsy showed no tuberculous focus to be present. Similar hæmoptysis does occur in croupous pneumonia, but not with the frequency it did in this epidemic.

Apart from actual hæmoptysis, blood-tinging of the sputum was common enough—as blood streaks; or general staining with bright red; or as a discolouration from altered blood—dull red, orange, rusty, brown, blackish-red, or almost tarry black; typical glairy rusty sputum was exceptional.

Hæmatemesis was observed many times; as a rule it was difficult to exclude the possibility of the blood from the stomach having been derived originally from the nose, swallowed and then vomited; but in a few cases it seemed almost certain that the blood was derived from the stomach itself—perhaps a diffuse oozing from an injected gastric mucosa. If blood was vomited at all it was generally of considerable amount—many ounces. There was no marked tendency to repetition of the hæmatemesis; the prognosis was not necessarily bad; no ulceration of the stomach was found in the fatal cases but acute congestion of the mucosa was common.

Bleeding per rectum was noticed in a few cases only. Dr. Lowe has recorded extensive ulceration of the colon in fatal influenza, so that the passage of blood per rectum is to be expected sometimes; but it was not commonly a sympton to attract notice. Whether occult blood was often present in the stools I cannot say; probably it was, if only because of the frequency of epistaxis and the liability of the nasal blood to be swallowed when the patient is recumbent. Pronounced melæna attributed to this cause was observed more than once.

Amongst women, a tendency to excessive uterine bleeding was exhibited by some when the menstruation came on during the attack; but there were many cases in which this tendency did not show itself at all; and although I have heard from others that spontaneous uterine bleeding occurred sometimes, unconnected with a menstrual period, this was not noticed in cases I saw myself.

Hæmaturia, sufficient to colour the urine with blood, was very exceptional. Nephritis was constant in the fatal cases, and albuminuria, suggesting nephritis was common in many that survived; red blood discs were sometimes found in the urine microscopically in such cases, but definite hæmaturia was rare.

I cannot say whether spontaneous retinal hæmorrhages occurred or not; there was little time or opportunity for retinoscopy during the epidemic and no obvious retinal changes were observed in the small number of cases thus examined.

Hemiplegia, followed by recovery, developed in one case during the acute influenzal attack apparently due to hæmorrhage; there was insufficient evidence, however to prove that the influenzal attack was the sole and essential cause of the seizure.

Delirium and Coma.

Delirium and coma occurred often enough amongst the bad cases, but far more striking than their occurrence was their entire absence almost to the very end in so many instances. Big strong men, heliotrope blue and breathing 50 to the minute, obviously dying, would be fully conscious, talking rationally on almost any subject, relatively clear-headed to within half-an-hour of death; often not realising in the least how dire their condition was.

When delirium did occur it was seldom of the noisy, trembling, shouting-out type, though cases of this sort occurred too. More often it was of the low muttering restless type, the patient picking at the bed clothes and at objects round about him, with rambling talk or incoherence ; getting out of bed, but readily getting back when asked to ; rational for a few moments when spoken to, able to answer questions, and yet lapsing quickly into a restless semi-conscious state when left. Another type of case became totally unconscious hours or even days before the end, restless in his coma, with head thrown back, mouth half open, a ghastly sallow pallor of the cyanosed face, purple lips and ears—a dreadful sight.

It is noteworthly that the Cheyne Stokes type of breathing was very seldom met with even in those deeply comatose; perhaps because of the extreme anoxhæmia.

Subsultus Tendinum.

Subsultus tendinum was a marked phenomenon in many cases quite apart from delirium or coma. The patient might be rational enough to be talking about himself and he might even himself call attention to the way he could not keep his legs or arms or back muscles quiet when he wanted to. A leg or an arm or the abdominal muscles or the back would give an involuntary twitch or jerk; or the face or one shoulder, or some other part ; the character of the condition approaching in a few instances almost to that of mild chorea. As a rule the prognosis proved bad, though the patient was by no means always in extremis when the symptom first developed.

Headache.

Headache was a pronounced symptom in nearly all the cases, the simple influenzal as well as the "pneumonic." Sometimes the whole head ached and throbbed ; sometimes the head did not ache if the patient kept quite still, but swam and ached all over if it was turned quickly or if the patient sat up or coughed. Besides this generalised headache, however, and often in addition to it, there was complaint of special aching "at the back of the eyes" ; or "inside the head in front," the patient generally putting his hand low down across the forehead to indicate its site. The more generalised headache was doubtless due to the toxæmic state, thus corresponding with the aching limbs and back ; but some light on the different nature of the ache "inside the head behind the eyes" is thrown by the autopsy findings in the sphenoidal and ethmoidal air cells. In 22 consecutive cases in which these were opened up they were found to be obviously infected in 21 ; and in over half of these the sphenoidal air cells were not merely inflamed, but they contained definite turbid fluid or actual opaque pus. It seems probable

that infection of these sinuses is common, and this may be the cause of the peculiar headache "right at the back of the eyes" that so many influenza patients complain of, either at the beginning of the illness, or during it, or even after the acute phase has passed off.

Nephritis.

In all the autopsies that I did or saw—between 100 and 200 in all—there was no instance in which the kidneys were not definitely inflamed; the common lesion being that which gives the swollen blood-oozing kidney. It would be unlikely that every case developing this acute nephritis should die; doubtless many recover, some completely, some with residual renal defects. Few cases, however, presented the ordinary clinical picture of acute nephritis for there was no œdema to attract notice. It is probably too much to say that no case of generalised nephritic œdema occurred at all; but I saw none, even when acute nephritis was demonstrated post-mortem. This raises the old difficulty of trying to decide whether, when albuminuria without œdema occurred in the patients who recovered, the condition was to be labelled merely "febrile albuminuria" or actual "nephritis." Albuminuria at some stage of the malady was common; all the cases that died seemed to have acute nephritis; the inference seems to be that at least some of the albuminuria cases that survived have some degree of actual nephritis also. One has not had the opportunity of tracing these cases since, so that one does not know what the condition of their urine may be now; but one feels that there is at least the possibility that some, regarded as cured, may come under notice again as chronic tubal nephritis cases when a few years have elapsed. If they do so the possible origin of their kidney disease may be unknown and they will then fall into the category of Rose Bradford kidney cases.

There being no œdema at the time of the acute disease, the urine has not always been tested; when practitioners were all so overwhelmed with cases that it was sometimes days before they could get to visit the patient at all, there was no time to test urines as a routine procedure, and how common, therefore, albuminuria was one cannot say; but it was certainly common in the severer type of cases. It would be thought perhaps that microscopical examination of the centrifugalized deposit for renal tube casts would have settled the questions of whether the albuminuria was "nephritic" or merely "febrile"; but this is not so, for even when acute nephritis was demonstrated post-mortem the urine during life had generally shown few casts, if any. Broken down renal cells with red blood discs were observed far oftener than casts; the lesion was so acute that the cast-forming stage had not been reached; hence, absence of tube casts will not exclude acute nephritis in the living, and

enable one to label the albuminuria merely "febrile." Personally, I have the feeling that acute nephritis was common in these cases; that it generally recovered quickly; but that in some cases it may have damaged the kidneys to an extent we do not yet know, the existence of nephritis during the influenzal illness having passed unnoticed because there was no œdema to attract attention.

Rarer Complications.

Acute Subcutaneous Emphysema of the Chest Wall.

Every now and then, perhaps once in 500 cases, one met with a very striking sympton, namely, spontaneous emphysema of the chest wall, with widespread and characteristic crackling beneath the palpitating hand. I saw 12 instances of this and all of them died, but I have heard of one or two which recovered. The patients were all of the severe type, but not necessarily in the cyanotic or hopeless stage when the accident happened. The escape of air into the subcutaneous tissues occurred over the front of the chest first in nearly all, spreading thence for variable distances until neck, shoulders, chest, back, and abdomen might all be crackling in a similar way, much as when the same accident results from a broken rib.

Sometimes the escape comes on immediately after a severe spasm of coughing, but it has also developed without obvious relationship to any coughing bout; the patient, for instance, may wake up to find himself crackling when he touched his chest.

The pathology of the condition is not infection by gas-producing organisms; it is due to escape of gas from within the lung through ulcerated apertures in the two layers of pleura, the ulceration in its turn being due, in my belief, to infection from minute abscesses in the underlying lung close beneath the visceral pleura. At autopsy one frequently finds half-a-dozen or more tiny abscesses, each the size of a pea or thereabouts, aggregated together in the central part of a hæmorrhagic and consolidated portion of lung; the pale abscesses, small though they are, contrasting markedly with the dark red consolidated lung in which they have formed, the infection being so acute and the abscesses so small, one of them now and then leads to an ulcerative puncture through both layers of pleura, the effect of which is to cause, not a pneumothorax but subcutaneous emphysema, just as a fractured rib causes not a pneumothorax as a rule but an escape of air into the subcutaneous tissues. Were the abscesses not so small, pneumothorax would be more likely perhaps. I have met with no case of spontaneous pneumothorax in this epidemic.

Hæmorrhage into, or Spontaneous Rupture of, Rectus Abdominis Muscle.

Quite a number of cases of spontaneous rupture of one or both rectus abdominis muscles have been met with, and in a still larger number the rectus abdominis muscle, short of actually rupturing, has been found at autopsy to be in a hæmorrhagic necrotic state such as precedes rupture. Almost invariably this lesion has affected only that quadrant of the muscle which is below the level of the umbilicus, and I have not met with similar hæmorrhage into, or rupture of, any other muscle, though I imagine that others besides the rectus abdominis must be affected sometimes. Possibly coughing efforts or some other mechanical reason is responsible for the rectus abdominis being most often involved; apparently the disorder is the result of vascular changes within the muscle—venous thrombosis it is thought. The affected muscle has much the appearance of the breast of a bird that has been badly shot at close quarters, the muscle being soft, pulpy, and infiltrated with dark extravasated blood. The lesion is not always obvious during life because the whole of the muscle sector is involved at once; one does not get, therefore, the drawing apart of the two normal ends with the depression in the centre, which is the characteristic sign of rupture of a healthy muscle.

If the patient is not already too ill to complain he tells one of acute pain in the lower part of the abdomen, either central between umbilicus and pubes, or to one or other side of this. In one such case, when the lesion affected the right rectus abdominis muscle only the pain was so severe, localised and associated with unilateral muscular rigidity over the right iliac fossa that it simulated acute appendicitis, and it was only after urgent operation had been performed that the error was found out. The appendix was normal; the right rectus abdominis muscle was crimson black from hæmorrhagic necrosis. This patient recovered completely; had it not been for the operation I should have had no idea of the real nature of his attack of acute and persistent pain, and I feel that the complication may have been even commoner than the ten or more cases in which I saw it demonstrated led me to suppose.

Jaundice.

Jaundice was quite uncommon, and when it was met with it was generally confined to a small percentage of cases in one particular district, other districts providing none. Its degree and type were similar to those of ordinary catarrhal jaundice; there was pallor of the stools and darkening of the urine, and the impression one got was that the jaundice was simple catarrhal-obstructive and not hæmolytic-toxic jaundice; the patients might do very well in spite of their jaundice, and although one might have expected to meet with hæmolytic-toxic jaundice of

grave omen in at least some cases of so serious a microbic disease, I met with none personally.

Parotitis.

Unilateral or bilateral parotid inflammation was not uncommon; it was associated with pain and tenderness over the swollen gland, but generally no reddening; it was quite exceptional for abscess formation in the gland requiring incision and drainage, to supervene. At first it was thought that the non-suppurating cases were instances of mumps coinciding with influenza, but this view was altered when the parotitis was found in cases that had had mumps before, and when, in case after case, the swelling was confined to the parotid glands, very seldom spreading to the submaxilliary and sublingual glands as mumps generally does. Apparently the parotitis was secondary to infection of the ducts from the mouth, corresponding to the similar parotitis that used to be common after abdominal operations before the toilet of the mouth was attended to more carefully than it is now. The parotitis was commoner in the severer cases than in the milder, so that many of the patients exhibiting the mumps-like facies died; but intrinsically it did not appear to be a grave sign.

Pericarditis and Peritonitis.

Some observers reported pericarditis as a complication, but I saw no case of this at autopsy, and observed no case of pericarditic friction during life. It was remarkable that this should be so, seeing how common acute pleurisy was, and how virulent the general infection; many cases having living micro-organisms in their blood stream. Peritonitis was equally rare. I saw no case with acute general pneumococcal or streptococcal peritonitis.

Panophthalmitis; Cancrum Oris; Noma.

Panophthalmitis was seen twice; cancrum oris and noma not at all. It speaks volumes for the general efficiency of the nursing of the cases that these dread results of uncleaned eyes and mouth were conspicuous by their absence.

Meningitis.

Acute meningitis occurred in a small but definite number of cases, its incidence being sufficient to attract special notice.

The cases in which it occurred may be classed under three headings, viz. :—

(*a*) Meningococcal cases, not influenzal at all, but mistaken at first for instances of the prevailing epidemic.

(*b*) Meningococcal cases, in which the primary illness was influenza, the meningococcal meningitis developing during the course of the influenza or before convalescence from the latter was complete.

(*c*) Acutc meningitis, not meningococcal, but due to one or other of the infecting organisms of the influenzal attack—Pfeiffer's bacillus, diplostreptococci, pneumococci or streptococci being recovered from the cerebrospinal fluid.

It was always difficult at the time to be sure into which of the three groups to put the cases ; and there may always be doubt as to whether groups (*a*) and (*b*) are distinguishable at all ; but one's impression, based on some scores of cases, is summarised above. The diagnosis was arrived at by lumbar puncture and bacteriological examination of the cerebrospinal fluid. Most of the meningitic cases proved to be meningococcal and it was only by studying the history of each and the charts and the course of the disease that one could see whether the disease was meningococcal meningitis from the start or whether there had been influenza first, meningococcal meningitis supervening during its course. That some were not meningococcal meningitis from the start seemed certain, because sometimes the patient had had typical influenza, with a subsidence of pyrexia and symptoms, and a partial convalescence before the meningitic symptoms developed. It is interesting moreover, that—although actual statistics are not available—one met with a larger number of these meningococcal meningitic cases amongst a given number of the influenza cases than one would expect to find even under conditions of crowding amongst an equal number of healthy individuals. For instance, in one hospital of 600 influenza patients four meningococcal meningitis cases developed in one week ; in all of these there seemed little doubt that their initial illness had been influenza ; and their previous history showed no common source from which they could all have derived the meningococcal infection. I saw over 20 instances of the association in all. The impression one formed was that the influenzal infection opened up the path for absorption of meningococci from the posterior nares and rendered carriers of meningococci more liable than usual to develop actual meningitis. I quite realise, however, that this is only an impression, without logical proof.

Apart, however, from meningococcal cases there were a few instances of acute fatal meningitis in which no meningococci were found, and the organism recovered from the cerebrospinal fluid was either Pfeiffer's bacillus or one of the

infective cocci associated with the latter in the patients' complex influenzal attack.

Otitis Media; Ethmoidal and Sphenoidal Sinus Infection.

Earache was a fairly common symptom, especially in younger patients; and when the epidemic swept through a boys' school, for example, there was considerable liability for one or more of the boys to develop acute otitis media followed by otorrhœa either during the acute influenzal attack itself or before recovery from it was complete. Amongst grown-up people the proportion of acute otitis media and otorrhœa cases was less, though temporary deafness of one or both ears, with pain suggesting temporary catarrh of the middle ear and mastoid cells, was not uncommon.

Infection of ethmoidal and sphenoidal air cells was rarely recognised during life; but that it must have been almost common seems probable from the post-mortem findings. These regions were not opened up in every fatal case, but they were in 22 consecutively, and in only one of these did the interior and lining membrane of the sphenoidal air cells look normal; in the remaining 21, the lining membrane was congested and red; in six there was definite opaque pale yellow pus filling the air spaces; and in the remainder there was turbid fluid which in every case yielded on culture the same micro-organisms as were recovered from the lungs.

The sphenoidal air cells were more constantly inflamed than the ethmoidal, to judge from the naked eye appearances; the frontal air spaces less often still; bacteriologically, infection was almost constant. This seems to have an important bearing on the nature of the headaches which accompany influenza and which trouble some patients long after the influenza itself is past.

Infective Endocarditis.

Infective endocarditis was met with in no case post-mortem, and during the epidemic at any rate was not observed during life, but it is noteworthy that in the months which succeeded the epidemic, and indeed throughout the year 1919, one was constantly meeting with cases of the chronic type of infective endocarditis, whose origin was obscure. In many of these cases there was an old-standing valvular lesion of the heart, but in some there was no known previous heart disease; so chronic were many of these cases that some survived for many months after the diagnosis became clear as the result of the changes in the cardiac bruits, the progressive anæmia, the multiple emboli and the development of a palpable spleen, all associated with more or less pyrexia of long continued type.

Whereas in ordinary times it is exceptional to have more than one case of infective endocarditis under one's care at Guy's Hospital, during 1919 one has had as many as five patients in the ward suffering from this disease simultaneously, and altogether I have seen over 70 cases in the course of twelve months, in many of which in addition to the typical clinical picture the diagnosis was confirmed by autopsy.

It would be a very difficult thing indeed to prove that the occurrence of so many cases of infective endocarditis in the year following the acute influenzal epidemic was really in any way due to that epidemic, but one began to meet with the cases in December 1918 and already by March and April 1919 one had become convinced that there was something remarkable about them and one wondered whether these chronic months-lasting infective endocarditis patients might not be, as it were, an aftermath of the virulent infections of the acute influenzal epidemic types. In none of these cases did one find influenza bacilli in the blood, a form of streptococcus being the usual micro-organism recovered. The patients had generally been "seedy" for weeks or months before they had to take to their beds and the diagnosis of infective endocarditis became clear; and one wondered whether the infecting micro-organism had not settled upon the heart valves at the time when there was the world-wide virulence of infecting micro-organisms which, taking in the main the form of an acute influenzo-streptococcal, or influenzo-pneumococcal illness, might in certain individuals have led to some focal infection, of which subacute or chronic infective endocarditis might have been one. One does not know whether there was any similar frequency of infective endocarditis after the virulent influenza outbreaks of 1889, 1890 and 1891, but it will be interesting to watch whether, after any future acute influenza epidemics, a similar frequency of infective endocarditis shows itself during the twelve months which succeed that future acute epidemic.

Localised Pus Formation.

Acute pleuritic effusions, generally small in amount and yielding turbid fluid rather than definite pus, were quite common in the "pneumonic" cases; this turbid fluid contained polymorphonuclear cells microscopically and yielded cultures of pneumococci, diplo-streptococci, or streptococci bacteriologically, but it was not very common for these effusions to develop into definite empyemata. Many such effusions, though containing turbid fluid, excess of leucocytes and micro-organisms resolved spontaneously; some were found post-mortem in the fatal cases, but a few became definite empyemata, and it was noted as a rather remarkable feature of such cases that once definite pus developed in the chest, the patient ultimately did well, no matter how ill he might appear to be at the time.

The same seemed true of other cases in which localised collections of pus developed elsewhere than in the chest. In two or three instances, for example, successive subcutaneous abscesses, pyæmic in type, had to be opened and drained with recurrence of fresh abscesses for several weeks, and yet, after a time of great anxiety, the patients made a complete recovery. In another type of case—quite exceptional—subcutaneous suppuration developed at the site of saline injection. Almost without exception any patient in whom any local abscess or empyema or similar focal suppuration occurred, ultimately did well. To such extent did this impress more than one observer that deliberate attempts to produce a spontaneous abscess, not by injecting extraneous organisms but by focalising the patients' own organisms, were made in the belief that if such "fixation" abscesses could be produced, the patients would do as well as those in whom spontaneous suppuration occurred. When, however, one tried to produce suppuration in this way by trauma, or by the subcutaneous injection of irritants such as turpentine, one did not once succeed in obtaining a local "fixation" abscess; and yet it is a point that merits special emphasis, that whenever spontaneous suppuration did develop in these influenzal "pneumonic" patients, one could almost invariably assume that the patient was going to pull through and get well, even though his actual state at the time might otherwise look precarious.

Age and Sex Incidence.

No age and neither sex was free from the danger of infection, and the "pneumonic" type of influenza case was met with in infants, children, boys, girls, young adults, full-grown people, and the aged. One's opportunities, however, brought one much more into touch with those of military age than with either the very young or the very old, and one formed the impression that the incidence of the disease—unlike that of 1890–91—was considerably greater in those between the ages of 20 and 50 than in those below this age period, in addition to which it was people of these ages who were mostly aggregated together in camps and barracks, juveniles and old people being spread wider apart and therefore less liable than the military to develop the disease by direct contagion when the first case in the neighbourhood fell sick. Apart, however, from the age incidence of the disease there was a very definite age incidence of the fatalities, and it was a striking feature of the epidemic that strong, healthy adults, especially those between 20 and 40, and more particularly between 25 and 35 years of age, were those who were most liable to succumb to the dreaded "pneumonic" type of the infection. Stricken with the same disease at the same time, and from the same source, with pneumonic complications, the chances of a man of 55 pulling through seemed

better than the chances of a man of 25 or 30. In this respect the epidemic of 1918-19 appeared to be entirely different to epidemics of the past in which, as a rule, the deaths have been largely among the elderly or the very young.

The Route of Infection.

It seems likely that the route of infection was not invariably the same, but one received a strong impression that the naso-pharynx and the nasal passages were a highly important focus from which the spread of the offending micro-organisms took place in a very considerable number of the cases. There is no absolute proof of this view perhaps, but there are several highly suggestive points which lead one to this opinion and, if correct, the view is of considerable importance in connection with the need for nasopharyngeal cleansing or antiseptic "toilet" as a preventive measure. Some of the points which would seem to indicate that the infection is at first localised in the naso-pharpnx, thence spreading to cause the more general disease, are as follows :—

In a very large proportion of cases epistaxis (p. 80) was the initial symptom, and occasionally this epistaxis was in itself quite severe. The occurrence of such epistaxis, spontaneously, in so many individuals suggests that there must have been some common cause for a breach of the surface of the nasal mucosa ; this breach occurring as a rule at the very commencement of the attack, or even before the patient realised that influenza was upon him. The likely common cause would seem to be acute congestion from a microbic invasion of the lining of the nose, in which case if the germs of the disease were the cause of the inflammation, there would be a ready path for themselves or their toxins to enter the blood stream and cause the acute disease.

In the next case the constancy with which acute infection of the sphenoidal air cells was found (p. 88) with actual pus in these cells in a considerable percentage of the fatal cases, would seem to afford strong evidence of there being posterior nasal infection tending to spread into the parts in this immediate neighbourhood.

The relative frequency of earache, otorrhœa (p. 88) and deafness indicating similar spread to the middle ear via the Eustachian tube, points in the same direction ; whilst from the mouth the way the micro-organisms tended to spread into the ducts connected with it, was shown by the development of parotitis (p. 86).

Again—though perhaps less cogent as an argument—the way in which meningococcal meningitis (p. 86) tended to occur in a larger proportion of these influenzal cases than one would expect from its incidence in the population generally, led one to think that this might be due to the influenzal infection in

the nose opening up channels through which the meningococci latent in the posterior nares could obtain a path of entry to cause their attack upon the meninges.

When bacteriological examination of the posterior nares of the apparently healthy contact cases was made the same varieties of organism as abounded in the tissues of infected patients would be found, but this, perhaps, is not an important argument in favour of the posterior nares being the primary focus from which the disease would subsequently develop, because even amongst the healthy individuals in non-epidemic times so many varieties of bacteria are found in the posterior nares that their recovery in influenzal contact cases need not necessarily have indicated a departure from the normal. The above points, however, and especially, perhaps, the common epistaxis and the frequency of infection of the sphenoidal cells led one to think that the nasal passages, and especially the posterior nares, were an important nidus for organisms which, by further spread, produced the disease; and one could not help feeling that an important means of checking development of the disease in contact cases was to wash out the nose and mouth and the posterior nares as adequately as possible at least twice each day, using for the purpose a bland antiseptic, mild enough not to irritate the mucous membrane, and yet sufficient to assist in keeping these parts clean.

The whole question of prophylaxis is dealt with elsewhere by others, but I wish to emphasize my belief in the importance of the nose, the naso-pharynx, mouth and fauces in this connection and in their relation to the route of infection in many at least.

Treatment.

I do not propose to go into the question of treatment at any great length, for once the influenza attack in any individual case had developed into the "pneumonic" type, it appeared that, no matter what treatment was adopted, it was extremely difficult, if possible at all, to modify the course of the disease in the least; and it was borne in upon one with the strongest emphasis throughout the epidemic that *the* thing to pay attention to was prevention rather than cure.

In the average case best results were obtained if the patient, directly he felt ill at all, went straight to bed and stopped there; those who tried to keep about in spite of having the disease upon them not only ran unnecessary risks themselves but also did harm, even if they did so with the best intentions, because, by keeping up and about, they were sources from which others became infected.

The patient should go to bed at once, take a tumbler of hot whiskey and water, 15 or 20 grains of aspirin, and cover himself up with a sufficiency of blankets in the hope of breaking out

into a good perspiration. There seemed little to be gained by giving any food at all; even milk was best avoided for 48 hours, provided that the patient during this time drank as much fluid as he could manage, either as plain water or as barley water, lime juice, lemonade, weak tea, soda water or iced water. Five pints a day would not be too much. If need be a simple aperient, such as castor oil, would be given at the beginning, and for the rest the treatment resolved itself into one of skilful nursing without the administration of any drugs, unless there were some symptom calling for individual treatment. Aspirin, for example, to relieve the headache, or to give sleep; a sedative mixture to relieve cough until such time as there is phlegm to be brought up when the sedative mixture might be changed for an expectorant one; a more potent hypnotic if there were sleeplessness in spite of aspirin. If definite bronchitic catarrh develops, leading to the dreaded "pneumonic" complications, one would be only too thankful if one knew of anything which would with any certainty check the disease process, but one met with nothing that was in the least degree successful in this respect. In a few cases, say a hundred or so consecutively, one might think that one particular remedial line one had adopted was giving undoubted benefit and then in the next two or three hundred cases treated in exactly the same way the same treatment would prove entirely disappointing. From experience, extending over thousands of cases, the general conclusion was that the nursing was of infinitely greater importance than the drugs administered. This applies not only to drugs but also to the use of antiseptics either by inhalation or by injection, and also to attempts at specific antibacterial treatment by means of vaccines or by sera. All kinds of antiseptics given by inhalation were tried and they were given continuously, or intermittently, for longer or shorter periods, and all without obvious benefit. In the belief that injections of antiseptics into the blood stream might do good, such things as flavin and eusol were given intravenously but without any apparent benefit. Patients were venesected without good. Infusion with normal saline either subcutaneously or intravenously or both, also gave no benefit. Some patients were both venesected and infused, but unavailingly; that is to say, the mortality was much the same amongst cases treated thus and cases in which practically no treatment other than nursing was adopted.

For the cyanotic cases venesection does no good, for the cyanosis is not due to cardiac failure but to anoxhæmia resulting from the albuminous exudate into the alveoli, interalveolar tissues, and tubes, in addition to whatever broncho-pneumonia or hæmorrhage has also developed. In theory, the correct treatment for this anoxhæmia would seem to be the continuous inhalation of oxygen, and repeated attempts to give this oxygen continuously by means of Professor Haldane's special apparatus were made; but in very few cases, even amongst those who were

willing to persist with the treatment, did the giving of oxygen in this way seem to really benefit the patient, whilst, on the other hand, in the great majority of cases it was only by urging and insisting that one could get the patient to submit to keeping the oxygen mask on; the wearing of it seemed more often than not actually to increase the patient's distress and he was generally only too thankful to have it taken off. At the same time one does feel that if oxygen could be given continuously to the cyanotic type of case, without at the same time creating a sense of distaste to the treatment on the patient's part, it ought to be beneficial and the right thing to use. One wonders whether in future cases, should they occur, it would not be a better plan to give the oxygen through soft rubber catheters inserted through the nostril so that a constant stream of oxygen might thereby pass into the patient's respiratory passages without producing that sense of oppression and suffocation that the patients complain of when wearing a face mask. I saw no cases treated in this way, but I think it would be a procedure worth a trial should any similar occasion arise, using the technique described by Stokes and Ryle (*Guy's Hospital Gazette*, 9th August 1919) in connection with cases of acute gas poisoning in France.

It is difficult to give any opinion as to whether the patients should be nursed in warm rooms or in cold, or even out of doors; at the beginning of the attack warmth, and the production of perspiration, certainly does seem to tend towards a favourable course in the disease, so that in the early stages at any rate it would seem advisable to have the patient in warm, though well ventilated, rooms or wards. On the other hand, when one had the opportunity of seeing the severe "pneumonic" cases verging upon cyanosis, or with the heliotrope colour actually developed, sometimes nursed in relatively warm wards and sometimes put right out of doors with waterproof canopies to keep off the rain, and warm clothing to keep their bodies warm, though their faces were exposed to the autumn or winter air, there could be little doubt that of these two alternative procedures, the outdoor and apparently more drastic line of treatment gave the better results. In some hospitals where there was immense strain upon the available beds, it became necessary to put the worst, and apparently hopeless cases, elsewhere, in order to make a maximum amount of room for less bad cases that seemed recoverable, and one saw scores of extremely bad cases transferred from the wards to the quadrangles out of doors, under waterproof canopies, and whereas at first one felt that this—though a necessary procedure—would at least not help any of these dire cases to get better, one found to one's surprise that a larger number of those very worst cases put out of doors did, as a matter of fact, recover than would have been the case, one felt sure, had they remained indoors One is therefore in this difficulty: one feels that at the beginning of the attack the

patient should be nursed in warmth; that if "pneumonic" cyanosis has developed he is better out of doors; but that one does not know just at what stage the change of conditions of nursing should be made for the best advantage of a bad case.

Although one formed no favourable impression of any of the vaccines or sera used by oneself during the epidemic, one equally feels that further researches by those skilled in connection with them should lead to the discovery of either a vaccine or a serum, or some similar product which would help to modify the attack and curtail its severity. One is very far from saying that no vaccine and no serum is likely to do good; one can only say that one was not at all impressed by any of those that one had to one's hand to use—whether autogenous or stock.

That serum treatment may possibly be of great value in future cases seems indicated by Dr. Huff-Hewitt's experiences. He reported four cases in the *British Medical Journal* for May 10th, 1919, page 575. His treatment needs trial in a very much larger number before one can give a final opinion upon its value, but it strikes one as being fraught with great possibilities. Briefly, it consisted in obtaining blood by the syringeful from a patient recently convalescent from a moderately severe attack of the disease, allowing it to clot and then injecting the resultant serum subcutaneously into the patient who is severely ill. In his case it was a convalescent mother who thus gave her serum to her child, and the latter though extremely ill at the time, forthwith improved and made a good recovery. The same result ensued in three other very serious cases treated in the same way, and one admires the resourcefulness of the practitioner who carried out this treatment without any laboratory to help him. It was not until the epidemic was over that one saw his publication, and one did not test the treatment oneself, but the possible value of making use of serum from convalescent individuals in the treatment of other patients still acutely ill seems clear, and one would advocate further use of the method in any future epidemic in the hope that thereby some more or less specific remedy will have been discovered for that which throughout the epidemic of 1918–19 baffled all other forms of treatment when once the "pneumonic" type of the disease had obtained a strong hold upon the patient.

Morbid Anatomy and Histology.

Three of the most striking points brought home to one by post-mortem examination in fatal cases are:—

(1) The fact that the lesion in the lungs in "pneumonic" cases is practically never a true croupous lobar pneumonia in the ordinary sense, but a complex and

variable mixture of inflammatory lung lesions, of which even broncho-pneumonia forms only a part.

(2) The fact that though the "pneumonic" lesions are very striking, changes elsewhere are also prominent, especially in the kidneys; suggesting that, although the condition is referred to generally as "pneumonic," the effect of the acute microbic toxæmia is much wider spread than this term would suggest. A general microbic toxæmia or even septicæmia rather than a purely pulmonary infection.

(3) The fact that even when the greater part of the changes produced seem to be in the respiratory tract, they are not confined to the lungs, but extend from the highest to the lowest parts of the track—from the sphenoidal air cells and nasopharynx above to the pleura below.

Having emphasized these three points, it is expedient to describe the chief changes found in the organs, seriatim.

The condition of the *sphenoidal air cells*, the ethmoidal and frontal sinuses, the significance of inflammation in the naso-pharynx, extensions thence to the Eustachian tubes and middle ear; and similar extensions by Stenson's ducts from the mouth to the parotid glands has been discussed above.

The *lymphatic glands* in the neck and thorax, and often those in the abdomen also, have generally been swollen and crimson from acute inflammatory hyperæmia. Those most swollen and inflamed have generally been those situated below the bifurcation of the trachea, these sometimes being found many times their normal size, occasionally looking as if about to suppurate in their central parts, although but few cases of real abscess in these glands was met with. Presumably the great swelling and hyperæmia of the bronchial glands was the result of microbic absorption into these glands from the inflammatory foci in the lungs. The glands in the hilum of each lung were generally hyperæmic and swollen from the same cause, and from the thorax up in to the neck the deeper glands, especially those in the sulcus between the œsophagus and trachea on either side, were crimson and inflamed as a rule, though their enlargement was less marked than was the case with the bronchial glands. The abdominal glands, notably the mesenteric and retroperitoneal, were less constantly inflamed and enlarged than were those in the thorax and neck, but not infrequently they attracted notice by their swollen size, very often by their dull red or crimson colour.

The *larynx* did not as a rule show œdema or naked-eye evidence of laryngitis, and no case of laryngeal exudation was noted, but the mucosa of the *trachea* from nearly the level of the larynx down to the first subdivision of the bronchi was nearly always dark red or crimson from acute inflammatory injection, the degree of which became more and more pronounced

as the tubes were traced from above downwards. It was exceptional to find much purulent phlegm within the larger tubes, and no great degree of superficial exudate was present, but it was common to find a glazed look of the surface as though the dark red and inflamed mucosa were covered by a fine pellicle of coagulable exudate, in addition to which multiple pin-point depressions or pits were generally obvious on close inspection, due either to exaggerated openings of the sub-mucous glands or else to minute shallow abrasions from superficial ulceration.

The finer *bronchioles*, as seen in the cut suface of the lungs, showed extreme reddening of their mucosa; variable amounts of muco-pus or pure pus was expressible from them, but only exceptionally to anything like the degree that characterised the "purulent bronchitis" cases of 1916 and 1917.

The *lungs* were invariably heavier than normal, often greatly so, the increased weight being a feature of the lower lobes rather than the upper. Acute *pleurisy*, was not always present, but it was common, generally of the "lack-lustre" type, or with a pellicle of granular lymph that could be peeled off, but seldom presenting anything like the "buttery" exudate often seen with croupous pneumonia. Large pleuritic effusions were exceptional, many of the acute pleurisies were dry, but with some there was effusion of a few ounces, or perhaps a pint or more, of slightly turbid colourless or pale yellow or blood-tinged fluid containing fine flakes or threads or shreds of detached fibrin. This fluid practically always contained micro-organisms and polymorphonuclear leucocytes in abundance, but it was not pus in the ordinary sense.

The acute pleurisy was commoner over the lower lobes than the upper, but it might be universal; beneath the inflamed visceral pleura there were generally angry-looking dark red petechial and larger hæmorrhages. Acute pleurisy might be present over lung that was not consolidated, but more often pleurisy and obvious lung inflammation coincided.

The lung lesions, complex or variable, struck one as being quite different in character to anything one had met with at all commonly in the thousands of autopsies one has performed during the last 20 years. Broncho-pneumonia was very frequently a part of the picture-complex, but it was not like the common broncho-pneumonia of ordinary years; lobar pneumonia was not represented in any of its ordinary phases—simple congestion, red hepatization, grey hepatization, purulent infiltration. It would be inadequate to describe the lesions as those of either broncho-pneumonia on the one hand or croupous lobar pneumonia on the other. To obtain a picture of what was met with one would need to mingle together in different

proportions in different cases that had presented similar clinical signs, symptoms, and course, any or all of the following:—

Acute congestion, giving a more or less dark red colour to the whole lung.

Diffuse hæmorrhage, producing still darker red, often almost black-red areas in the already deep red lung, these hæmorrhagic areas being of all sizes and shapes from miliary to massive and scattered at randum throughout the lungs.

Broncho-pneumonia, sometimes recognisable only on careful search, sometimes widely disseminated and occasionally confluent; most marked as a rule in cases that had survived more than a day or two and generally giving one the impression of being rather a super-added development, almost inevitable in lungs so affected, than the primary and essential lesion.

Hæmorrhagic infarcts, similar in colour to diffuse intra-pulmonary hæmorrhages but differing from the latter in their pyramidal shape, with the broad base of the pyram beneath the pleura.

Miliary abscesses, often aggregated together in little focalised groups of from three or four to a score or more, showing up as paler foci in the dark red background, often situated in the midst of a deep crimson hæmorrhage, or an infarct, and not infrequently visible as a group of little abscesses beneath the pleura before the lung was cut. These grouped abscesses were similar to those seen in the midst of septic infarcts due to infected emboli such as result, for instance, from lateral sinus thrombosis.

Collapse, sometimes superficial only, sometimes associated with multiple areas of broncho-pneumonia, sometimes massive and independent of broncho-pneumonia, but adding an important factor to the lobar distribution of the signs of consolidation.

Croupous pneumonia, met with very rarely indeed in macroscopically recognisable degree, but occasionally forming part of general mixture of lesions.

Compression of one or other lower lobe by pleuritic exudate.

Purulent bronchiolitis, with thick pus expressible from the bronchioles seen in the cut lung.

Passive œdema of the bases.

Active œdema with extensive albuminous exudate into all parts of the lungs, not definable by the naked eye, but shown to be extreme in many histological sections; a peculiar and apparently highly important feature of these cases (*see* below).

Interstitial emphysema, often widespread throughout the lung tissue, but less easy to detect here with the naked eye

than when the typical gas bubbles formed the familiar streaks beneath the pleura.

It is difficult to make a word picture which adequately describes what was the average admixture of the above diverse ingredients. Many writers have referred to them conjointly as "pneumonia," giving the impression that "croupous pneumonia" prevailed; this was emphatically not the case; others have spoken of the condition as in the main "broncho-pneumonia"; but this again gives a wrong impression because broncho-pneumonia was only a part of the whole; congestion, hæmorrhage, infarction, inflammatory œdema, pleurisy and collapse might all be extensive in a case showing so little broncho-pneumonia that the latter had to be sought for carefully to be found at all. One feels tempted to coin a new word altogether to express so complete a type of lung-inflammation, and to term it "pneumonitis"; for no part of the lung tissue seemed to escape. One could then speak of the lesions as being of such varying types as the following:—

"Pneumonitis," with preponderance of congestion and œdema, but with little or no consolidation;

"Pneumonitis," with much congestion, bronchitis and some broncho-pneumonia in the lower lobes;

"Pneumonitis," with preponderance of congestion, interstitial hæmorrhage, œdema, and collapse, but little broncho-pneumonia;

"Pneumonitis," with congestion, hæmorrhages, collapse, and extensive broncho-pneumonia;

and so on.

It is worthy of particular note that here and there one came across a case with the same clinical picture as the rest, and yet with lungs so little altered to the naked eye that one might easily have passed them as almost normal—no consolidation in any part of any lobe; microscopically there would be bronchiolitis, peribronchiolitis, and diffused inflammatory albuminous exudate, both insterstitial and intra-alveolar; yet without any discernible broncho-pneumonia, and no obvious consolidation anywhere; no part of any lobe, larger than a minute fragment, would sink in water, and yet the clinical picture of the case was indistinguishable from that in which extensive broncho-pneumonia would be found at autopsy. In short, though broncho-pneumonia was a common thing to find post-mortem in little or greater degree it was only part of a much more complex mixture of lesions and, though usual, it was not absolutely an essential part of the mixture.

Microscopically the lung lesions were found to be just as protean as the macroscopic appearances would suggest. Sections taken from different parts of the same lung would often look so totally different that at first one could hardly believe that they were from the same case. Some showed

typical broncho-pneumonia, the alveoli being crowded with desquamated swollen epithelial cells, leucocytes and red corpuscles, and the interalveolar wall thinned by compression so as to be hardly more than linear; others, extensive hæmorrhage with myriads of red cells bulging the interalveolar walls and leaking into the interior of the alveoli; or again some alveoli might be full of extravasated blood bulging into nucleated cells of broncho-pneumonic origin, and others with fibrillated fibrin like that of croupous pneumonia; in another place the alveoli and the interalveolar walls might look normal, but a bronchiole would be found to be full of exuded small round cells and epithelial cells derived from partial disintegration of the mucosa with diffuse small-round-celled infiltration of the peribronchial connective tissue; in another section it might be difficult to recognise lung tissue at all, the whole being densely infiltrated with inflammatory cells, and the interalveolar walls apparently necrotic and destroyed; but none of the above seemed so remarkable as the "Gruyère cheese" changes which were so common and which were so entirely unlike what is met with in any ordinary forms of pneumonia that they seemed to be of essential importance, the other changes—hæmorrhages, broncho-pneumonia and so on—being super additions. The condition has been illustrated in Special Report Series No. 36 of the Medical Research Committee, and it is not very dissimilar to the initial results of the action of acute irritant gasses on the lungs. The first impression a typical section gives one is that the paraffin wax may have been but partially dissolved out when the block was being prepared. All through the section—filling the alveoli in some places, distending the interalveolar walls in others or the peri-bronchial connective tissue, or blocking the bronchioles, or infiltrating all parts of the section simultaneously—there is a hyaline or homogeneous material, staining faintly pink with eosin, but containing few cells; resulting, one supposes, from the rapid out-pouring of an albumiuous, non-cellular, coagulable exudate which, in the process of fixation of the tissues, becomes converted into what looks like hyaline material.

Amid this one discovers outlines of normal alveoli in some places, alveoli whose walls are disintegrating in other places, and in yet other places, spaces which are more or less obviously not alveolar spaces at all—round or ovoid holes of varying sizes without any defined walls, but reminiscent of the air holes which characterise a Gruyère cheese. Some of these may be the result of breaking down of interalveolar walls so that two, three, four, five or more original alveoli have been thrown together into one larger one, looking like a microscopic cavity in the hyaline matrix; some, on the other hand, appear to be gas-bubbles—microscopic interstitial emphysema—in the albuminous intra-pulmonary exudate.

Precisely similar non-cellular exudate into gas-holes is seen after acute gas poisoning. It seems likely that it is this acute inflammatory œdema of the lung tissue which, preventing inspired air from gaining access to the intra-capillary blood, accounts for the anoxhæmia and heliotrope cyanosis of the worst cases.

That broncho-pneumonia, hæmorrhage and other secondary changes should supervene on this lesion is what one would expect; they did so after acute gassing in France if the patient was able to survive long enough; but just as the lesion caused by gassing is not primarily broncho-pneumonia, but primarily and acute albuminous inflammatory œdema followed by broncho-pneumonia, so in these influenzo-"pneumonic" cases the primary effect was, in a large number of cases at any rate, an acute inflammatory albuminous exudate; broncho-pneumonia developing subsequently if the patient survived long enough.

The *kidneys* in almost all the fatal cases showed acute non-suppurative nephritis, milder in degree perhaps than that met with in scarlatina, but similar in type. The kidneys were uniformly enlarged, often weighing from 10 to 12 ounces the pair. The capsules peeled easily.

In colour they were sometimes irregularly blotched and mottled with alternate pallor and dark red; when cut the cortex looked swollen, with varying degree of lack of sharp definition between cortex and medulla; but most striking of all was the way in which, when the organ cut open was laid on the table with the cut surface upwards, the small vessels everywhere oozed out dark red blood without squeezing until in a brief space there was a film of blood obscuring everything; the condition was not quite the acute red blood-*dripping* kidney of scarlatina, but was approaching this condition and might be described as one of large red blood-*oozing* kidney.

Microscopically there were varying degrees of congestion of capillaries, swelling of glomerular tufts and epithelial cells, interstitial blood-extravasations and cloudy swelling of tubular epithelial cells, with desquamation of broken down cells into the lumen of the tubules; definite small-round-celled infiltration was less common.

The *liver* was generally of fairly normal colour and consistence; occasionally rather on the pale side, the pallor when present being fairly uniform. The whole liver looked swollen and enlarged in most cases, but otherwise not materially affected to the naked eye. On histological examination, however, nearly all the liver showed acute degenerative changes of many of the parenchymatous cells, particularly in the central parts of the lobules; cloudy swelling and lack of nuclear staining-power being more pronounced than actual fatty change. There was little or no tendency to small-round-celled infiltration of the portal canals.

The *heart* showed neither pericarditis nor endocarditis in any case I saw, nor any special tendency to supercardial ecchymoses. Many observers have reported dilatation of the heart in these cases, but what struck me most of all was the remarkable constancy with which the heart showed hardly any departure from the normal at all. When post-mortems were performed early, the heart muscle was firm and of good colour, rigor mortis in it was good, and there was little tendency to dilatation of any of the cavities; the right auricle and ventricle might be moderately distended with dark blood clot and, in comparison wish the empty and contracted left side, looked a little dilated; but the degree of distension was seldom great, and acute dilatation of the heart was quite unusual.

The *thyroid gland* was uniformly enlarged in nearly every case, a phenomenon which attracted attention though its causation was not obvious. The gland was sometimes quite three times the average size, and the isthmus was swelled as well as the lateral lobes, much in the same way that it is in Graves' disease. This change occurred as much in those who had never been overseas as in those who had been abroad, so that it could not be attributed to previous illness such as trench fever which may cause similar enlargement, notably in cases associated with D.A.H. The swollen gland was firm, and uniform in consistence, generally of its ordinary dull-red colour, and microscopically it did not show evidence of being acutely infected. The condition seemed to be one of simple uniform swelling of the gland secondary to the acute toxæmia of the general disease.

The *supra-renal capsules* were in the main of normal size and external appearance; but their medullary portions were generally dark red and pulpy from breaking down.

Under the conditions that existed at the time it was not easy to make special observations upon them and one would not care to say how much of the change in them was due to rapid post-mortem disintegration and how much to disorder caused by the infective disease itself.

The *alimentary canal* seldom presented macroscopic evidence of infection; but there was a group of cases observed and recorded by Lowe in which the colon was in a state of extensive and acute ulcerative colitis, with destruction of the mucosa, similar in type and degree to that which results from acute dysentery. The cæcum and ascending colon were the parts most affected, though no portion of the colon appeared exempt. Whatever the special factor may have been to cause this bowel ulceration in Lowe's cases one does not know; apparently it was not the use of calomel or similar drastic purge. Pneumococcal or streptococcal colitis are familiar enough in other circumstances, and one is not surprised that cases of this kind did occur during the epidemic; rather is one surprised that they were observed so seldom.

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CHAPTER IV.

The Bacteriology of Influenza.

BY

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In the previous chapters of this report the general features of the epidemics of influenza have been described. In discussing those features which are conventionally termed epidemiological, the assumption has been implicit that some living organism is the essential "materies morbi" of influenza, and we have not attempted to define its precise nature. In the present chapter this question must be explicitly raised, and a review will be attempted of the bacteriological findings in the recent epidemic.

Nearly 30 years have elapsed since the last world-wide outbreak of the disease, and during this period notable advances have been made, not only in the technique of bacteriology, but in the interpretation of the data which it reveals. It will be convenient, in the first place, to note certain of the factors which have contributed to widen the outlook of the pathologist in face of the problems offered by epidemic influenza, and especially those which are relevant to the discussion which will follow.

In 1890, the bacteriologist who sought to establish the connection between a given microbe and a given disease was restricted to the fulfilment of what were traditionally known as Koch's four postulates. These conditions, laid down at a time when the "germ theory" of disease was on its trial and was the subject of hostile criticism, were designed to remove all possibility of doubt as to the causal relationship of a microbe to an infective disease. They have lost none of their force with the advance in our knowledge; indeed the germ theory of disease is now so firmly established that we are in danger of too readily accepting a microbe as the causal agent of a disease, and of neglecting the rigid proofs which should be demanded. The conditions laid down by Koch are to-day easier of fulfilment than they were in 1890: our means of cultivation have been greatly improved, and our range of experimental animals has been extended not merely to monkeys and anthropoid apes, but, with increasing daring, to man himself where the risk could be justified. We are, further, able to employ lines of evidence unrecognised when the postulates were laid down and lying beyond their scope. Since 1890, the whole science of "immunology" has been developed; all the data which arise from the study of immunity reactions, from the results of specific serum treatment and of prophylactic inoculation, may

now justly be used in support of the causal relation of a microbe to a disease.

A second direction in which the horizon of the bacteriologist has widened lies in the fact that he is no longer restricted to the bacteria proper in his search for a pathogenic microbe. Many infective diseases have been found due to animal micro-parasites, while in not a few of our commonest fevers the causal agent is, on good evidence, though usually without the strict fulfilment of Koch's postulates, referred to the so-called "filter-passing" group of microbes; that is to say, to organisms so small that they are invisible, or barely visible, with the highest powers of the microscope, and are capable of passing through the pores of a filter which will keep back most, if not all, known bacteria. It need scarcely be said that evidence as to such invisible agents of disease must be even more jealously scrutinised than where a visible and readily-cultivable virus is in question.

There is a third direction in which much has been learned, and it is one of particular importance in relation to influenza. Even when the primary microbic cause of a disease has been established beyond question, it by no means follows that all the customary clinical phenomena of the disease are to be traced to this cause. On the contrary, they are often due to what are termed "secondary infections" by microbes other than that which is the primary cause, and such secondary infections may be fraught with greater danger to life than the primary infection itself. It is now well known that the healthy human body harbours on its cutaneous and especially on its mucous surfaces, a teeming and variable bacterial flora, many members of which are potential causes of disease. That they habitually produce no such effect depends upon two inversely-related factors—the resistance of the healthy body and the virulence of the microbes. Equilibrium of this nature is, however, readily upset: the resistance of the body may become lowered to a point at which even feebly-virulent organisms can invade it, or the virulence of the saprophytic flora may be raised to a point at which even the healthy body may be invaded. We know, too, that this saprophytic flora, and notably that of the respiratory tract, is in a constant state of flux. In crowded centres of population, individuals are constantly exchanging bacteria by a mechanism identical with that by which ordinary infections spread—in the main, so far as respiratory diseases are concerned, by the channel of "mouth-spray." If proof of this be needed, it is furnished by modern investigations into the prevalence of meningococcus carriers when cerebro-spinal fever is rife. The importance of secondary infections is well exemplified in such diseases as scarlet fever and measles. We do not, it is true, certainly know the primary cause of either of these fevers, though there is strong evidence in each case that it is a filter passer. But we have good reason for believing that neither of the

diseases, in an uncomplicated form, is a very serious affection, at least in this country. Their mortality is due to their complications, which depend upon secondary infections; streptococcal and pneumococcal invasions are the usual direct causes of death. The effect of the primary virus is so to depress the resistance of the body as to enable these almost ubiquitous saprophytes to gain a foothold. Their virulence is enhanced by successful invasion of the tissues, and when such fevers are epidemic there is little reason for doubting that the very means by which the primary virus spreads from case to case serves, at the same time, to convey the dangerous agents of secondary complications in a state of increased potency for harm.

Such considerations as these must be kept clearly in view in a discussion of the primary cause of influenza, in which it is also true that it is the complications which kill—complications, too, which are largely identical with, and due to the same secondary infecting agents as, those of such a disease as measles. For where the primary virus is obscure and difficult of detection, it may easily happen that a secondary invader masquerades as a primary cause. A well-known example of such an error is offered by swine fever. Salmon and Smith described in this disease an organism which they termed the "hog-cholera bacillus," now known as B. suipestifer or B. choleræ suis. This organism, undoubtedly pathogenic for the pig, long passed as the cause of swine fever, till Dorset and Bolton demonstrated that the true virus was a filter-passer and B. suipestifer a secondary invader from the intestine. The latter organism will produce in the pig certain of the phenomena of swine fever, but the disease so caused does not spread to other pigs after the fashion of the genuine disease.

The preceding remarks have not been made at random, but to illustrate certain of the advances in knowledge, gained during the past few decades, which have deeply influenced the mental attitude of pathologists and bacteriologists in face of such a problem as that presented by epidemic influenza.

The History of Pfeiffer's Bacillus.

From a bacteriological point of view the outstanding result of the previous great epidemic was R. Pfeiffer's discovery of the organism associated with his name and which he called the "influenza bacillus." This discovery was not made at the time of the primary outbreak, but during a secondary epidemic wave in 1892; it was published in that year, but a more complete account was furnished by Pfeiffer in a paper entitled "The Aetiology of Influenza" in the *Zeitschrift für Hygiene* in 1893. In this admirably lucid and well-written paper he describes how he found, in the sputum and pulmonary lesions of the cases which he studied, a minute Gram-negative bacillus in prodigious numbers. The difficulties attending the cultivation

of the organism were successfully overcome, and it was shown to require haemoglobin for its artificial growth. Pfeiffer failed to cultivate it from the blood of human cases, and he discredits Canon's observations to the contrary. He demonstrated the toxic effect of cultures upon the rabbit; guinea pigs and mice were found more resistant to the toxin: he endeavoured, though with indifferent success, to reproduce the disease in monkeys. He was unable to satisfy himself of any multiplication of the bacillus in his experimental animals; when death occurred it seemed due to a purely toxic effect.

Pfeiffer's observations aroused wide-spread interest, and his results were confirmed by others, but opportunity to put the causal relationship of his bacillus to the disease to more crucial tests disappeared, for the epidemic was at an end. The matter was therefore left in this position:—a new bacillus had been discovered, in connection with a disease not previously studied by bacteriologists, though only in a secondary wave of the epidemic. It had been shown to be toxic for laboratory animals, but the disease had not truly been reproduced in them: Koch's postulates had not been fulfilled. Nevertheless the close association of the organism with the lesions of the respiratory tract in human influenza and the profusion with which it was found in the sputum were so suggestive as to make out a *primâ facie* case for Pfeiffer's bacillus as the cause of the disease. This conclusion was widely accepted and the "bacillus influenzæ" took its place in bacteriological text books, perhaps with an admission that full proof of its relation to the malady was still wanting.

As commonly happens after a severe visitation of influenza, localised outbreaks of influenza-like disease, varying in extent and severity, have recurred at irregular intervals since the pandemic of 1890. Pfeiffer's bacillus was naturally sought for in such outbreaks, and not infrequently found. But often it was not found, and other pathogenic bacteria became recognised as the apparent causal agents in many of the minor epidemics, the evidence being of much the same order of validity as that attaching to Pfeiffer's bacillus in 1892. One has only to look at a list of the microbes which it is now deemed right to include in a "polyvalent catarrh vaccine" to realise how many are the organisms which now appear to be associated, and commonly on fair circumstantial evidence, though without the strict fulfilment of Koch's postulates, with febrile catarrhs of a more or less "influenzal" nature. Amongst the ingredients of such a vaccine we find not only Pfeiffer's bacillus, but streptococci, pneumococci, micrococcus catarrhalis and its allies, staphylococci, Friedländer's bacillus and B. septus. And we may well begin to wonder whether an affection, which has some claim to be regarded as a clinical entity, can possibly own such a variety of causal agents; for it is clearly possible that they may be of only secondary importance, the primary cause of febrile catarrh

being still unknown. The latter view is supported by Kruse's experiments on the transmission of "common cold" by material which has passed through a Berkefeld filter; similar experiments have more recently been carried out by Foster, and with even greater success. Should febrile catarrhs be proved primarily due to a filter-passing virus, the importance of the bacteria just enumerated would not be disproved, though they would be relegated to a secondary position. The prophylactic value of polyvalent anti-catarrh vaccines is thought by many to be not inconsiderable, though no trustworthy statistics on the subject are forthcoming.

The question, however, which is here at issue concerns the relation of Pfeiffer's bacillus to influenza and the light which has been shed on the subject since its discovery. A primary difficulty with which we are confronted at the outset lies in the difficulty of defining influenza. What is the relation between pandemic influenza and the minor epidemics of somewhat similar character, which occur irregularly during inter-pandemic periods, and between these, again, and febrile catarrhs? It is probable that the most likely person to answer this question is the epidemiologist, for it is in the "natural history" of these respective visitations that their most conspicuous differences lie. The clinician is baffled by the fact that the affections shade off into one another in such fashion that he can draw no clear line between them. The bacteriologist, who ought to be able to answer, is on the horns of a dilemma: for until the clinician can tell him the difference between epidemic influenza and a febrile cold he cannot sit in judgment on Pfeiffer's bacillus; and until he knows whether or not this organism is the primary cause of influenza, he cannot, by bacteriological means, decide between the two affections.

The following facts concerning Pfeiffer's bacillus may, however, be regarded as having been established in the course of the last five and twenty years:—

(1) It is not infrequently cultivated from swabbings taken from the pharynx of normal persons, living there as a temporary and apparently harmless saprophyte.

(2) It is still more commonly found in catarrhal conditions of the respiratory tract, and its presence, under such circumstances, may, or may not, be associated with fever and constitutional symptoms.

(3) It is familiar to laryngologists and rhinologists as the apparent causal agent of many chronic inflammatory conditions of the throat, nose and accessory sinuses.

(4) Certain cases of meningitis have been associated with the presence of this bacillus or of one very closely related to it.

(5) In rare instances it has been found to be the exciting cause of malignant vegetative endocarditis.

These facts at least show that Pfeiffer's organism is a not uncommon pathogenic agent in man, and one more especially related to catarrhal affections of the respiratory tract. They do not, on the other hand, disprove its causal relation to epidemic influenza. It might be argued that the original bacillus discovered by Pfeiffer was only one of a group of closely-related organisms, that it was the true cause of epidemic influenza and that the febrile catarrhs of inter-epidemic periods were due to other members of the group. Such an opinion, was, indeed, put forward by Pfeiffer himself, who found in certain catarrhs and broncho-pneumonias not associated with influenza, strains of bacilli related to B. influenzæ, but in his opinion separable from it by certain slight morphological and cultural differences: he called such strains "pseudo-influenza bacilli." Many bacteriologists would agree that we may be confounding under the common term, B. influenzæ, a number of closely related species. Bordet's "whooping cough bacillus" would fall into such a group, and the bacillus described from meningitis cases is said to present certain differences from the classical B. influenzæ. Very little serious systematic work has so far been attempted on this group of minute hæmophilous bacilli.

Or again, it might be argued that, even if all the forms of the bacillus encountered in non-epidemic times were referable to a single species, this might comprise a number of serological races, and that true epidemic influenza was due to the spread of some race of peculiar virulence. Such a hypothesis would be supported by the facts which have come to light concerning cerebro-spinal fever, in which epidemic outbreaks have been shown to be associated with the spread of certain types of the meningococcus (Gordon), or again by the relation of certain definite types of pneumococci with lobar pneumonia. It would, however, be pure hypothesis, supported only by such analogies as these, for the "influenza group" of bacilli does not readily lend itself to serological study: until the recent outbreak no facts were forthcoming as to the existence of well marked serological races amongst these organisms.

Looking back upon the experience gained between 1890 and 1918 it cannot be said that the status of Pfeiffer's bacillus as the cause of epidemic influenza had materially altered. No further confirmation had been obtained, nor had the observed facts disproved a possible relationship. Nevertheless the establishment of its widespread occurrence in non-epidemic times, often under conditions having no apparent relation to influenza, had rendered bacteriologists somewhat more critical of its claims—claims which, it was felt, could only be decided when a new pandemic arose.

Pfeiffer's Bacillus in the Outbreak of 1918.

It was therefore with considerable interest that the bacteriological results in the recent epidemic were awaited. They have

been conflicting, especially so during the summer outbreak, but it is now possible to take a general survey of the reports published from many parts of the world. There is a striking lack of unanimity in the opinions expressed by different workers, in different countries, and, it may be added, at different periods of the epidemic. Competent bacteriologists in France, America, Germany and a few in this country, either failed to find the bacillus or found it in a proportion of cases so small as seriously to damage its reputation. This was particularly the case during the outbreak in the summer of 1918: in the autumn outbreak it was more commonly found. On the other hand many British bacteriologists, working both in this country and with our forces in France, were able to find the bacillus in so many cases that they were convinced of its aetiological rôle in the disease.

The first question which arises concerns the validity of the negative results recorded. Pfeiffer's bacillus is admittedly not an easy organism to cultivate, and its colonies on most media are small and readily overlooked; but thanks to Pfeiffer's researches and to his discovery of its hæmophilic proclivities, the methods of successful culture had long been well known. Shortly before the summer outbreak, Levinthal had published an account of a new medium, made with boiled blood agar, which greatly facilitated the cultivation of the bacillus, while Matthews, a little later, also described a medium, made by the addition of trypsinised blood to agar, on which the bacillus grows with exceptional luxuriance. Though some workers may have used these media during the summer epidemic, it is probable that the majority employed ordinary human blood agar. Some of the failures to grow Pfeiffer's bacillus are perhaps due to this cause: it is certain that during the autumn outbreak, when the new media were more widely known and used, the proportion of cases in which the bacillus was found and of observers who found it, was greater than had been the case in the summer.

Such an explanation of the facts recorded is, however, unconvincing. Pfeiffer's bacillus can be cultivated by an experienced bacteriologist without the aid of the new media, useful as they have proved. Apart from cultivation, the bacillus, when present in influenza sputum or in the lung-juices, is usually there in immense numbers, and is so readily found in well stained films that a competent observer could hardly overlook it. Amongst these who have recorded their failure to find the organism are bacteriologists of the highest repute: the failures were perhaps more numerous in Germany than elsewhere: such men as Kolle, Gruber, Friedemann and many others failed to find it, while Uhlenhuth and Pfeiffer himself were puzzled at their want of regularity in finding it. It is expressly stated by Sobernheim that, using precisely the same technique, he uniformly failed to find it in the summer epidemic and almost uniformly succeeded in October.

But even in Germany some observers reported the common presence of Pfeiffer's bacillus, for instance, Simmonds at Hamburg, and Dietrich in the German Army, while in the autumn many found it. In England it was commonly found, even from the first: Matthews got it in every one of a dozen cases, and McIntosh found it frequently. In the British Armies in France it was the organism most regularly and abundantly present, though the figures available refer more particularly to the autumn epidemic. Gibson and Bowman record it in 68 per cent. of the bronchial fluids examined, 32 per cent. of the lungs, 85 per cent. of the tracheal scrapings, and 20 per cent. of the pleural fluids. Tytler, Janes and Dobbin found it in 56 out of 67 cases examined, and Patterson, Little and Williams, in 46 cases, failed only once to find it in the bronchi and lungs. The French bacteriologists, in Paris and elsewhere, found it commonly in autumn, though by no means always in the summer. In the United States observers were divided in their opinions. The Danish observers, in a report published by the Serum Institute at Copenhagen sum up against Pfeiffer's bacillus as a cause of influenza. They found it in only 37 per cent. of the cases. In Buda-Pesth it was found in abundance, and in Italy it was frequently met with. In the sudden outbreak at Johannesburg in South Africa, Lister found it in the lungs in 53 out of 56 fatal cases. It may be added that Pfeiffer's bacillus has repeatedly been cultivated from the blood, during the epidemic, but only in a small proportion of cases.

The foregoing is only a brief summary of some of the more important records. It makes no pretence at completeness, but it suffices to give a fair idea of the findings. Put still more shortly, it may be said that during the earlier outbreak of the disease in the summer of 1918 Pfeiffer's bacillus was found in some countries in a large proportion of the cases, sometimes in nearly all. In other countries it was sometimes found and sometimes not; in one country—Germany—it was found only exceptionally during the summer. Our troops in Mesopotamia experienced both the summer and autumn epidemics in 1918, yet Dr. Ledingham, who was then acting as pathological consultant, informs us that Pfeiffer's bacillus was not found during the epidemic in that country. In the autumn epidemic it was found more regularly everywhere, even in Germany.

The evidence does not carry conviction as to the primary causal relationship of Pfeiffer's bacillus to epidemic influenza. The varying results cannot be correlated with the competence of the observers. The observed facts, notably the earlier negative findings in certain localities, and the more general positive results in the autumn outbreak, would be better explained on the assumption that the bacillus played a very important secondary rôle in the disease, and was not the primary infecting agent. So far as the evidence derived from association with influenza can take us, the case for Pfeiffer's bacillus must still

be deemed unproven as regards its primary causal agency, though its intimate connection with the disease is even more firmly established than before.

Association with the disease is not, however, the only field in which evidence may be sought. Attempts have been made to support the ætiological rôle of Pfeiffer's bacillus by a study of the specific immunity reactions. It has been shown that specific antibodies arise in the blood during the course of influenza. The evidence is clear that, both by agglutination and complement fixation, such antibodies, specific for Pfeiffer's bacillus, are demonstrable in a considerable proportion of sufferers and convalescents from influenza. Thus Hartley examined 42 cases, and the serum of 27 of these agglutinated Pfeiffer's bacillus in a dilution of at least 1 in 50. Control normal sera failed to do this. Gibson and Bowman obtained very similar results ; several of their cases showed agglutination with a serum dilution of 1 in 640. What is true of agglutination is true also of complement fixation, in regard to which Wollstein's observations may be cited. In the case of many diseases such observations would be of high significance, but we cannot attribute to them much importance as evidence of the primary rôle of Pfeiffer's organism in influenza. For it is of such common occurrence in the disease, as a definitely pathogenic invader, that it would be strange indeed if antibodies were not formed against it. As a matter of fact similar antibodies have been found in influenza, when looked for, against other bacteria, such as streptococci, which are admitted to be merely secondary infecting agents.

In another direction, however, these immunological studies in influenza have proved of interest, for they distinctly suggest a wide range of antigenic variation in the bacillus recovered from different cases in the same epidemic. Of Hartley's cases some agglutinated certain strains and not others. Valentine and Cooper, reporting on agglutination and absorption tests on 171 strains in New York, could hardly find two which were serologically alike—a result which suggests that their methods were too delicate to be of practical value. The results of the complement fixation test have also indicated more than one race of the bacillus.

Further evidence may be sought in the effects of attempted immunisation against influenza by means of vaccines containing Pfeiffer's bacillus. The results of some of these efforts, on a sufficiently large scale, have now been published, *e.g.*, figures from Australia, from the Home Forces of the British Army, and from New Orleans. The true significance of the figures, which at first sight are striking, is a matter for expert statistical analysis, and will be discussed elsewhere in this report. The figures appear to show a considerable decrease in the attack rate amongst the inoculated, as compared with uninoculated controls, a still greater decrease in the liability to serious pulmonary complications, and an extraordinary diminution in the fatality of

the disease which, in the British Army, was only 0·12 per 1,000 in the inoculated as against 2·25 per 1,000 in the uninoculated. The death rate figures cannot be held relevant to the primary causal rôle of Pfeiffer's bacillus, which is admittedly responsible for a large proportion of the fatal pulmonary complications of influenza, even if it is held to be a secondary invader. If, however, a due statistical analysis of the figures for the attack rate shows them to be significant, they will constitute the strongest argument yet brought forward in support of the bacillus as the primary cause of influenza.*

Attempts to convey influenza to human volunteers by spraying the nose and pharynx with pure cultures of Pfeiffer's bacillus, recently isolated from fatal cases of the disease, have been carried out by Lister and Taylor in South Africa, and by Wahl, White, and Lyall in America. The experiments were on such a small scale that the negative results recorded can hardly be regarded as of serious significance, especially as the subjects of the experiments were, in both cases, for one reason or another, unsuitable ones.

Evidence as regards a Filter-passing Virus in Influenza.

When, as many believed, Pfeiffer's bacillus was discredited by the findings in the summer outbreak in 1918, the attention of numerous observers was directed to the possibility of a filter-passer as the primary cause of the disease. It was, indeed, difficult to think of any other alternative, for Pfeiffer's bacillus was the only known organism with any serious claim to this status. The characters of the so-called "Diplococcus epidemicus," described by some bacteriologists, can hardly be regarded as differentiating it from previously known inhabitants of the normal respiratory tract.

During the year 1918 a number of different observers recorded experiments with material from influenzal cases filtered free from ordinary bacteria. Nicolle and Lebailly first tested the unfiltered sputum from a case by introducing it into the conjunctiva and nostrils of two monkeys. After an incubation period of six days the animals developed fever lasting three days, after which they remained thin and depressed. The blood of the monkeys, injected into a man, produced no effect. The sputum from influenza cases, diluted with saline, centrifuged and passed through a Chamberland L 2 filter, was injected into one man subcutaneously and into another intravenously. No effect was produced by the intravenous injection, but the man subcutaneously inoculated, after an incubation period of six days, developed headache and an evening rise of temperature lasting 11 days. The blood of this case, injected into another man, produced no effect.

De la Rivière took blood from four influenza cases, proved its sterility on culture, and passed it through a Chamberland

* *Vide* pp. 64–5, 149, 175–6, 194.

L 3 filter ; he injected 4 c.c. of the filtrate under his own skin, and after three days got headache, muscular pains, and slight fever lasting a few days ; later his pulse became irregular.

Selter sprayed his own throat, and that of another person, with a saline filtrate of material from the throats of influenza cases ; both developed mild but typical influenza.

Leschke filtered sputum, lung juice, and similar material from influenza cases, diluted with saline, first through three layers of hardened filter paper and then through a Chamberland or Berkefeld filter. The experiments were controlled by cultures on ordinary media. He claimed to cultivate a filter-passer which grew as masses of small round bodies ; he could not grow it beyond the first generation. After many unsuccessful attempts, he finally succeeded in infecting human beings by spraying. Typical influenza was produced the same day or on the day following, with fever and tracheitis. On one occasion two people who nursed the experimental subject developed influenza.

Gibson, Bowman, and Connor carried out an elaborate series of experiments at Abbéville during the height of the epidemic amongst the British Expeditionary Force. They used animal experiment only, justly remarking that human experiments during the epidemic would have been of little value. As a criterion of a positive result they relied on hæmorrhagic and inflammatory changes in the animals' lungs identical with those seen in early fatal cases of influenza in man. The animals employed were monkeys, baboons, rabbits, guinea-pigs, and mice, and, with all these, successful results were obtained. The technique of the filtration experiments was carefully controlled. A Chamberland L 1 bis or F was employed. In monkeys, the inoculations were carried out subconjunctivally or by nasal instillation, or both ; in rabbits, intravenously or subcutaneously ; in guinea-pigs and mice, subcutaneously. With filtered material, free from ordinary bacteria, positive results were obtained with sputum from early cases, but less easily in later ones ; controls with sputum from ordinary acute bronchitis were negative. Filtered blood from influenza cases gave a positive result in a mouse and a doubtful one in a monkey ; unfiltered blood readily produced positive results on mice. Passage experiments from animal to animal were successful ; three rabbits in series yielded positive results, and an increase of virulence during passage was noted. The virus was also submitted to culture by Noguchi's method, and apparent growth of small coccoid bodies (from 0·1 to 0·2 μ in diameter) was obtained and carried on in subculture ; with these cultures positive results were obtained on a baboon, a monkey, rabbits, guinea-pigs, and mice, filtered and unfiltered extracts of the lungs of some of the animals thus affected again yielding positive results in further animals. The coccoid bodies were cultivated from the kidneys of infected

animals, filtrates of lung tissue, and filtered sputum from cases of influenza. In all successful animal inoculations the symptoms appeared on the fifth to the seventh day; the clinical symptoms were usually slight in comparison with the severity of the pulmonary lesions found after death; the main lesion was a patchy hæmorrhagic condition, with œdema. Broncho-pneumonia was never observed.

In Japan, Yamanouchi, Sakakimi, and Iwashima, made an emulsion of the sputum of 43 influenza cases in Ringer's solution. This was injected, unfiltered, into the throat and nose in 12 healthy persons, and a Berkefeld filtrate of the emulsion into 12 other healthy persons in a similar manner. All, except six who had recently suffered from influenza, developed the disease after an incubation period of two or three days, those receiving the filtered emulsion equally with the others. A filtrate of the blood of influenza cases, injected into the throat and nose of six healthy persons, produced influenza in all. Subcutaneous injection of filtrates of blood and sputum into eight persons equally produced the disease, except in one person who had recently had it. On the contrary emulsions of Pfeiffer's bacillus, alone and mixed with pneumococci, streptococci, &c., injected into the nose and throat of 14 normal persons, produced no effect at all.

Lister and Taylor experimented in South Africa, in February and March 1919, with filtered and unfiltered nasal washings and sputum from cases of influenza, and also with cultures of Pfeiffer's bacillus, using human volunteers and monkeys as the subjects of the experiments. The epidemic had already swept the country, and the 11 volunteers, who had escaped the disease, were presumably men of high resistance. With unfiltered sputum they succeeded in infecting two men out of five, the incubation being 36 hours, but neither men nor monkeys were affected when filtered material was used; nor did the application of living cultures of Pfeiffer's bacillus yield more than a doubtful indisposition in a single case only.

In Germany, v. Angerer claimed to find and grow a filter-passer in influenza, but he does not record experiment on man or animals. Binder and Prell also claim to have found and grown a filter-passer which they term "Aenigmo-plasma influenzæ."

While it is apparent that some of the observations summarised above are suggestive of a filter-passer as the cause of influenza, it cannot be said that any of them offer conclusive proof of such a proposition. Human experiment, in presence of an epidemic, is a somewhat dangerous guide, while animal experiments, however similar the appearances produced, leave us in some doubt as to whether the disease caused was truly epidemic influenza. If we compare the description of the pulmonary lesions upon which Gibson, Bowman, and Connor relied as evidence that they had reproduced the disease in

animals with material which had passed through a filter, with that given by Wollstein of the lesions caused in her experimental animals with filtrates of cultures of Pfeiffer's bacillus, it is noticeable that they are largely identical. True, there was no long incubation period in Wollstein's experiments, but she injected large doses of her toxic filtrates intravenously. Oedema, and hæmorrhagic patches in the lungs, and congested or hæmorrhagic trachea and bronchi, were present in the one set of experiments as in the other. Indeed, all who have experimented with the toxin of Pfeiffer's bacillus, which appears to be a soluble one, bear witness to its selective hæmorrhagic action upon the lungs.

The strength of the case argued by Gibson and his colleagues lies in the facts that there was a definite incubation period in their experiments, that they succeeded in carrying on the effects through more than one generation of animals, and that they produced them with cultures. The weakness lies in the fact that the lesions in their animals do not afford convincing proof of influenza. The incubation period in their experiments was longer than in the naturally occurring human disease, and this is true also of the experiments of Nicolle and Lebailly, though not in those of de la Rivière, of Leschke, and of the Japanese observers.

The cultivation of filter-passing organisms is notoriously a difficult thing, and beset with many fallacies, as Arkwright has well pointed out in his recent criticism of the subject. The results so far recorded in influenza must be received with caution.

It is plain that far more data are required before we can fully accept a filter-passing virus as the primary cause of influenza. The most that can be said at the present moment is that a fair case has been made out for granting the possibility, some would even say the probability, of such a cause.

Secondary Infections in Influenza.

Whatever opinion may be held as to the primary cause of influenza, the organisms responsible for the infections of the respiratory tract, to which its chief terrors are due, are well known. They are Pfeiffer's bacillus, the pneumococcus, streptococci, and especially streptococci of hæmolytic type, and more occasionally staphylococci, and other organisms to be mentioned in due course. Pfeiffer's bacillus may be placed in this category without prejudice to its possible claims to be regarded also as the primary cause of the disease.

Uncomplicated influenza, *i.e.*, fever and toxic constitutional symptoms without serious involvement of the lower respiratory tract, is not uncommonly seen in individual cases during an epidemic, though some degree of catarrh is the rule, so that

bronchitis would be included in any account of the symptomatology of the affection. The uncomplicated disease, though annoying and painful, and often attended by prostration and followed by prolonged cardiac debility, is rarely of itself fatal. Exactly the same is true of measles and scarlet fever; death from these affections is almost invariably due to complications set up by secondary infections with microbes which are well known, and are certainly not the primary infecting agents. In all cases, including influenza, the secondarily infecting bacteria are species commonly found as saprophytes of the respiratory tract or fauces in normal persons. The normal resistance of the healthy body prevents any harmful invasion. But in the presence of acute infection of the respiratory tract by the specific microbes of the zymotic diseases in question the lowered resistance of the body is apt to permit of local, or even general invasion by these saprophytes, which are potentially pathogenic; and the invading bacteria will be those which chance to be present, or predominant, in the affected individual. That is to say, the secondary invasion may be a different one in two separate cases of the same disease, different in two different localities during the same epidemic, different, on the whole, in different epidemics. Moreover, when once such a secondary invader has achieved success, it tends to become exalted in virulence by the ordinary mechanism of animal passage, and, by repetition of the process, still further exaltation of virulence may be brought about till it can attack healthy persons. For the secondary agents which lead to the respiratory complications of influenza are present in the bronchial secretions and have the same opportunities for spreading from case to case as the primary causal agent. There is even reason for the belief that the secondary infecting agents may, under favourable conditions, go on spreading in epidemic fashion, after the primary infecting cause has disappeared. In the opinion of Macallum, the pneumonias which devastated some of the military camps in the United States began as complications of epidemic measles, but later spread as epidemic pneumonias after the measles had ceased. It is thus clear that the phenomena presented by the recent pandemic of influenza should be regarded from a double point of view; the outbreak of influenza, *per se*, is not necessarily the same thing as the outbreaks of respiratory complications which accompanied it. There were, so to speak, epidemics within the epidemic. It is precisely such considerations as these that render it so extremely difficult to determine the position of Pfeiffer's bacillus in relation to the outbreak as a whole.

Whether or not we assign to Pfeiffer's bacillus the primary or secondary rôle, there can be no doubt that it played a part of vast importance in the recent epidemic of influenza. Though we know it, at normal times, as a mere agent of catarrh, the more virulent types which prevail in epidemic times are more

highly toxic and seem especially to lead to escape of blood from the smaller vessels in the lungs and bronchi. It seems probable that we must regard Pfeiffer's bacillus as the chief cause of the hæmorrhagic œdema and the localised hæmorrhagic areas in the lungs which have been so conspicuous a feature of the late epidemic.

Next in frequency and importance, as secondary infections in influenza, come the *streptococci* and here all are agreed that at least two types are concerned. If, as most bacteriologists have done, we group them according to Schottmüller's classification by their characters on blood agar, we find that his *streptococcus viridans*, representing the types of feeble virulence habitually present in the respiratory tract, has been very prominent, often exceeding in abundance any other organism in the pulmonary secretions. It is unlikely that streptococci of this type have played a part of much importance in the phenomena of the epidemic; they are notoriously of low pathogenic power, and their abundance merely reflects their normal frequency in the bronchial secretions. It is otherwise with the *streptococcus hæmolyticus*, an organism which probably corresponds, in great part, with that more usually known as streptococcus pyogenes. The presence of this streptococcus in the pulmonary lesions has been a distinguishing feature of the epidemic, especially in the autumn when the disease exhibited its highest fatality, and it was found in the worst cases. It was responsible, in conjunction with Pfeiffer's bacillus, for the hæmorrhagic lesions in the lungs, and it was more frequently found than Pfeiffer's bacillus in the hæmorrhagic pleurisies and empyemata which were of common occurrence.

The *pneumococcus* does not appear, from most reports, to have played such an important part, as a secondary infection, as either of the preceding, though it was not infrequently found in some countries. Although pneumonias, catarrhal, interstitial and sometimes lobar, were not uncommon, the pulmonary lesion regarded by most histologists as characteristic of the epidemic was a hæmorrhagic œdema rather than a pneumonia. Considerable variation occurred, however, at different centres, in the frequency with which pneumococci were found.

Staphylococci were noted by Tytler and his colleagues at No. 3 Canadian General Hospital in France, as the most abundant organisms in the bronchi after Pfeiffer's bacillus. This experience does not seem to have been a general one, though these common organisms were naturally found at times elsewhere. The same observers note the infrequency of hæmolytic streptococci in their findings.

The *meningococcus* was noted in a certain number of cases in this country, and notably in a group of cases in Scotland and in cases landed from American transports at Southampton. It is probable that no great significance attaches to this finding;

when influenza attacks a person who happens to be harbouring the meningococcus in his nasopharynx, it is not unnatural that the organism should multiply in the respiratory tract, but there is no evidence that it played any important part in the pathological processes set up.

This list of secondary infecting agents does not, of course, represent all the organisms isolated from the lungs during the epidemic, but it includes all those which can be regarded as of serious import in the respiratory complications of the disease. Our information about the bacteriology of other complications is too fragmentary to be summarised here, though it is probable that the toxins of Pfeiffer's bacillus exercised an important influence on certain organs such as the heart.

Before leaving the subject of secondary infections in influenza, mention must be made of the terminal septicæmias which were not infrequently observed. Blood cultures were carried out by large numbers of observers. The majority of results were by all accounts negative, but the larger amounts of blood now taken, as compared with the practice in 1890, yielded a certain harvest of positive results. The organisms usually found in the blood were streptococci, both of the viridans and hæmolytic type, pneumococci and Pfeiffer's bacillus. Several writers explicitly state that their positive results were most frequent in advanced and severe cases, and it is a fair conclusion that they represent an overflow into the blood from the pulmonary lesions rather than a primary septicæmia. It is perhaps, too, not without significance that Pfeiffer's bacillus was not more often found in the blood than other organisms admitted to be secondary invaders.

Summary.

A survey of the evidence obtained during the epidemic of 1918 as to the bacteriology of influenza appears to lead to the following conclusions:—

(1) The position of Pfeiffer's bacillus as the primary cause of the disease has been in no way strengthened. Its case remains unproven, and the crucial tests to which it has been submitted seem to indicate it rather as a secondary infection of the highest importance and significance than as the primary "materies morbi." At the same time it cannot be asserted that, as a primary cause, it is wholly out of court.

(2) The evidence for a filter-passing virus as the primary cause of the disease is suggestive, but at present a final verdict cannot be given.

(3) The complications to which the epidemic has owed its abnormal fatality have been due to secondary infections, in which Pfeiffer's bacillus and the haemolytic streptococcus have played a predominant part.

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CHAPTER V.

The Infectivity of Influenza.

Few living epidemiologists doubt that influenza is an infectious disease. (*a*) The occurrence of secondary cases after the introduction of a sick person into a previously untainted community; (*b*) the non-random distribution of cases in houses, seen in the excess of multiple cases, the small average interval in time between successive cases in the same house; and (*c*) a host of clinical observations, although severally not logical proofs of contagion have a cumulatively convincing effect.

Yet the most distinguished epidemiologists of an earlier generation categorically declined to recognise influenza as an infectious disease and held it to be an outstanding example of an epidemic disease covered by the ancient definitions. It will be recalled that the ancient masters of our art unanimously characterised an *epidemic* disease as one generated by the action of the atmosphere upon men's bodies. Such was the view of Hippocrates, and in his commentary upon the Hippocratic writings, Galen rationalised the conception. In the introduction to his comments on Hippocrates' books of Epidemics, Galen observed:—

> "It does not often happen that a disease takes hold upon a city, a nation, or an army, through their food supply nor even by virtue of community of occupation or labour. But if we are immoderately heated or chilled, dried, or moistened, by the all-embracing air, the symmetry of our bodies—which is health—is confused and destroyed. The air is all around us, and we breathe it. To no other influences are we all simultaneously subject and throughout the whole day. It cannot be, but that the properties of living bodies are influenced and modified by atmospheric change."

It is implicit in this argument that an epidemic (the remark just quoted applies exclusively to epidemics) does really occur as an explosive phenomenon, differences of diet, housing and occupation being without influence. When these conditions *are* fulfilled we can hardly deny the cogency of the argument. But in fact they have never yet been fulfilled by any epidemic disease. Chroniclers spoke of plague in just the terms more recently applied to influenza, uniformly exaggerating both the local explosiveness and the velocity of diffusion. Plague has been said to outstrip in its progress the swiftest horse, even although the writer recorded facts which proved that the extension was really quite gradual. The final magnitude

of the calamity has led to foreshortening. In plague the error has been easily recognised, since the warning epizootic has been observed, or, where no record of an epizootic exists, bills of mortality record the scattered deaths (as in the great plague of London) which happened sometime before the storm gathered head. The relatively low fatality of influenza deprives us of such warning while the increased velocity of human travel is but slowly realised in epidemiological studies. Thus we can understand why influenza lingered in the ancient class as a cataclysmal or telluric phenomenon. We need scarcely hesitate now to rank influenza amongst the other infectious or contagious diseases such as measles, scarlet fever, whooping-cough and small-pox.

Accepting this general doctrine, we are called upon to inquire whether personal infectivity varies sensibly through the clinical stages of the disease, to learn when the sick man is most dangerous to his associates. We must also learn whether the infective matter is capable of conveyance through articles soiled by the discharges of the patient or by objects with which he has been in contact. Precise answers to such questions could only be furnished by controlled experiments and to plan and conduct such experiments in the midst of an epidemic have proved impossible. We, therefore, reluctantly fall back upon the essentially imperfect test of personal opinion and common fame.

The Ministry circulated a questionnaire to the medical personnel of the home command and among the matters upon which an expression of opinion was invited were (1) Evidence as to the stage when influenza was most infectious and the duration of infectivity. Ninety-three replies were made to this question and 72 or 77·4 per cent. asserted that the early stage of the disease was the most infectious; 12 thought the most infectious period to be during the pyrexia, 7 during the early catarrhal stage; these two sets might safely be included amongst the supporters of early infectivity and if they are so included, only two of the 93 informants denied that the most infectious stage was early, alleging it to be seven days from the onset, but without assigning any reason. Of the evidence in support of early infectiousness we may cite such instances as the following:—One officer remarks that 100 cases were admitted to hospital on the second or third day of illness, but no secondary ward infections occurred, although in the camp of 613 men which gave origin to the cases 413 contracted the disease. Another officer notes that a patient while in billet infected house mates, but after removal to hospital infected none.

While we recognise the need of caution in drawing conclusions from expressions of opinion which are necessarily influenced unconsciously by current medical beliefs and sometimes merely rationalise what has been irrationally and

uncritically accepted, it may be remarked that they are concordant with our *a priori* unexpected discovery that domestic overcrowding is not a demonstrably important factor of varying incidence rates, and our surmise that acute and transitory overcrowding in trains, omnibuses, and places of entertainment is more important. This would be so were the very early or prodromal stage much more infectious than the period of prostration, since the later period contributes only to intra-domestic or institutional risks.

Passing to the second point, viz., the respective importance of direct personal and indirect infection, *per fomites*, the question elicited 79 replies. Six offered no opinion, one believed infection to be entirely air-borne, one thought indirect infection the more important, stating that visitors to influenza wards suffered less than those handling soiled linen, five thought both methods of equal efficacy, stress was laid upon the equal incidence of influenza upon nursing and laundry staff, while 66, or 90·4 per cent., of these committing themselves to any answer, believed direct infection the more important; several of this majority relied upon precisely the same kind of evidence as the minority, viz., a comparison of nursing and laundry staff, evidence which, without further information, is unconvincing, and we cannot say that any particular train of circumstances cited by any informant could be deemed crucial. It is, however, proper to remark that nobody was able to bring forward positive testimony that the disease had been transmitted under conditions from which direct personal contagion could be excluded.

Turning to numerical details, viz., as to the range of infection, only 31 answers were received, and so many of these betrayed a confusion between evidence and speculative opinion (one informant, for instance, would put no limit to the range of infection since "micro-organisms are floating in the air and are distributed indiscriminately by the wind"; another categorically demanded close contact as a *causa essentialis*, because "the "B. influenza is kept alive with difficulty away from its "hosts") that we are unable to draw any numerical conclusions as to the range of infective discharge.

Although the subject must be mentioned elsewhere, it is convenient to take note now of the military medical officers' experience relative to overcrowding. No reporter doubted that overcrowding was important, but hardly any evidence of the relative shares of domestic and extra-domestic overcrowding was forthcoming. The only numerical statements have reference to extra domestic overcrowding. Thus it was reported from the Irish Command that a fall in the death-rate occurred at Cork immediately after music halls and theatres were put out of bounds, and that at Londonderry, where the same order was in force, only eight cases occurred amongst 1,770 troops. From Chatham we were told that only 8 per cent. of a brigade of 10,000 men fell ill when the disease was prevalent in the

town ; places of amusement had been put out of bounds. The average incidence on the district was alleged to be "more than tenfold" as great. These statements do not, we think, carry conviction. We should know whether the closure of places of amusements was effected early or late in the local epidemic, an essential datum not furnished, while the alleged incidence of more than 80 per cent. upon the civilian population of Chatham may be disregarded as altogether incredible and discordant with the direct evidence collected in other places.

The general inferences to be drawn from the evidence submitted are that while influenza is surely contagious from person to person, the infection being perhaps conveyed by the expulsion of fine particles of mucus from the mouth and nose, no indirect method of dissemination has been proved to possess epidemiological importance. It also appears probable that the patient is most dangerous in the early stages of his illness ; it may even be that there is infectivity in the prodromal stage before the patient experiences any physical inconvenience.

Whether the disease is more infectious than measles or smallpox is a question we cannot answer. That it prevails more extensively during a given time proves nothing, since we are dealing with a disease in which the limitation of susceptibility by age and previous attack is greatly less than for measles or (in a well-vaccinated community) smallpox.

The reader may perhaps be surprised that we have been able to add so little to the evidence provided by Dr. Parsons nearly thirty years ago. The reason is that to pass from a qualitative demonstration of the *fact* of infection to a measurement of the *intensity* of infective power is a difficult undertaking. It might, perhaps, be supposed that a comparison between the final proportion of attacked persons to persons exposed to risk in isolated households or communities within which an infected person had been received would give the measure we seek. We might compare such percentages with a similar measurement upon households into which scarlet fever or diphtheria had been imported. Actually the number of such instances available is very small in pandemic times, while the proportion of the exposed to risk partly protected by previous attack is almost never available. In other words, we have not been able to institute comparisons strictly *in pari materia* and, on that account, do not feel justified in attempting to pass beyond the affirmation that influenza is an infectious disease, to the measurement of *how* infectious it is. That the power of transmission from person to person is not constant but a function of the "epidemic constitution," perhaps also of individual physiological variations, follows from the evidence submitted in other chapters of this report.

CHAPTER VI.

Natural Immunity.

It is important for both general and particular reasons to know whether passing through an attack of influenza renders the victim less liable to be again successfully attacked. The general reason is that if a large proportion of the victims is rendered immune from second attacks then, where the disease has been widely prevalent, it should follow that an appreciable proportion of the inhabitants might become insusceptible, a proportion sufficiently large to diminish the chance of another outbreak attaining considerable dimensions. The particular reason is that successful natural immunisation would guide us in attempts to confer an artificial immunity by inoculation.

Owing to the importance of the matter, efforts were made to secure data over a wide field. The kind of material desired was samples of persons living under various conditions of whom the influenzal records through two or three periods were accurate, *i.e.*, we desired to know how many of the group were attacked in the summer and how many not: how many of each of these classes were attacked in the autumn and similar information respecting the third wave.

That the effect of differences in environment and geographical situation might be noted, sample censuses were made in several cities and inquiries were also directed to some boarding schools. Data relating to members of the University of Cambridge, and to a police force were also secured. The original material so far as relevant to the present inquiry is recorded in Table I. (further particulars of certain data are given in the appendices). The appraisement of these results is a matter of considerable difficulty and it will therefore be proper to discuss in detail the methods employed.

All statistical inquiries involve two orders of difficulty, material and analytical. The material difficulties are those due to imperfections of the crude data themselves. Such imperfections, or errors, are of two kinds, systematic and unsystematic. Systematic errors are introduced in two ways. The first, and less important, is deliberate or unconscious bias of recorders. Thus a recorder who believed *a priori* that an attack of influenza did confer immunity, might accept evidence that a person who was not attacked in the autumn had been attacked in the summer with less criticism

than he would apply in other cases. This source of systematic error (another form of which is to reject observations making against a theory with greater readiness than those making in its favour) has, we think, not occurred in our series. A more prominent source of systematic error is introduced by the method of sampling used. If a house to house visitation be the source of the statistics it may be easier to obtain information from particular types of household and it was actually found that this happened. Families containing young children are proportionally over-represented in the censuses because tenements occupied exclusively by adults were more likely to be empty at the times of day when the particulars were collected. Hence the samples are not random samples of all households, but only of households of a particular class. The effect of this upon our investigation is not, however, of serious importance.

In the school inquiries it is possible that some systematic error may have been introduced by a failure to record accurately the changes of the population, *i.e.*, we have an insufficient guarantee that all *said* to be exposed to risk throughout the period of observation were really so exposed and that no others were exposed. It was obviously not possible to subject such data to the rigid control practicable when the whole investigation was guided by the staff of the Ministry or by experienced medical officers of health. There is, however, no reason to believe that any important error of this kind has occurred. Upon the whole it is felt that the prejudicial effect of systematic error has been sufficiently slight to disregard. Unsystematic errors are those due to carelessness or random inaccuracy on the part of the recorders or to inherent difficulties in making exact specifications. These have indubitably entered largely into the matter. Many of the particulars have been gathered by untrained or partly trained observers working often under pressure, while, even for the expert clinician or epidemiologist, to decide upon the evidence of an uneducated witness whether he or a member of his family had had an attack of "influenza" is a matter of delicacy. Hence we do not suppose that a high standard of accuracy has been reached.

There is, however, an important difference between the statistical consequences of systematic and unsystematic errors of record. The former might lead to a totally false conclusion: the latter would in general merely blur the analytical results, although they might lead to a negative conclusion where good data would warrant a positive conclusion. In some inquiries, notably those of psychologists, allowance is made for the attenuation of the statistical results due to such unsystematic errors. We shall not, however, attempt any such refinements here, since, for reasons about to be discussed, the range of fluctuation in results deduced from perfectly accurate data of the present class is so great that it is unprofitable to pause upon minutiæ.

We now turn to the analysis of the data and the expression of their meaning in the form of statistical averages. From the outset, two points are obvious. One is that if a first attack of influenza does confer any protection, then the case rate of those previously attacked will be less than that of those not before attacked. The other point is that variations of case rate might and do occur quite irrespective of previous history.

We can display the principle with the help of a time honoured illustration. If we draw counters at random out of a bag containing equal number of white and red counters, we shall rarely draw precisely equal numbers of each kind. If in a series of trials, or in a single trial, there is a clear majority of red counters, we shall not infer that it is really easier mechanically to extract the reds, unless we can show that the discrepancy is much greater than would be likely to arise by mere chance in the extraction of samples of the given size from a bag containing perfectly similar and equally accessible red and white counters. The arithmetical criterion applicable to such a case is simple. If the proportion of red counters is p, and of white counters $1 - p = q$, then if n are drawn (it is assumed that either the total number in the bag is indefinitely great or that each counter is replaced after drawing) we shall *on the average* draw np red counters per sample of n, and the chances are that a considerable majority of the drawings will not differ from this average by more than two or three times $\sqrt{npq}$. Why then should we not apply this method to the case of influenza? If a previous attack confers no immunity, the n persons attacked in the summer, and the m persons not attacked in the summer may be likened to samples drawn from a bag, which bag is to be composed of red and white counters in the proportions representing the total attacked and total not attacked *during the autumn*, and, if there be no immunity, the difference between the proportions of attacked in the two samples should not greatly surpass the expected fluctuation determined by the formula quoted above.

There are two objections to this process. The first, and more important, is that the formula measuring the extent of fluctuations is deduced on the assumption that the drawings are independent, that if the first counter drawn in any trial happens to be red it is neither more nor less likely that the second will be red than if the first had been white. Suppose the bag of counters subject to other conditions. That, for instance, the colours in the bag were changing, sometimes white counters turning red, sometimes red counters turning white. Suppose that the mere drawing of a counter and its replacement made a difference, that when a white counter was drawn and thrown back into the bag, any red counter it impinged upon turned white. Clearly the law of fluctuations in sampling such a bag as this might be very unlike those of

the time honoured and static "universe" of counters which provides our simple formula. But a moment's thought shows that the statistical nightmare we have just conjured up is more nearly in point when we are discussing an infectious disease than the orthodox scheme. The chance of contracting an infectious disease is ill represented by taking the proportion of those affected by the disease in the "universe," and using that proportion as the constant measure of risk for each person. The "counters" are *not* all alike in shape and smoothness, and the drawings are *not* independent. These two departures from the scheme will affect the variations of the composition of samples in different ways, but, in the general case, the combined bias will deprive the formula of much practical value. In fact, a very good statistical criterion of infectivity is to note that the fluctuations of samples, *e.g.*, the numbers of houses with 0, 1, 2, &c. cases of diseases in different samples *are* quite unlike those which would occur upon the hypothesis from which we started.

Are we, then, to conclude that arithmetical computations based on the theory of simple sampling are mere waste of time in the present connection? The answer is that such calculations although of very limited value, are not entirely worthless. The present differs materially from such a statistical record as, for instance, the number of cases of infectious disease in each of a series of years or the distribution of cases in houses. In either of these the method is valueless. It would be useless to use it to measure the probability that the attack rate in Manchester (measured on a sample of the inhabitants of Manchester) differed significantly from a sample in Leicester. But the data we are to analyse consists, in each sample, of persons under a common environment all exposed to a considerable average risk of infection. Those previously attacked were not segregated in any way from those not previously attacked, while the evidence we have analysed in another chapter makes it a matter of doubt whether variations of domestic conditions (which in any event do not apply to the schools) are very compelling factors. Hence it may well appear that the time-honoured test is not altogether useless, but may at least serve to indicate a lower margin of fluctuation, so that deviations found by it to be within the limits of chance fluctuations may certainly be dismissed as insignificant. It may still be objected that there is another difficulty; even were the counter-drawing analogy tolerable, we do not actually know the contents of the bag. We know the numbers attacked by influenza *in our samples* not in the universe of which they *are* samples. This objection is, however, much less formidable than the former because the combined totals of the samples are large and if the analogy were valid, the risk of assuming the proportion obtained in the combined samples to be that of the whole universe does not import any great error into our calculations.

If we are to use this method as a preliminary test, the question next arises as to whether we can render our results comparable by the eye, if we use some function of the proportions instead of the proportions themselves, since the latter will vary with the severity of the epidemic in the particular town or community studied. A method which suggests itself is to compare $100(p_1-p_2)/p_1$ for the different samples where p_1 is the proportion attacked in the second epidemic of those not attacked before, and p_2 is the proportion attacked amongst those who had been attacked before. This expression, which has been termed the percentage efficiency of protection, has the merit of varying from 0 to 100, when p_1 is equal to or greater than p_2, but it has various demerits. For instance if p_1 is less than p_2 the range of negative efficiencies is unlimited and to reach a correct mean value needs a series of comparisons strictly *in pari materia.**

Consequently this method of comparison is not very good. Actually we provide the following statistical deductions based on the method of simple sampling.

(1) The number attacked in the second of each pair of epidemics from amongst the previously attacked, and the ratio of the difference between this and the "expected" number to the latter's probable error on the assumption of independent liability, the chance of attack being defined by the ratio of attacks to persons at risk in the combined second samples.

(2) A comparison of percentage attack rates.

(3) Percentage efficiencies, together with the probable fluctuation of such a measure around the value zero which would be almost its mean value in an undifferentiated population (the probable fluctuation has been calculated on the hypothesis of independent chances).†

This method being admittedly imperfect although easy to grasp we turn to others. The most usual is to tabulate the data in such a form as the following:—

1st Epidemic.	2nd Epidemic. Attacked.	2nd Epidemic. Not Attacked.
Attacked - -	a	b
Not attacked -	c	d

* For a full discussion of this and other points, touched on in the text, *see* Greenwood and Yule, Proc. Royal Soc. Med. (Section of Epidemiology and State Medicine) VIII., 1915, pp. 113–190.

† *I.e.*, if p were the proportion attacked in the second epidemic, and n and m the number previously attacked or not attacked, we have—

$$67{\cdot}449 \times \sqrt{\frac{1-p}{p}\left(\frac{1}{n}+\frac{1}{m}\right)}$$

for the approximate probable error of zero efficiency. It is assumed that squares of deviations from the mean values are small in comparison with the squares of the means themselves, otherwise (3) *supra* is inexact.

One then calculates the probability that such a distribution into the four subdivisions might have arisen by chance, adding some coefficient designed to measure the tendency to co-variation, or correlation, of the attributes.

This method is free of some of the more serious objections urged against the counters' scheme of our previous argument. It is quite true that the measures of improbability deduced from the tables 2A–2N are based upon theoretical reasoning perfectly akin to that involved in the deduction of the formulæ of simple sampling. But the statistical "universe" sampled is different. It is in fact the universe of "attacked" and "not attacked"; the law of variation postulated of *that* universe is not subject to quite such damaging criticism as when employed above. In fact so far as the measure of mere improbability is concerned, the theoretical objection is rather that it somewhat *overestimates* the likelihood that the samples are homologous. Hence any results which pass this test may be received without hesitation as something more that a mere freak of sampling a universe where the incidence of a disease is ideally equal upon the two classes compared. Hence the values of the entries in Tables 3, 4, 5 (column 11) are a sufficient rough and ready criterion of the likelihood that

TABLE 1.

Locality.	Population at Risk.	Numbers having Influenza in						
		Summer.	Autumn.	Summer and Autumn.	Winter.	Summer and Winter.	Autumn and Winter.	Summer, Autumn, and Winter.
South Shields -	462	14	27	1	31	1	3	0
Leicester - -	4,619	325	662	16	323	22	37	2
Wigan - -	1,075	45	81	1	117	0	2	1
Newcastle - on - Tyne.	4,461	277	225	4	326	39	17	2
Manchester -	4,686	709	403	70	73	26	11	3
Blackburn - -	1,284	111	82	8	83	6	10	1
Widnes - -	3,417	423	274	14	340	21	8	0
Cambridge University.	1,766	423	406	41	130	17	38	8
City of London Police.	746	50	113	4	24	0	5	0
Clifton College -	451	162	99	22	85	71	61	13
Haileybury -	515	180	73	41	106	22	25	10
Eton - - -	753	393	172	29	*	—	—	—
Harrow - -	429	90	258	29	*	—	—	—
Finchley Elementary Schools.	1,224	134	387	18	29	5	4	0

* No returns for the Winter Epidemic.

there is a real difference; so long as the figure in this column exceeds seven or eight, we may infer that differentiation has been rendered highly probable. To devise an average or coefficient based ultimately upon this function and comparable from sample to sample yet free from the grave objections urged against the coefficient entered in column (5) is difficult and the value of such coefficients is a matter of controversy. For present purposes we believe that the cogency of the individual results should be judged by the record of column (11)—column (12) merely translates the result of column (11) into a scale of probabilities—while column (5) may be used to compare one experience with another but always subject to the restrictions and criticisms above noted. The significance of the remaining columns has been explained; the various "probable error" tests are of subordinate value although actually there is in the series no instance of conflict, *i.e.*, no sample which passing the imperfect test would be rejected by the more reliable one (the autumn and winter comparison of Wigan is a partial exception).

With these necessarily long yet incomplete explanations we pass to the numerical results shown in Tables 3, 4, and 5 based upon the material tabulated in No. 1.

TABLE 2A.

SOUTH SHIELDS. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	1	13	14	Summer +	1	13	14	Summer +	3	25	28
−	27	421	448	−	34	414	448	−	32	402	434
	28	434	462		35	427	462		35	427	462

TABLE 2B.

LEICESTER. *Four-fold Table.*

-—	Autumn.			-—	Winter.			—-	Winter.		
	+	−			+	−			+	−	
Summer +	16	309	325	Summer +	24	301	325	Summer +	39	639	678
−	662	3,632	4,294	−	360	3,934	4,294	−	345	3,596	3,941
	678	3,941	4,619		384	4,235	4,619		384	4,235	4,619

Table 2c.

Wigan. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	1	44	45	Summer+	1	44	45	Autumn+	3	79	82
−	81	949	1,030	−	119	911	1,030	−	117	876	993
	82	993	1,075		120	955	1,075		120	955	1,075

Table 2d.

Newcastle. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	4	273	277	Summer+	41	236	277	Autumn+	19	210	229
−	225	3,959	4,184	−	343	3,841	4,184	−	365	3,867	4,232
	229	4,232	4,461		384	4,077	4,461		384	4,077	4,461

Table 2e.

Manchester. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer+	70	639	709	Summer+	29	680	709	Autumn+	14	459	473
−	403	3,574	3,977	−	84	3,893	3,977	−	99	4,113	4,213
	473	4,213	4,686		113	4,573	4,686		113	4,573	4,686

Table 2f.

Blackburn. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer+	8	103	111	Summer+	7	104	111	Autumn+	11	79	90
−	82	1,091	1,173	−	93	1,080	1,173	−	89	1,105	1,194
	90	1,194	1,284		100	1,184	1,284		100	1,184	1,284

Table 2G.

Widnes. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	14	409	423	Summer +	21	402	423	Autumn +	8	280	288
−	274	2,720	2,994	−	348	2,646	2,994	−	361	2,768	3,129
	288	3,129	3,417		369	3,048	3,417		369	3,048	3,417

Table 2H.

Cambridge University. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	41	382	423	Summer +	25	398	423	Autumn +	46	401	447
−	406	937	1,343	−	168	1,175	1,343	−	147	1,172	1,319
	447	1,319	1,766		193	1,573	1,766		193	1,573	1,766

Table 2I.

City of London Police. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	4	46	50	Summer +	0	50	50	Autumn +	5	112	117
−	113	583	696	−	29	667	696	−	24	605	629
	117	629	746		29	717	746		29	717	746

Table 2J.

Clifton College. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	22	140	162	Summer +	84	78	162	Autumn +	74	47	121
−	99	190	289	−	146	143	289	−	156	174	330
	121	330	451		230	221	451		230	221	451

TABLE 2K.

HAILEYBURY. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	41	139	180	Summer +	32	148	180	Autumn +	35	79	114
−	73	262	335	−	131	204	335	−	128	273	401
	114	401	515		163	352	515		163	352	515

TABLE 2L.

FINCHLEY ELEMENTARY SCHOOLS. *Four-fold Table.*

—	Autumn.			—	Winter.			—	Winter.		
	+	−			+	−			+	−	
Summer +	18	116	134	Summer +	5	129	134	Autumn +	4	401	405
−	387	703	1,090	−	33	1,057	1,090	−	34	785	819
	405	819	1,224		38	1,186	1,224		38	1,186	1,224

TABLE 2M.

ETON.

—	Autumn.		
	+	−	
Summer +	29	364	393
−	172	188	360
	201	552	753

TABLE 2N.

HARROW.

—	Autumn.		
	+	−	
Summer +	29	61	90
−	258	81	339
	287	142	429

We consider first the influence of summer attack upon the fates of the samples in the autumn wave of influenza. In five of the 14 instances, viz., South Shields, Wigan, Manchester, Blackburn, the City of London police, and Haileybury College such difference of percentage attack rates as appears cannot be regarded as beyond the range of chance having regard to the size of the samples. In two of these, Wigan and the City police the difference of attack rates is greatly in favour of those who had had summer influenza, but the magnitude of the epidemic was too small to enable the results to pass our test. In the three other cases there is not even a *prima facie* advantage on the side of the summer victims. The balance, six samples, all show a considerable and statistically significant superiority of

TABLE 3.

Summer and Autumn.

—	(1) Total investigated.	(2) Per Cent. attacked amongst those previously attacked.	(3) Per Cent. attacked amongst those *not* previously attacked.	(4) Probable Error of the Difference (2) and (3).	(5) "Efficiency" of Protection.	(6) Probable Error of Zero Efficiency.	(7) Ratio of (5) to (6).	(8) Actual Number attacked twice.	(9) Calculated Number attacked twice.	(10) Probable Error of (9).	(11) Value of χ^2 for corresponding four-fold Table.	(12) Value of P.
South Shields	462	7·1	6·0	±4·37	−18·4	±72·07	0·26	1	1	±0·60	0·02970	0·994
Leicester	4,619	4·9	15·4	±1·37	68·1	± 9·36	7·28	16	48	±4·30	26·56571	—
Wigan	1,075	2·2	7·9	±2·73	71·8	±35·40	2·03	1	3	±1·20	1·94778	0·58
Newcastle	4,461	1·4	5·4	±0·92	73·2	±17·97	4·07	4	14	±2·48	8·25467	0·04
Manchester	4,686	9·9	10·1	±0·83	2·6	± 8·21	0·32	70	72	±5·41	0·04489	0·991
Blackburn	1,284	7·2	7·0	±1·71	− 3·1	±24·40	0·13	8	8	±1·81	0·00730	0·999
Widnes	3,417	3·3	9·2	±0·97	63·8	±11·55	5·52	14	36	±3·85	16·38905	0·001
Cambridge University	1,766	9·7	30·2	±1·58	67·9	± 6·76	10·04	41	96	±5·81	71·77573	—
City of London Police	746	8·0	16·2	±3·59	50·7	±22·90	2·21	4	8	±1·73	2·39263	0·50
Clifton	451	13·6	34·3	±2·93	60·4	±10·90	5·54	22	44	±3·80	22·60555	—
Haileybury	515	22·8	21·8	±2·59	− 4·5	±11·69	0·38	41	40	±3·69	0·06614	0·987
Eton	753	7·4	47·8	±2·18	84·6	± 8·15	10·38	29	105	±5·91	156·70702	—
Harrow	429	32·0	76·1	±3·76	57·7	± 5·63	10·25	29	60	±3·01	62·60082	—
Finchley School	1,224	13·4	35·5	±2·91	62·2	± 8·78	7·08	18	44	±3·67	24·58404	—

Table 4.

Summer and Winter.

—	(1) Total investigated.	(2) Per Cent. attacked amongst those previously attacked.	(3) Per cent. attacked amongst those *not* previously attacked.	(4) Probable Error of the Difference (2) and (3).	(5) "Efficiency" of Protection.	(6) Probable Error of Zero Efficiency.	(7) Ratio of (5) to (6).	(8) Actual Number attacked twice.	(9) Calculated Number attacked twice.	(10) Probable Error of (9).	(11) Value of χ^2 for corresponding four-fold Table.	(12) Value of P.
South Shields - -	462	7·1	7·6	±4·84	5·9	±63·94	0·09	1	1	±0·67	0·00386	0·999
Leicester - -	4,619	7·4	8·4	±1·07	11·9	±12·89	0·92	24	27	±3·36	0·39572	0·92
Wigan - - -	1,075	2·2	11·6	±2·93	80·8	±28·98	3·45	1	5	±1·42	3·70907	0·30
Newcastle - - -	4,461	14·8	8·2	±1·17	−80·5	±13·63	5·91	41	24	±3·21	14·40079	0·002
Manchester - -	4,686	4·1	2·1	±0·42	−93·8	±17·41	5·39	29	17	±2·75	10·00533	0·019
Blackburn - - -	1,284	6·3	7·9	±1·79	20·4	±23·05	0·89	7	9	±1·90	0·37152	0·93
Widnes - - -	3,417	5·0	11·6	±1·09	57·3	±10·70	5·36	21	46	±4·31	17·05987	0·001
Cambridge University -	1,766	5·9	12·5	±1·17	52·8	±10·74	4·92	25	46	±4·59	14·39117	0·002
City of London Police	746	0	4·2	±1·91	100·0	±49·10	2·04	0	2	±0·92	0·21676	0·96
Clifton - - -	451	51·9	50·5	±3·31	2·6	± 6·49	0·40	84	83	±4·29	0·07379	0·995
Haileybury - -	515	17·8	39·1	±2·90	54·5	± 9·16	5·95	32	57	±4·21	24·61737	—
Finchley School - -	1,224	3·7	3·0	±1·70	−23·1	±34·50	0·67	5	4	±1·35	0·19650	0·96

Table 5.

Autumn and Winter.

—	(1) Total investigated.	(2) Per Cent. attacked amongst those previously attacked.	(3) Per Cent. attacked amongst those *not* previously attacked.	(4) Probable Error of the Difference (2) and (3).	(5) "Efficiency" of Protection.	(6) Probable Error of Zero Efficiency.	(7) Ratio of (5) to (6).	(8) Actual Number attacked twice.	(9) Calculated Number attacked twice.	(10) Probable Error of (9).	(11) Value of χ^2 for corresponding four-fold Table.	(12) Value of P.
South Shields	462	10·7	7·4	±3·48	−45·3	±45·88	0·99	3	2	±0·94	0·41932	0·92
Leicester	4,619	5·8	8·8	±0·78	34·3	± 9·31	3·68	39	56	±4·85	6·74839	0·08
Wigan	1,075	3·7	11·8	±2·44	68·9	±21·86	3·15	3	9	±1·92	5·04104	0·17
Newcastle	4,461	8·3	8·6	±1·28	3·7	±14·91	0·25	19	20	±2·86	0·02968	0·994
Manchester	4,686	3·0	2·3	±0·50	−26·0	±20·81	1·25	14	11	±2·25	0·67247	0·866
Blackburn	1,284	12·2	7·5	±1·98	−64·0	±25·37	2·52	11	7	±1·71	2·64963	0·46
Widnes	3,417	2·8	11·5	±1·29	75·9	±11·94	6·36	8	31	±3·55	21·00660	—
Cambridge University	1,766	10·3	11·1	±1·15	7·6	±10·54	0·72	46	49	±4·45	0·25012	0·95
City of London Police	746	4·3	3·8	±1·31	−11·8	±33·77	0·35	5	5	±1·41	0·05537	0·99
Clifton	451	61·2	47·3	±4·99	29·4	± 7·02	4·19	74	62	±3·71	6·82973	0·08
Haileybury	515	30·7	31·9	±3·33	3·8	±10·52	0·36	35	36	±3·35	0·06092	0·988
Finchley School	1,224	1·0	4·2	±0·71	76·1	±22·89	3·32	4	12	±2·35	51·52726	—

the summer patients in face of the autumn epidemic. If we average the so-called efficiencies using as weights the squares of reciprocals of the "probable errors"—we find that the average value is 54·3 per cent. which would mean, were the severity of the different epidemics constant that the previously attacked person had only half the risk of contracting the disease incurred by his unprotected neighbour (for reasons above explained, this is a crude approximation to the unattainable truth). When we compare the winter experience of the persons attacked or not attacked in the summer, the proportion of undifferentiated samples increases; in fact Haileybury (the single school which showed no measurable difference in the previous comparison), Newcastle, Widnes, and Cambridge University give significant values; 36 per cent. of the samples against 57 per cent. in the previous comparison. The numerical measure of average efficiency sinks to 16·0 per cent. The autumn-winter comparison leads to results not much better than the last; four out of 11 are significant, the average efficiency 25·2 per cent.

From this comparison we may infer that, on the average, summer influenza did confer a measure of protection against the autumn and a smaller protection against the winter disease. (2) that the diminished value of the summer attacks in protecting against the winter form was not entirely a matter of the wearing out of an acquired immunity, since the autumn exposure did not confer a much greater benefit than did that of the summer, but more probably due to some immunological differentiation of the third from the first and second waves, a surmise which is concordant with the clinical experience that winter influenza was unlike that of the autumn.

Although we may be reasonably certain that the advantage —however measured—of those previously attacked is in a majority of the instances a real phenomenon, the results as a whole are unsatisfactorily discordant. Why should we find such a difference between Leicester and Manchester or between Eton and Haileybury? Had we merely been furnished with one or other set of data we should have been led to seriously erroneous conclusions as to the epidemiological importance of naturally acquired immunity, have dismissed it as of no moment or have attributed to it a general importance it is far from possessing. Before considering the matter in detail, we may meet a criticism which has no doubt already occurred to the reader.

We remarked in an earlier chapter that the age incidence of the autumn influenza was different from that of the summer, hence, in town censuses, judging immunity without reference to the ages of the exposed may lead to a fallacious result (it cannot of course do so when, as in schools, the population is of uniform age). This point was taken early in the enquiry and it was found that a separate evaluation of age groups both at Manchester and Liecester did

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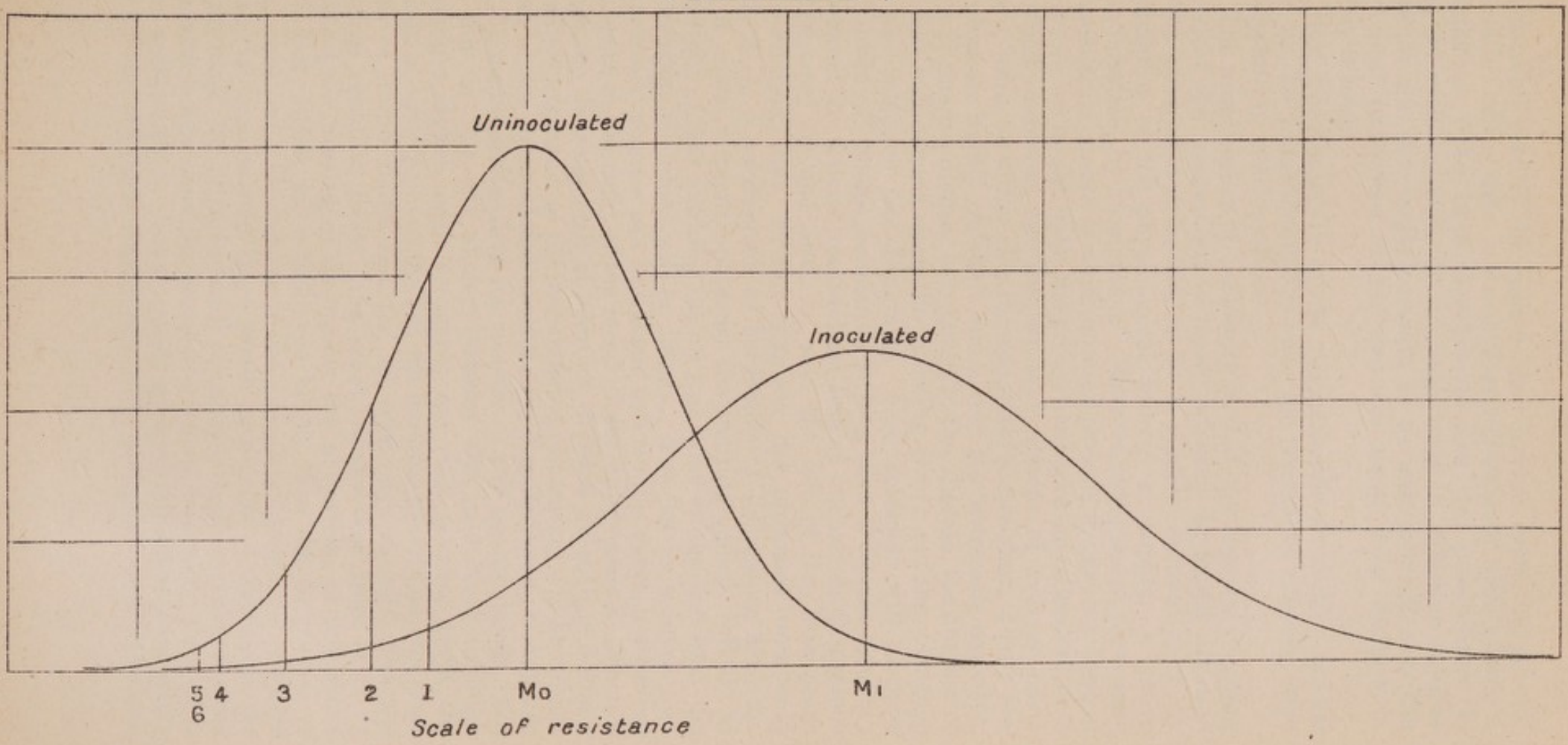

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not sensibly modify the conclusions derived from the general tabulation of the data. No obvious explanation such as this is practicable. We have therefore to face two possibilities (1) that the wide variation of the case rates in different samples accounts for the varying results as to immunity, (2) that the biological properties of the *materies morbi* were distinct in different cases.

How hypothesis (1) might reconcile the conflicting results may not be immediately obvious; the possibility reposes upon the following considerations.

The usual theory of immunity is that the *average* resisting power of the immunised is raised, whether by actual addition to the store of anti-bodies, as in a passive immunity, or by stimulation of the physiological sources of supply, in active immunisation, by vaccination. Were we to represent pictorially the resisting powers of unimmunised persons to varying doses of infection we might liken the group to a curve the tallest ordinate of which measures the frequency of persons of average resistance, while on either side the ordinates diminished, a very few persons are highly resistant and a very few extremely sensitive. Now if this population is exposed to the disease the number of victims will depend upon the resisting power and the virulence of the infection (*see* Diagram). One epidemic will perhaps cut down all whose resistance is less than a certain amount; measured by a point on the base line of our curve, all persons represented by the area to, say, the left of the base point fall victims, thus, in our diagram the ordinates numbered 1, 2, 3, 4, 5 correspond to epidemics of decreasing severity. In a more virulent epidemic the limiting points will lie to the right of that marking the critical value in the first case, the proportion of victims will be larger. Now let us suppose that immunised persons can be likened to a similar curve, but having its mean to the right of the mean of the uninoculated. Then if we make the same critical point of division we shall find that a smaller proportion of the inoculated or immunised will be victims than of the uninoculated provided the distribution on either side of the means is identical for inoculated and uninoculated. But if this is not so; if for instance the inoculated although having a higher mean resistance are more variable about the mean, at some point to the left the curve of inoculated will cross that of uninoculated and for all epidemics so mild that the critical resisting power (the point on the base line) is at or beyond the crossing of the curves, the proportion of inoculated who fall victims will actually be larger than the proportion of uninoculated. Conversely if the inoculated were less variable than the uninoculated there would be a point of crossing to the right hand, for all epidemics of greater than a certain severity the attack rate upon the inoculated would be greater than upon the uninoculated. These purely geometrical consequences are of course perfectly compatible with biological considerations,

since a prophylactic measure conferring a great average benefit may place a small minority at a disadvantage. But Greenwood and Yule, who extended this conception—originally due to Maynard—to the comparison of inoculation data found no instance in which the critical point fell within the range of practical importance, the differentiation against the inoculated only commencing at (in the case of a more variable inoculated population) a mildness of attack tantamount to no real epidemic at all, thus, on the scale of our diagram, it is impossible to indicate the position of the critical point which is reached when the attack rate is less than ·04 per 1,000. Still, since the theoretical possibility existed, it seemed well to test the influenza data in its light. The result of using the method described in the memoir of Greenwood and Yule cited above on the statistics of towns was to demonstrate that the discrepancies could not be interpreted in this way. The correlation of attack rates upon immunised and unimmunised, which on the theory would be very large was found to be but moderate. We have failed to reconcile our heterogeneous results in this way and reject the explanation.

We now pass to the second hypothesis.

It is known that different strains of an organism equally lethal for experimental animals may differ in their utility for stimulating the production of anti-bodies. Even in the instance of *b. typhosus*, Hooker* and Weiss,† have thought that a classification might be based upon antigenic powers. It might be that, even were the precise *materies morbi* common to two localities, its immunising efficiency varied, the Leicester strain might be more potent than the Manchester variety.

In a recent discussion of the epidemiology of phthisis‡ Dr. Brownlee has offered reasons for thinking that epidemiologically considered the disease breaks up into three types, one having its maximum incidence upon early life, a second form prevailing in middle life, a third form chiefly affecting the declining years.

It was shown that the gross death rates from phthisis in various English registration areas could be reproduced by combining the three typical rates in different proportions, and that in those districts in which the middle-age type was predominant, phthisis as a cause of death was sensitive to variations of the social environment (overcrowding, poverty, &c.). It should be understood that the analysis into three types does not imply that no young persons die of "middle age" phthisis, or that no old persons die of "young adult phthisis." The nomenclature is derived from the position of the maximum in the several types. Thus, the decennial death rate per thousand was estimated to be at age 20–25, 459 from the

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* *Journal, Immunology*, 1916, II., 1.

† *Journal, Medical Research*, 1917, XXXVI., 135.

‡ Medical Research Committee, Special Report Series, No. 18.

"young adult," 211 from the "middle age," and 23 from the "old age" type; at ages 25–35 the three contributions would be at the rate of 422,365 and 36. Were two communities to receive infection of, in one, "middle age," in the other of "young adults" type, deaths would occur at all ages in both, but the position of the maximum rate of mortality would not be the same in both. From the point of view of immunity it would appear that a community chiefly infected with "young adult" phthisis might derive little or no benefit from, say, tuberculin derived from cultures of the variety of bacillus responsible for "middle age" disease; Brownlee has suggested that a reconciliation of the conflicting testimonies as to the value of tuberculin treatment might be sought in these epidemiological results.

It is evident that, on this hypothesis, one indication of differentiation would be provided were the age distributions of attack significantly different in homologous epidemics.

TABLE 6.

Manchester and Leicester.

Age Group.	Manchester.	Leicester.*
	SUMMER.	
0–5	44	23
5–15	180	82
15–25	131	73
25–45	261	117
45 and over	93	30
Total	709	325
	AUTUMN.	
0–5	84	93
5–15	136	198
15–25	54	108
25–45	151	214
45 and over	48	65
Total	473	678

* This table has been constructed as follows:—

The Leicester rates at ages have been applied to the Manchester age groups, and the totals proportionately increased or reduced to bring the total number of cases at Leicester into agreement with the observed total. The probability that the two distributions might have arisen by sampling a common population is, for the summer pair, ·30, for the autumn pair, ·11.

In other words, there is no strong evidence of age differentiation in attack rates as a whole, we cannot assert that the observed difference is too great to be plausibly credited to chance fluctuations.

Thus, there is a great divergence between the immunological results in Manchester and Leicester; if we found either that the two summer epidemics or the two autumn epidemics were not *in pari materia* from the point of view of age incidence, then it would be *a priori* probable that we were dealing with different epidemiological facts, and Brownlee's case would be in point. If the test of homogeneity due to Pearson* be applied to the pair of summer and also to the pair of autumn epidemics of these samples (Table 6, p. 147) it is found that the chances that what difference there is might be due to random error of sampling are quite substantial. Hence, so far as the age incidence test is concerned, no differentiation can be established. It is, however, a noteworthy fact that Haileybury College, the one public school which recorded no sensible protection in the autumn as a consequence of summer attack, is in striking contrast to Clifton (which showed a summer–autumn advantage) by virtue of conferring an apparently considerable immunity against winter illness. This result is explicable in terms of a type differentiation, although naturally one such contrast is not enough to prove the proposition. Since quite as great differences emerge amongst the towns or amongst the schools as separate any town from any school, it is plain that variations of immunity cannot be due to the difference of environment between an urban working class population and the pupils of a public school or to the difference between age homogeneity and heterogeneity of contacts.

It is much to be regretted that the extent and nature of our material do not suffice either to prove or disprove this hypothesis of type distinctions, since it is of capital importance. If the difference between Clifton and Haileybury or between Manchester and Leicester is not due to some methodological error and cannot be explained by some environmental cause which has escaped us, but really depends upon an essential variation of the *materies morbi*, then vaccination with the Manchester or Haileybury material derived from summer cases would have had very different results from those of the exploitation of the Leicester or Clifton material. It may even be that the variations of vaccinal results reported by different observers do not mean that all were without value and the differences merely random or explicable as gross methodological errors (which often arise by contrasting case rates derived from different stages of an epidemic).

It may be that the immunological value of the inoculant was not the same for different epidemics.† This doubt will only be resolved by a combination of experimental and epidemiological research, attention being paid to the particular biological characters of material derived from different local outbreaks.

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* *Biometrika*, 1911, VIII., p. 250. † *Vide supra*, p. 118.

Measures have been taken to clear up this point, although we naturally hope that the opportunity for so doing, only provided by a widespread epidemic, will not be afforded.

It may now be asked whether, in view of this divergence, we are of opinion that on the average it is likely that some protection is usually conferred, whether in terms of our unproven but plausible explanation, homologous strains are commoner than heterologous strains. It is a delicate task to pick and choose data, but had we to give a preference we should suppose that the data collected at Cambridge by Dr. S. M. Copeman, F.R.S., are most likely to represent *average* conditions. These data were compiled with especial care, the informants were educated men, and their places of exposure to risk in the summer epidemic were widely scattered (only a small minority were in residence at Cambridge throughout the epidemics). These data show a considerable immunising power in the summer attacks, and we conclude, although with natural hesitation, that it is probable on the average that an appreciable degree of active immunity was attained by those who passed through an attack of influenza in its first and mildest manifestation. There is less evidence that the active immunity afforded by an autumn attack was of real value.

A corollary of these deductions is that, although a naturally acquired immunity is often a valuable safeguard, and its existence a justification for the further study and application of artificial immunisation, the communal value of any immunisation process is not sufficient for us to reckon it as at all likely to prevent the recurrence of epidemics in the future of the surviving generation.

We should add, with reference to the question asked by Sir Frederick Andrewes (*supra*, pp. 118–119), that the lower incidence of influenza upon the inoculated strength of the Home Forces is *not* cogent statistical evidence that inoculation did in fact confer an immunity. Three inoculations appear to have been carried out during the epidemic, and, of course, those attacked were not subsequently inoculated; the period of exposure to risk was not the same for the classes compared, being, on the average, less for the inoculated. No conclusion therefore can properly be drawn from the fact that the attack rate upon inoculated men was below the average.

CHAPTER VII.

The Weather and Epidemic Disease, with particular Reference to Influenza.

A belief that the states of the weather influence the prevalence of disease is amongst the oldest of popular doctrines, and, in a general way, there can be no doubt of its truth. Not only is it certain that "common colds" are not equally frequent in all seasons of the year, but the same remark applies to outbreaks of other endemic diseases.

A specific relation between epidemic outbursts of disease and atmospheric changes has also been asserted to exist, and, down to a recent epoch, this doctrine greatly influenced epidemiological thought in general* and the aetiological theory of influenza in particular. Modern physicians have, perhaps, usually derived their inspiration from the teaching of Sydenham or from the writings of those epidemiologists who were influenced by Sydenham's example, but the observations upon which the theory was based belong to Greek medicine, and it will be convenient here to summarise the older teaching, because so much of it has become part and parcel of our half conscious or even wholly unconscious stock of beliefs, and is still influential.

The historical source of our traditions is the collection of writings bearing the name of Hippocrates which are rather *disjecta membra gigantis* than a co-ordinated system; however, from those tractates having the best claim to be regarded as authentic, the following conceptions emerge.

The aetiology of disease involves three prime factors: (1) the character of the individual, his *crasis* or *temperament;* (2) his habits of life in the widest sense, including his diet, occupation, &c.; (3) the character and variations of the atmosphere, its *katastastis* or *status*, to which he is exposed.

In the Hippocratic writings, the words "epidemic" and "endemic" are not used quite in the sense of modern writers, and Hippocrates brings under the connotation of an epidemic prevalence of sickness which we, using the term less widely, should not so designate. He studied disease from two sides. On the one hand he linked up the general type of sickness prevailing endemically from year to year and generation to generation in any one place with the enduring and permanent characteristics of its inhabitants and its climatic peculiarities. On the other hand he also studied the diseases of the same locality in different seasons of the same year and in the same seasons of different years, and associated variations of the quantity or quality of prevalent (epidemic) sickness with

* *Cf.* Dr. Ballard's discussion of the subject in his report on the epidemic of pneumonia at Middlesbrough in 1888. (Reports of Med. Off. Local Govt. Bd., 1888, App. A, No. 18, C. 5813.)

variations of atmospheric status. It is this second investigation with which we are principally concerned. It was the model of Sydenham's books on the epidemic constitutions of London in the 17th century.

Hippocrates provided a series of observations covering some years, and these are not welded together by any detailed theory. This may partly be due to the fragmentary character of the surviving books, but a more probable explanation is that the author, evidently a man of rare sagacity, had a vivid sense of the complexity of his task, and felt that—as one of his most famous aphorisms puts it—judgment was difficult and experience deceptive.

In the hands of Galen the observations and provisional theories of the older, and wiser, physician were moulded into a system which has, both for good and evil, largely determined the subsequent development of medical ideas.

From the commentaries of Galen upon the Epidemics of Hippocrates, from his treatise upon Temperaments, and, especially, from his study of Fevers, we can form a tolerably clear idea of the Galenical doctrine, of how Galen elaborated a consistent theory of the three factors of disease.

He assigned the personal or innate character (temperament) to one of nine types, each dependent upon a different blending of four elementary characters, the Hot, the Cold, the Moist, and the Dry. The ideal or eucrasic temperament is that in which these elementary qualities are perfectly harmonised. A discord may arise in eight ways ; four of these are due to simple excess of one quality, four others to linked excess of Hot with Moist or Dry or of Cold with Moist or Dry. This is an exhaustive classification because Hot and Cold, Moist and Dry are taken to be incompatibles, co-existent excess of both members of each pair is deemed impossible, so that no linked excesses of *three* qualities are permissible.

The object of this classification—which is not intrinsically so ridiculous as the naif physical associations of the words themselves make it seem to us—is to explain the varying response of the individual to changes of environment and to what we should now term exposure to infection. A diet or an occupation appropriate to one temperament is harmful to another ; the *form* of the injury inflicted upon one temperament by any pathogenic agent will depend upon the temperament, and, as the quantitative range of each class is continuous and there are eight non-physiological groups, the scope of clinical varieties is immense.

The second group of causes, viz., the habits of the individual, is related to the first by a pathological system which we need not expound, since it has long since lost such importance as it possessed, and we pass to the third factor.

Galen posited an extra-corporeal cause of pestilence which he located in the atmosphere. He thought that this might

possibly be a mere physical property, such as excessive heat, but indicated that something more tangible, veritable "seeds of pestilence" could never be excluded and might usually be predicated. Whatever the precise origin of these "seeds," their existence depended upon a warm and moist state of the atmosphere. Thus the necessary conditions of a pestilence (*any* disease which, in our usage, is epidemic and produces a great mortality is a pestilence in Galen's usage of the term) are seeds of pestilence existing in a moist and humid atmosphere. But these conditions, although necessary, are not sufficient; they may exist and yet no epidemic may follow, because the "chief "factor in the production of a disease is the preparation of the "body to endure it."*

Hence the paramount importance of the temperamental and dietetic, or occupational, factors above discussed; the external invader, the seed of pestilence, excites a change, a "putrefaction" in one or more humours of the body.

The extent of the mischief done will depend upon the state of these humours (which will be affected by habits, &c.) and their original disposition (which will depend upon temperament). "Let us suppose that the atmosphere is bearing certain seeds "of pestilence and that some of the bodies exposed to this "atmosphere are full of waste products apt to putrify and other "bodies are pure . . . that some have lived in sloth, sexual "excess, and gluttony, others have taken exercise and eaten "in moderation. . . . Judge then which class is the more "likely to be affected by the inspiration of the putrid air. . . . "So often as the status of the air deviates from its proper norm "into the hot and humid, pestilential diseases must needs "arise, but the victims will be mainly such as were beforehand "full of impurities, while those who labour moderately and are "temperate in diet remain refractory."†

Such was the teaching of the Greek physicians. Part of it was of great value, viz., the emphasis put upon individual predisposition and habit in modifying the corporeal response to infection; we have seen that an adequate theory of influenza must take full note of this. On the other hand, although an enthusiastic Galenist might make out a case for regarding the Pergamite as a pioneer of the bacteriological school with his seeds of pestilence, there can be little doubt that he had nothing of the sort in mind and even less that the notion of

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* *De Differentiis Febrium*, Bk. I., Cap. VI. (The text with Latin translation is printed in Vol. VII. of Kuehn's edition of Galen's works, pp. 289 *et seq.*)

† *Ibid.* The first seven chapters of this work and the commentaries on Hippocrates' epidemics contain the more important epidemiological observations of Galen. The latter are printed in Vol. XVII. (*a*) of Kuehn, *see* especially p. [illegible], *et seq.*, p. 30, pp. 43–4, pp. 96–7, and p. 667. Vol. XV., p. 121, *et seq.*, are also of some interest.

putrefaction due to an atmospheric something fostered vague speculation and hypotheses which discouraged useful experimental and observational research. So far as the specific object of this chapter is concerned, that of meteorological influences, Galen added little of importance to the teaching of Hippocrates; he merely formulated in precise language the opinion that a moist and humid atmosphere forebodes pestilence. But even if this opinion were solidly based upon observation, it is not very illuminating, because the facts were derived from a study of diseases observed in south-eastern Europe and Asia Minor (Galen provides no exact epidemiological observations of Rome). Littré* pointed out that the clinical characters of the cases detailed by Hippocrates were unlike those of current diseases at Paris, but very like the accounts of practitioners in tropical and semi-tropical lands. Consequently, even were the doctrine of a moist and warm pestilential status or constitution appropriate to the general aetiology of "fevers" in tropical or subtropical lands, it does not follow that a warm moist constitution is noxious in other climes.

The error of applying literally the results of the ancients to wholly different conditions is one reason why little profit has been drawn from a study of their writings. It is the spirit, not the letter, which refreshes.

The remarks just made of course apply to the post-classical writers whose observations were not made in these latitudes; the details of their results, or alleged results, do not concern us; the only question is whether they improved upon the general theory. They did not do so. Avicenna† in his account of the subject incorporates the whole substance of the passage from Galen to which we have referred; the only additions are an amplification of the theory of a putrefaction due to the moist warm air, and some remarks upon forebodings of pestilence to be derived from the appearance on the surface of animals which usually lurk in holes (here we have the famous reference to the deaths of rats in plague times).

Finally, we reach Sydenham, who abandoned the superficially attractive system of Galen and reverted to the less formally systematic but more philosophical method of Hippocrates. Sydenham asserted roundly that it was impossible to "describe specific forms of epidemics as arising out of specific "changes in the atmosphere; easy as such a proceeding may "appear to those who can theorise about fevers and christen "the disease accordingly, speculating laxly upon those alterations which can take place in our blood and humours through "the degeneration of this or that principle."‡

Yet Sydenham did not abandon the belief that the atmosphere was ultimately responsible for epidemic disease; he

* *Œuvres d'Hippocrate*, Vol. 2 (introd.).
† *Liber Canonis*, Bk. IV., tract. 4.
‡ *Obs. Med.*, I. 2, 22.

rejected perceptible atmospheric influences, but accepted occult atmospheric influences (*nisi in quantum secretae aeris influentiae illa deberetur*). His quarrel, in fact, is rather with the Galenists than with Galen, because, as remarked above, it is hardly fair to attribute to Galen the quite crude conceptions of hot and cold moist and dry which the actual words suggest; put otherwise, we may doubt whether Galen himself really supposed that a moist and warm katastasis, status, or constitution meant no more than that the weather was warm and rainy.

We have thought ourselves authorised to devote some pages to the exploration of this ancient history, because the theories in question, whatever may be said against them, have had much influence, and their exposition displays some of the difficulties of the inquiry and why little progress has been made.

There have been several reasons for a failure to advance. One was literal acceptance of the dogma of a pestilential constitution tending to warm and moist. Since this did not seem to correspond to experience, but yet *must* be true, being classical, it followed that the whole doctrine was esoteric and of no practical value. This, no doubt, accounts for an absence of records in early modern times. After the decline of the Galenical tradition as an influential code, the epidemiological and meteorological observations of such writers as Sydenham and Huxham made it appear, not that the weather was of no importance to the epidemiologists, but that its influence was neither direct nor apt to be revealed without the application of methods and the collection of data which even now are incomplete and a century ago were almost non-existent. Hence it is that down to nearly our own time no important progress was achieved.

It was to be expected that after the establishment of the General Register Office Dr. Farr would devote attention to this matter of weather and disease, and he did so as early as in the Third Annual Report. This first note on the subject only deals with temperature and disease in London during the years 1838–41, but brings out the effect of cold upon "the pulmonary "class and the cerebral diseases of the aged." Farr asked, *inter alia*, how long the effects of excessive cold persisted, and found that the mortality rose at once and continued to be above the normal for so long as 30 or 40 days.

In the 28th Annual Report Farr made a remark which is an appropriate criticism of much statistical work (including that contained in this chapter). He observed that averages taken over extended periods might give very incomplete information as to the state of weather experienced. "The tempera-
"ture, weight, humidity of the atmosphere, and other physical
"forces should not be masked under mean values, but
"laboriously traced throughout their course from day to day,
"and if it were possible, from morning to night and from

" night to morning, and observed in connection with the con-
" temporaneous facts that relate to human life, as these also
" are successively recorded, if the sway which they exercise is
" to be appreciated in its full significance."*

In one of the weekly returns (No. 51, 1874) Farr made an assessment of the excess mortality due to cold in the severe winters of 1855 and 1874. He found that the excess mortality increased with age in geometrical progression. Thus, subtracting from the deaths at ages the corrected average number, the excesses were such that, starting from the age group 20–40, the rate doubled every 9·21 years of life in 1855, and every 8·77 years of life in 1874. It will be noticed that Farr envisaged the subject of weather and disease from a lower altitude than did the ancients. He did not attempt to connect the *ultimate* origin of epidemic or endemic disease with particular atmospheric constitutions, but contented himself with associating quantitative alterations of the variables. Herein he displayed the true modern spirit. Aiming at too much our ancestors failed to reach what was attainable; Farr and his successors with humbler aims arrived at valuable ends. In the happy phrase of Macaulay, " Plato drew a good bow, but, like
" Acestes in Virgil, he aimed at the stars, and therefore, though
" there was no want of strength or skill, the shot was thrown
" away. . . . Bacon fixed his eye on a mark which was
" placed on the earth, and within bowshot, and hit it in the
" white." Farr's successors were of his school, and discovered many important relations between weather and disease. We may instance Ballard's work on the correlation of summer diarrhœa and the earth temperature,† and Newsholme's work on diphtheria and deficiency of rainfall.‡

In the interpretation of the results obtained by these methods some difficulty is experienced when we have to do with epidemic diseases owing to the fact that many such diseases exhibit a periodicity which may have nothing to do with variations of weather. For instance, if we associate the weekly or monthly variations of, say, temperature throughout the year with the corresponding weekly or monthly death or morbidity rates, a close correspondence of the graphs may not be due to the high or low temperature determining the incidence of the disease, but merely express a seasonal periodicity; the curve of average temperature throughout the year is regular. This, however, has been the usual plan.§

If on the other hand we ignore the cyclical position of the months and associate months from a series of years, the mean

* 28th Annual Report, Registrar Gen., p. xix., also reprinted in *Vital Statistics* (Farr Memorial Vol. pub. by Sanitary Institute in 1885), p. 415.

† On Epidemic Diarrhœa, *Rep. Med. Off. L.G.B.*, 1887.

‡ The Origin and Spread of Pandemic Diphtheria, *Lancet*, I., 1898.

§ *See* Jessen, *Zeits. f. Hygiene*, 1896, XXI., 287.

temperatures of which fall within certain limits, with the corresponding disease rates* we commit another error, or at least, make another assumption. It is unreasonable to attribute the same weight to the same absolute temperature in March and January. Again, we do not really avoid the before-mentioned difficulty, because on the average the same months are likely to fall into the same groups of temperature limits.

It appears that the simplest method of securing an unexceptional comparison is to confine the scrutiny to meteorological variations of the same, or approximately the same, element in the seasonal cycle, *e.g.*, to compare the temperature and the incidence of disease within the same month over a series of years, the plan adopted by Greenwood and Theodore Thompson in their paper upon the influence of weather upon the incidence of rheumatic fever.†

The application of this method to the problem of respiratory disease will presently be shown. Before passing to this we must refer to the few attempts made to explain the actual origin of epidemic sickness in terms of meteorology. Influenza, as we mentioned in an earlier chapter, is the one great pestilence which a modern writer of genius has referred to meteorological, or more properly speaking, telluric influences.

Dr. Charles Creighton devoted a section of his classical treatise to this subject. He began by pointing out that influenza almost alone of epidemic diseases has conserved its type. "To " have lasted unchanged through so many mutations of things, " from mediæval to modern, and from modern to ultra-modern, " and to have become more inveterate or protracted at the end " of the 19th century than it had ever been is unique in this " history. Influenza appears to correspond with something " broadly the same in human life at all times."‡

Creighton notices with approval the work of Noah Webster (the lexicographer) who "saw that influenza was the crux of epidemiology." In Creighton's opinion, "From Boyle we may " take the great principle of a progressive infection through " regions of air (or leagues of ground). . . . From " Arbuthnot we may take the organic source and nature of the " influenzal miasmata, and the association with changes in " the level of the water in the soil. From Webster we may " take the idea that the historic influenzas, having been sudden, " occasional, or phenomenal, must have had phenomenal causes " somewhere in either hemisphere."§

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* *See* Behrens, *Arch. f. Hygiene*, 1901, XL., 1.
† *Journ. Hygiene*, 1907, VII., 171.
‡ *History of Epidemics in Britain*, II., 399.
§ *Ibid.*, p. 408.

Creighton collects a large number of instances in which epidemics of influenza have followed more or less closely upon terrestrial disturbances, and concludes in the following terms. "A theory of influenza constructed from such generalities as "those of Boyle, Arbuthnot, and Webster will have attractions "for many over the theory that influenza is always present in "some remote country and becomes dispersed now and then "over the world by contagion from person to person: it will "have superior attractions, for the reason that influenza is a "phenomenal thing which needs a phenomenal cause to "account for it. But if anyone were to attempt to fit each "historic wave of influenza with its particular earthquake, or "to find the precise locality where clouds of infective matter "had arisen, or the particular circumstances in which they "arose, he would certainly find his fragile structure of proba-"bilities pulled to pieces by the professed discouragers and "depravers. I make no such attempt, but I am not the less "persuaded of the direction in which the true theory of "influenza lies."*

In other chapters of this report we have referred to some of the arguments and facts just quoted, and have attempted to associate them by means of a different, although not necessarily better, hypothesis. We have not now to criticise, but merely to record the reasons for devoting attention to the weather and allied phenomena in a report upon the pandemics.

The statistical work of Farr and his successors encourages us to look for some correlation between variations of the meteorological elements and variations of the incidence of epidemic disease, especially of respiratory disease. The well deserved reputation of Creighton requires us to ask whether in the events of 1918 any meteorological happenings were so much out of the common run as to characterise the period and justify the adjective phenomenal.

Our discussion must be confined to the British Isles, and we are deeply indebted to Sir Napier Shaw, F.R.S., Director of the Meteorological Office, for the valuable advice and records which he has placed at our disposal.

The second of the two questions proposed, will not detain us long. No two years are the same and, in that sense, the weather of each is peculiar to itself. But no event of the year 1918 was of such a character that we could fairly bring it into the class of occurrences which would make it a reasonable basis from which to explore further the telluric theories of Boyle, Arbuthnot, Webster, or Creighton. We do not say that this negative result

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* *Ibid*, p. 425.

is fatal to their theories, but it absolves us from their discussion. For the same reason we have no basis whatever for the reconstitution of a doctrine of pestilential katastasis which would explain any of the epidemiological facts. Such connection between the influenzas and the weather as our study has helped to elucidate is of a lower order of importance and to it we now turn.

We shall first and more particularly consider the meteorological conditions of the London district. The three waves of the pandemic were experienced in the metropolis between the 26th and 32nd week of 1918 (first wave), from the 41st week of 1918 to the 2nd week of 1919 (second wave), and from the 5th to 18th week of 1919 (third wave). The maximum weeks of mortality were the 28th of 1918, the 44th of 1918, and the 9th of 1919. An examination of rainfall and of sunshine records does not bring out any variation from the normal which is in the same sense in each period, but there is a certain similarity between all in the matter of temperature.

It is the practice of the Meteorological Office to tabulate accumulated temperature, a base being an air temperature of 42° F. For instance, the report for the 28th week of 1919 notes that in S. E. England the accumulated number of day-degrees above 42 was 1,344, and that this was 134 below normal; in the 29th week the return is 1,460, 155 below normal. This is to say, that down to the 28th week the balance of warmth above 42 was below the average (the normal standard is the mean of the years 1881–1915), and that in the 29th week the adverse balance was increased, that week being below the standard. If now we plot the weekly variations from the standard for a series of weeks, the heights of its ordinates will measure the general situation, while the slope from point to point measures the change of position. Diagram 1 shows the deviations of accumulated temperature from the standard from the 20th week of 1918 to the 18th week of 1919 (Kew readings) and the positions of the waves of influenza in London are indicated. The broken lines correspond to the week of maximum mortality in each wave. In the 20th week of 1918 a credit balance of day-degrees over 42 F. had been amassed, and this was increased up to the 22nd week, and but slightly diminished by the 24th. Then a sharp fall began and by the 26th week nearly the whole of the credit balance of more than 70 degrees had been spent. This sharp fall immediately preceeded the manifestation of the epidemic in our mortality returns and occurred during the first stage of the pandemic in London. A similar phenomenon will be observed before the second wave; from the 38th to 40th week there is a steep decline. We see the same course before the third wave, temperature is lost steeply from the 3rd week of 1919 to the 7th, *i.e.*, until well into the period of epidemic prevalence.

To face p. 158.

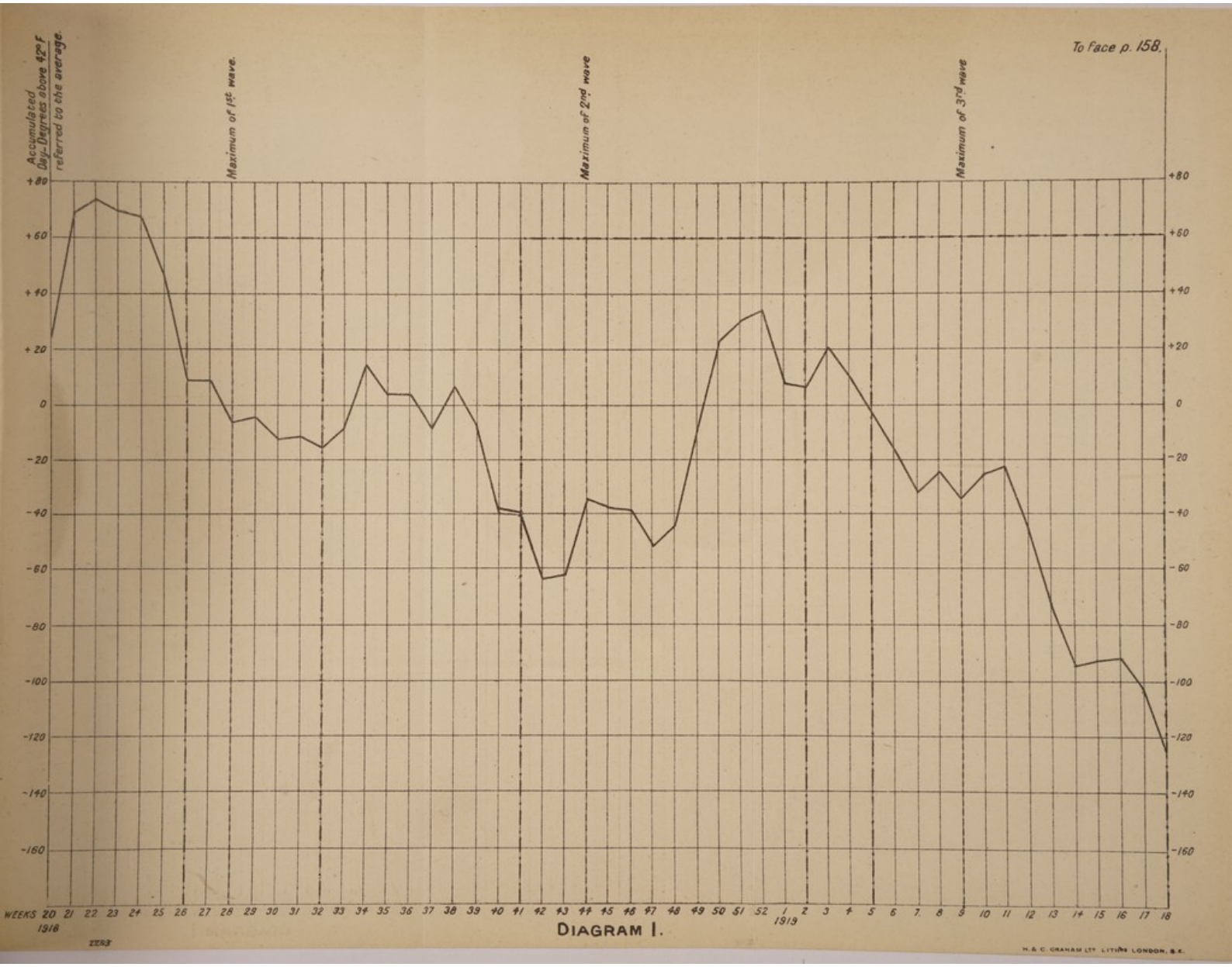

DIAGRAM I.

H. & C. GRAHAM LTD. LITHO LONDON, S.E.

To Face p. 158.

Accumulated Day-Degrees above 42° F referred to the average.

S. E. DISTRICT OF ENGLAND.
FIRST 10 WEEKS OF 1918–1919–1920.

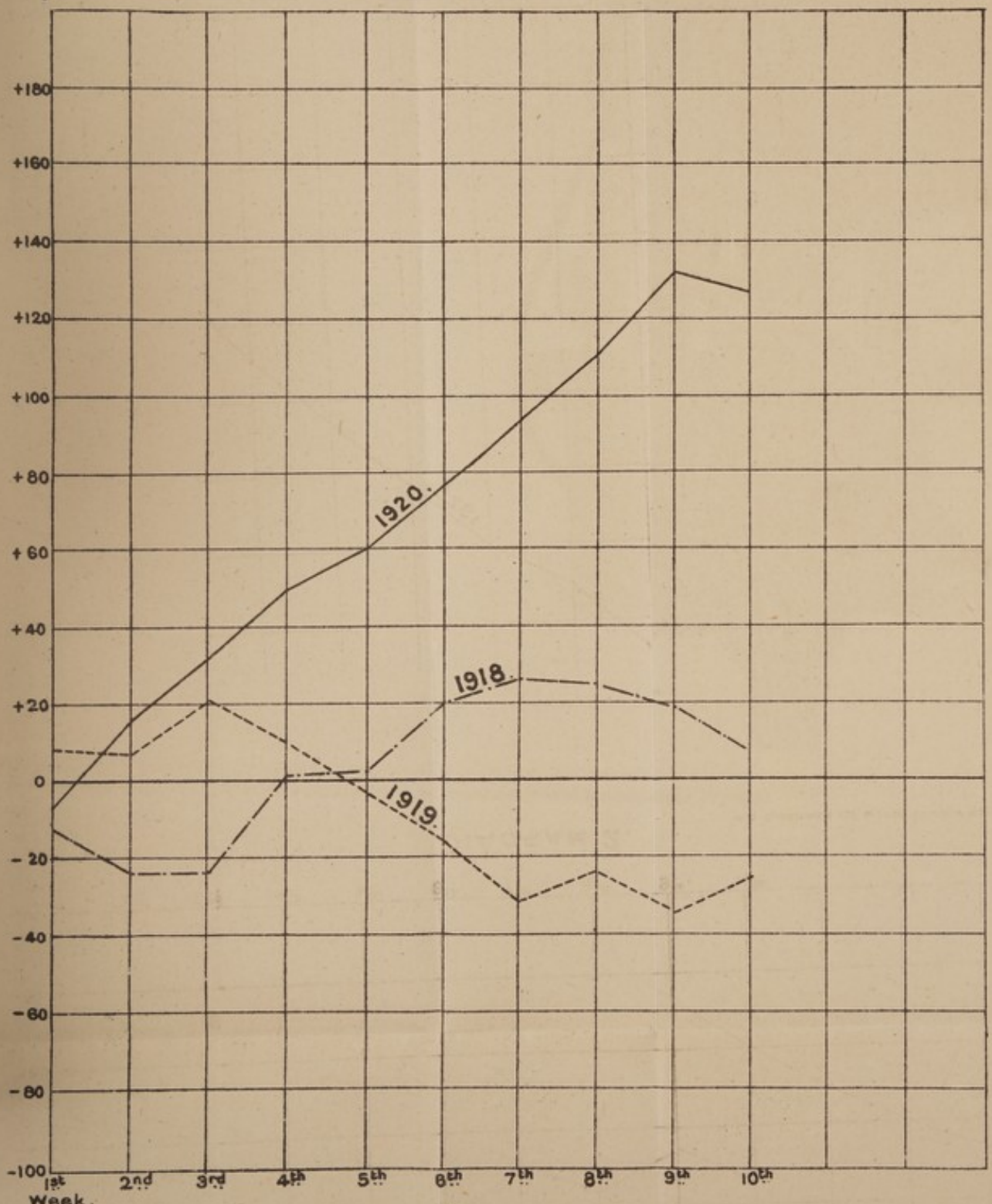

DIAGRAM 2.

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R. & G. GRAHAM LTD LITHO LONDON, S.E.

Turning to the general facts of the situation, we find that in the 25th week of 1918 the mean temperature in all stations was below normal, and strong south-westerly to westerly winds were experienced. In the following week warmth was "very deficient" in 10 districts, and "deficient" in the remaining two. In the 39th week, eight districts reported "deficient," and four "moderate" warmth. In the 40th week, S.E. England reported "very deficient" warmth, 10 others "deficient," and only one (East Scotland) "moderate" warmth. It may be mentioned that "very deficient" warmth is such a departure beneath the average as is likely to occur once in 12 years, while "deficient" warmth will occur on the average three times in 12.

In the 4th week of 1919, six districts were "deficient," and six "moderate"; in the 5th week, S.E. England was "very deficient," and all the others "deficient"; in the 6th week, S.E. England was still "very deficient," and all other districts, except Ireland, "deficient." In the 7th week, the Midlands were "very deficient," and all the others, again excepting Ireland, "deficient."

We see then that the state of affairs in London was fairly typical of conditions prevailing over most of the British Isles.

This uniformity of deficient temperature before and in the first stage of each outburst of influenza may be contrasted with the diversity of the rainfall conditions. Prior to the summer wave, the weather was exceptionally dry. The report for the 25th week of 1918 refers to the almost continuous deficiency through seven weeks, S.E. England having but 39 mm. as compared with a normal of 77 mm. In the 38th week, the rainfall was "very heavy" in 10 districts, and "heavy" in the remaining two. In the 40th week it was "very heavy" in three districts, "heavy" in seven districts, and "moderate" in only two. Thus there is agreement in the matter of deficient warmth; flat disagreement with respect to moisture.

In diagram 2 a further comparison is instituted. We give the differences of accumulated temperatures above 42° F. for the whole S.E. District of England in the first 10 weeks of the three years 1918, 1919, and 1920. In 1918, influenza was not epidemic in the first 10 weeks of the year; in 1919 there was a great outbreak, the third wave of the pandemic; in 1920 influenza was epidemic, but the epidemic was of minor importance. Only in 1919, the year of great mortality, is there a long continued downward trend, the curve from the 3rd to the 7th week was almost a straight line and more than 40 day degrees were lost in the period.

Somewhat similar observations were made in Paris, and in the report issued by the Direction d'Hygiène* the following remarks occur :—

> "Without pre-judging the relation which may exist between meteorological phenomena and the invasion of influenza, we observe that the mortality in 1918 was at first low in the course of a period of remarkable dryness from May to the end of June coincidently with a lowering of the temperature prolonged for five or six weeks. From this point the mortality began to rise slowly, and then diminished as the temperature rose and more than the average rain fell.
>
> "August was characterised by a period of four weeks' dryness and ended with a sensible increase of deaths seeming to prepare the way for the critical period from mid-September to mid-October, in the course of a phase of drought which endured for six weeks during which great deviations were noted. A lowering of the temperature, which was below normal for five weeks, coincided with this exceptional dryness. It was during this period that the percentage of deaths from influenza were most numerous ; 32·66, 45·27, 49·22, 46·59.
>
> "In November, the temperature became normal again and rain fell in abundance, the rain gauge showing a considerable positive deviation ; the deaths from influenza decreased, falling from 1,263 to 629, and then to 309 (absolute numbers) or to 42·22, 39·84, 26·45 per cent.
>
> "The curve of mortality rose again coincidently with a new fall of temperature. It reached 25 per cent. During this period, rain fell in abundance, then at the first increase of temperature, influenza declined ; this outburst lasted, with a slight inflection, three weeks.
>
> "Is there correlation between the increase of mortality from influenza, and a fall of temperature coinciding with a period of drought? Or is influenza favoured by one only of these meteorological phenomena, low temperature or absence of rain?"

These results suffice to establish a *primâ facie* case in favour of the view that an unseasonable deviation of weather towards the side of coldness played some part in the mortality of the epidemics. They not less clearly testify against any claim that weather conditions rank among the primary factors of the pandemic or epidemic prevalences. Diagram 2 makes this clear, for, were deficient temperature primary, we should expect that 1920 would have less fatal influenza than 1918, since it

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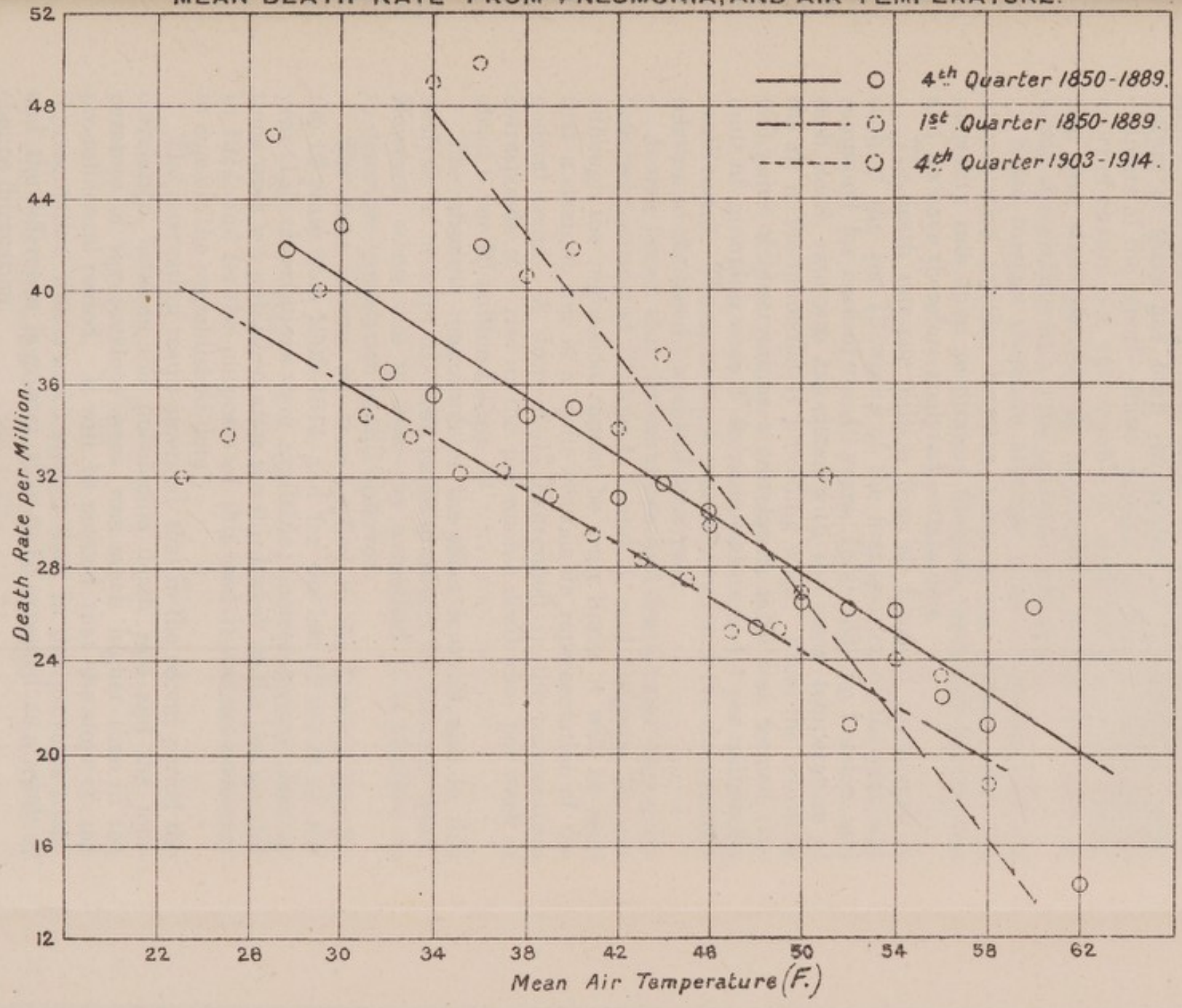
* *Epidémie de Grippe à Paris.* Recueil de Statistique de la Ville de Paris et du Départment de la Seine. 1919, p. 13.

MEAN DEATH-RATE FROM PNEUMONIA, AND AIR TEMPERATURE.



To Face p. 160.

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H. & C. GRAHAM LTD LITHOGR LONDON. S.E.

accumulated steadily for weeks a credit balance of warm days. Actually there was more influenza in 1920 than in 1918 within the periods compared. But it is hardly necessary to labour the point, since it is not here contended that deficiency of temperature was more than an accessory cause. Yet such a subordinate rôle is not without importance. We may in fact suppose that, given the necessary conditions of epidemic prevalence—the nature of these has been the subject of discussion in other chapters of the report—those climatic conditions which are the sport of chance but yet capable of moderately accurate prediction over short periods, do appreciably modify the price in terms of mortality which the epidemic will exact.

Some interest therefore attaches to an investigation of the quantitative relation between temperature changes and the mortality rate from respiratory diseases, especially pneumonia, and we have therefore analysed certain data.

The death rate per million from all forms of pneumonia in each of the last 12 weeks of the first quarter of the year was computed for each of the 40 years, 1850–1889, in London, and each death rate was associated with the mean temperature of the air in the immediately preceding week. From the resulting 480 pairs of observations a correlation table was formed (the units of grouping were 2° for temperature and 4 per million for death rates). The same course was followed with the observations relating to the last 12 weeks of each year.

It was found that temperatures and death rates were quite definitely correlated (Tables 1, 2, and 3, and Diagram 3), and although the regression cannot be truly linear it will be seen that a straight line is a quite reasonable representation of the general trend. A drop of 1° Fahrenheit in the temperature corresponds to a rise in the pneumonia death rate per week of about 6 per 10 million living.

The absolute magnitude of the effect is small, and in this analysis no attempt has been made to correct for seasonal trend. However, when this is done—by a method it is needless to to describe—correlation is still observed.

The same process was then applied to the London data for the 12 years from 1903–1914, and for the last 12 weeks of the year (135 observations were available; meteorological observations were not published after the third week of the last quarter in 1914, and for the purposes of this trial it was not necessary to consult the unpublished data).

The interesting result emerged that in the recent period the correlation between the pneumonia death rate and the temperature of the previous week was much higher than in the pre-influenzal period. It will be noticed that the slope of the representative line is much steeper than in the previous trials, and the difference is greater than to be expected as a result of chance fluctuation.

The absolute magnitude of the variation of death rate with temperature is indeed still small, and the irregularity of the trend considerable, but it is hard to escape the suspicion that the pneumonia of recent years has varied more closely with temperature changes than was the case in the pre-influenzal period. The limitation of the analysis to the last quarter of the year which, prior to the great pandemic, has not, in the present generation, been much affected by epidemic influenza avoids an obvious source of fallacy.

The object of the present remarks is not to expose the general relation between weather and respiratory diseases—a problem which it is intended to examine in detail in another report—but to illustrate the fact that inferences drawn from one period cannot be safely transferred to another. A further inference is that, although the measurable fluctuations of mortality from pneumonia and of such a criterion as air temperature have been much too small to warrant any certain inferences from the peculiarities of 1918–19 noticed above, there is still room for further study of the problem.

We may, indeed, have complete confidence that the cruder generalisations of the old hypotheses are unsound, and that the part played by the meteorological peculiarities of the pandemic years was a minor one; it is not, however, improbable that some importance attaches to the features to which we have directed attention.

TABLE 1.

Relation between the Weekly Death-rate from Pneumonia in London and the Average Temperature of the previous Week.

—	1st Quarter, 1850–89.	4th Quarter, 1850–89.	4th Quarter, 1903–14.
Mean temperature (x)	39·57	44·49	46·15
Mean death-rate (y)	30·48	30·77	31·69
Standard deviation of temperature.	5·13	6·26	5·51
Standard deviation of death-rate	8·08	9·37	10·11
Coefficient of correlation	−·371	−·458	−·707
Regression equation	$y=53·59$ $-·584x.$	$y=61·27$ $-·686x.$	$y=91·57$ $-1·298x.$
No. of observations	480	480	135

Difference of regression coefficients in the two samples of 4th quarters, $·612 \pm ·086$.

TABLE 2.

A Comparison of the observed Mean Death-rates for particular Ranges of Temperature with the Values assigned by the Equations for 4th Quarters.

Mean Temperature (F.).	1850–89.			1903–14.		
	No. of Observations.	Mean of observed Death-rates.	Calculated Death-rate.	No. of Observations.	Mean of observed Death-rates.	Calculated Death-rate.
28	4	41·5	42·1	—	—	—
30	3	42·5	40·7	—	—	—
32	14	36·6	39·3	—	—	—
34	12	35·7	38·0	3	49·2	47·5
36	18	41·8	36·6	3·5	49·9	44·9
38	42·5	34·7	35·2	5	40·5	42·3
40	45	35·2	33·8	13	41·9	39·7
42	58	31·3	32·5	19	34·1	37·1
44	57	31·5	31·1	14·5	37·3	34·5
46	58·5	30·8	29·7	13	29·9	31·9
48	47·5	25·6	28·4	25·5	28·7	29·3
50	48	26·8	27·0	11·5	26·8	26·7
52	29	26·2	25·6	12·5	21·2	24·1
54	22·5	26·2	24·3	5·5	24·0	21·5
56	13	22·5	22·9	5	23·3	18·9
58	6	21·2	21·5	4	18·5	16·3
60	1	26·5	20·1	—	—	—
62	1	14·5	18·8	—	—	—

TABLE 3.

A Comparison of the observed Mean Death-rates for particular Ranges of Temperature with the Values assigned by the Equation for 1st Quarters (1850–89).

Mean Temperature (F.).	No. of Observations.	Mean of observed Death-rates.	Calculated Death-rate.
23	1	32·0	40·1
25	3·5	34·3	39·0
27	4	46·5	37·8
29	10·5	40·0	36·6
31	22	34·5	35·5
33	26	33·9	34·3
35	51	32·1	33·1
37	63	32·4	32·0
39	58·5	31·1	30·8
41	63·5	29·6	29·6
43	80·5	28·4	28·5
45	49·5	27·8	27·3
47	35·5	25·2	26·1
49	10·5	25·5	25·0
51	1	32·0	23·8

CHAPTER VIII.

Domestic Overcrowding and Influenza.

In an earlier chapter we affirmed the conclusion that influenza is an infectious disease, and we now pass to discuss the environmental factors which might be supposed to favour

Table I.

Manchester Block Census.

District.	No. of Families.	No. of Families Attacked in First Wave.	Percentage.	No. of Families Attacked in Second Wave.	Percentage.	Persons per Family.	Percentage of Males Aged 15—45 to Inhabitants.
Ancoats C.	36	17	47.2	12	35·3	4·8	15·8
„ E.	33	12	36·4	10	30·3	4·1	18·7
„ N.	21	11	52·4	3	14·3	4·6	11·5
„ S.	37	5	13·5	8	21·6	4·7	13·9
„ W.	47	2	4·3	6	12·8	4·8	12·5
Ardwick E.	31	12	38.7	2	6·5	4·4	18·5
„ N.	30	10	33.3	9	30·0	4·4	14·3
„ S.	40	8	20·0	6	15·0	4·5	17·7
Beswick	37	12	32·4	10	27·0	4·6	15·8
Blackley	35	10	28·6	9	25·7	3·5	14·8
Bradford	32	7	21·9	8	25·0	5·4	14·0
„ No. 1	32	9	28·1	10	31·3	4·6	17·1
C./M. E.	42	11	26·2	14	33·3	4·9	12·7
„ S.	35	11	31·4	9	25.7	4·1	16·2
Gorton	36	14	38·9	14	38·9	5·1	19·2
„ S.	33	4	12·1	12	36·4	5·2	20·2
„ W.	38	14	36·8	10	26·3	4·5	18·2
Harpurley	36	15	41·7	9	25·0	3·6	22·3
Hulme C.	37	10	27·0	9	24·3	5·2	13·1
„ E.	22	10	45·5	4	18·2	4·2	10·8
„ N.	38	5	13·2	8	21·1	3·9	23·1
„ S.	35	15	42·9	7	20·0	4·3	15·3
„ W.	36	13	36·1	16	44·4	4·7	14·8
London Road	36	9	25·0	8	22·2	4·9	14·3
Newton	36	18	50·0	11	30·5	4·3	19·4
„ E.	12	6	50·0	1	8·3	5·3	21·9
St. George C.	36	20	55·6	12	33·3	5·8	12·9
„ E.	34	10	29·4	8	23·5	4·4	16·8
„ N.	28	22	78·5	7	38·9	4·8	13·3
St. George Honsell	37	16	43·2	16	43·3	4·6	16·6
Total	1,018	338	33·2	268	26·3	4·6	16·0

the transference of infection from person to person. Of these the one having the strongest *a priori* claim to consideration is domestic overcrowding. It is an old and well authenticated observation that the death rate of pulmonary tuberculosis is highly correlated with the proportion of inhabitants per room in tenements; a similar remark applies to the distribution of measles. In both instances, one of a chronic, the other of an acute infection, those crowded together in dwellings suffer a relatively heavier incidence than persons better and more amply housed. We had anticipated that the distribution of influenzal infection would exhibit the same phenomenon, and the subject was examined in detail at Manchester, Leicester, Newcastle-on-Tyne, and South Shields.

The salient features of the Manchester inquiry are exposed in Table I. (p. 164). This table has been analysed in various ways. In the first instance the distribution around the mean percentage of infected households was compared with that expected were the density per house a matter of indifference, and the chance of any household becoming infected constant. It was found that the resulting distribution did not agree with the observations, a finding which at once suggested that housing density *was* important. But when account was taken of the number of persons per family the distribution still evaded explanation. For instance, if the number of persons per family is correlated with the proportion of families attacked in each district the resulting co-efficient is nearly zero in value. As a particular instance it may be noted that the number of persons per family in West Ancoats with the lowest attack rate is the same as in North St. George's with the highest attack rate. A larger factor in producing the fluctuations of household attack rates would seem to be the variations of age constitutions. Thus

TABLE II.

MANCHESTER.

Age Distribution of West Ancoats and North St. George (Percentage).

Age Groups.	West Ancoats.	North St. George.	Whole Census.
0–5	15·6	20·7	14·1
5–15	24·1	29·6	27·1
15–25	18·8	11·9	14·0
25–45	22·8	28·2	29·0
45–65	13·8	9·6	13·0
65 and over	4·9	·0	2·8
Total	100·0	100·0	100·0

the age constitutions of the two last-named districts are different, and significantly so (Table II.), and we have already remarked that in each wave a special age incidence prevailed. To form some notion of the magnitude of this disturbance we correlated the proportion of inhabitants aged 15–25 with the attack rate, and compared the attack rates predicted from the resulting equation with those observed. This led to a better accord than the hypothesis of either independent chance or of dependence upon density, but was still far from satisfactory, hence some source of disturbance not revealed by the figures has operated (the possibility that the proportion of males at ages 15–45, *i.e.*, persons certainly mixing with the outside world, would be correlated with the attack rate was examined, and the correlation found to have no significance). But it seems clear that the number of persons per family has not been an important determining cause.

It is, of course, reasonable to contend that the average number of persons per family is not a good measure of domestic overcrowding, and we pass to evidence concerning the number of persons per room and the attack rate.

In his Leicester census Dr. Arnold found that 2,122 persons were housed in tenements providing one or more persons per room, and amongst these 655 cases occurred, or 30·9 per cent. (all waves). The number housed less than one person per room was 2,480. Of these 725 were attacked, 29·2 per cent.; the attack rates are sensibly equal. In houses with eight or more persons to the house (627 persons), the attack rate was 27·4 per cent. In houses with two or less persons (318 in all) 26·7 per

TABLE III.

NEWCASTLE-ON-TYNE.

Households containing Persons per Room.	Total Number of Households Investigated.	Number of Households affected in the whole Epidemic Period.	Number of Households not affected throughout.	Proportion of affected Households to Totals.	Total Number of Cases.	Average Number of Cases per affected Household.	Percentage of Cases to Immediate Contacts in affected Households.
				Per cent.			Per cent.
Less than 1 person -	101	36	65	35·6	49	1·36	47·1
1–2 - - - -	388	166	222	42·8	270	·63	40·6
2–3 - - - -	292	142	150	48·6	322	2·27	41·4
3–4 - - - -	138	77	61	55·8	192	2·50	39·8
4 and over - - -	51	22	29	43·1	60	2·72	37·3
Total - -	970	443	527	45·7	893	1·15	40·8

cent., again a sensible equality of incidence. The data furnished by the assistant medical officer of health, Newcastle-on-Tyne, are shown in Table III., p. 166. No evident association between housing density and attack rate is discernible.

In South Shields information was obtained which is summarised in Table IV. There being some suggestion of a higher

TABLE IV.

SOUTH SHIELDS.

Rooms per Person.	Percentage of Cases.
1·52	15·9
·33	25·0
·49	8·1
1·43	21·4
·49	43·9
·57	4·3
·21	19·6
·95	4·7
1·63	13·5
·46	15·8

attack rate upon the more congested streets, the correlation between rooms per person and attack rate was computed, but although negative, *i.e.*, the attack rate declined as the rooms per person increased—its absolute value did not exceed its probable error and indeed without any calculation it is apparent that the relation is not uniform or striking. Dr. Joseph, M.O.H., Warrington, found that of 777 persons living in infected houses with one or more persons to a room, 327 (42·1 per cent.) contracted influenza. Amongst 112 persons housed at a rate of less than one person per room, 42 or 35·7 per cent. were attacked. From these and other tabulations (*see* Table V.), it is seen that the association between density and attack rate is of little importance.

In a valuable report on the epidemic in Paris issued by the Prefecture of the Seine,* it is inferred, from a graph of the death rates from influenza plotted against an index of housing conditions, that "overcrowding and insufficiency of housing are principal factors of the frequency and gravity of influenza." The writers point out that the particulars of housing and population are inadequate, since they depend upon pre-war enumeration, but we do not think that even *primâ facie* they suggest any causal association between housing and mortality from influenza. From the tables inserted in the report we have extracted:—(1) The proportion of the population who "vivent

* *Receuil de Statistique de la Ville de Paris—Epidémie de Grippe* 30 *Juin* 1918–26 *Avril* 1919. Paris, 1919.

dans des logements défectueux," the death rate from influenza and the death rate from other causes than influenza in each

TABLE V.

Influenza and Housing Conditions at Warrington.

	251 Houses with one or more Persons per Room.	102 Houses with less than one Person per Room.
	Per cent.	Per cent.
Not-infected	123 = 49·0	60 = 58·8
Infected	128 = 51·0	42 = 41·2
No. of occupants in the infected houses.	777	112
No. of persons who contracted influenza.	327 = 42·1	42 = 37·5

Houses with two or more Persons per Room.	Houses with less than two Persons per Room.
There were 66 houses occupied by 467 persons.	There were 287 houses occupied by 1,159 persons.
The No. of persons affected was 118, or 25·2 per cent.	The No. of persons affected was 251, or 21·6 per cent.

	66 Houses with two or more Persons per Room.	287 Houses with less than two Persons per Room.
	Per cent.	Per cent.
Not-infected	23 = 34·8	160 = 55·7
Infected	43 = 65·1	127 = 44·2
No. of occupants in the infected houses.	314	575
No. of persons who contracted influenza.	118 = 37·6	251 = 43·6

quartier (omitting Salpêtrière, the enormous general death rate of which may depend upon the presence of institutions). Tables V.A, V.B, V.C contain these particulars in as fine groupings as the small total number of observations admits. Working out the correlations in the ordinary way, it appears that the correlation between the death rate from all causes except

influenza and the measure of bad housing for a constant influenza rate is ·57 ± ·05; the correlation between the influenzal death rate and bad housing for a constant death rate from other causes is − ·06 ± ·08 and the correlation between the death rates from influenza and from other causes, for housing conditions constant is ·19 ± ·07. In other words there is no measurable association between bad housing conditions and the death rate from influenza, the result we have found in our own experience.

TABLE Va.

Death Rate per 10,000, *other Causes than Influenza.*

Death Rate per 10,000, Influenza.	50–75.	75–100.	100–125.	125–150.	150–175.	175–200.	200–225.	225–250.	250–275.	Totals.
15–20	—	—	1	1	1	—	—	—	—	3
20–25	1	4	6	4	2	—	—	—	—	17
25–30	—	7	6	11	3	—	—	—	—	27
30–35	1	2	4	8	3	—	—	—	1	19
35–40	1	—	3	2	2	—	—	—	—	8
40–45	—	—	1	1	1	—	—	—	—	3
45–50	—	—	—	—	—	—	—	—	—	—
50–55	—	—	—	1	1	—	—	—	—	2
Totals	3	13	21	28	13	—	—	—	1	79

TABLE Vb.

Death Rate per 10,000, *other Causes than Influenza.*

Proportion per 1,000 Defectively Housed.	50–75.	75–100.	100–125.	125–150.	150–175.	175–200.	200–225.	225–250.	250–275.	Totals.
100–150	1	—	—	—	—	—	—	—	—	1
150–200	1	2	—	—	—	—	—	—	—	3
200–250	1	7	1	—	—	—	—	—	—	9
250–300	—	3	2	—	3	—	—	—	—	8
300–350	—	—	4	1	—	—	—	—	—	5
350–400	—	—	4	4	1	—	—	—	1	10
400–450	—	1	5	2	1	—	—	—	—	9
450–500	—	—	2	9	1	—	—	—	—	12
500–550	—	—	3	4	3	—	—	—	—	10
550–600	—	—	—	5	1	—	—	—	—	6
600–650	—	—	—	3	3	—	—	—	—	6
Totals	3	13	21	28	13	—	—	—	1	79

TABLE Vc.

Proportion per 1,000 *Defectively Housed.*

Death Rate per 10,000 Influenza. —	100–150.	150–200.	200–250.	250–300.	300–350.	350–400.	400–450.	450–500.	500–550.	550–600.	600–650.	Totals.
15–20 -	—	—	—	—	—	1	—	—	1	1	—	3
20–25 -	—	1	4	1	1	2	3	—	1	2	2	17
25–30 -	—	—	4	3	2	3	4	6	3	—	2	27
30–35 -	1	1	1	2	—	3	1	4	3	2	1	19
35–40 -	—	1	—	2	1	—	—	2	1	1	—	8
40–45 -	—	—	—	—	1	1	—	—	1	—	—	3
45–50 -	—	—	—	—	—	—	—	—	—	—	—	—
50–55 -	—	—	—	—	—	—	1	—	—	—	1	2
55–60 -	—	—	—	—	—	—	—	—	—	—	—	—
Totals	1	3	9	8	5	10	9	12	10	6	6	79

The limitations attaching to these negative results are to be emphasised. To infer from them that the intensity of aggregation in dwellings is without influence upon the spread—*a fortiori* to suppose that it does not affect the fatality—would be unwarrantable. The proper conclusion is that such variations of congestion as occur in an ordinary sample of working class communities or can be gauged by statistics of inhabitants per room or of persons per family are not factors of the first order of importance in the dissemination of the disease. The most tragic events in the history of the recent influenzas, those witnessed on certain transports bringing great numbers of troops to Europe, happened under conditions of serious overcrowding, probably much more serious than found in any statistically significant proportion of a civil population. But it must be added that even on ships of war, greatly congested and peopled with men at the most susceptible age, the attack rate did not usually exceed what we have found in civilian groupings. Surgeon Commander Sutcliffe estimated the summer attack rate on officers and men of the Grand Fleet at 11·5 per cent. of the average strength, a not excessive figure.

Superficially regarded, the result reached is so irrational that it requires further justification. It will be asked whether the finding is not a *reductio ad absurdum*, since we have admitted and even emphasised the infectious character of the disease; surely, it may be said, the most favourable conditions for the evolution of an infective disease are afforded by overcrowded dwellings, and if one finds no decisive relation between overcrowding and case incidence, should not this be taken to prove not that no such relationship exists, but that the data are unsuitable.

It is perfectly true that the consequence of errors of observation is to conceal manifestations of a statistical law, and, without doubt, errors inhere in the material discussed. But it has been found that the data still provide evidence of infectivity, that the distribution of cases is not a random one, although density in houses does not seem to be a source of disturbance. Two other tests have been applied. The distribution of houses containing 0, 1, 2, &c. infected persons has proved to be unlike a simple chance distribution, and—what is, perhaps, a more useful criterion—the average interval between successive deaths in the same house or, more generally, the average interval between the members of all possible pairs of deaths, when deaths have occurred in a house, is much shorter than one would expect to find as a matter of chance. If within a period of m days, a number of pairs of events have happened at random, the mean interval between members of such pairs would be about $m/3$ days. An application of this test to deaths in the C.B. of Blackburn showed that in November and December the average interval was about three days, the defect from the chance expectation being too great to be reasonably attributable to error of sampling. Hence, the conclusion is to be drawn, verified by much non-statistical evidence, that successive deaths or cases are not independent but contingent, and, therefore, that direct infection was probably a factor.

The explanation of the relative unimportance of the house density factor seems to us to be the following:—In the first place, as we have remarked above, the natural infectivity of the germ may be so high that the necessary exposures and contacts of *all* persons living under urban conditions are sufficiently numerous to provide opportunities of transfer so effective that any increase above the average is relatively a factor of negligible order. In the second place, since this will clearly not explain why some districts, or certain houses, suffer much more than others, we must postulate the existence of a variety of strains of infective organism or a considerable variation of resisting power to attack (the latter possibility seems less probable, since we cannot suppose that the inhabitants of a particular district—although we might perhaps predicate it of the inmates of a particular house—all chance to be naturally immune). The former supposition has in its favour other evidence. We saw that the amount of immunity enjoyed during a second exposure to infection by those who had taken the disease before has varied from place to place, even when the circumstances, whether of age or housing conditions, were identical. To explain such discrepancies, we might suppose that the infective properties of the organism have not been uniform, that in some instances the biological type of the second invader has agreed in important particulars with that of the first assailant, that in others this is not so. On this assumption, we could understand why the disease should behave like other

infective diseases in some respects (non-random distribution through houses and districts, short interval between successive cases), and, in others, deviate from the usual course. A thoroughly stable, not highly infective organism, the properties of which vary but little, should certainly cause a greater relative incidence upon overcrowded tenements.

Finally, these results do *not* lead to the conclusion that overcrowding is a matter of indifference from the administrative side. It is, for instance, possible, or even probable, that the incidence of complications and, therefore, the death toll is correlated with density of population. The data used in the present analysis although extensive enough to bear an argument concerned with incidence are much too slender to admit of analysis from the standpoint of mortality in correlation with density. The analysis of the mortality figures of the great towns in chapter 2 suggested that the influenzal death rate was substantially correlated with the death rate from other causes; the latter is well known to be associated with density (whether through the directly prejudicial effects of overcrowding upon health, or because the poorest and physically least fit are *ab initio* overcrowded, is not here to be discussed); further, the very serious mortality experienced on several of the United States, Transports hurrying men to this country at the crisis of the war is *primâ facie* evidence of the importance of this factor when mortality is to be assessed. In ordinary times, we should be able to explore the matter, as did Dr. Farr in 1847, by contrasting the death rates of various quarters of the metropolis which exhibit extreme variations of housing density. But our present inability to assign moderately accurate population totals or criteria of housing density would render such comparison misleading, and it has not been attempted. We conceive that what has been adduced does no more than make it improbable that domestic overcrowding can be deemed a *principal* factor of the spread of epidemic influenza.

CHAPTER IX.

The Prophylaxis of Influenza.

Some derision has been excited amongst the laity by the prescription of rules for avoiding infection, which none but a small minority of the population can obey. The derision, although natural, is illogical, and serious mistakes in the history of preventive medicine can be attributed to the desire of physicians to provide not only a panacea, but a panacea which will be agreeable to or within the reach of the general public. We may add that not only do practically unrealisable measures excite derision, but, in those measures which might be carried out, congruity with popular notions of medical fitness (as in the leading case of Naaman the Syrian) has always been a principal factor of acceptance. In our own generation the traditionary belief in a specific pill or potion is not extinct, but it has to struggle with a more recently engendered faith in the value of laboratory methods leading to the injection of some substance beneath the skin. Like most popular beliefs these are not unwarranted—such great triumphs as the bark treatment for malaria in the past or anti-typhoid inoculation in the present are sufficient examples—but there will be found reason to suppose that half knowledge has created difficulties in the way of a clear apprehension of the prerequisites of success.

From what has been said in this report it follows that to avoid crowds, to shield the mouth and nose when coughing or sneezing, not wantonly to thrust one's face into the face of one's interlocutor, are essentially rational and appropriate methods of reducing the risk to take influenza. It even appears probable that the one form of overcrowding which in some little measure *can* be controlled by the individual is the form most potent to spread the disease. In the present state of the nation to advise the avoidance of overcrowded tenements and lodgings is, indeed, a council of perfection; it is likewise a vain quest to seek for public conveyances not grossly overcrowded; but it is quite easy not to frequent theatres, music halls, and picture houses; it is even possible to avoid bargain sales, political meetings, and assemblies. These forms of congestion belong to the extra-domestic group which, by a process of exclusion, we have discovered to be more influential than intra-domestic conditions admittedly hard to control now.

It follows that the derided advice to avoid crowds is neither so trivial nor so absurd as has been believed. In our opinion the public ought not to be deterred from following it either by

newspaper ridicule or by the thought that in any event they cannot escape some crowding when engaged in active pursuits.

Passing to the prophylactic measures more congruent with the popular sense of fitness, we have first to consider the claims of the traditional pharmacopia and of the products of synthetic chemistry which, attested by recommendations of some medical men, have been pressed on the attention of the public.

So far as prophylaxis is concerned, in no instance has even a *prima facie* case been made out on behalf of any drug. It is surely obvious that the fact that a group of individuals to whom some prophylactic has been exhibited did not contract a disease only merits consideration when it can be shown that an essentially similar sample, submitted to the same or similar conditions of risk, but not using the prophylactic, suffered from the disease. This elementary precaution has not been observed by the bulk of adherents of specific prophylactics; certain exceptions will be noticed below. We do not think there is any doubt that the oral administration of drugs is quite ineffective in warding off attacks of influenza.

Passing to the second approved method of defence, by means of vaccination, the following are the chief points to be had in mind.

When the *materies morbi* has been isolated, and the existence of immunising responses to inoculation with such *materies* or some derivative thereof has been observed under laboratory conditions, the antecedent probability that a method of inoculation will be useful is great. All that remains is to determine whether the extent of practical advantage conferred exceeds any risk or inconvenience attending the application of the process. The second part of the inquiry demands time and care; the essential point is to be sure that trials on the practical scale are made *in pari materia*, inoculated differing from uninoculated solely in the matter of inoculation, or, when this is impossible, as it nearly always is, to make quantitive allowance for the disturbing influences present. The history of anti-typhoid inoculation illustrates all phases of the investigation, from the isolation of the specific microbe, through the experimental stage, up to the field inquiry. It was possible before the outbreak of the war to claim a high degree of probability in favour of the view that anti-typhoid inoculation was a prophylactic measure of the highest importance, and nothing which has since occurred casts doubt upon the validity of the arguments then used or the expediency and efficiency of the administrative actions to which they gave rise.

When the *materies morbi* is unknown in the sense that it cannot either be physically identified or cultivated *in vitro* it is evident that a truly specific "vaccine" cannot be prepared, although it may happen that a method of vaccinal prophylaxis is practically useful. This proved true in connection

with the disease known as swine fever here and hog cholera in America, the essential organism of which is ultra microscopic and incapable of cultivation *in vitro*.

The inoculation of blood from infected animals coincidently with that of the serum of treated or recovered animals was found empirically to confer a large measure of immunity against the natural disease. In such a case the antecedent probability of success is slighter than when the vaccine is derived from a well authenticated *causa essentialis*, but, provided the field testing be carried out under the necessary conditions of stringency, the final practical result may be of equal importance. In the matter of influenza, not only do we start without the antecedent probability assured by the isolation of the essential microbe, but field testing has to be carried out under conditions of exceptional difficulty.

The bacteriological position of the subject is explored in another chapter; here all that we need to remark is that the materials used in the preparation of vaccines consist of organisms admitted by most to be important sources of damage in the assault of influenza, and by some to be the only serious assailants. But those who admit these arguments recognise that the biological properties of the different organisms may vary from strain to strain, and some skilful bacteriologists even doubt whether the reduction of individual strains to a relatively small number of differentiated types, as has been possible for meningococci and pneumococci, is practicable. Hence there is no great *a priori* probability that if the inoculated subject do in the course of his exposure to infection encounter the micro-organism which has furnished the vaccine, such micro-organism will be of a type having immunological affinity to the source of the prophylactic.

Finally, the total failure in some instances and the partial failure in all instances of a natural attack of the disease called influenza to confer immunity against exposure to a second wave of epidemiologically similar disease is not encouraging to those who are at best imitating, *longo intervallo*, the processes of nature.

These various considerations combined to diminish any hopes of a dramatic success in the use of anti-influenza vaccines such as crowned the anti-typhoid inoculation campaign, and the consequent pessimism has been justified. The Ministry of Health assisted those willing to employ prophylactic inoculations and collected statistics which, however, have so far been scanty and unreliable. It is natural enough that during the very strenuous time of the epidemics, the pressure due to the epidemic itself being superadded to the general dislocation of war time, the statistical data provided should lack exactitude; in any event the returns were so incomplete, evidence of true comparability*

* No inferences can be drawn from a lower attack rate upon inoculated than upon uninoculated persons if the inoculations were carried out during the epidemic, without due allowance for variations of length of exposure.

in the few instances in which a large measure of protection seemed to have been conferred upon the inoculated, so slight and untrustworthy, that we are unable to say that the arguments in favour of prophylactic inoculation as a general measure have been at all strengthened by our experience of the 1918–19 pandemic. Such evidence as we have in favour of any practical advantage points to the efficacy of vaccines derived from organisms cultivated from cases existing in the neighbourhood of the tested population. This is illustrated by the example quoted in the section of influenza in the annual report of the Chief Medical Officer. A pure Pfeiffer vaccine was used for the inoculation of employees (volunteers) in a London business house, and the inoculated escaped the disease, while their co-workers experienced a normal incidence. It appears doubtful whether any simple stock vaccine derived from a limited number of strains of micro-organisms implicated in the clinical evolution of influenza is likely to be of value. Whether vaccines derived from the special strains isolated in each community are more beneficial; if so, what geographical and population groupings might be the best units for vaccinal purposes, are questions which we cannot yet answer. It may even be that the specificity of strains is carried in nature to so fine a point that no local sub-division upon a scale practically possible would be of service. This is a problem of the future; here we can but record the conclusion that vaccinal prophylaxis is not yet a reliable shield.*

It thus appears that neither of the methods which would *a priori* commend themselves to the public, that of exhibiting specific drugs or that of inoculating specific vaccines is a measure which in the event of a recrudescence of the disease can be relied upon. Yet we must draw a distinction between the two in so far as the use of potent drugs, especially perhaps alcohol, may quite conceivably do serious harm while the inoculation of a vaccine, provided the operation be performed under conditions of surgical cleanliness and the source of the material above suspicion, can do no harm and may do great good.

We now come to simpler measures of prophylaxis which, chiefly on that account, need stronger credentials before they are likely to commend themselves to the public. The most important is nasal disinfection. The usual source of infection being the naso-pharynx and the toxic influence upon life of gaseous fumes painfully familiar, it was to be expected that nasal disinfection by gas would have supporters. The matter was put upon a scientific foundation by Dr. Benjamin Moore, F.R.S., who, in conjunction with Dr. A. Gregor, carried out a number of observations upon the sterilising action of nitrogen peroxide or sulphur dioxide in high dilutions (1 in

* *See also* pp. 64 and 195.

30,000 volumes). These observers obtained (*a*) a partial disinfection of the naso-pharynx commencing one or two hours after exposure and lasting for 8–12 hours; (*b*) they believed the hydrogen-ion concentration of the nasal secretion increased, the secretion becoming acid to litmus and remaining so for some hours. When the fumes included nitrogen peroxide, starch and potassium iodide rapidly turned blue in an atmosphere which could be borne without discomfort, while, a few hours later, the nasal and bronchial secretions became orange yellow from the xantho-protein reaction with mucin.

These experimental results were a motive for epidemiological comparison of the incidence of influenza upon workers exposed to nitrous or sulphuric fumes with that on normal controls, and in a few instances there appeared to be a very much smaller incidence upon the workers exposed to gas than amongst controls. The importance of the matter led to a series of special investigations by officers of the Ministry and extensive statistics were collected at Widnes, in the Swansea Valley, and in a Metropolitan gas works. A précis of the results was included in the report of the Chief Medical Officer and the complete protocolls are appended to this report. From these it appears that the prophylactic value of such exposures as occur in the normal course of industrial life is inappreciable. These negative conclusions throw no suspicion upon the cogency of the original experiments since there is no reason to doubt that the concentration of the fumes was often, perhaps always, less than in the conditions of those experiments, but they perhaps make it doubtful whether any method of fume disinfection suitable for wide employment is likely to be a success*; the essential point of the experiments, viz., an attempt to heighten local resistance in the nasal-pharynx raises wider issues.

Dr. Leonard Hill, F.R.S., has recently published an important study of the relation between the condition of the nasal mucous membrane and susceptibility to coryza.†

Dr. Hill first drew attention to the long noted relative immunity from colds of persons living under open air conditions. Dr. Hill argued that for the realisation of infection, not only must a *materies morbi* be present, but also the naso-pharyngeal tissues must in some way be rendered sensitive. He writes: "F. F. Muecke and I examined the appearance of " the nasal mucous membrane when the subject is exposed " first to outdoor conditions, and secondly, to those conditions " of heating and ventilation which are not uncommon in rooms. " Out of doors the wind moves at a much greater velocity at " head than at foot levels, owing to friction of the moving air " against the ground; the ground, too, is warmed by the sun.

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* *See also* Parsons II., p. 58.

† The Science of Ventilation and Open Air Treatment, Pt. I., pp. 141, *et seq.* (London, 1919, published by the Medical Research Committee.)

" As a rule then, excepting such unpleasant conditions as " pertain to thawing snow, the head is cooled out of doors at a " greater rate than the feet. Cool breezes blowing round the " head, the radiant heat of the sun, and a warm ground to " stand on, are the ideal outdoor conditions. The healthy " mucous membrane of the nose, examined by aid of a " speculum, under these conditions appeared pale and taut. " If touched with a probe, it did not pit, and there is no thick " secretion upon it. Indoors in these conditions which are " unfortunately only too common, when the feet are chilled by " draught blowing over a cold floor, and the head immersed in " warm stagnant air, the nasal membrane appeared in those " examined swollen, congested, and covered with thick secre- " tion. A probe pushed into the swollen membrane formed a " pit, showing how boggy it was, and the airway was so " diminished that I, with a deflected septum, could not breathe " through the affected nostril. Warming the feet at a fire at " once relieved the congestion and did away with the obstruc- " tion, so that the airway became free. It is the congestion " and swelling of the nasal membrane, and no doubt that of " the air sinuses opening into the nose, which largely causes " the feelings of stuffiness in the head and headache felt in " crowded, overheated, places of assembly. This headache is " generally, but erroneously, attributed to the absorption of " chemical poisons supposed to be present in the stuffy " air. . . . Suppose the nasal membrane has become " swollen, congested, and covered with thick secretion in these " bad conditions, and the subjects go outside again into the " cold air, then the membrane at once becomes pale, owing to " vaso-constriction, but for some little time it remains swollen " and boggy, pitting on the touch of the probe. I believe that " these conditions of the nasal membrane have a great deal to " do with catching colds. In crowded rooms infection takes " place from mucous spray, sneezed, coughed, or spluttered out " in speaking. The inhaled bacteria will be caught by the " swollen mucous membrane covered with thick secretion. In " those who live out of doors not only is the membrane kept " taut, but the flow of arterial blood through it is rapid, for the " inhaled air has to be warmed up and moisture rapidly " evaporated from the membrane so as to saturate this air at " body temperature. Thus more lymph comes out into the " membrane from the blood vessels, and this contains pro- " tective substances. Offensive bacteria are either washed " away or destroyed, and thus kept out. On the other hand, a " membrane covered with thick secretion, and congested, offers " a medium more suitable for the bacteria to settle and grow " on, for it is boggy and stagnant, not flooded with fresh " lymph, particularly when vaso-constriction takes place on " passing from an over-hot moist atmosphere into the wintry " outside air. The ciliated cells, white corpuscles, and lymph

" may be chilled and the velocity of vital reaction reduced " when the blood vessels constrict. Colds may not always be " a question of re-infection, but of lowered resistance to the " strains of pneumococci or streptococci which people happen " to carry in their noses. Stagnant conditions of the nose and " its sinuses may exalt the virulence of organisms and so " spread infections. Epidemics may thus be started by " carriers whose humoral conditions are altered by unhealthy " enviroment. It is recognised that measles and influenza " lower the resistance to other organisms, *e.g.*, pneumococci, " tubercle bacillus; mustard gas likewise lowers the resistance " to infection, why not then the conditions set up by the " change from heated to cold atmosphere. The washing away " of bacteria by the outflow of secretion must be one of the " most important methods of defence. T. H. C. Benians " suggests that mucin is probably the first line of anti-bacterial " defence in the nose and throat, as throughout the rest of the " alimentary canal. In most acute influenzal throat infections, " as well as other acute septic throats, the mucosa appears red, " dry, and with an absence of visible mucin. Many such " throats when tested are strongly acid to litmus. The natural " reaction is alkaline. As acidity is against the growth of " catarrhal organisms, these must escape the acid reaction " of the surface by invading the membrane. In the running " stage of a cold the secretion is often found to be sterile."

Recently Wahl, White, and Lyall* report that influenza bacilli experimentally implanted in the nares disappear in 24–72 hours, but when implanted on the throat persist and multiply for weeks. The original observations of Hill and Muecke on the changes in the nasal mucous membrane have been confirmed by Cook and Winslow, and it will appear that the general ætiological theory of Hill is in accord with the epidemiological results we have described and reconciles some anomalies of everyday experience. Thus the evidence as to the relative incidence of influenza upon indoor and out of door workers has been conflicting; it has not been found uniformly that the outdoor workers were less affected; omnibus conductors have been said to suffer greatly from the disease. The explanation is that the *conditions* of exposure are of paramount importance, everything depending upon the manner in which the local tissue reacts.

If a stagnant condition of the mucous membrane concomitant with a slow rate of local circulation and imperfect secretion of lymph be generated by temporary exposure to unfavourable conditions (such as arise in the course of transport workers' duty) intermittent exposure to cold may actually further reduce the local resisting power. In the end we reach the conclusion that it is not the presence in the nostrils of any specific chemical, but the realisation of the physiologically most favourable conditions, whether as a result of

* *Journ. of Infect. Diseases*, XXV., 1919, p. 419.

specific medication or otherwise, which is the true method of prophylaxis. Thus we might explain the success said to have been met with through the use of various snuffs in warding off infection. It was no specific property of the snuffs but the stimulation of the local secretion and the circulatory conditions favouring the secretion which was responsible for favourable results. Were the snuff not to produce this reaction its presence in the nasal cavity would be unavailing. It might be asked whether there is any evidence that inveterate snuff takers are less sensitive to nasal infection than others, but a moment's reflection shows that no information of value could be obtained in such a way. Persons who are addicted to this old fashioned method of using tobacco, resort to it with such frequency that it is probable that direct mechanical obstruction of the nasal passages might counterbalance any advantage which should in theory result.

The apparently great danger of spreading influenza by extra domestic crowding might be inferred from Hill's theory, since the crowded railway carriage with its humid warm atmosphere and draughty floor is precisely the condition needed to reduce the defensive power of the nasal mucous membrane to its minimum. The practical inference deducible from the results described is that attention to the hygiene of the nose may be a most useful prophylactic. Douching of the nose morning and night with a faintly antiseptic solution and occasional resort either to smelling bottle (which Dr. Hill suggests might contain sodium bisulphite) or snuff box (which may contain any of the high dried tobacco snuffs, or, if preferred, the menthol snuff sold by most chemists) are recommendable measures. The snuff or smelling bottle should be used when the subject finds that he or she cannot inspire or expire through the nose with customary ease. As remarked, the formation of a snuff habit, like that of the traditional antiquary or Scotch grandfather, would defeat the end in view, viz., to use the stimulant only when needed by the mucous membrane. We are naturally led by these considerations to the question of masks as prophylactics. From the point of view of the general population it would seem that the cogency of the arguments just recited is decisive against masks. The probability of rendering a mask, habitually and frequently worn in everyday life, a trustworthy bacterial filter is very small, of its causing sufficient obstruction to nasal respiration to disturb the physiological harmony desired, very great. None of the sensational statements in the Press that epidemics of influenza abroad have been controlled by the compulsory wearing of masks in public have been found trustworthy. In our opinion the general wearing of masks is neither expedient nor scientifically defensible.

On the other hand, when the question is of affording extra protection to the attendants upon the sick in hospital wards, the objections just adduced lose validity. It even appears desirable that such precautions be taken, since the ritual would

be intelligently carried out and its existence inculcate upon the subordinate staff a sound belief that influenza in hospital is a dangerous infective disease.

We have now reviewed the principal measures of prophylaxis which have been advocated and tested, and it seems that the simple non-mystical methods which come within the scope of personal hygiene are those which both epidemiological and physiological research sanction. It is permissible to hope that further study of the micro-biological problems presented by epidemic influenza will enable us to establish vaccine prophylaxis upon a securer foundation than it yet possesses; the efforts of both the Ministry of Health and of the Medical Research Council are continuously directed to that end; but the public should not suppose either that nothing of service can be attempted until the authentic *materies morbi* of influenza has been unequivocally defined or that the recognition and successful isolation of the authentic microbe would necessarily be followed by elimination of the influenza scourge. The history of all epidemic diseases is a witness to the truth that much may be done when the essential factor of a disease is undefined, and, *per contra*, that complete certainty as to biological ætiology is compatible with imperfect control of a disease. Brilliant as have been the improvements effected by the introduction of vaccinal prophylaxis for typhoid, the successes achieved by sanitarians in the 19th century, before the bacteriological era, were even greater. It is not true that without bacteriological certainty nothing important can be achieved in the direction of rational prophylaxis.

CHAPTER X.

A General Discussion of the Epidemiology of Influenza.

In previous chapters we have described the observations upon the late epidemics made by ourselves or communicated to us by other British epidemiologists and the clinical and bacteriological facts have been recounted by experts. It remains to redeem the promise of our preliminary chapter, to indicate the modifications of general epidemiological theory relative to influenzas which are imposed by the events of the last three years. This chapter, then, will be devoted to a general study of the events, and is intended not to expound any "official" theory, but to suggest to other epidemiologists trains of thought, the pursuit of which may lead to the attainment of important truths and will at least tend to a clarification of ideas. It is the merest sciolism to imagine that the necessarily partial and incomplete investigations of a few months or even years can authorise the promulgation of a symmetrical and comprehensive theory of such events as we have witnessed. The problem of influenza is still unsolved; its solution will be one of the great events in the history of medicine.

Notwithstanding the prominence accorded to the epidemics both in the newspapers and in common talk, it may be doubted whether the general public or even the medical profession have realised that the epidemics of 1918–19 rank in respect not only of absolute but even of relative mortality not lower than third and perhaps second upon the roll of great pestilences. No epidemic of smallpox or cholera, not even the typhus periods of the earlier years of the 19th century, can vie with the influenza of 1918–19 as agents of destruction. Some of them, indeed, were more deadly within a narrow circle—the famine typhus of Ireland is an example—and in the worst years of the new cycle of Indian plague some parts of that Empire suffered more than in 1918–19. But the gross mortality due to influenza in the autumn of 1918 far exceeded that of any year or of any three or four years in the recent annals of the bubonic plague. There are in fact only two loimological events which can be brought into comparison with the late pandemic, the plague of Justinian's reign and the 14th century Black Death.

Of the pestilence in Justinian's time, little can be said. "In "time, its first malignity was abated and dispersed; the disease "alternately languished and revived; but it was not till the "end of a calamitous period of 52 years that mankind recovered "their health or the air resumed its pure and salubrious quality. "No facts have been preserved to sustain an account, or even a "conjecture, of the numbers that perished in this extraordinary

" mortality. I only find that during three months 5,000, and " at length, 10,000 persons died each day at Constantinople ; " that many cities of the East were left vacant, and that in " several districts of Italy the harvest and the vintage withered " on the ground. The triple scourge of war, pestilence, and " famine afflicted the subjects of Justinian, and his reign is " disgraced by a visible decrease of the human species, which " has never been repaired in some of the fairest countries of " the globe."*

Our knowledge of the 14th century pandemic is a little more exact, and, for reasons which will appear in the sequel, we shall devote some paragraphs to the study of this pestilence which may fairly claim the first place among the " captains of the men of death."

The European position in 1347 with respect to plague differed from that relating to influenza in 1918 in one important particular. While, as we have seen, *an* influenza had been domiciled for some time before 1918, there is no certain evidence of localised plague and no evidence at all of pandemic plague in Europe between 1348 and the century following the pandemic of Justinian's reign. It is, however, known that for generations before 1348 plague had been endemic both in China and in foci to the north of Hindustan ; it is equally sure that commercial intercourse between Europe and the East, carried on by the overland route, had been constant and frequent for at least a century ; indeed there was more intercourse between the two civilisations than obtained during the two succeeding centuries, when, for a variety of reasons, the overland trade was suspended. The subsequent identification of China with Cathay and of Peking with Marco Polo's Cambulec was a product of maritime explorations in the Elizabethan era. We have, therefore, to learn why plague was only effectively imported into Europe so late, and the natural explanation would be that towards the middle of the 14th century the plague in China became especially infective, and as a token of this we should expect to find the years immediately before 1348 thick set with records of pestilence in the Chinese annals.

These annals have been studied by several epidemiologists, with a result entirely contrary to the expectation. The events which secure pride of place in the annals of the half-century before 1348 are not pestilences, but famines and earthquakes. It is not until 1352, *i.e.*, after the great European death, that plagues become conspicuous in the Chinese records. As Creighton observes : " Every year from 1352 to 1363, except " 1355, has an entry of ' great pestilence ' or ' great plague ' " (yi-li), in one province or another, although the old tale of

* Gibbon, *Decline and Fall of the Roman Empire*, Chapter XLIII. (Gibbon derived his medical knowledge from Mead ; the duration in time of the plague was probably much greater than 52 years).

" floods and famines has come to an end in the annals. The " last of the nearly continuous series of great pestilences is in " 1369, when there was a great pest in Fukien, and 'the dead " lay in heaps on the ground.' "*

It is, therefore, not true that before the Great Death epidemic plague was exceptionally virulent in its original home. Another point to mark is that the Great Death at its inception was an epidemiological type of plague which has never since appeared in epidemic or pandemic form upon European soil. The plague experience of London in later times and of much of Western Europe towards the end of the 17th century was strictly *in pari materia* with that of modern India, a typical bubonic plague. Of the original form of the Black Death and of its subsequent change of type we have the witness of Gui de Chauliac, the surgeon of Pope Clement VI. at Avignon, where the plague began in January 1348. "It was," he says, "of two kinds; the first lasted two months, with con- " stant fever and blood-spitting, and of this people died in " three days. The second lasted for the rest of the time with " continuous fever, buboes, and carbuncles (*apostematibus et* " *anthracibus*) in the outward parts, especially the armpits and " groins. These died within five days. Such was the con- " tagiousness, especially when there was a spitting of blood, " that not only by remaining with but merely by gazing upon " the sick, one took the disease, so that they died without " attendants and were buried without priests. A father would " not visit his son nor a son his father; charity was dead and " hope prostrate."†

Even if we could adopt a sceptical attitude towards the statement that the clinical form was a *primary* plague pneumonia, we must hesitate to suppose that infection was conveyed only indirectly as in modern instances. How special the modification of biological characters in the *bacillus pestis* must be to agree with the epidemiological facts need not be enlarged upon. Most epidemiologists, especially perhaps Sticker, have recognised that the formula, rat-flea-man, does not describe the primary pandemic of 1348.

We have then to account for:—(1) The importation of the disease at a time not *prima facie* favourable to importation, certainly no more favourable than any year for a century before; (2) A very special clinical and epidemiological type of disease after importation. Let us turn to the immediate circumstances attending its introduction. These are furnished in the contemporary narrative of Gabriel de Mussi, to whose testimony

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* *History of Epidemics in Britain*, vol. I., p. 153. It is of some interest to note that in 1888 and 1889 serious floods occurred in China, and large numbers of persons were rendered homeless. (*Influenza*, E. Symes Thompson, London, 1890, p. 415.)

† Transcripts of all the important original descriptions of the Black Death are printed by Haeser, *Geschichte der Medicin*, Bd. III., pp. 157–182.

Haeser, Creighton, and Gasquet all attach importance. De Mussi traces the direct origin of the European plague to a company of Italian traders who, with their merchandise, had taken refuge from predatory hordes of "Tartars" in the Crimean town of Caffa. In this town the merchants stood a siege of, he says, three years. They were, it seems, grossly overcrowded, but not famished. (Ibique hostium exercitu infinito vallati, vix poterant respirare, licet navigio alimenta ferrente, illud talle (sic) subsidium intrinsecis spem modicam exhyberet.) Eventually a plague broke out amongst the besiegers, from whom it was communicated (it is said by hurling corpses into the town) to the townsfolk. So great was the mortality that the siege was raised. The clinical form of disease amongst the besiegers was *bubonic* plague—the expressive words are :-"" signati corporibus in juncturis, humore coagulato in inguinibus, febre putrida subsequente," and the surviving merchants took ship to Europe. Wherever survivors landed, first at Genoa, subsequently at Venice and Marseilles, the plague broke out, spreading under the forms and with the mortality which are historic.*

The epidemiological importance of this train of events is considerable. Intercalated between the plague in its primordial home and the western pandemic is a period of maturation within a besieged city; from the fugitives of that city the subsequent extension of the plague through Europe can be can be followed unequivocally. It is difficult to resist the conclusion that the specialised exaltation of virulence sustained by the *materies morbi* was a consequence of frequent passage from host to host within the beleaguered town. Of significance, too, is the fact that those in contact with the group of persons who, by hypothesis, provided the field of variation and development, experienced the sickness at least as severely as—perhaps more severely than—the original hosts. The "Tartars" were ravaged and the mortality in Europe was prodigious.

It will naturally be objected that inferences from the handful of statements not characterised by obvious rhetorical exaggeration, which are our sole criterion of the 14th century plague, are dangerous, and that we should at least bring into comparison some modern epidemic of like nature. We cannot, of course, cite any epidemic on the same scale, but in the winter of 1910–11, Manchuria and China experienced an outbreak of primary pneumonic plague resembling in its broad features the great death of the 14th century, although its dispersive power was much smaller.

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* Gabriele de Mussi was a notary, of Piacenza, and died in 1356; he was not present at Caffa during the events he described. Caffa, sometimes known as S. Feodosia, was an important trading centre of the Genoese and the cathedral city of a diocese. At the time of the plague it was the centre of almost all commerce between Asia and Europe—vid. Canale, *Della Crimea, del suo commercio e dei suoi dominatori*, I., p. 208, cited after Gasquet, *The Black Death*, 2nd edition, London: 1908, p. 5.

It is full of epidemiological interest to notice that, although experts differed as to whether the original source were the conveyance from a rodent, the "Tarbagan," to man of a primarily bubonic infection subsequently modified by passage, or whether the primary source were naturally occurring sporadic pneumonic plague, there is no doubt that one stage of the epidemiological evolution was completed in a temporarily overcrowded town. The epidemic was traced to Manchouli, an Asiatic town, the normal population of which was 500 Russians and 2,000 Chinese, but, during the Tarbagan season, the Chinese increased to 10,000, and "they crowd into very poor hovels or inns, where, with "piles of raw pelts, they may often be found living, sleeping, "and eating, from 20 to 40 in the smallest of badly-ventilated "rooms."* Dr. Petrie adds the significant remark: "Curiously "enough, it is stated that the hunters are free from the disease "during their sojourn in the open country, and that it is only "when they congregate in the overcrowded caravansaries of "Manchouli and Hailar that they are subject to attack from "Plague."

In other words, a period of intensive overcrowding separated the endemic from the pandemic stage in Manchuria within our own memory, precisely as occurred 570 years ago in the Crimea. We do not, of course, press this analogy far. We have no evidence that the overcrowding in the hunters' settlement was more intense in 1910 than in any of the previous years which did not witness a dispersive plague. Yet, as we think, the parallelism is illuminating.

The inferences to be drawn from this history are that the infective power of a *materies morbi* may be specialised and exalted if it be given an opportunity of repeated passage through a considerable number of hosts, and that when the newly-acquired property has become temporarily stable, dissemination of the organism will lead to a pandemic, and that for the realisation of a pandemic it is not essential that the conditions initially needed to produce the modification shall be present.

We have now to consider whether the facts elicited respecting a disease essentially unlike influenza in one respect, viz., the specific determination of its *materies morbi* and its usual clinical evolution, similar to it on this present occasion by virtue of its extreme diffusibility and original predilection for causing death by an apparently primary pneumonia, throw any light upon the recent pandemics.

We must first of all contrast as sharply as we can the epidemiological findings of 1918–19 with those of earlier pandemics. The first wave, that of, in England and Wales, the summer of 1918, both with regard to the form of the case curve plotted against time as an abscissa, and with regard to the fatality rate, differed little, if at all, from the experience of

* Report of the International Plague Conference held at Mukden, April 1911, printed at Manila, 1912, p. 413.

1889–90. True it occurred not in winter but summer, but the summer influenza of 1782 is a sufficiently recent precedent on a large scale to admit of our judging season to be a non-essential. On the other hand, the age incidence of the disease was different —we think significantly different—from that of previous local epidemics or of the pandemics of earlier years, such as 1889–1893 or of 1907–8. In this primary manifestation we detect a shade of difference. It already appears that the *materies morbi* had suffered a change, making it apt to develop within the bodies of the young adults, not yet, however, prone to destroy them.

The autumn wave accentuates the distinctions and minimises the agreements between past and present. As points of agreement, we still have a reasonable concordance between the form of the wave and that of earlier secondaries, but even here it is to be observed that the interval was shorter between the sucessive phases than on any previous occasion. The distinctions are a continuance of heavy incidence upon the young adults and an exacerbation of the rate of mortality. The proportional death rate at ages 20–40 continued to rise; hence, as the incidence rate upon these ages was relatively slighter than in the summer, the fatality rate must have increased. The third wave, although it exhibited some tendency to revert toward the earlier type—that of the 90's—was more like the secondary than any predecessor.

It follows, then, that we have no complete parallel with the Black Death; the train of events is a longer one. If we bring the two later waves of influenza into comparison with the European history of the Great Death, there is a similarity. The first of these secondary waves corresponds to the initial form of the plague, its first few months of severity. The second resembles the subsequent manifestations, which, although greatly more fatal than later epidemics (save a few special instances, such as the London plague of 1665), conformed more nearly to the natural type of epidemic bubonic plague, as the last wave of influenza differed less from the classical form (although absolutely it differed much) than its predecessor.

If, then, there be any ætiological similarity, we must discover in the circumstances immediately prior to and during the first wave of influenza something akin to what took place within the walls of Caffa in 1346–47.

But this is no difficult task. The circumstances of the ancient Crimean town were reproduced in countless foci throughout the world. Whether it be larger or smaller aggregations of troops in the theatres of war, training units in the belligerent states, aggregations of operatives within the factories of both belligerents and neutrals, temporary or long-continued congestions of humanity due to dislocation of transport or to altered distribution of population, all these were occurring, not in one state but universally.

But there remains a broad distinction between the two pandemics, inasmuch as throughout the whole of western Europe, in the early summer of 1918, influenza was epidemic, with a modified age incidence, but retaining its former low fatality. The distribution seems not to have been so widespread in the new world, but, over and above such camp outbreaks as that of Funston, it is plain that in various States of the North American Union a mild influenza was epidemic in the spring of 1918. It might therefore seem otiose to seek in the disseminated foci of human concentration the source of that exaltation of virulence which characterised the second and really calamitous outbreak rather than in the general sanitary and economic circumstances of the populations of the different centres. We think that an explanation of the sort last indicated is impossible, for the following reasons: There is no evidence in the general vital statistics collected by Dr. Low and discussed elsewhere in this report that the mortality caused by the second influenzal wave was proportionally higher in those countries or populations especially affected by war privations. We have called attention to the case of Ireland, and we would note that of New Zealand. In New Zealand the havoc wrought by influenza much exceeded that experienced in the crowded cities of Great Britain. We have also pointed out in an earlier chapter that the correlation of the influenzal death rates in the first wave with the pre-war standardised death rates of the great towns in England was substantial, but very much lower in the second wave. The interpretation of these statistics is not, indeed, simple. There is some reason to think that the distribution of mortality rates in the second wave was influenced by the acquirement of a partial immunity, but a broad view of the whole of the facts inclines us to suppose that the intensification of virulence must be referred, not to the general conditions of the population so far as these pertain to domestic housing or nutrition, but to the continued operation of some special factors.

If we suppose that the first modification of the *materies morbi* which called into being the epidemiological type of the first wave, differing from previous influenzas in its selective affinity for young lives, but not highly virulent, was indeed the widely scattered distribution of centres of acute demographic congestion, in camps, factories, &c.; then, as these conditions were not relieved but intensified as the war approached its crisis, it is natural to think that a further modification of the virus which had already become adapted to multiply in the tissues of that section of the population which had previously exhibited great resistance, a modification importing not merely high infectivity but extreme toxicity was generated by the conditions still acting. This hypothesis will account for the almost incredible fatality of the second influenza, not only in transports but also in some civilian communities, such as New Zealand, into which it was directly introduced from crowded

ships, *i.e.*, from the very milieu which we suppose to be a prime factor of the imposed modifications both of infectivity and toxicity. We shall also understand why there is little evidence that *domestic* conditions of overcrowding affected the distribution of the disease within an infected town.

Our general theory of the epidemiology of influenza may now be enunciated.

It will appear that the *materies morbi* of the disease is and has long been wide spread but that neither in dispersiveness nor toxicity was it often able to maintain itself upon a high level of efficiency. At irregular intervals some strain or strains of the organism acquired a high power of diffusibility. Since, at least in English experience prior to 1889, the development of an influenzal wave was preceded by peculiar changes in the public health, in particular a prevalence, sometimes for more than a year of anomalous fevers and nervous illnesses, we can suppose either that the *materies morbi* was undergoing a series of modifications, of which these strange illnesses were the clinical manifestations, or that the acquirement of effective dispersiveness was not so much a consequence of bacterial modification as of change in the properties of the human host. The former explanation, in view of our general epidemiological history and the special researches into morbid periodicity which we owe to Brownlee, is the more probable.

Towards the close of the 19th century some general change took effect, either the *materies morbi* was able to retain, whether generally or locally a higher grade of diffusiveness than it had formally possessed save at long intervals, or the demographical conditions which lead to the evolution of that property were stabilised, with a consequence that, subsequently to 1889, epidemics or pandemics have recurred at much shorter intervals and have been characterised by a higher virulence than before. It is probable that both factors have concurred. The comparatively abrupt change in the year 1889 pointed to a biological modification or sport, the subsequent events to the continued operation of the cause group, perhaps decentralised housing and the expansion of means of communication, of a demographic order. The causative organism having now acquired a relatively high and stable power of diffusibility due to the co-operation of factors mentioned, there remained the possibility of a further modification in the direction of increased virulence. The historical facts of the Great Plague of 1347–50 suggest that a very remarkable intensification of virulence may be effected if *materies morbi* is afforded frequent opportunities of transfer amongst a number of hosts confined within narrow limits but absolutely numerous enough to afford considerable variations of soil. In effect, the virulent and diffusive infection which destroyed half the inhabitants of Europe in the middle years of the 14th century seems to have passed through a Crimean ante-chamber wherein it developed the special properties

which characterised it during the subsequent pandemic and differentiated it from either earlier (in the east) or later (both in east and west) manifestations of epidemic plague. It appears that the part played by the Crimean focus in that pandemic has been played by many different foci throughout Europe and America during the year 1918, that to these we must attribute the temporary fixation by the organism or organisms of influenza of its second and deadly property.

The story then of the germ of influenza is divided into three phases. The first which lasted for many centuries was, if we may be permitted to use teleological language, a series of attempts to maintain a high level of infectivity or dispersiveness, which attempts were unsuccessful. The second phase ushered in by the year 1889 is marked by a partial victory of the germ, a fairly constant infective power has been secured and much infection is produced throughout the world at frequent intervals, but the toxicity relatively to the infectivity is still slight. The final phase is of complete victory, infective power is maintained, even enhanced, and to this is added a toxicity surpassed by few epidemiological competitors*. Viewed as a contest between man and "germ," it would seem that in the congestion of public transport and the multiplication of public assemblies and entertainments, features which increasingly characterise the development of the European type of civilisation, a strategical advantage was given to the enemy. Finally in the provision of countless incubators, whether in garrisons, war-time factories or abnormally overcrowded and ill-ventilated means of transport and places of entertainment, the opportunity was afforded for the development of destructive powers which secured to the enemy a decisive and overwhelming victory.

Such is the general theory of epidemiology which we are led to adopt as a working hypothesis. We must distinctly and categorically assert that it is *only* a working hypothesis and one which further research may altogether displace. Confronted with the usual explanation, viz., the random occurrence of an extreme variation, a sport of the *materies morbi* in an unidentified nidus, it does, we think, offer advantages. The latter explanation does not fit in the other parts of the puzzle; it leaves as merely fortuitous concurrences, so far as influenza is concerned, both the general demographic changes of the past 40 years and the special alterations of human life which have been imposed by the war; in a word it regards the pandemic as "an act of God." Our hypothesis does, at least, assign to works of man an intelligible role in the evolution of the drama. Man first by facilitating the intercourse and close temporary aggregation of human beings with various biological

* If the Middlesbrough epidemic (*supra*, p. 17, footnote) be regarded as an "influenza," considerable toxicity and slight dispersive power have been combined in particular outbreaks before 1890.

aptitudes, has afforded opportunities of bacterial modification and selection, has done so with steady increasing persistence for many years. Since the war he has involuntarily experimented in intensive cultivation ; both these actions have, in our view, been essential links of the chain of causality. But that what we have postulated is no more than a working hypothesis is sufficiently demonstrated by the fact that the very events in the history of the Black Death which fit so aptly into our scheme were used by Creighton skilfully and successfully to interpret the Black Death in the sense of Pariset's hypothesis that plague is a soil disease due to putrefactive changes in the earth dependent upon the presence of corpses and engendered by the soil disturbances of the east following a succession of great floods and earthquakes. No reader of Creighton's chapter on this subject unacquainted with the subsequent history of epidemiological and bacterial investigations into plague could deny that the hypothesis was an excellent working hypothesis, describing adequately the facts then known. Equally no reader familiar with subsequent events can doubt that the hypothesis is entirely false. Such may be the fate of our own speculation. Yet it is false science to neglect the teaching of a working hypothesis, the practical inferences deducible from it, because it may ultimately have to be rejected. It has often happened in the history of the human mind that a hypothesis subsequently discarded has prompted inferences both true and valuable, as happened in the instance of the corpuscular theory of light or even the phlogistic theory of heat. Not all the inferences of Galen and Aristotle from faulty physiological premises have perished with the hypothesis originally authorising them.

The first inference from our hypothesis is that in the seeming conflict between man and his microscopic competitors, there can never be a time when man is securely master of the universe. Intoxicated by the victories achieved over the plague (in Europe) over the enteric group, over typhus (in western Europe) and over small-pox, we are too apt to suppose that the campaign has ended in our favour, that we have little more to fear from the typically epidemic diseases and may concentrate against the endemic group. That we have just passed through one of the great sicknesses of history, a plague which within a few months has destroyed more lives than were directly sacrificed in four years of a destructive war, is an experience which should dispel any easy optimism of the kind. No instructed epidemiologist can say that the world may not have to endure during the next half century other plagues of the first order of severity.

The second inference is the essential solidarity of all mankind in the matter of epidemic sickness. In a narrow sense, this solidarity has been realised since the beginnings of western civilisation. The conception of a sanitary cordon, the barring out or sealing up of an infected territory is, indeed, an old

notion. But our hypothesis extends this conception greatly; and enables us to see that the sanitary cordon is but a very small part indeed of a supra-national system of preventive medicine. The dangers to the world from epidemic sickness in this matter of influenza are enhanced in two ways. The inevitable trend of the movement of population will keep the infectivity of the organism at a high level. This we may face with equanimity. But if anywhere in the world there be large collections of men, whether through war or economic strife, or through that dissolution of civil society, which a certain degree of collective misery and disorganisation entails, herded together *en masse*, there will be opportunities for the other modification of the *materies morbi* which renders it apt to conquer the world. No sanitary cordon, no quarantine, will shield us from this danger. The porters of the infection may not be sick; to exclude even the sick has often been found a task beyond the powers of a quarantine authority; land quarantine has, in fact, never yet succeeded. To realise that the material well-being of the inhabitants of a foreign—perhaps even a hostile—country is a pressing concern of ours is very hard. Yet the teaching of this pandemic is that it is a hard truth. Any supra-national organisation for the control of epidemics will need to face it. The popular belief that misery breeds disease is strictly true, and the influenza of 1918–19 is no exception to the rule The history of the world has never yet provided an instance of a mortal and highly-dispersive illness among the antecedents of which human misery did not assume a prominent place. Not necessarily, indeed, a universal misery; the town of Caffa, in 1347, was not typical of mediæval Europe, nor was Ireland of the potato famine, nor were the industrial towns of Lancashire at the end of the 18th century generally characteristic of their time. Yet the absolute extension of misery has always been great before a plague, and has assumed a form different from that afflicting the populations in settled times.

If our general diagnosis be correct, what is the world's outlook upon future pestilences or dangers of pestilence? It is, we think, gloomy. The conclusion to which we are led is that the generation of a great pestilence such as influenza or pneumonic plague is dependent upon disturbance of social order involving for absolutely large numbers of human beings the endurance of conditions of insalubrity which afford for invading parasites a suitable field of modification. So soon as the new properties have been stabilised no barrier against the pandemic or epidemic extension will avail, nor will those individuals or nations who have not suffered the primary evils be more resistant to the disease than their fellows.

No impartial spectator can doubt that at the present time, and almost certainly for a generation to come, there will exist in many nations and over wide tracts of country precisely the type of misery which we suspect to be the

appropriate forcing house of a virulent and dispersive germ. In ancient times, at least after the breakdown of the primary civilisations, when there was nearly as much misery in the world as now, the non-centralised character and relative insignificance of manufacturing industries hindered the development of urban aggregations upon a large absolute scale (relatively, in terms of inhabitants per house or per room, mediæval cities were probably more overcrowded than most modern towns). At present the poor and miserable must herd together, and will not die quietly in their hovels. These are evils the removal of which is not within the province of epidemiologists to discuss. We can but note them and remark that no technical device of the sanitarian, no resource of the laboratory, can have any effect in the reduction of death and sickness from epidemic or even endemic disease at all commensurate with the consequences which must follow a *universal* improvement of the standard and conditions of life.

Admitting, then, as highly probable that destructive epidemics or pandemics of respiratory disease will recur during the present generation, it remains to inquire what conjectures we may properly make as to their time of occurrence and the form likely to be taken, and then finally to consider what palliatives are available.

Epidemiologists owe a considerable debt of gratitude to Dr. John Brownlee, Statistician to the Medical Research Committee, for his introduction to epidemiological research of the method of harmonic analysis. When a long series of data is available it is possible by arithmetical methods to determine (*a*) whether the phenomenon is periodic, recurrent, and (*b*), if periodic, what is the probable interval between successive maxima or minima. Hence, if the conditions as to data be realised, it is possible to determine beforehand when an assigned epidemic disease may be expected to prevail with a more than average intensity. Some who have not sufficiently attended to the matter have objected that the conception is fatalist, that it amounts to postulating of epidemic diseases an inevitableness which deprives sanitary administration of any hope or basis of success. The very reverse of this is the proper inference. What periodic analysis suggests is that the *materies morbi*, like other forms of life, pass through cycles of change, rendering them more or less apt to succeed in the struggle for successful existence which is the law of all life. In the most favourable case, we shall be able to predict, not that a great and devastating epidemic will occur at an assigned time, but that then the conditions will be most favourable for the generation of an epidemic. This being known, we can so act, not that there will be no epidemic, but that its ravages may be mitigated, perhaps altogether checked. If, for instance, we could suppose that the conditions favourable to the eruption of an influenza would be exceptional, say, during the month of February 1921, we should take pains to

limit the occasions of exultation of virulence upon which we have dwelt above; we should also bring into operation the other palliatives of which we have yet to speak.

In an appendix to this report we have reproduced Dr. Brownlee's contribution to the subject, and we have elsewhere indicated the material limitations to which it is necessarily subject. We may say generally that the principal practical weakness of the method in its application to the study of influenza is that the *materies morbi* of the disease is evidently subject to rapid evolution of type, and that we have no sufficient grounds for supposing that the characters acquired have now become so stable that deductions from past records will afford a secure basis of prediction of future events. Hence, although we consider that the epochs of emergence predicted by the method should be looked upon as times worthy of special administrative attention, we do not think the occurrence of devastating epidemics at times not predictable by the process can be deemed very unlikely.

We have next to consider the probable fatality and age of attack in future epidemics.

History teaches us that, both in interpandemic periods and in pandemics before the late events, influenza endangers the extremities of life. There is also evidence that in the late pandemic the final phase was indicating some reversion to what had been the normal type. No doubt we cannot put much weight upon this, since the season of the third wave in this country was a time of year normally fatal to the very young and very old; but it is permissible to think that manifestations of epidemic influenza in the near future are likely to revert still more towards the previously stable type of age incidence, and that toll will be taken of the very aged. We must not, however, omit to notice that if our general hypothesis be correct, and if the opportunities of modification are still provided lavishly throughout the world, we may again witness an intensification of the young adult and early middle age type of infection which has done so much mischief.

Thirdly, we must inquire whether any material limitation of invalidity or mortality is likely to follow the survival of a large number of persons attacked in the recent pandemic.

We have discussed this matter of naturally acquired immunity at such length in Chapter VI. that we can express our general conclusions briefly. It is, we think, probable that, on the average and in the majority of districts, a previous attack of influenza confers some protection upon those again exposed. But from the complete failure to discern any protection in particular districts and the large number of second attacks within a short interval in most districts, it seems probable that the prophylactic value of a previous attack depreciates rapidly and cannot be depended upon to create any national or communal protection against a second or third visitation.

Summarising our conclusions, we think that recurrences of influenza in epidemic or even pandemic form during the next few years are very likely to be seen; that they will exhibit a partial reversion to the age type known before 1918; and that naturally acquired immunity, although real, is inconstant.

Finally we shall discuss the palliatives which seem of most value. Amongst measures of public sanitation, we have remarked above that limitation of the occasions of casual crowding are probably the most important. Improvement of means of public conveyance, whether by additions to rolling stock or by acceleration of service, should at least mitigate the congestion of the suburban railways. It is a matter of common observation that there has been no improvement, rather a deterioration, in this respect since the armistice. We view the conditions under which outworkers are still conveyed to and from London and other large cities with anxiety.

The thronging of theatres and picture houses is, we think, a source of danger, although we are unable to say that this is a more serious danger than that the travelling public is forced to endure. It may perhaps be doubted whether exhibitions of moving pictures ought to form so large a fraction of the amusements of our urban populations; but the prohibition of such spectacles, even were it practicable, would leave so much casual overcrowding untouched, and the actual effect of closing picture houses and theatres upon the progress of an epidemic has been so doubtful, that we do not expect that much can be usefully done in this way. It is, however, reasonable to demand that a high standard of ventilation should be attained.

A national resort to prophylactic inoculation is a measure upon which we cannot yet express an opinion. It is plainly desirable that the medical profession and the public should have ample opportunities to test a method which, at the worst, can do no harm, and steps to that end have already been taken by the Ministry of Health. Until the resistance of the inoculated has been tested by exposure to the disease, judgment must be reserved.

The possibility of keeping a virulent infection at bay by imposition of quarantine has been considered. The Medical Director of the Quarantine Service of the Australian Commonwealth, Dr. J. H. L. Cumpston* has collected data which suggest that the application of strict quarantine to vessels bound from infected countries delayed the outbreak of influenza within the Commonwealth, the disease not appearing in epidemic form until January 1919 and causing proportionally and absolutely many fewer deaths than in New Zealand where similar rules were not enforced.

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* Influenza and Maritime Quarantine in Australia. Service Publication No. 18, Quarantine Service. Melbourne, 1919.

The recorded facts are certainly consistent with Dr. Cumpston's hypothesis, but in view of the great variations of mortality and appreciable variations of date of incidence in states which did not and could not apply the Australian system in its full rigour, it may be doubted whether any certain inferences should be drawn. So far as the passenger traffic of this country is concerned it may be regarded as probable that no methods of general quarantine would be of the least effect. In the event, however, of the arrival of a vessel upon which a severe outbreak had occurred and which harboured persons still gravely ill it is a matter for consideration whether a few day's isolation might not be enforced.

We have dealt elsewhere with the question of hospital treatment and think there is no doubt that a carefully arranged scheme of nursing and hospital accommodation would diminish the number of deaths attributable to an epidemic. This, however, in so far as it has not been dealt with elsewhere, is a question of administration the details of which must vary with local needs and does not, therefore, come within the scope of this general discussion.

Thus far we have considered prophylactic or palliative measures a responsibility for the application of which must chiefly rest upon the community or its executive officers. There remain the things which each individual can do for himself. We have no doubt that faithful attention to the petty details of personal hygiene, measures directed to securing hygienic habits of life, the avoidance of excess, in a word obedience to the plain rules of physiology are as important as any more specific measures we can devise.

With these remarks we may conclude our examination of the events of the great pandemic. We hope that the information collected in this report will be of service to the student both of medicine and of sociology and that the record of phenomena has not been obscured by the avowedly provisional interpretations which we have offered. Much still remains to be done and perhaps the next decade will provide the key of mysteries still locked securely against us. We hope that the mechanism of pandemic and epidemic sickness will be unveiled and that a far more precise description of the successive phases of its evolution will be rendered, such a description as is possible for but few diseases now, perhaps for malaria and bubonic plague alone. Yet we do not doubt that the basal conception, the notion inhering in the very word plague will remain unshaken. Not that a plague is the arbitrary stroke of some supernatural power, but that it is the inevitable reaction of human society to a disturbance of social hygiene and is, therefore, ultimately within our control, not through the utilisation of specifics but by an harmonious adjustment of living between the members of all the human family.

PART II.

Influenza in Foreign Countries, 1918–19.

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The Incidence of Epidemic Influenza during 1918–19 in Europe and in the Western Hemisphere.

BY

R. Bruce Low, C.B., M.D.

Introduction.

When it was first recognised that influenza was spreading over Europe in 1918, the source of the epidemic was attributed to Spain, and the disease was, on this account, termed "the Spanish Influenza." Although the presence of epidemic influenza in Spain was recognised in the early months of 1918, it is beyond doubt that at the same time outbreaks were occurring in other European countries, including France and Germany and in the Russian war zones. It cannot, therefore, be admitted that the European epidemic began in Spain. A recent American report contains the statement that: "It seems "probable that the earliest appearance of the 1918 influenza "pandemic was in Eastern Europe, and that by April it was "spreading in the war zones of Western Europe." It may be remembered that the 1889 epidemic was believed to have originated in China, and spread thence to Russia, where it was spoken of as the "Chinese" influenza. From Russia the disease spread westward into Europe, where it was named the "Russian" influenza. From Europe the infection was carried to North America by shipping, and there it was called the "European" influenza; and when it spread from North America across the Pacific to Japan it was known there as the "American" influenza.

As will be seen subsequently, there were a series of waves of epidemic influenza in Europe during 1918–19. The first of these, though attacking large numbers of persons in the spring and summer of 1918, caused comparatively few deaths; but the autumn wave was most serious, and gave rise to a high mortality, not only in Europe but also in America and other parts of the world. The third main wave of influenza in Europe occurred in the early part of 1919.

It has been suggested that one of the best ways of judging of the fatal ravages of epidemic influenza is to take the general mortality rate, especially when comparing the effects of the epidemic in different towns. In mild epidemics of influenza the information gained from mortality statistics is not of so much value as that obtained from the more severe outbreaks. Some statistics published in the weekly Bulletin of the City of Amsterdam give the death rates of a number of Continental towns—French, German, Austrian, Hungarian, and Swiss, showing the week in which, during the autumn

epidemic of 1918, the general mortality rate reached its highest point in each of these cities, and it is interesting to note that in places as widely apart as Paris, Berlin, Hamburg, Dresden, Breslau, Münich, and Bern the general death rate reached its maximum in the same week, namely, that ended on October 26th. The death rate in that week for Paris was 46·1 per 1,000, Berlin 41·7, Hamburg 47·7, Cologne 51·5, Leipzig 43·2, Dresden 42·2, Chemnitz 59·4, Stettin 50·7, Breslau 72·5, Nürnberg 60·8, Zurich 45·3, and Bern 34·8. It is curious that the influenza wave apparently struck Austria and Hungary most severely a week earlier, that is, if we reckon the height of the epidemic to be the time when the general mortality rate rose to its highest point. It was in the week ended October 19th (a week before Paris, Berlin, Zurich, and other cities showed their highest death rate) that Vienna, Prague, Trieste, and Budapest reached their maximum, Vienna having for that week a death rate of 58·6, Prague 94·4, Trieste 136·5, and Budapest 58·4. It will be observed further on that it was in the week ended October 26th, 1918, that the highest general mortality rate was also recorded in the city of New York, namely, 60·2 per 1,000. It is significant that the general death rate should show its highest point during the same week in such widely separated places as Paris, Berlin, Hamburg, Breslau, and New York. Unfortunately, it has not been possible to obtain the correct mortality rates for all continental cities, so that a complete comparison of the dates of the maximum general death rates during the influenza epidemic of the autumn of 1918 cannot be made in this report. These particulars will, no doubt, be forthcoming shortly, and in that case a comparison will then be possible.

For the purposes of this report comparatively little difficulty has been experienced in obtaining, from some countries, a certain amount of statistical and other information respecting the rise and fall of epidemic influenza within their borders; but in certain other countries, and particularly those in which during 1918 hostilities were still in progress in connection with the great European war, the details received were scanty and infrequent. This was particularly the case with Russia, Belgium, Austria, and Turkey, and perhaps, to a less degree, with Germany and the Balkan States. From the Western hemisphere there were also differences in the amount of information available from the various republics. While, on the one hand, from the United States ample details were forthcoming, from the South and Central American republics very little was reported respecting the course of their influenza epidemics.

To trace the early manifestations of the pandemic in various countries has been extremely difficult, and in some instances the search has been unsuccessful. As the first wave in 1918 was very mild in character, it was probably not recognised at all in some localities, and the temporary discomforts which

were experienced were attributed at the time to other causes. In some other places the existence of an influenza epidemic was not recognised until the malady had reached its maximum. As influenza has no pathognomonic initiatory sign by which it can be recognised, and as, up to the present, no specific microbe has been identified as being invariably present in all cases of the disease, it is not easy to make a correct diagnosis, especially before there is evidence of any epidemic. So that it has to be admitted that sometimes other illnesses are diagnosed as influenza, and, on the other hand, influenza may be pronounced to be some other malady altogether, and therefore in this way the beginnings of an epidemic may pass unnoticed.

In the following pages is given, as far as the available material has permitted, a historical and geographical account of the influenza pandemic of 1918–19, so far as it was observed in Europe and America, the incidence of epidemic influenza in Asia and Africa being dealt with separately by my colleague, Dr. James. The course of the pandemic in Great Britain and Ireland has been described in Part I. of this Report.

1.—Epidemic Influenza in Europe, 1918–19.

(Not including the British Islands.)

Sweden.

Influenza is included in the list of diseases the notification of which is obligatory in Sweden. In the year 1913, for example, 24,746 influenza cases were notified; 25,275 in 1914; 39,467 in 1915; and 29,414 in 1916.* The corresponding figures for 1917 have, unfortunately, not been obtained. (The population of Sweden is now about 6 millions.) It appears, therefore, that a disease regarded as "influenza" has been present for some years in Sweden, but whether the epidemic disease that swept over the country in 1918 was precisely the same malady as that which previously occurred, it is impossible to say. There were two separate and distinct prevalences of influenza in Sweden during 1918, the first, beginning towards the end of July, lasted about six weeks; while the second, commencing towards the latter part of September, that is, after an interval of about five weeks, lasted to the end of November, a period of some 12 weeks. As is well known, Sweden is a sparsely-populated country, except as regards some large towns, the chief of which are Stockholm the capital, with a population of 413,163, and Gothenburg (or Goteborg) with 196,943 inhabitants. The behaviour of influenza in these two cities was quite different both in incidence and fatality, for the fatality rate was considerably higher in Stockholm than in Gothenburg, while more attacks were recorded in the latter than in the former.

* 300 deaths were attributed to "grippe," or influenza, in Sweden during 1914.

For the 10 weeks from June 30th to September 7th, there were in Stockholm 1,316 notified influenza cases, with 96 deaths, giving a case mortality rate, 7·3 per cent.; in the same period, 73 cases of pneumonia were reported, with 50 deaths. In Gothenburg, during the same 10 weeks' period, there were 4,812 cases of influenza with only 29 deaths, giving a fatality rate of 0·6 per cent.; the deaths from pneumonia numbered 30 (the number of pneumonia attacks has not been ascertained). The figures for these two Swedish cities, in the first epidemic wave are shown in comparison in the appended table :—

1918. Week ended.	Stockholm. (Population, 413,163.)				Gothenburg. (Population, 196,943.)		
	Influenza.		Pneumonia.		Influenza.		Pneumonia.
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Deaths.
6th July - -	2	—	4	2	1	—	4
13th ,, - -	6	—	4	3	18	—	3
20th ,, - -	13	—	3	2	573	—	4
27th ,, - -	111	2	3	3	1,084	3	1
3rd August - -	358	17	12	11	1,120	3	3
10th ,, - -	411	38	20	12	815	10	4
17th ,, - -	248	23	12	6	517	5	2
24th ,, - -	102	12	9	4	344	5	1
31st ,, - -	42	2	4	4	185	2	4
7th September -	23	2	2	3	155	1	4
Total - -	1,316	96	73	50	4,812	29	30

From this table, it would seem that the incidence of influenza in Gothenburg during this first epidemic wave was greater than that in Stockholm. The facts as to the number of deaths from influenza and from pneumonia in each town considered separately would lead to an opposite conclusion; it appears likely that the notification of cases of influenza is carried out more completely in Gothenburg than in Stockholm.

The second wave of epidemic influenza in Sweden was of a more severe character, for during the 10 weeks beginning September 8th and ending November 16th, 3,379 cases were notified in Stockholm, of which 993, or 29·4 per cent. died. In Gothenburg, during the same period, 14,411 cases were reported, of which 605, or 4·2 per cent., died. More details of these waves of influenza appear to be necessary before a complete explanation can be offered for the apparent differences in the experiences of those two towns as regards the prevalence and fatality of influenza. In reply to a request for an explanation of the above, the medical officer of health for Gothenburg wrote to say that he put every case of pneumonia which was in any way connected with influenza under the heading of influenza; he suggested that in some other towns the same course was not followed, and to judge by their statistics pneumonias are not classed under the heading of influenza, but given as "septic pneumonia," or simply as "pneumonia."

The following table gives week by week the incidence and mortality from the epidemic in Stockholm and Gothenburg during the second wave of the influenza infection :—

1918. Week ended	Stockholm.				Gothenburg.		
	Influenza.		Pneumonia.		Influenza.		Pneumonia
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Deaths.
14th September -	34	3	5	4	181	—	2
21st „ -	79	8	14	8	464	7	1
28th „ -	153	30	16	11	1,181	26	4
5th October - -	285	53	21	12	2,196	47	1
12th „ - -	646	128	42	29	3,287	95	9
19th „ - -	746	178	70	18	2,555	163	23
26th „ - -	524	221	52	22	1,606	104	13
2nd November -	414	160	37	10	1,297	67	9
9th „ -	305	118	30	5	907	47	5
16th „ -	193	94	26	7	737	49	1
Total - -	3,379	993	313	126	14,411	605	68

There was no conspicuous rise in the incidence of influenza either in Stockholm or in Gothenburg during the remainder of 1918; but the notifications of influenza continued to be higher in the latter city than in the former. The following table gives the incidence of influenza and pneumonia in Stockholm and Gothenburg during the last six weeks of 1918 and the first four weeks of 1919, *i.e.*, for a further period of 10 weeks :—

Week ended	Stockholm.				Gothenburg.		
	Influenza.		Pneumonia.		Influenza.		Pneumonia.
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Deaths.
1918.							
23rd November -	149	71	8	6	752	33	
30th „ -	158	55	18	8	727	24	
7th December -	89	49	18	11	630	23	?
14th „ -	72	33	18	7	573	17	
21st „ -	84	53	13	13	430	19	
28th „ -	45	42	18	17	368	27	1
1919.							
4th January -	66	56	16	5	466	16	4
11th „ -	80	44	10	11	440	12	3
18th „ -	41	24	7	4	317	8	3
25th „ -	31	20	10	6	215	19	8
Total - -	815	447	136	88	4,918	198	19*

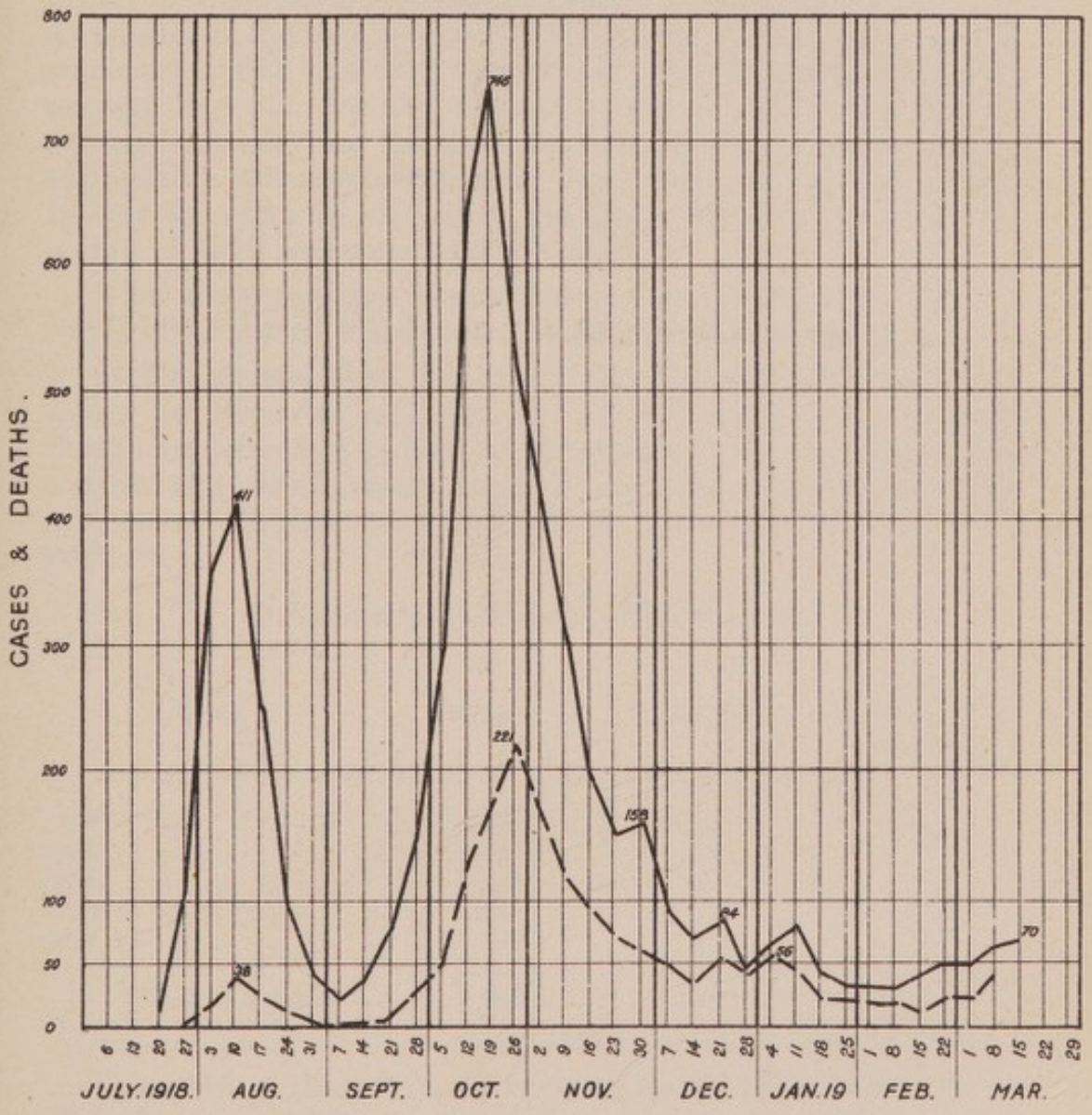
* Incomplete.

STOCKHOLM

INCIDENCE OF INFLUENZA.

——— CASES.

— — — DEATHS.



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In Stockholm the number of fatal influenza cases continued to fall until the end of February when a small increase again occurred, but this was in no way of a serious character; the general death rate remained comparatively low until the week ended 15th March, when the total deaths from all causes rose to 148, of which 38 were ascribed to influenza and only three to pneumonia, the general death rate for the week being 18·6 per 1,000, as compared with an average of 14·1 for the preceding six weeks. After April the death rate fell to normal. In Gothenburg the incidence of influenza continued to fall during February, and about the middle of March a fresh rise began in the number of reported cases. This increase reached its height in the two weeks ended 5th and 12th April in which respectively 642 and 518 cases were notified against a weekly average for 255 for the preceding nine weeks. Neither the general mortality nor the number of deaths from influenza were appreciably increased during the four weeks ended 12th April. It may therefore be said that the third influenza wave in the spring of 1919 was comparatively mild.

The accompanying chart shows the progress of the two waves of influenza which passed over Stockholm during 1918.

It has already been remarked that influenza has been prevalent in Sweden year by year before 1918. As regards Stockholm, concerning which figures are available, cases of the disease and deaths were reported in each of the five years previous to the recent epidemic, as shown in the appended table :—

Influenza in Stockholm, 1913 *to* 1917.

Year.	Cases.	Deaths.	Case Mortality Rate Per Cent.
1913 - - - -	531	14	2·6
1914 - - - -	1,031	61	5·9
1915 - - - -	468	30	6·4
1916 - - - -	848	18	2·1
1917 - - - -	389	14	3·6
Total in 5 years -	3,267	137	4·2

Comparing these figures with those for 1918 and up to 25th January 1919, namely, 5,510 attacks and 1,536 deaths, giving a fatality rate of over 24 per cent., it will be seen how much more serious was the recent epidemic in Stockholm, that is, if all cases were duly notified.

As regards influenza, elsewhere than in the two principal cities, it may be mentioned that the malady was exceptionally severe among the young conscripts called up for military service. According to a report by the Swedish Army Medical Department there had been amongst these conscripts up to the

end of September 34,000 cases and 444 deaths; nearly all of the fatal cases occurred among young and vigorous men between the ages of 20 and 30. This report led to an agitation, begun by medical men and taken up by the Socialistic party, to suspend the military training of the conscripts until the danger from the epidemic had passed. A demand was made that an extraordinary session of the Riksdag should be summoned to deal with the question, and threats were made that if this demand were not complied with a general strike against military service would be ordered. On 16th October an announcement was made in the press that the Swedish Government, in response to medical representations, had decided to postpone the military training of the conscripts until a future date, which would be fixed later on, when the danger from epidemic influenza had passed away.

Some aggravation of the suffering from the epidemic was caused in September by the fact that there was a serious scarcity of certain drugs required in the treatment of influenza patients, further supplies not being easily obtainable from abroad. When the epidemic was at its height in Stockholm there was a certain amount of panic owing to an assertion made by some irresponsible persons that the current malady was in reality pneumonic plague. Some people on this belief hastened to increase their life insurance. It was also suggested that the food difficulties, and especially the deficiency of fats in the dietary, had rendered the population more susceptible to the infection of influenza.

Influenza began to be epidemic in August in the seaport town of Malmö, the third largest town in Sweden, having a population of 112,700, situated on the Sound opposite Copenhagen. From August to the end of November 11,617 cases were notified, of which 395 or 3·4 per cent., were fatal.

Norway.

Influenza is a notifiable disease in the kingdom of Norway, as in Sweden; 16,671 cases were reported during 1913, 11,161 in 1914, and 22,930 in 1915.* In July 1918 an epidemic developed. In the month of June 526 cases and one death had been recorded in the urban districts of Norway; in July the number of cases rose suddenly to 38,604 with 203 deaths. There was a comparative lull in August, the number of reported cases and deaths falling to 6,251 and 37 respectively. A second epidemic wave began in September, when 15,468 cases and 256 deaths were reported, and in October the epidemic attained its highest point, 49,174 cases and 672 deaths being recorded. The month of November showed a marked

* During 1916, 14,650 cases were reported in the Norwegian urban districts and 6,606 in 1917.

decrease, there being 26,058 notified cases with 579 deaths. There was a further decline during December, when the cases fell to 9,389 and the deaths to 189. There was another rise in March 1919, after which the disease gradually subsided. The following table gives, for purposes of comparison, the number of influenza cases and deaths month by month for the four years 1916, 1917, 1918, and 1919 in the urban districts of Norway for which alone statistics are at present available.

Influenza in Norwegian Urban Districts in 1916, 1917, 1918, *and* 1919.

—	1916.		1917.		1918.		1919.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
January - -	3,168	12	1,734	10	573	1	7,138	143
February - -	5,477	25	1,713	11	549	2	3,797	63
March - -	2,769	14	920	9	396	—	8,015	237
April - -	774	5	457	3	309	1	3,423	81
May - - -	332	2	260	1	224	—	977	19
June - -	148	2	121	—	524	1	387	6
July - -	64	—	90	1	38,604	203	192	2
August - -	80	1	99	—	6,251	37	135	2
September -	154	—	144	1	15,458	256	305	—
October - -	239	—	241	—	49.174	672	375	—
November -	801	5	343	—	26,058	579		
December - -	644	4	484	—	9,389	189		
Total - -	14,650	70	6,606	36	147,509	1,941	24,744*	553*

* 10 months only.

Calculated on these figures the case mortality of influenza in the Norwegian towns was 0·5 per cent. in 1916, 0·5 in 1917, and 1·3 in 1918.

Generally, the influenza notified in previous years has been a disease of the winter months, beginning in November and reaching its maximum in the coldest period of the year, January and February; its lowest incidence occurs usually during the summer months of July and August. The outburst in July, and that of October, 1918, were, therefore, quite unusual, if the "influenza" of 1918 is regarded as the same as that which has been present in the Norwegian towns during previous years.

Of the 14,650 cases (70 deaths) reported in 1916 in the town districts of Norway, 1,644 (24) were referred to Christiania and 1,734 (13) to Bergen. Of the 6,606 cases (and 36 deaths) from influenza reported in Norwegian town districts during 1917, 950 (18) were referred to Christiania, 372 (4) to Bergen, and 712 (3) to Stavangar.

The course of an influenza epidemic can generally be followed in the mortality returns of pneumonia. In Norway pneumonia, like influenza, is notifiable. The following table gives the number of notified pneumonia cases and deaths

reported in the Norwegian towns during 1916, 1917, and 1918:—

Pneumonia in Norwegian Towns.

—	1916.		1917.		1918.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
January - -	483	68	527	94	290	55
February - -	481	81	453	87	228	29
March - -	426	58	351	58	232	45
April - - -	401	52	328	55	291	43
May - - -	434	59	453	55	389	70
June - - -	286	40	298	27	287	50
July - - -	271	18	181	29	537	80
August - -	127	18	101	15	187	31
September - -	207	26	149	12	655	91
October - -	281	41	208	31	2,843	470
November - -	303	52	244	28	1,246	234
December - -	334	52	317	35	741	104
Total -	4,034	565	3,610	526	7,926	1,302

There was, as shown above, an increase of pneumonia cases and deaths coincidently with the outbursts of influenza which began in July and September 1918.

Christiania, the capital and chief commercial town of Norway, has a population of 259,627; it is a port, and its chief staple industry is its shipping trade. Influenza has been present in it for some years, though not in large amount. The following table gives for each month of 1918 the number of cases and deaths certified in Christiania, as due respectively to influenza and pneumonia:—

1918.	Influenza.		Pneumonia.	
	Cases.	Deaths.	Cases.	Deaths.
January - - - -	78	—	32	21
February - - - -	91	1	37	16
March - - - -	65	—	45	24
April - - - - -	22	1	46	21
May - - - - -	33	—	73	37
June - - - - -	246	—	35	23
July - - - - -	15,024	129	135	37
August - - - -	347	16	15	13
September - - - -	1,064	40	75	10
October - - - -	6,605	248	200	32
November - - - -	2,973	242	122	16
December - - - -	1,048	88	44	20
Total - -	27,596	765	859	270

In a London newspaper of 1st November 1918, a statement was made that in the kingdom of Norway, up to the end of September 1918, about 100,000 persons had been attacked by influenza, and that 2,000 of them had died.

It has been said that the influenza epidemic really began in Christiania about the middle of June 1918. Through the courtesy of Dr. Bentzen, Medical Officer of Health for the city, the following figures are available as to the weekly incidence of the disease, beginning with the first week in July.

The following table shows the weekly incidence and mortality of influenza in Christiania during each week of the second half of 1918 :—

The Incidence of Influenza in Christiania Week by Week in the Second Half of 1918.

Week ended	Influenza.	
	Cases.	Deaths.
6th July	2,165	3
13th ,,	6,485	19
20th ,,	7,940	66
27th ,,	1,554	35
3rd August	326	11
10th ,,	117	7
17th ,,	26	2
24th ,,	31	3
31st ,,	32	3
7th September	67	—
14th ,,	151	9
21st ,,	319	10
28th ,,	354	23
5th October	603	16
12th ,,	1,132	24
19th ,,	1,497	33
26th ,,	2,006	92
2nd November	1,993	133
9th ,,	1,216	81
16th ,,	502	67
23rd ,,	426	37
30th ,,	255	20
7th December	309	22
14th ,,	213	27
21st ,,	196	17
28th ,,	192	14
Total	30,107	774

Dr. Bentzen's totals are rather higher than those reported in the previous table, but they are probably nearer the truth than the others.

Calculated on Dr. Bentzen's figures the case mortality of influenza in Christiania was 2·5 per cent. The first wave of influenza which began in mid-June reached its height in the 3rd week in July. August was comparatively free from the disease but in September the number of cases began to increase until they reached the maximum at the end of October and beginning of November. The incidence gradually "tailed off" up to the end of December. The autumn prevalence was attended with a higher proportion of fatal cases than the summer outbreak.

During January 1919, the deaths from influenza in Christiania continued to decrease, but about the middle of March there was a slight increase. Up to 1st March, the weekly average number of influenza deaths had been about seven for the eight preceding weeks, but for the week ended 8th March, the number rose to 26, and in the two following weeks the influenza deaths were respectively 46 and 59; there was a dimunition in the next two weeks, namely, 30 and 32, and for the week ended 12th April, there were 23 influenza deaths, after this the number greatly lessened, only 27 altogether being registered during the following 11 weeks. Thus the spring outbreak was of comparatively small dimensions.

The accompanying chart shows the progress of the two prevalences of influenza in Christiania during 1918.

Bergen is a seaport town in the west of Norway and is the second city in the kingdom, having a population of about 80,000. As in Christiania and elsewhere in the country, influenza in previous years has often been reported. In 1917, for example, 372 cases and 4 deaths from this cause were certified in Bergen. But nothing in any way comparing with the outburst of 1918 had previously been observed in recent years. Beginning in July with 4,003 cases and 27 deaths, there was a decrease in August, the cases numbering only 856 with 5 deaths; but in September the disease again increased, the notifications rising to 1,497 with 10 deaths. Thus, in the three months July, August, September, 6,356 cases and 42 deaths from influenza were recorded. The second wave of influenza reached its height in October when 8,300 cases and 148 deaths came under notice, with 3,074 and 98 respectively in November. Pneumonia is also notifiable, and in July 51 cases and 25 deaths were reported, the corresponding numbers for June being respectively 28 and 5. In August only 18 pneumonia cases were notified with a single death, but in September the cases rose to 61, of which 7 were fatal. The October pneumonia attacks numbered 637 with 46 deaths, and in November 176, 14 of which died. The following table gives

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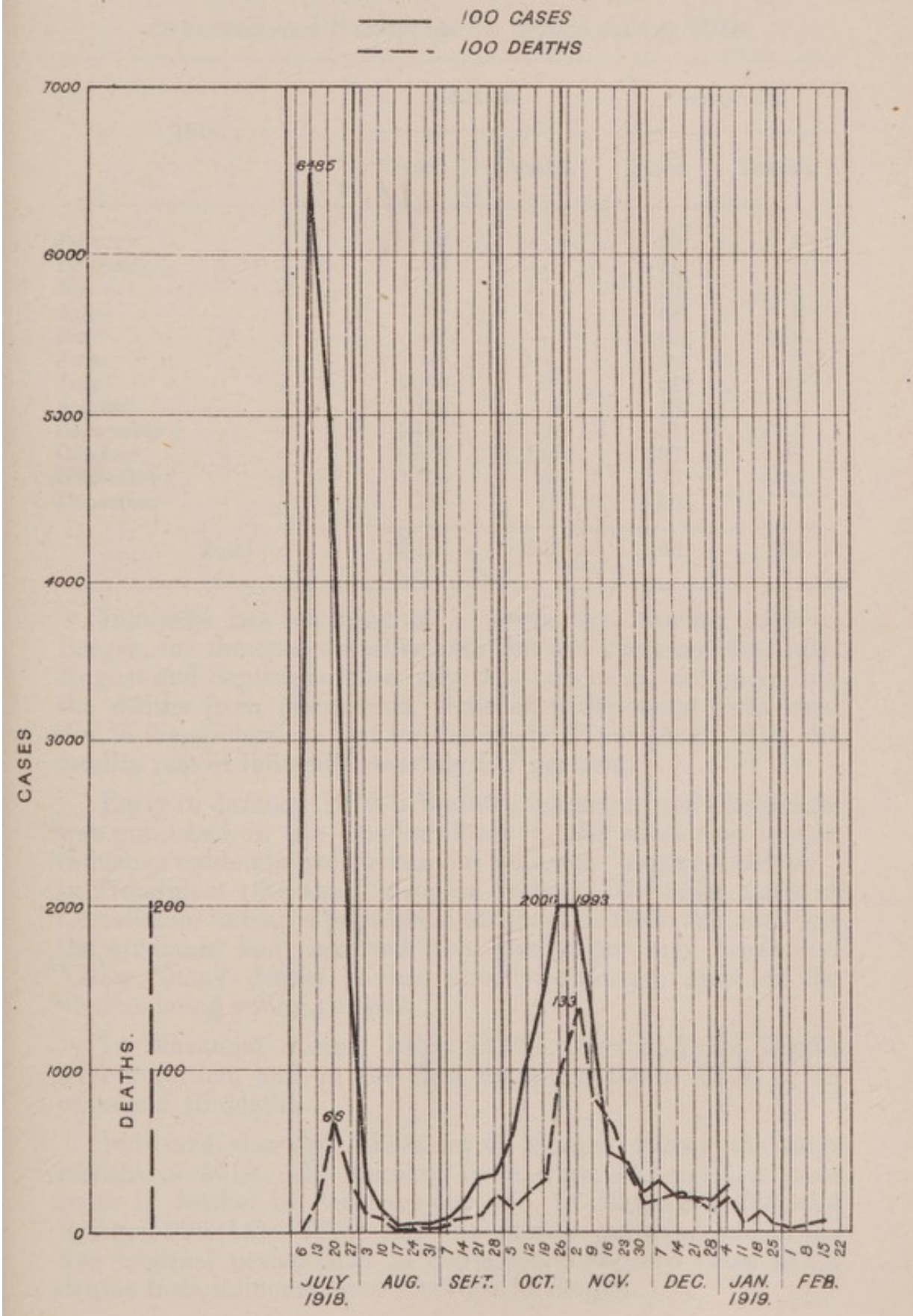
CHRISTIANIA

INCIDENCE OF INFLUENZA

WEEK ENDED 6 JULY 1918.

WEEK ENDED 1918.



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the number of cases notified and deaths from influenza and pneumonia in Bergen month by month during 1918:—

Influenza and Pneumonia in Bergen during 1918.

1918.	Influenza.		Pneumonia.	
	Cases.	Deaths.	Cases.	Deaths.
January	43	—	30	6
February	32	—	21	2
March	50	—	23	4
April	19	—	28	6
May	23	—	45	14
June	46	—	28	5
July	4,003	27	51	25
August	856	5	18	1
September	1,497	10	61	7
October	8,300	148	637	46
November	3,074	98	176	14
December	255	14	50	7
Total	18,198	302	1,168	137

Influenza has not been of a severe type during 1918 in Bergen, for the case-mortality rate for the three months, July, August and September, was only 0·6 per cent., not including the deaths from pneumonia, some of which must have been due to the epidemic. But for the whole 12 months of 1918, the fatality rate of influenza was only 1·7 per cent.

Early in January 1919, a Reuter's telegram from Christiania was published in the London Press to the effect that a fresh influenza epidemic had broken out and was "raging violently" in Trondhjem (the third town in Norway, 250 miles north of Christiania, having a population of about 50,000), and also that the epidemic had involved the Hardanger and Gudbrand Valley, many deaths having already occurred, most of the victims being young persons.

In Stavangar during 1918, 14,284 cases and 108 deaths were recorded, and in the first three months of 1919, 1,148 cases and 13 deaths.

Influenza was still prevalent in Bergen during the early months of 1919. In January there were 360 notified cases (with 17 deaths) in February 696 (20), in March 962 (39), and in April 296 (18). After this the disease subsided and in the five months' period May to September only 115 cases and 8 deaths from influenza were recorded in Bergen.

Denmark.

In Denmark, as in Sweden and Norway, influenza has been for some years on the list of diseases the notification of which is compulsory by law. The notified cases of influenza in Denmark year by year from 1906 to 1917 are given in the following table:—

Influenza Cases notified in Denmark 1906 *to* 1917.

Denmark.	1906.	1907.	1908.	1909.	1910.	1911.
Notifications of influenza.	11,281	51,390	65,359	32,592	29,438	25,665

Denmark.	1912.	1913	1914.	1915.	1916.	1917.
Notifications of influenza.	28,442	27,434	18,566	45,951	39,028	34,775

Unfortunately the mortality of influenza for the whole of Denmark for past years has not been obtained, only the figures for the towns being available. These are shown in the subjoined table for the 12 years period 1906 to 1917.

Deaths from Influenza in Danish Urban Districts 1906 *to* 1917.

Danish Towns.	1906.	1907.	1908.	1909.	1910.	1911.
Deaths reported from influenza.	58	244	299	146	149	99

Danish Towns	1912.	1913.	1914.	1915.	1916.	1917.
Deaths reported from influenza.	120	124	62	235	179	199

From 1894 to 1905 the yearly average number of influenza deaths was 220 in the Danish urban districts.

Some practitioners in Denmark recognise a "true" and a "false" influenza, the latter resembling the former in many respects. It is not possible now to say how much of the influenza reported in Denmark during past years was of the "true" or the "false" variety.

So far as can be ascertained the first wave of epidemic influenza in 1918 appeared in Denmark during July, and though there was a marked remission in September the disease was very prevalent during the whole of the second half of 1918, as may be seen in the appended table which gives the number of cases notified in 1918 month by month in the Kingdom of Denmark :—

The Incidence of Influenza in Denmark during 1918.

1918.	Influenza Cases.	1918.	Influenza Cases.
January - - -	2,081	July - - - -	38,021
February - - -	2,026	August - - -	30,754
March - - -	1,848	September - -	10,057
April - - -	1,452	October - - -	113,651
May - - - -	941	November - -	157,864
June - - -	626	December - - -	135,618

Total cases 1918 - - - 494,939

The total number of deaths registered from influenza during 1918 in Danish urban districts was 4,150.

The population of Denmark is estimated at 3,018,000.

From the above table it will be seen that there were two great waves of influenza in Denmark during 1918; the first began suddenly in July and was over by September, while the second, which was far more severe and widespread, began in October persisting to the end of the year, and well into 1919. In January 1919, 109,253 influenza cases were notified, 43,850 in February, 36,523 in March, and 23,048 in April. There was a decided fall in the number during May only 7,441 cases being notified. A further reduction occurred in the next three months the cases numbering 2,227 for June, 2,029 in July, and 2,214 in August.

It was expected that there would be considerable remission of the epidemic during December, but it appears from statements made in the Danish press that the influx of prisoners of war into Denmark from Germany served to renew the force of the infection. During January 1919 the disease was rife among French and Belgian prisoners of war quartered in Jutland.

In November 1918 influenza had spread to Iceland and the Faroe Islands. The course of the epidemic in Denmark can best be studied from the behaviour of the disease in Copenhagen the capital, with a population of 539,000.

Copenhagen.—The incidence curve of the influenza epidemic in Copenhagen followed closely that of Denmark. From the

weekly bulletin issued by the Copenhagen municipal sanitary department the weekly number of cases and deaths is available. The first influenza wave of 1918 in Copenhagen began in the second week of July, and may be regarded as having subsided by the end of August, though the usual "trail" of the disease was still observable in the first half of September. The following table gives the influenza cases and deaths, as well as those from pneumonia reported in Copenhagen during the 11 weeks period June 30th to September 14th :—

First Wave of the Influenza Epidemic of 1918 *in Copenhagen.*

1918.	Influenza.		Pneumonia.	
	Cases.	Deaths.	Cases.	Deaths.
Week ended 6th July - - -	68	—	20	4
,, ,, 13th ,, - - -	846	1	12	3
,, ,, 20th ,, - - -	3,868	2	32	3
,, ,, 27th ,, - - -	8,514	3	57	12
,, ,, 3rd August - -	6,141	5	52	6
,, ,, 10th ,, - -	2,961	4	24	5
,, ,, 17th ,, - -	1,491	1	17	2
,, ,, 24th ,, - -	863	—	14	—
,, ,, 31st ,, - -	395	—	10	2
,, ,, 7th September - -	213	—	8	2
,, ,, 14th ,, - -	273	—	7	1
11 weeks - -	25,633	16	253	40

From later information received from the medical officer of health of the city, it appears that the mortality from the epidemic was greater than is shown in the weekly returns as will be seen later.

The second wave began towards the end of September and reached its height in the second half of October, gradually decreasing up to the end of the year. In the 11 weeks period ended 30th November 1918, the notified cases of influenza in Copenhagen amounted to 60,952 with 107 deaths as compared with 25,633 attacks in the previous 11 weeks with only 16 deaths. The highest point in the curve of the first wave was reached in the week ended 27th July, the operation of the real active period being about five or six weeks. Between the highest points of the first and second waves there was an interval of about 12 weeks; the second wave was much more extensive and more fatal than the first. Comparing the fatality of influenza in Copenhagen with that of other European towns it is evident that the type of the disease was mild.

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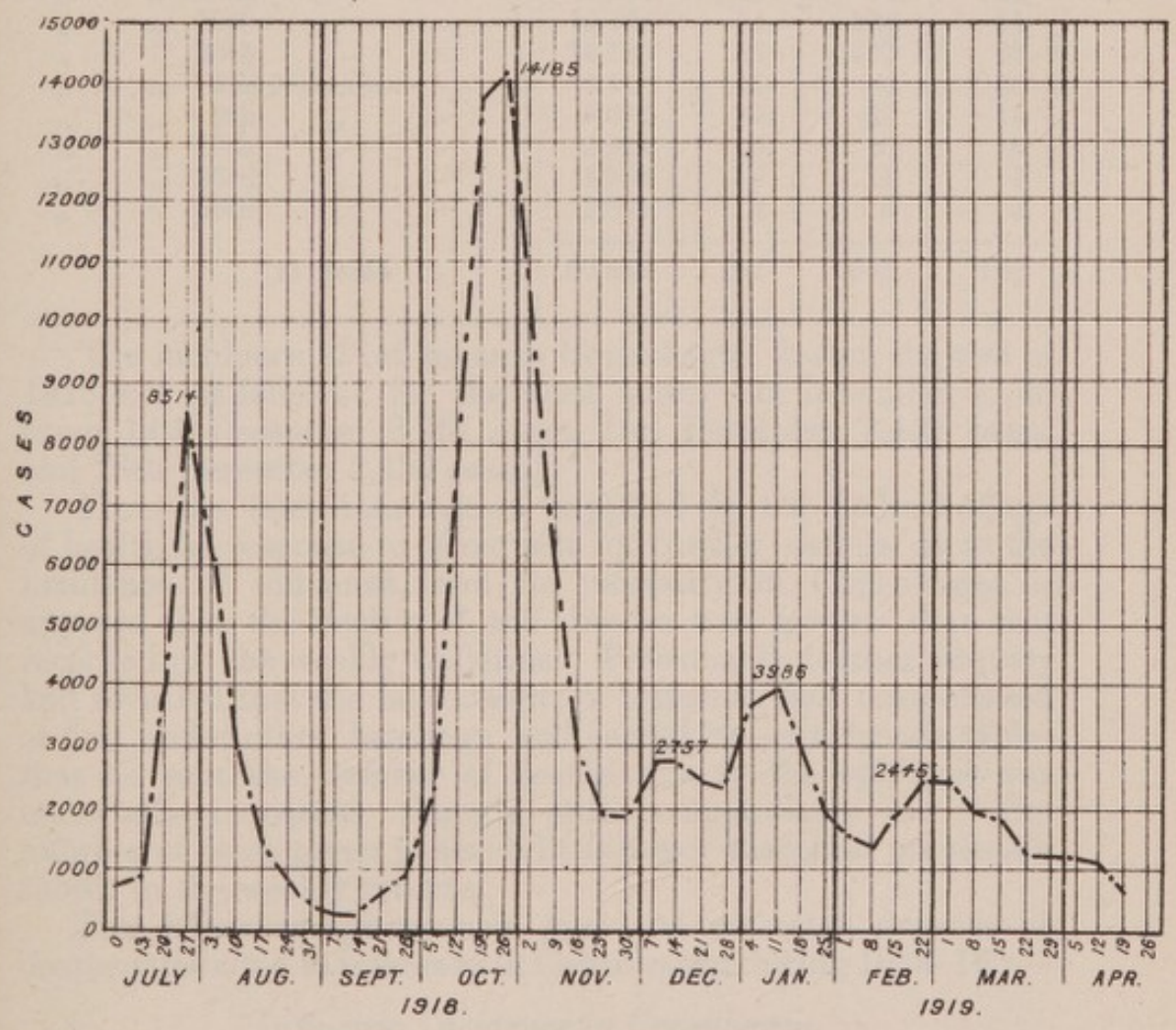
COPENHAGEN.

CASES OF INFLUENZA DURING LATTER PART 1918 AND BEGINNING OF 1919.



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The following table gives the course of the second epidemic week by week from September to the end of October 1918 :—

Second Wave of the Influenza Epidemic in Copenhagen.

1918.	Influenza.		Pneumonia.	
	Cases.	Deaths.	Cases.	Deaths.
Week ended 21st September - -	618	—	18	5
„ „ 28th „ - -	893	—	18	6
„ „ 5th October - -	2,171	—	28	11
„ „ 12th „ - -	7,504	10	74	22
„ „ 19th „ - -	13,786	11	204	11
„ „ 26th „ - -	14,185	26	177	10
„ „ 2nd November - -	9,412	15	60	30
„ „ 9th „ - -	5,672	25	67	15
„ „ 16th „ - -	2,924	8	10	10
„ „ 23rd „ - -	1,932	6	11	4
„ „ 30th „ - -	1,855	6	5	2
11 weeks - -	60,952	107	672	126

The incidence of influenza in Copenhagen during the rest of 1918 was as follows : for the week ended 7th December 2,746 cases, 14th December 2,751 cases, 21st December 2,442 cases, and 28th December 2,330 cases.

From the later information supplied by the medical officer of health in response to a request for further details as to the incidence of influenza, and its mortality in Copenhagen it appears that the fatality of the disease was greater than that recorded in the weekly bulletins. Presumably further inquiry had revealed that the fatal results of influenza had been classed at first under other headings and as will be seen by the tables that he sent the number of deaths due to the epidemic was materially increased, though still comparatively low. The number of cases given is also a little larger than that previously shown in the weekly returns.

The following figures now give the corrected number of deaths attributed to influenza in Copenhagen during 1918–19 :—

Influenza Incidence in Copenhagen.

Period.	Cases.	Deaths.	Case Mortality.
			Per cent.
1st. July 1st to September 7th, 1918 - -	25,360	85	0·3
2nd. September 8th to November 30th, 1918	61,285	1,293	2·1
3rd. December 1st to March 23rd, 1919 -	37,722	616	1·6
Total - - - -	124,367	1,994	1·6

As to the age and sex incidence, the distribution per 1,000 cases was as follows :—

——	0–1.	1–5.	5–15.	15–65.		65 and over		Total.
				M.	F.	M.	F.	
1st period - -	2	31	104	539	317	3	4	1,000
2nd period - -	6	73	283	268	365	2	3	1,000
3rd period - -	8	78	222	305	380	3	4	1,000

For the second six months of 1918, the following table has been supplied by the medical officer of health for Copenhagen with reference to the age and sex distribution of 1,483 cases investigated :—

Age and Sex Distribution of Influenza Cases in Copenhagen in the latter half of 1918.

1918.	Under 1.		1–5.		5–15.		15–25.		25–35.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
July - -	0	1	0	1	1	1	9	1	7	3
August - -	—		—		0	2	14	1	10	9
September -	—		—		0	3	4	1	2	6
October - -	10	7	17	15	21	28	102	81	149	151
November -	11	8	10	4	4	8	71	55	112	119
December -	1	3	7	4	4	7	33	19	57	43

1918.	35–45.		45–55.		55-65.		65 and upwards.		All Ages.		Total, both Sexes.
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
July - -	3	2	3	0	—		2	2	25	11	36
August - -	2	6	1	2	0	1	1	0	28	21	49
September -	—		0	1	1	0	—		7	11	18
October - -	64	65	14	12	5	7	1	7	383	373	756
November -	39	35	12	11	3	4	9	4	271	278	519
December -	24	12	7	8	1	4	3	5	134	105	239

The conclusions arrived at by the Copenhagen medical officer of health are that the mortality from influenza in the city seems relatively low, and the reason for this is probably that the notification of cases is more complete than in other great towns. Not only the age incidence has altered during the course of the epidemic, but also the sex incidence, and to

a considerable degree the mortality. The immunity against subsequent attacks conferred by a primary attack is in his experience by no means absolute, but he adds that on this point no statistical evidence is at present available.

During October, when the second wave was at its height, the schools, theatres, and picture palaces were closed and public meetings prohibited. The epidemic involved all classes of the population. During the third week of November, as the epidemic was rapidly decreasing, the schools were re-opened and other restrictions removed. It may be added the medical society of Copenhagen, with their organisation, and with the help of volunteer drivers and motor cars, visiting nurses, and other assistance, were able to do much to relieve the pressure of the epidemic. Financial aid was also furnished by the municipality. School kitchens were utilised in the preparation of food for the sick at their homes. A subscription list was opened by one of the principal newspapers to provide funds for the relief of the most pressing necessities of the stricken families.

The following table gives for comparison the notified cases of influenza in Copenhagen month by month during the two years 1917 and 1918 :—

Influenza Cases Notified.

——	1917.	1918.
January	2,388	298
February	1,726	293
March	1,080	240
April	653	172
May	262	79
June	66	68
July	47	16,798
August	50	8,341
September	106	2,617
October	119	43,809
November	152	15,072
December	160	11,848
Total cases	6,809	99,635
Total deaths	51	1,623
Cases mortality rate	0·75 per cent.	1·63 per cent.

Influenza continued to be prevalent in Copenhagen during the first part of 1919, as will be seen in the following table,

which gives for each month of the first half of 1919 the number of reported cases and deaths from influenza in Copenhagen :—

Influenza in Copenhagen, 1919.

——	Notified Cases.	Deaths.
January - - - - - - - -	12,337	327
February - - - - - - - -	8,064	145
March - - - - - - - -	7,074	163
April - - - - - - - -	3,658	75
May - - - - - - - -	1,228	32
June - - - - - - - -	258	5
Total - - -	32,619	747

Reckoned on the above figures the case mortality in the first half of 1919 was 2·3 per cent.

Iceland.—A serious epidemic of influenza occurred in Iceland in the latter months of 1918. In the town of Reykjavik, the capital of the island, with a population of about 8,000, during the early part of November a consular report stated that 70 per cent. of the inhabitants were prostrate with the disease. A ship which left Iceland early in January 1919 brought the intelligence that there had been 600 deaths from the epidemic in Reykjavik and its vicinity. The demand for coffins had been so great that undertakers, though working day and night, could not keep up with the demand. Druggists could not serve their customers fast enough, so great was the number of persons seeking medicines for their sick friends and relations at home.

Recently the Icelandic Government, with a view to protect the island against influenza, have issued a notice as regards the treatment of ships arriving from foreign countries. The main provisions of this notice are that no communications with ships arriving from abroad are to be permitted unless authorised by a quarantine officer. This officer has to make inquiries as to the health of persons on all such ships, and also as to whether influenza has been occurring at the port of departure. If five days have passed without the vessel having communicated with infected or suspected persons, and if no one on board has been suffering from suspicious symptoms, the ship is not considered dangerous, and those on board may be allowed to land. If a ship has been at sea for less than five days, or if it has communicated with infected or suspected persons *en route*, it is to be submitted to quarantine at the port of arrival until five days have elapsed from the date of exposure to infection. The special quarantine committee at Reykjavik may, however, modify the procedure, and can authorise the loading or unloading of a vessel before the full period has

elapsed if the district medical officer considers that these operations can be effected without risk being incurred. If the ship carries sick, or if there is a dead body on board, and if the district medical officer considers it likely that there is influenza on board the vessel, it will be directed to the nearest quarantine station, only strictly medical assistance being given. Infected persons arriving on vessels are to be isolated in lazarets which are to be kept ready for use. When such infected persons have been landed the ship is to be "isolated" for five days, but the special committee at Reykjavik may authorise loading or unloading of the ship if the district medical officer agrees.

Holland.

In Holland as in other parts of Europe influenza became epidemic during 1918. The prevalence of the disease was first observed in July and reached its height in August. There was a diminished incidence during September, but a fresh outburst occurred in October reaching its height in November. There were thus two distinct waves of the disease. So far as can be ascertained the total deaths caused by influenza in Holland during 1918 amounted to 17,734 distributed over the 11 provinces as shown in the following table:—

Deaths from Epidemic Influenza in the Dutch Provinces during 1918.

Provinces.	Population.	Deaths from Epidemic Influenza in 1918.
North Brabant	719,799	1,699
Gelderland	726,741	1,860
South Holland	1,645,920	3,562
North Holland	1,278,812	2,641
Zeeland	247,205	694
Utrecht	330,088	654
Friesland	384,760	1,100
Overyssel	433,114	1,622
Groningen	359,627	1,390
Drenthe	203,082	1,394
Limburg	438,198	1,118
Total	6,767,346	17,734

Among the places at which influenza appeared in July was an internment camp for British soldiers at Groningen, also at Losser in the province of Overyssel, at Sluys in the province of Zeeland, at Dalen in the province of Drenthe, and in the cities of Rotterdam and Amsterdam. From July to September, which includes the period of the first influenza wave, the deaths attributed to the epidemic amounted to 815 of which 89

occurred in July, 534 in August, and 183 in September. The deaths recorded from acute respiratory diseases during the same three months numbered 3,225. In October the deaths due to influenza rose to 3,017 and those from acute respiratory diseases to 5,237. November showed a still greater increase, the influenza deaths numbering 10,676 and the acute respiratory diseases 16,960; in December the mortality declined, 2,886 deaths being ascribed to influenza and 5,226 to acute respiratory diseases. The appended table gives, as far as can be ascertained, the incidence month by month of the influenza mortality: —

Deaths from Influenza and Respiratory Diseases Month by Month in the Dutch Provinces in 1918.*

Provinces.	Influenza.				Acute Respiratory Diseases.			
	July to Sept.	Oct.	Nov.	Dec.	July to Sept.	Oct.	Nov.	Dec.
North Brabant - -	89	166	1,204	218	427	390	2,075	527
Gelderland - - -	91	411	1,129	217	376	771	1,960	406
South Holland - -	217	421	1,895	794	693	687	2,901	1,367
North Holland - -	147	825	1,348	293	598	1,297	2,274	623
Zeeland - - - -	13	94	453	124	60	140	637	202
Utrecht - - - -	39	121	375	124	153	241	781	303
Friesland- - - -	18	50	765	260	76	112	1,104	384
Overyssel - - -	83	546	774	214	291	824	1,077	363
Groningen - - -	23	154	959	242	128	300	1,474	361
Drenthe - - - -	28	100	1,043	213	93	144	1,285	291
Limburg - - - -	67	129	731	187	330	331	1,392	399
Total - -	815	3,017	10,676	2,886	3,225	5,237	16,960	5,226

Summarising the figures in the table, it appears that during the third quarter of 1918, there were 815 recorded deaths from influenza and 3,225 from acute respiratory diseases in the eleven provinces of Holland. In the fourth quarter of the year the deaths from influenza amounted to 16,579 and those from the acute respiratory diseases 27,423. The coincident increase of the number of deaths from the latter class of diseases with that observed as regards epidemic influenza especially in the month of November suggests that deaths certified as due to pneumonia, broncho-pneumonia or other acute respiratory disease were in reality due to epidemic influenza. So that the actual mortality recorded in Holland from influenza was greater, probably much greater, than that given in the official returns of the disease.

In the city of Amsterdam from July to December 1918, there were 1,239 deaths from influenza; in January 1919, there were 24, in February 18, and in March 23.

* The total number of influenza deaths given in this table is 17,394, that is, some 350 less than the number given in the previous table. This discrepancy is probably accounted for by the fact that the figures given in the first table are from a more recent report than those given in the second table, and therefore, including some deaths not previously reported.

In the city of Rotterdam (population over 450,000) in the six months ended December 31st, 1918, 1,099 deaths were certified from influenza; in January 1919 there were 110 deaths and in February the number fell to 24, and in March to 17. In the Hague (population 270,000) during the second half of 1918 the deaths from influenza amounted to 530. In the chief towns of Holland the peak of the epidemic was not reached simultaneously judging by the death rate from all causes; for in Amsterdam the general death rate was at its highest point in the week ended November 2nd when the rate was 52·7 per 1,000. In Groningen the highest mortality rate occurred in the week ended November 9th when the rate was 81·2 per 1,000. In Rotterdam, the Hague and Utrecht, the highest mortality rate was reached in each instance in the week ended November 30th, when the rates were respectively 36·9, 32·2, and 39·0 per 1,000.

In the city of Amsterdam "influenza" has been appearing for some years in the mortality returns, 29 deaths being certified from this cause in 1914, 25 in 1915, 78 in 1916, and 36 in 1917. In the second half of 1918 the deaths recorded from this disease in Amsterdam numbered 1,239, along with 578 others from pneumonia, and 315 from broncho-pneumonia. In 1917, the number of new cases of "grippe" treated by the municipal medical service of Amsterdam was 835, of which 297 were men, 464 women, and 74 children.

The following table gives the weekly mortality from influenza and pneumonia in Amsterdam from June 30th to December 31st, 1918:—

Deaths from Influenza and Pneumonia in Amsterdam in the Second Half of 1918.

1918. Week ended	Deaths from Influenza.	Deaths from Pneumonia.
6th July	—	3
13th „	—	7
20th „	—	8
27th „	15	4
3rd August	21	16
10th „	24	28
17th „	9	29
24th „	2	22
31st „	2	7
7th September	—	9
14th „	1	2
21th „	2	6
28th „	3	9
5th October	8	4
12th „	25	18
19th „	101	38
26th „	225	63
2nd November	290	78
9th „	193	61

1918. Week ended	Deaths from Influenza.	Deaths from Pneumonia.
16th November	110	55
23rd „	48	22
30th „	52	23
7th December	31	22
14th „	30	12
21st „	23	18
28th „	14	6
29th to 31st December	4	8
Total	1,239	578

The estimated population of Amsterdam in December 1918 was 643,841.

The municipal health authority did not consider school closure an efficacious measure for controlling the epidemic, as many of the inhabitants of the town (and their children) live under conditions of overcrowding in their homes, though it was admitted that this measure in country districts would prove useful. Most of the individuals attacked by influenza in Amsterdam belonged to the age group 20 to 40 years. A circular was issued by the Central Board of Health to medical men requesting prompt notification of all influenza cases coming under their notice, as well as the reporting of any epidemiological facts concerning the malady.

Whether the infection reached Holland in the first instance from its landward side where five of its provinces abut upon the German frontier, or was brought by shipping to one or other of its ports on its western shores, it is not at present possible to say. It is, however, asserted by a Dutch authority that the introduction of influenza into Holland in 1918 was due to the return home of large numbers of workmen to Holland who had been employed in Western Germany where the disease was very prevalent at the time.

The incidence of fatal influenza on the cities of Amsterdam, Rotterdam, and The Hague, month by month, from July 1918 to May 1919, is shown in the following table:—

City.	Population.	1918.						1919.				
		July.	August.	September.	October.	November.	December.	January.	February.	March.	April.	May.
Amsterdam	643,841	5	72	11	572	477	102	24	18	23	39	2
Rotterdam	450,000	9	67	14	110	542	357	110	24	17	8	9
The Hague	270,000	4	44	3	41	292	146	22	14	11	12	—

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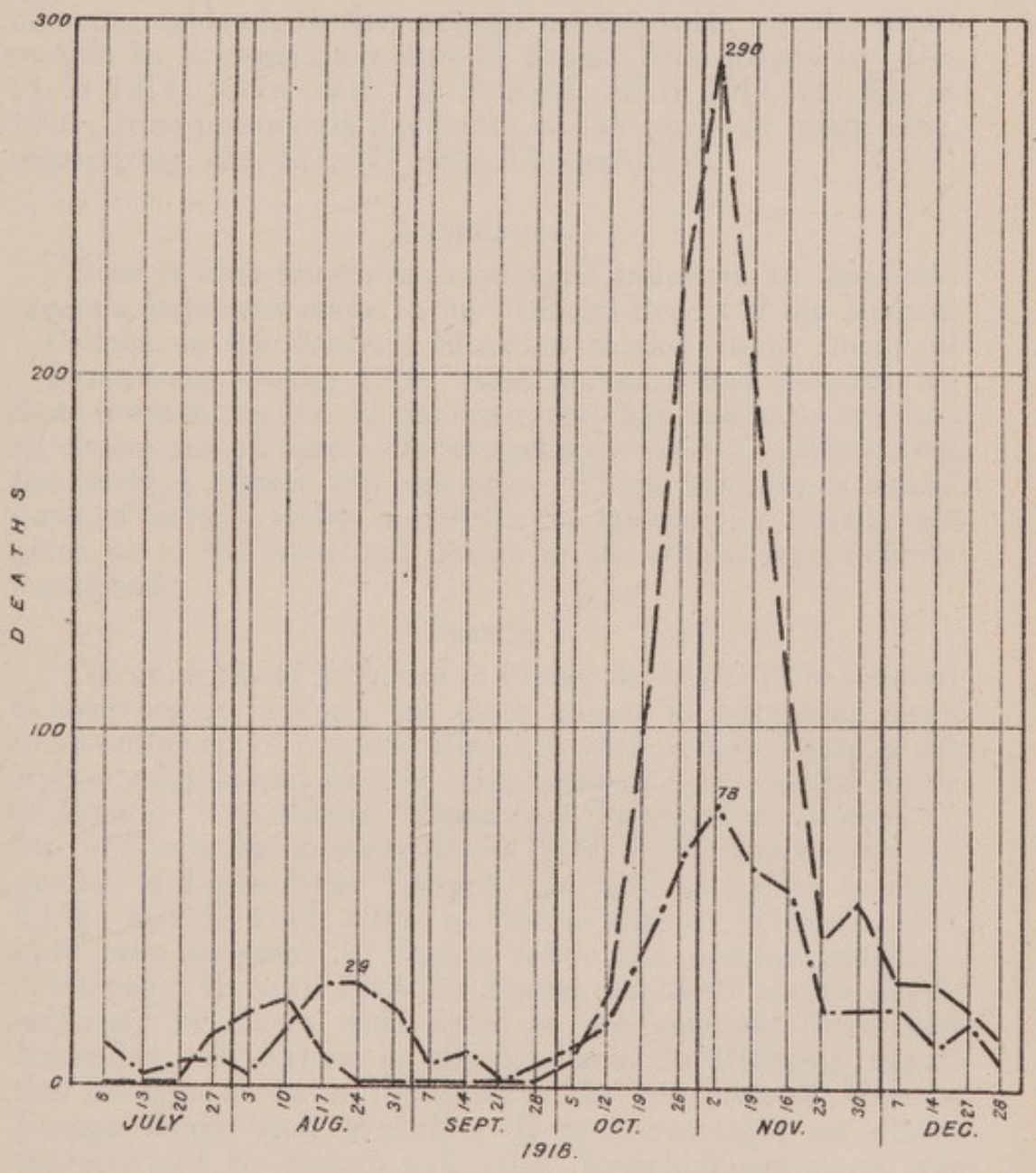
AMSTERDAM.

CASES OF INFLUENZA AND PNEUMONIA DURING LATTER HALF 1918.



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It has been suggested that the deaths caused by influenza in Holland during 1918 were considerably more than has been given in the statistics. The *Centrale Gezondheidsraad Maanblad*, for November 1919, gives the deaths caused by influenza, broncho-pneumonia, &c., in Holland during 1918 as amounting to 36,317, a mortality four times higher than the average of the previous eight years.

Belgium.

Owing to the war and the occupation of Belgium by the Germans, there is little information obtainable as to the influenza epidemic in that country during 1918. In the death records for Brussels there were 21 deaths from grippe in 1913, 14 in 1914, 20 in 1915, 45 in 1916, 65 in 1917, and 696 in 1918; from pneumonia the deaths in the same six years were, respectively, 452, 431, 377, 489, 703, and 1,242.

Luxemburg.

There is little known as to epidemic influenza in this State except a statement made in an "Official Report of the Medical "College on the Sanitary Situation in the Grand Duchy of "Luxemburg during 1918," that influenza had invaded the State towards the end of the year, and had suddenly attacked all classes and all ages. Its ravages were evident chiefly among the working classes and the poor. There was also a second wave of some severity in 1919. No statistics or details are given as to the cases and deaths in the official report above-mentioned.

France.

The epidemic of influenza in France during 1918 is believed to have begun in April, but there appear to have been some small outbreaks of a similar illness in 1916 and 1917 among the civil population and also in some military camps in the North of France. The official "Statistique Sanitaire de la France" for 1914 (relating to towns above 5,000 inhabitants) gives the number of deaths from "grippe" in 1912 as 4,835; in 1913, 6,158; and in 1914, 2,105, so that a form of influenza must have been endemic in France before the present pandemic developed. In April 1918 the disease appeared almost simultaneously in places wide apart as, for example, Paris and Marseilles. M. Netter, in the Académie de Médecine, stated that the appearance of influenza in the war zone of Western Europe dated back to 1917. It is known that an illness characterised by catarrh and other bronchial symptoms was prevalent in the military hospitals of France in the early part of 1917.

There was at one time a prevalent opinion that the influenza infection was introduced into France from Spain. Another view was that it came to Paris and other places from the war front, and there was also a belief by some that the disease was brought into France from Switzerland by repatriated prisoners

of war. On the other hand, Dr. Legroux asserts that influenza made its first appearance in France in the region of Dunkirk and travelled thence to the south.

The outbreak in April 1918 was in all respects similar to the epidemic of 1889. It began suddenly unfitting the patient for work of any kind; it was characterised by fever, intense pains in the head and limbs, lasting about three days, and leaving the sufferer weak and prostrate for some time. In this first wave of the epidemic, though many persons were attacked, there was comparatively little mortality. After an interval the disease reappeared in Paris and elsewhere in July, still of a mild type, but it was not till the end of September that the infection displayed more serious activities causing many deaths. The height of this wave in Paris was reached in the last week of October when the deaths from all causes amounted to 2,566, giving a death rate of 46·1 per 1,000 of the inhabitants. Of the 2,566 deaths from all causes, 1,473 were certified as due to influenza and 435 to pneumonia (all forms). In the week ended 2nd November, the total deaths registered in Paris were 2,402, of which 1,329 were attributed to influenza and 344 to pneumonia, the general death-rate being 43·2. From this date the epidemic declined. There was another epidemic wave in February 1919, though not of so serious a kind as that in October and November. The following table gives the weekly number of deaths from influenza, pneumonia, and from all causes from the beginning of September 1918 to the end of May 1919:—

Deaths from Influenza, Pneumonia, and from all Causes in Paris, Week by Week, from September 1918 *to* 31*st May* 1919.

Week ended	Deaths in Paris from			
	Influenza.	Pneumonia, all Forms.	All Causes.	Death Rate per 1,000 of the Population.
1918.				
7th September	21	55	666	11·9
14th „	24	92	718	12·9
21st „	64	101	873	15·7
28th „	123	142	888	15·9
5th October	240	166	989	17·8
12th „	616	230	1,445	26·0
19th „	1,046	268	1,944	34·9
26th „	1,473	435	2,566	46·1
2nd November	1,329	344	2,402	43·2
9th „	771	221	1,579	28·4
16th „	370	147	1,168	21·0
23rd „	226	125	997	17·9
30th „	243	158	1,080	19·4
7th December	291	172	1,145	20·6
14th „	221	143	1,098	19·7
21st „	202	114	885	15·9
28th „	172	128	933	16·8

Week ended	Deaths in Paris from			
	Influenza.	Pneumonia, all Forms.	All Causes	Death Rate per 1,000 of the Population.
1919.				
4th January - - -	170	155	992	17·8
11th „ - - -	185	168	1,046	18·8
18th „ - - -	109	169	1,025	18·5
25th „ - - -	93	177	1,002	18·0
1st February - - -	97	165	1,009	18·1
8th „ - - -	115	238	1,213	21·8
15th „ - - -	195	387	1,457	26·2
22nd „ - - -	426	513	1,894	31·7
1st March - - -	496	396	1,687	30·3
8th „ - - -	368	298	1,440	25·9
15th „ - - -	197	209	1,133	20·4
22nd „ - - -	114	146	998	17·9
29th „ - - -	62	123	939	16·9
5th April - - -	41	114	894	16·1
12th „ - - -	27	154	942	16·9
19th „ - - -	26	134	930	16·7
26th „ - - -	16	129	908	16·3
3rd May - - - -	6	111	821	14·7
10th „ - - - -	11	114	890	16·0
17th „ - - - -	9	113	826	14 8
24th „ - - - -	1	84	739	13·3
31st „ - - - -	4	91	747	13·4

It has been suggested that the official statistics for Paris do not show the whole of the ravages of the influenza epidemic in that city. A statement appeared in the London press which seems to favour the above suggestion for the number of victims that perished in Paris from the epidemic between 1st October 1918 and 28th February 1919 is given as 19,650.

The following table has been compiled from the weekly mortality returns for Paris, arranged for months from April 1918 to May 1919, showing separately the deaths certified from influenza, pneumonia, and "all causes" :—

Deaths in Paris from Influenza, Pneumonia, and all Causes, Month by Month, from April to May 1919.

——	Influenza.	Pneumonia, all Forms.	All Causes.
1918.			
April - - - - - - - -	8	515	3,757
May - - - - - - - -	14	464	3,851
June - - - - - - - -	11	204	2,499
July - - - - - - - -	44	293	2,583

	Influenza.	Pneumonia, all Forms.	All Causes.
1918.			
August	68	276	3,003
September	232	390	3,145
October	3,475	1,099	6,944
November	2,939	995	7,226
December	886	557	4,061
1919.			
January	654	834	5,015
February	1,142	1,534	6,251
March	741	776	4,510
April	116	642	4,492
May	25	402	3,202

The influenza prevalences in April and July do not appear to have swelled the monthly mortality returns, but the effect of the third wave began to be evident in September, while its greatest ravages were apparent in October and November. The fourth wave can be observed in the mortality returns for February 1919. An explanation* offered for the autumn recrudescence of influenza in Paris was the return at the end of September and beginning of October of many families to the city from the country and seaside districts at the termination of the holidays.

Many of the larger towns of France suffered from the autumn epidemic, including Marseilles, Lyons, Bordeaux, St. Etienne, Nantes, &c., and while the civil population were being affected the military also suffered. In the Marseilles district influenza was reported in April and May, again in July and August, and once again in September and October. Nearly 4,000 soldiers were attacked; the mortality rate among them was 9·6 per cent.

Influenza and pneumonia were prevalent in the late autumn of 1918 among the British and American troops in an equally severe form as that which was present among the civil and military populations of France. It was reported in the public press that in a period of about six weeks 50,000 cases with 3,000 deaths had occurred among the troops of the British Expeditionary Force in France.

In the American Expeditionary Force influenza and pneumonia were very prevalent from the middle of September to the middle of October. The following table is taken from the *Journal of the American Medical Association* for 16th November 1918, and gives the cases of influenza and pneumonia and other respiratory disease as well as meningitis;

* Dr. P. Lereboullet in the *Paris Médical* for 16th November 1918.

also the percentage of deaths among the pneumonia patients during the five weeks' period ended 11th October :—

Cases reported in the American Expeditionary Force in France during the Five Weeks ended 11th October 1918.

1918. Week ended	Influenza.	Acute Bronchitis.	Broncho Pneumonia.	Lobar Pneumonia.	Meningitis.	Percentage of Deaths among Pneumonia Cases.
13th September -	3,066	495	78	401	21	14·5
20th „ -	4,279	476	101	376	18	29·3
27th „ -	3,921	524	154	427	47	31·9
4th October -	9,285	988	364	671	88	34·9
11th „ -	5,794	890	194	401	134	45·3

It has been observed in France that when infections of the upper respiratory tract are prevalent the incidence of meningitis in the community soon increases. It has also been observed that when the infection of pneumonia passes rapidly through a succession of hosts an increasing severity develops in the symptoms. The arrival of fresh drafts of young troops to the American Expeditionary force added the heaviest percentage of infected men per strength, and gave the highest percentage of complicating pneumonias. The areas of heaviest infection were the base ports, the depôt divisions and such training areas as had received recent reinforcements from home, including men who had been exposed to the massive infection that had prevailed on the transports and in the crowded troop trains.

At Brest, which has a population of about 120,000, influenza was more or less prevalent during the second half of 1918. The following table gives the deaths reported from influenza and from all causes during each of the last six months of the year :—

Deaths from Influenza and from all Causes in Brest, July to December 1918.

1918.	Influenza.	All Causes.	———
July - - -	9	226	47 deaths in the naval hospital.
August - - -	51	235	75 deaths among the garrison troops.
September - -	433	640	364 deaths in the military hospitals.
October - - -	249	409	31 deaths in the military hospitals.
November - -	60	206	
December - - -	86	268	

During January 1918 there were only 15 deaths reported from influenza in Brest, but in February the number rose to 48 and fell again in March, when 24 fatal cases were recorded.

Dr. Louis Martin in *La Presse Médicale* for 24th October 1918 states that the influenza epidemic in Brest began in the Depôt de la Marine after the arrival of large numbers of young adults from all parts of France to begin their training. Most of them arrived fatigued by their long journey, and some of them were already ill of the disease. The railway carriages were overcrowded, and under the circumstances influenza infection probably acquired an added virulence.

There is no doubt that from time to time fresh strains of the influenza infection were introduced into French ports. For example, on 17th October 1918 a vessel arrived at the port of Havre having 74 cases of the illness on board among a crew of 78.

The following table gives the weekly number of influenza cases, and also the cases and deaths from pneumonia, in the American troops at Brest, from the middle of September 1918 to the end of April 1919.

Week ended	Influenza Cases.	Pneumonia.	
		Cases.	Deaths.
1918.			
16th September	603	179	29
23rd „	304	55	59
30th „	1,010	274	70
7th October	581	642	285
14th „	1,322	479	219
21st „	567	208	125
28th „	270	103	36
4th November	246	21	23
11th „	279	118	8
18th „	179	19	15
25th „	103	159	6
2nd December	86	24	10
9th „	143	8	4
16th „	119	24	7
23rd „	135	18	11
30th „	121	112	4
1919.			
6th January	196	39	2
13th „	153	53	14
20th „	156	54	19
27th „	183	47	7
3rd February	258	136	16
10th „	259	74	13
17th „	233	124	18
24th „	209	93	17

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· AMERICAN TROOPS · BASE. SECTION # 5 · BREST ·

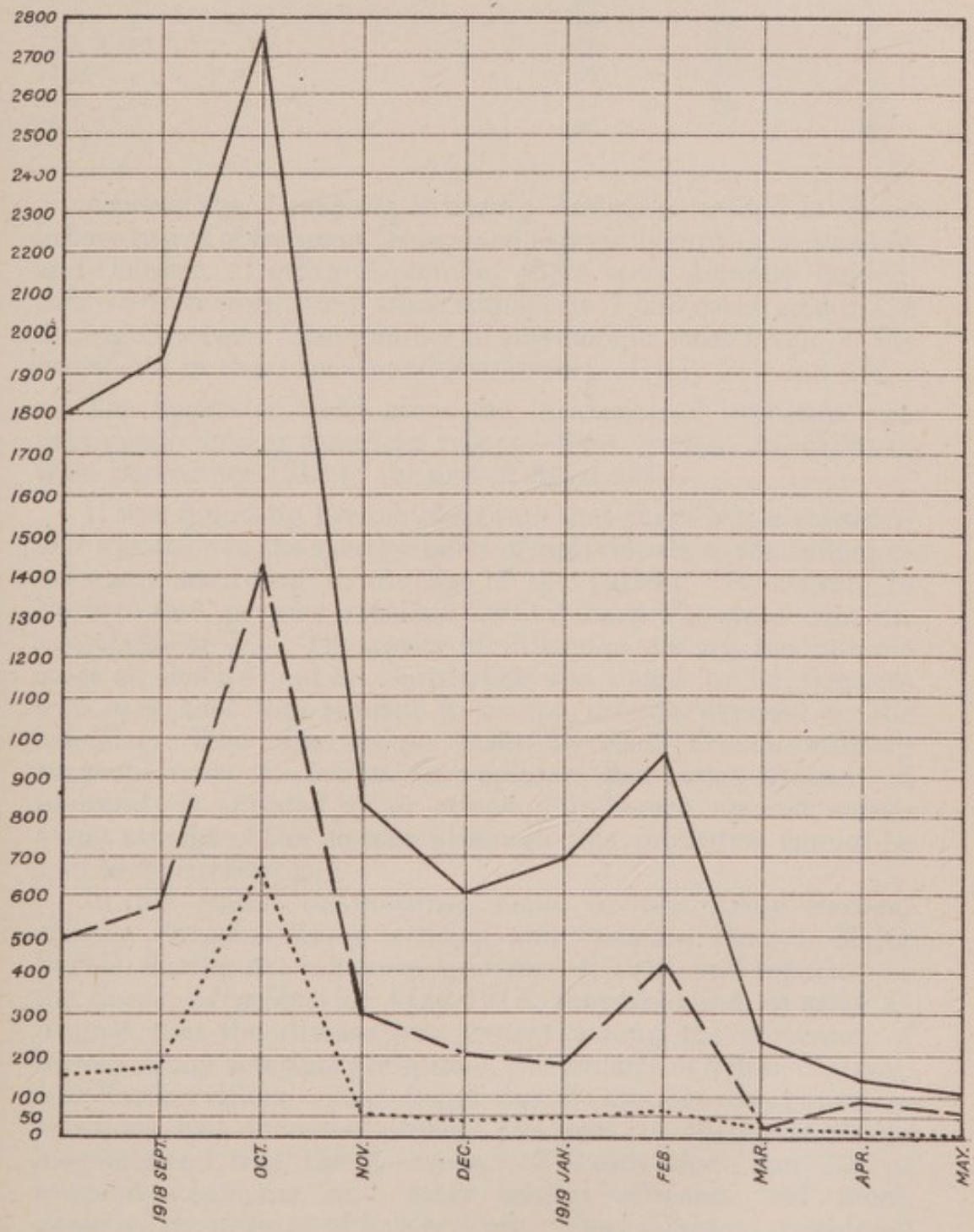
CHART SHOWING

— — — *ADMISSIONS* } *PNEUMONIA*

..... *DEATHS* }

——— *ADMISSIONS — INFLUENZA*

FROM SEPT. 1918 TO JUNE 1919 BY MONTHS.



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Week ended	Influenza Cases.	Pneumonia.	
		Cases.	Deaths.
1919.			
3rd March - - - - -	72	9	12
10th „ - - - - -	58	4	7
17th „ - - - - -	39	20	5
24th „ - - - - -	63	13	4
31st „ - - - - -	30	17	2
7th April - - - - - -	35	13	2
14th „ - - - - - -	52	28	1
21st „ - - - - - -	19	13	3
28th „ - - - - - -	20	24	2

Among the transports bringing American troops to Brest influenza was of frequent occurrence, especially during September and October. During September there were actually landed, sick of influenza, from these transports 1,693 cases, and 2,329 during October. The number of pneumonia cases taken off the transports in these two months were respectively 219 and 674.

The appended chart gives the incidence of influenza and pneumonia among American troops—Base, Section 5—at Brest, from September 1918 to the end of April 1919.

It was found by French observers that there was a considerable variation in the susceptibility of individuals to the influenza infection, according to the age of the patient. It was rare in Paris to find persons attacked by influenza who were over the age of 45 or 50. The rarity of influenza among the chronic cases at Bicêtre and La Salpêtrière was noted by Dr. Souques who says that they seemed to escape, though exposed to the infection. The observations made by some French military surgeons seem to favour the opinion that some amount of immunity is afforded by an attack of influenza against subsequent attacks of the disease, although this protection cannot be said to be invariable.

In the course of inquiries made by two Army medical officers, Surgeon Major Orticoni and Assistant Surgeon-Major Barbiè, during the influenza epidemic of 1918, and reported in the *Revue d'Hygiène* for April 1918, they observed, as early as August, that the disease was present among the personnel of certain Army veterinary hospitals, particularly at Vittel, Clairval, and Rambervillers. They found that the number of suppurative affections had increased during the year in these veterinary hospitals and that the percentage of horses which had died of broncho-pneumonia and other similar ailments had much exceeded the average of former years. They therefore considered

the question whether the pandemic of human influenza and certain affections in horses diagnosed as distemper or "gourme" were related in any way. "Gourme" is defined in *Littrés Dictionnaire de la Langue Francaise* as a "disease of horses, and "particularly of young horses, characterised by inflammation of "the mucous membrane of the respiratory track with swelling of "the submaxillary glands and tumefaction of the surrounding "cellular tissue." The "gourme" observed in 1918 was a more severe disease than usual and was marked by a period of fever, with infections from the nasopharynx, anasarca and pulmonary complications; the disease was more than usually prevalent among horses. In the veterinary hospital at Ravenel, near Mirecourt, the number of such cases had in previous years been insignificant, but from April to October 1918, more than 400 horses, ill of this prevalent "gourme," were admitted. Neighbouring Army veterinary hospitals had a similar experience. The horses belonging to the civil population did not escape, and according to trustworthy reports in some localities nearly every horse was attacked by the prevalent malady. In certain villages around Mirecourt, all the horses suffered from bad coughs at the very time that human influenza was raging at its worst in the locality. The horse epizootic began in April, it attained its height in August and September, and was declining in November; its curve was similar to that of the human epidemic. M. L. Martin, Assistant Director of the Paris Pasteur Institute, has stated that the mortality of the horses in the stables of that Institute in 1918 had been much higher than usual. It was mentioned by some of the older French veterinary surgeons that in 1889 there was a recrudescence of the "gourme" affections among horses at the time that influenza was epidemic among the human population. The term "gourme" no doubt includes ordinarily a number of different affections, but in 1918 the prevalence was marked uniformly by the same characteristic symptoms. In ordinary times this "gourme" is an affection of young horses, rarely attacking those over the age of six years; it is, moreover, generally a disease of the winter or early spring months. In 1918, however, the disease was widely prevalent during the summer, and attacked indiscriminately horses of all ages. In the Ravenel Army veterinary hospital in 1918, about 5 per cent. of the horses died from "gourme." Majors Orticoni and Barbiè are therefore disposed to regard the "gourme" of 1918 as having been etiologically different from the form that is usually observed in France, though in some way resembling it. They isolated from the blood of certain horses, attacked by fever and serious pulmonary congestion, a bacillus having, they said, the same characters of morphology, coloration, and culture, as the bacillus isolated from human influenza patients. Whether these bacilli are identical, or are only related, these observers are not yet in a position to decide. The micro-

organism obtained from the sick horses was a cocco-bacillus, aerobic, immobile, decolorised by Gram, having the characters of poly-morphism of the pasteurellæ, giving like the human bacillus all forms short and long, and not growing on ordinary culture media (bouillon and agar gelose). It is therefore concluded that this bacillus from the horse, if not identical, is closely related to the human influenza germ ; the only difference observed was that the bacillus from the horse could be passed into the organs of inoculated mice, while the human bacillus only in exceptional instances, passed into the organs of the experimental animal.

In severe cases of human influenza with pulmonary complications in France, three organisms were generally isolated, namely, the pneumococcus, the streptococcus, and Pfeiffer's bacillus. The pathogenic action of the cocco-bacillus, isolated from the blood of human patients during the 1918 epidemic was nil for guinea pigs and mice, and but feeble and variable for rabbits. By hemoculture the presence in the blood of equine cases of a cocco-bacillus resembling Pfeiffer's bacillus, associated in certain instances with the pneumococcus and the streptococcus, was demonstrated. As in human cases, the serum of convalescent horses is stated to have had therapeutic action which appears to be efficacious against the septicæmic form of human influenza, and when employed at the onset of the attack it appeared to neutralise the influenza virus, " ameliorating the patient's general condition, arresting pul- " monary and pleural complications, and rendering benign and " without gravity an illness in which the complications are " very often mortal."*

Before the Académie des Sciences in October 1918, Dr. Roux, Director of the Pasteur Institute, reported the results of experimental research on the comparative pathogenesis of influenza. This work was done on monkeys and then on human subjects by Drs. Nicolle and Lebailly of the Pasteur Institute of Tunis. Their observations showed that the infection is carried during the active stage of the disease by the nasal and bronchial mucus of influenza patients. The monkey can be inoculated with the virus applied to the nasal mucous membrane, or injected under the conjunctiva. The symptoms of influenza develop in about six days. A man developed the symptoms in about the same time when inoculated subcutaneously with bronchial mucus diluted 10 times with physiological saline solution. The

* La Pandemie Grippale de 1918 ; Relations de la Grippe humaine avec certaines epizootics animales, par le Dr. Orticoni, Médecin-Major, Médecin-chef d'un Laboratoire de bacteriologie d'Armée, et le médecin-Aide-Major L. Barbié, attaché au Laboratoire.
Revue d'Hygiène, April 1918.

infecting agent according to Dr. Roux seems to be a very small micro-organism which passes through a Chamberland filter and which is distinct from other microbes hitherto described, and notably from the bacillus of Pfeiffer. The subcutaneous injection of this organism into the human subject reproduces the disease. Intravenous injection of it, however, does not convey the disease to man or to the monkey.

As regards anti-influenza measures, a Commission appointed by the Académie de Médecine, in addition to compulsory notification, recommended the following :—

Individual Prophylaxis.

(1) As influenza is transmitted by the discharges from the respiratory tract, individual prophylaxis should include a careful disinfection of the mouth and nose.

(2) During the epidemic the throat should be gargled at least twice a day with some antiseptic, *e.g.*, a teaspoonful of "Liqueur de Labarraque" in a glass of water.

(3) The antisepsis of the nasal fossæ should be maintained by the application of a vaseline ointment with 1 per cent. of resorcin, or 2 per cent. of eucalyptus.

(4) The hands and face should be washed before each meal.

(5) In the presence of actual influenza or even suspected influenza, all the usual sanitary precautions should be taken.

(6) Late hours should be carefully avoided; and every means should be taken to prevent chills, or overwork.

(7) At the first indication of illness, suspected to be influenza, the temperature of the patient should be ascertained, and this should be repeated several times during the day; and if any elevation of temperature be discovered the sufferer should be put to bed and isolated from the other members of the household.

General Prophylaxis.

(1) Places in which many people crowd together should be carefully supervised so as to secure healthy surroundings and proper ventilation.

(2) The accumulation of dust containing micro-organisms should be prevented. Wet sweeping should be used, and dusting should be done with damp cloths.

(3) All overcrowding should be suppressed, since it is a great factor in the spread of influenza and other diseases.

(4) Special precautions should be taken in barracks and camps to prevent overcrowding.

(5) In case of extension of the epidemic, the Minister of the Interior should forbid the holding of fairs, the opening of theatres, cinemas, concert rooms, and assemblies, after consultation with the Departmental Council of Hygiene. So long as places of public amusement are open, free ventilation should be provided, and dry sweeping should be forbidden.

(6) The public themselves should avoid crowds and public gatherings.

(7) Public conveyances are often the means of spreading influenza; therefore trams, district railway carriages and other public conveyances, owing to the overcrowding that commonly takes place in them, should be frequently washed and disinfected.

(8) School-closure is of great use, especially if any of the pupils have been attacked by the infection. Free ventilation and frequent cleansing are very neccessary in schools.

With further reference to the precautions to be used in French schools against influenza the Prefect of the Seine, on the advice of various authorities, issued for the guidance of directors of schools in Paris and the adjoining departments, the following recommendations:—

(1) Stop all meetings in school buildings.

(2) Inquire into reason for absence of any pupil from school without delay, and inform the Bureau of Medical Inspection of Schools as to the result; exclude any child who presents any symptoms of indisposition and send home under supervision, requesting the head of the family to obtain medical advice.

(3) Advise parents to keep their children at home if they show any signs of illness.

(4) Exclude from school the brothers and sisters of those attacked, and inform the directors or directresses of any schools attended by other members of the affected family.

(5) Frequently wash the floors, blackboards, and desks with a solution of "Javelle water," or creolin, or a 20 per cent. solution of formyl or anodorine.

(6) Do not close the whole school, but dismiss for a period of 15 days any class if three-quarters of the members are absent from influenza. If, however, the situation is regarded as grave by the school medical inspector, he may close the school.

(7) Advise the use of antiseptics for the nasal fossæ, such as "vaseline gomenolee"* or a solution of collargol.

Spain.

A disease termed influenza or "grippe" has appeared in the mortality records of Spain for some years. For example, in 1916 the deaths from "grippe" numbered 6,485 and in nine months of 1917, there were 5,568. In February 1918 "grippe" is said to have been present at San Sebastian (population about 50,000) a seaport, and health resort, situated in the North of Spain about 11 miles from the French frontier, but it did not, apparently, become epidemic in that town until May. It is, however, possible that it may have been more prevalent in the early part of 1918 than was admitted at the time, and allegation is made that the municipal authorities of San Sebastian purposely concealed the prevalence of influenza as far as they were able, so as not to prejudice the summer season of this health resort, and stop the coming of the expected visitors.

The Spanish authorities strongly maintain that the first epidemic wave of influenza that swept over Spain in May and June 1918, was due to infection introduced from France. The French authorities on the other hand as stoutly contend that France received its infection from Spain. Other experts attributed the origin of the infection of Spain to the Levant, while others again contend that influenza was in the early months of 1918 already epidemic in Central Europe. The first wave of the influenza epidemic of 1918 in Spain was comparatively mild, but very widespread; one estimate is that some eight millions of persons were attacked during May and June. One of the places that suffered much from this first epidemic wave was the city of Madrid (population 648,760) where it is asserted some 30 per cent. of the population suffered, including King Alfonso and a number of his Cabinet Ministers. The effects of the epidemic were felt severely among the public officials, and some Government Offices had in consequence to be closed, and the municipal tram services suspended.

* Gomenol is a proprietary French remedy obtained from a plant of the Myrtle tribe.

Bacteriological investigations proved the presence of Pfeiffer's bacillus in almost all cases, thus setting aside the alarming reports in circulation about the "new disease" which some persons at first asserted was phlebotomus or sand fly fever; others said it was plague, while yet others regarded it as a form of "typhus" fever. Abdominal symptoms were observed in a large number of cases, diarrhœa being common. Dr. Mancel Martin de Salazar, of the Ministry of the Interior, stated that the development of the influenza epidemic in Madrid during May, coincided with the presence of a large number of visitors, the inference apparently being that the latter had introduced the infection into the city. The early summer epidemic was characterised by its great infectivity, its short duration and the relatively benign character of the symptoms. Nevertheless, in Madrid 56 persons died from the disease in the month of May, and 220 in June. In the mortality records of Madrid for January 1918, 29 deaths were attributed to "grippe," 14 in February, 20 in March, and 6 in April, so that unless the "grippe" which was epidemic in Madrid in May and June was different from that already existing in the city, it would not be necessary to ascribe the origin of the infection to France, the Levant or Central Europe.

Along with the deaths from influenza there was a corresponding increase in the deaths due to respiratory diseases, tuberculous affections, and heart diseases. The great power of diffusion of the infection was most marked in circumstances where persons assembled together, as in barracks, theatres, postal and telegraph offices, factories, and workshops. The greatest incidence was among those in the age group 20 to 30 years, while children under five years and old persons were comparatively rarely attacked.

The second wave of influenza during 1918 in Spain began in September, reaching its maximum in October and decreasing in November. Again it was asserted that a fresh strain of infection had been imported from France, and to prevent a recurrence the frontier was closed for some weeks.

The character of the second epidemic was severe and there was a considerable mortality. So serious indeed was the prevalence of the disease in some localities that the public deserted the theatres, music halls, and public restaurants. Public dances were forbidden, and many of the people only ventured forth when they could carry with them the materials for inhaling eucalyptus, creosote and other disinfectants or germicides. Great difficulty was experienced in burying the dead, coffins at one time being almost unobtainable. The mortality was highest in the age group 20 to 39, and those least affected were young children and elderly people. The following table gives a summary of the fatal incidence of influenza during

the three months, September, October, and November 1918, in the kingdom of Spain :—

Mortality from Influenza in Spain in the Second Epidemic of 1918.

1918.	Deaths from Influenza and its Complications.
September - - - - -	7,857
October - - - - - -	79,484
November - - - - -	40,478

The population of Spain is 20,747,893. As in the summer prevalence, the disease carried off many chronic invalids, such as those suffering from pulmonary tubercle, organic disease of the heart, acute nephritis and Bright's disease.

The following table shows the increase of such deaths during the epidemic period :—

1918.	Deaths from		
	Pulmonary Tuberculosis.	Organic Disease of the Heart.	Acute Nephritis and Bright's Disease.
September - - - -	2,655	2,833	896
October - - - - -	4,394	5,596	1,239
November - - - -	3,354	4,355	1,213

The figures in the above two tables are taken from the November number of the *Boletin Mensual de Estadistica Demografico-Sanitaria*, issued by the Spanish Government. From the same publication a table has been extracted showing the number of deaths from influenza during September, October, and November in each of five age groups.

The age distribution of deaths from influenza in Spain during the months of September, October and November, 1918.

Age Groups.	September	October.	November.
Under 4 years - - - -	899	11,312	6,173
5 to 19 years - - - -	1,362	15,623	8,517
20 to 39 years - - - -	3,756	34,828	16,490
40 to 59 years - - - -	1,121	11,212	6,092
60 and upwards - - -	695	6,109	3,209
Total -	7,833	79,084	40,481

The monthly totals given in this table are not quite the same as those given in one of the previous tables, but the discrepancy may be accounted for by the assumption that the precise age was not ascertained in some 421 of the fatal cases included in the former table.

There are 49 provinces in Spain (including the Canaries and the Balearic Islands) and the statistical records published officially give the deaths in each of the capitals of these provinces. These capital towns did not all suffer from influenza in the same degree, and some suffered during the summer prevalence while others were attacked in the autumn epidemic.

The cities which suffered much were Barcelona, Valencia, Madrid, Murcia, Viscaya, Oviedo and Saragossa. The next table shows the distribution of the fatal incidence of influenza in these cities during 1918 :—

Spanish Provincial Capital Cities.	Population.	Deaths from Influenza in Seven Spanish Cities during 1918.												
		January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
Barcelona -	621,419	37	20	18	7	16	28	8	6	65	1,178	152	*	1,535
Valencia -	245,871	10	7	8	5	3	15	11	7	71	676	264	*	1,077
Madrid - -	648,760	29	14	20	6	56	220	10	10	30	194	214	150	953
Murcia - -	138,012	—	2	7	5	4	11	3	7	31	495	183	*	748
Viscaya -	100,461	—	—	5	—	1	16	3	—	8	480	146	*	659
Oviedo - -	55,913	—	3	3	3	1	15	2	—	15	349	20	*	411
Saragossa -	124,455	8	10	6	5	4	28	7	7	5	205	129	*	414

* No information received.

The influenza death rate, calculated on the above figures, per 1,000 of the population of the City of Madrid with its 953 deaths, is lowest with a rate of 1·4. Barcelona comes next in order with a rate of 2·4, then Saragossa 3·3, Valencia 4·3, Murcia 5·4, Viscaya 6·6 and Oviedo 7·4 per 1,000 of the inhabitants.

Barcelona, on the Mediterranean Coast of Spain, is the second city in point of population, and next to Cadiz the principal port of the kingdom. Judging from the number of deaths, month by month, influenza appears to be endemic in it. A correspondent states that influenza and pneumonia occur more or less all the year round in Barcelona, but incidence is increased in the winter months. The local authority are always unwilling to furnish immediate and accurate information on the subject of vital statistics. There were 75 deaths from influenza (grippe) in the first quarter of 1918, a period during which there was no suspicion of an epidemic. The same remark applies to Madrid, where, in the first three months of 1918, 63 deaths

were ascribed to influenza. In the summer epidemic, Madrid, in May and June, had 276 fatal cases recorded, while Barcelona had only 44 in the same two months.

The progress of epidemic influenza in the 49 provincial capitals of Spain during each month of 1918 is shown in the appended table :—

Deaths in the Capital Cities of Provinces of Spain in 1918.

1918.	All Causes.	Influenza.	Pneumonia.	Other Respiratory Diseases.*
January - - - -	10,598	167	495	2,144
February - - - -	8,534	103	348	1,633
March - - - -	9,069	139	387	1,723
April - - - -	7,341	75	247	1,188
May - - - - -	7,711	139	254	1,770
June - - - -	10,202	709	397	1,581
July - - - - -	8,379	183	154	781
August - - - -	7,922	81	146	650
September - - -	8,896	747	286	1,009
October - - - -	24,196	7,980	1,293	4,979
November - - - -	12,407	2,541	409	2,104

* Excluding pulmonary tuberculosis.

In the town of San Sebastian, which is not one of the provincial capitals, the autumn epidemic began in August, causing 96 deaths : 264 in September : and 284 in the first 10 days of October.

There was an outbreak of influenza in the Balearic Islands in September, and this continued during October and November. So far as can be ascertained, 429 deaths resulted ; but this is probably below the actual number.

In the Canary Islands (regarded as a province of Spain), influenza was also prevalent during October 1918, especially in Grand Canary, La Palma, and Teneriffe.

The epidemic seriously affected members of the medical profession and the *Medicina Iberia* for January 18th, 1919, published at Madrid, states that some 200 medical men died from the disease in Spain, the result of their close contact with grave cases. In this connection, the Spanish Government voted a sum of money to be distributed among the widows and orphans of the medical practitioners who fell victims to the epidemic.

Attention was drawn in the Spanish medical journals to the common occurrence, during the epidemic, of abortion or premature delivery among pregnant women attacked by

influenza. Dr. Valesco accounted for this by the congested state of the uterus leading to hæmorrhage, which acted like foreign body, leading to expulsion of the uterine contents.

An instance of the methods sometimes adopted in Spain by high officials towards medical men when reporting on outbreaks of epidemic disease is worth recording here ; it was mentioned in one of the medical journals.

An outbreak of influenza occurred in the prison at Almeira in the summer of 1918, the inmates of this institution being among the earliest attacked in the town. Dr. Gomez Casas, the medical officer of the prison, in due course, reported to the authorities the circumstances associated with the influenza outbreak. The information was very unwelcome to the authorities, and, subsequently, when it reached the governor of the province, he sent for Dr. Casas, and directed him to make a written statement for publication withdrawing his diagnosis of influenza and denying the existence of the disease in Almeira prison. This Dr. Casas refused to do, and he was, in consequence, dealt with by the governor and fined. His medical friends were very indignant, and the provincial "Colegio Medico" paid the fine, but sent an official protest to the central authority, the Consejo de Sanidad, at Madrid, with what result was not yet known at the time of writing.

In order to meet the many opinions expressed at first as to the true nature of the epidemic, a Spanish Medical Commission was sent to Paris to collect information, particularly as to the identity of the epidemic in Spain with that occurring in France and other countries of Europe. Their conclusions were that the Spanish epidemic and that in France were identical, not only from the clinical, but also from the bacteriological and epidemiological points of view. In both countries there were more females attacked than males, and this was explained by the tendency of women in both to lead an indoor existence.

Portugal.

In Portugal, as in Spain, there is an illness termed "influenza" which appears in the death records apart from epidemic periods. It is more frequent in the winter than in the summer. In Lisbon, for example, during 1917, there were 120 deaths certified from this cause, and 41 in Oporto ; and in the first quarter of 1918, 23 were certified in Lisbon and 16 in Oporto. The epidemic of 1918, as in Spain, is stated to have begun in May, and soon extended over the whole of Portugal. At first the epidemic malady was thought to be phlebotomus fever, but this was soon disproved. The Portuguese authorities assert that the infection was derived from Spain. The first epidemic of 1918 in Portugal lasted until the end of July. So far, no trustworthy statistics are available as to either the

number of cases, or deaths, but it is generally stated that the disease was present throughout the country in May and June in a mild form, and caused a comparatively small mortality. In August there was evidence of a second wave beginning in the neighbourhood of Oporto. This second epidemic was of a far more serious character than the first. So severe were the pulmonary complications, and so rapid the fatal onset, that for a time the malady was regarded by some as pneumonic plague. By the end of September most of Portugal had been invaded by the "new" strain of infection, which caused many deaths, "especially among the semi-starving population in the north." During October influenza continued to be prevalent throughout Portugal, and is said to have caused about 200 deaths daily in Lisbon at the height of the epidemic. It is probable that details of these two epidemics will be published later. Some cases of influenza were landed from America and other ships at Lisbon in the autumn.

Professor Ricardo Jorge, of the Superior Council of Hygiene, for Portugal, in discussing the influenza epidemic in the *Medicina Contemporanea* (Vol. XXXVI.), recalls the fact that the last pandemic which struck Portugal in 1890 developed during the summer months. He admits that all attempts to enforce prophylactic measures were in vain, and nothing seemed to arrest in any way the progress of the epidemic. Great efforts, however, were made to meet the demands for medical assistance among the poor, to provide hospital accommodation for the serious cases, and for the distribution of food and medicine to the necessitous poor.

Gibraltar.

There were two separate and distinct outbreaks of influenza in Gibraltar among the civil population during 1918.

The first wave began in the latter part of May, reached its height in June and subsided early in July, having lasted for a period of six or seven weeks. Some difficulty has been experienced in obtaining definite figures as to the first influenza epidemic; one estimate gives 600 cases and another 800 out of a population of 16,549. This outbreak was mild in character, and the mortality small. Many cases never came under medical observation. The weather at the time was hot and dry. The infection is believed to have entered Gibraltar from Spain.

The second wave which began in September was of a more severe kind. It began at the season of the onset of the autumn rains, the weather being inclement, causing the people to remain indoors under conditions of deficient ventilation. This wave of influenza reached its maximum in October gradually subsiding in November. The number of attacks has been variously estimated, one authority mentions 3,000 cases, but it has been suggested that the probable number of attacks was nearer 6,000

including the mild cases. There were 126 influenza deaths. According to a report by Colonel G. Dansey-Browning, A.M.S., Medical Officer of Health for Gibraltar, relapses were fairly frequent especially among the milder attacks. Only a small percentage of those who suffered in the first epidemic of 1918 experienced another attack during the second wave of the infection.

The interval between the two waves was about six weeks and during this interval some cases of epidemic catarrh were reported, but it is said the second outbreak was due to a fresh importation of the infection from Spain. The disease was at the time very prevalent in the neighbouring Spanish towns of San Roque and La Linea, and thousands of the inhabitants of these towns were entering Gibraltar each day to work alongside the residents. Subsequent to this there were repeated introductions of the infection by shipping. Ships, for example, coming to Gibraltar from Genoa landed in September 47 persons suffering from influenza. Other ships landed influenza cases from Biserta, Sierra Leone, Barry, Cardiff, Aquillas, Dakar, Newport (Mon.), and New York.

At the Colonial Hospital 361 influenza cases, 337 landed from merchant ships, were treated during 1918, and 57 of them died giving a case mortality rate of 15·8 per cent. These 361 cases represented 31 nationalities, 100 being British, 45 Indian, 38 Italian, and 30 Spaniards.

Colonel Dansey-Browning states that Pfeiffer's bacillus was isolated in all primary influenza cases which were examined, and the pneumococcus in the pneumonic cases. Owing to the stress and strain of the second epidemic and the amount of work devolving upon the Health Department, only a relatively small number of bacteriological examinations were carried out. Comparatively little action was necessary during the first outbreak, but in the second the Health Department prohibited indoor public assembles and wakes, restrictions being placed on funeral ceremonies in churches: influenza was added to the list of notifiable diseases, and when it was thought necessary schools were closed.

Malta and Gozo.

Although "influenza" is stated to be endemic in Malta it has seldom appeared of late years to any extent in the mortality records of the island: rarely more than 10 deaths in a year have been ascribed to it since the pandemic of 1889–90; but in 1890 there were 64 deaths reported from influenza, 65 in 1892, and 78 in 1900. The population of the island is about 200,000. The past experience in Malta is that the incidence of this disease was limited to the winter and spring months, so that the occurrence of an epidemic in the summer months, as happened in 1918, was quite unusual. It appears from a report by the Chief

Government Medical Officer, Dr. A. Critien, dated December 12th, 1918, that there had been some cases in the early part of the year, but this was regarded as only being the ordinary seasonal appearance of "influenza." In June, nine cases were reported, 33 in July, and 50 in August. In September the malady became epidemic both in Malta and in the island of Gozo, which is only 4 miles distant. The height of the outbreak was reached in October when 4,651, cases were reported in Malta, and 1,203 in Gozo. There was a great decrease in the number of cases in November, and by mid-December the epidemic had subsided. The following table gives, for the civil population of Malta, the number of influenza and pneumonia cases reported month by month from June to December, together with the number of deaths recorded in that period due to the epidemic. The incidence of influenza on the naval and military forces stationed at Malta will be discussed separately :—

The Incidence of Epidemic Influenza at Malta, in 1918.

1918.	Influenza.		Pneumonia and Broncho-pneumonia.		Total due to the Epidemic.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
June - - -	9	—	—	—	9	—
July - - -	33	31†	—	—	33	} 31†
August - -	50	—	—	—	50	
September - -	3,197	65	83	34	3,280	99
October - -	4,651	294	280	56	4,931	350
November - -	1,036	63	104	39	1,140	102
December* -	195	4	14	2	209	6
Total -	9,171	457	481	131	9,652	588

* From 1st to 10th only.

† Including deaths from influenza and pneumonia, the number of each not being distinguished in the official report.

As regards the period prior to September when there was a widespread prevalence of simple cases running a short course to spontaneous recovery, but not officially reported, it is impossible to estimate their number. It is generally believed in official quarters that the reported cases of influenza in Malta during the epidemic, represent only about half of the number of persons attacked. Reckoning the fatality rate of the disease on the figures given in the table, namely a total of 9,652 attacks, and 588 deaths, the result is a case mortality rate of 6 per cent. According to Dr. Critien, if the missed cases were considered in the calculation the rate would be about 3·7 per cent. It is

believed that the infection was brought to Malta by shipping: The first districts to suffer were those in which the inhabitants were closely connected with the docks and other Government establishments. It is reported that early in June some ships of the Royal Navy and others of the Mercantile Marine had arrived with influenza cases on board. As Malta is the halfway port between Europe and the East, ships from infected ports are extremely likely to bring with them infection of influenza and other diseases to the island.

As soon as the development of the epidemic began in Malta leaflets and posters were issued calling public attention to the extreme infectiousness of the disease, its methods of transmission, and the value of free ventilation and fresh air in its prevention, as well as the necessity of personal and domestic cleanliness. Cases were isolated at their homes when possible, or when severe in hospital, and rooms, bedding and linen were disinfected Measures were taken to prevent overcrowding in public places, cinemas, theatres, and other places of amusement, as well as the enforcement of free ventilation. The opportunities for visiting hospitals and charitable institutions were curtailed; railway carriages were disinfected and Government schools were closed; orders were issued to discontinue the pawning of clothes. All persons arriving from abroad were subjected to temporary surveillance and their personal effects were disinfected in certain cases.*

The incidence of epidemic influenza in Gozo during the Autumn of 1918 was similar to that observed in Malta.

Gozo is an island situated 4 miles from Malta and having a population of about 20,000. The epidemic began in September in which month 232 cases were reported, with 1,203 in October, and 510 in November. The incidence of the disease in Gozo is shown month by month in the following table:—

The Incidence of Influenza in Gozo in 1918.

—	Influenza.		Pneumonia and Broncho-pneumonia.		Total Cases and Deaths due to the Epidemic.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
September -	232	3	2	1	234	4
October - -	1,203	39	31	2	1,234	41
November- -	510	32	53	2	563	34
December†- -	100	6	—	—	100	6
Total -	2,045	80	86	5	2,131	85

* Preliminary report on the influenza epidemic in Malta by Dr. A. Critien, Chief Government Medical Officer.

† From 1st December to 10th only.

Calculated on these figures, the case mortality rate in Gozo was 4 per cent.

Epidemic Influenza in the Population under Military Command at Malta.—The population under military command at Malta is estimated at about 14,500, made up of the garrison averaging 7,400 troops, patients of the Mediterranean Expeditionary Force in the 11 war hospitals in Malta, averaging about 4,000 for the period in question; the remainder include the Royal Air Force. Details: Awaiting Passage Depôt, Prisoners of War (2,100 in number), women and children, Red Cross personnel, and women employed by the Army and the Navy.

According to a report by Major G. R. Bruce, R.A.M.C., Specialist Sanitary Officer Malta Command, influenza began to appear among the ships in Malta harbour towards the end of May and beginning of June. The prevalence that followed is divided into two phases, the first extending from June 8th to August 8th, and the second from August 14th to November 30th. Altogether 4,192 cases were reported, 696 cases and 1 death occurring in the first period and 3,496 attacks with 66 deaths in the second. The first outbreak was of a very mild character with only one death, but the second was of a more serious nature. It began, apparently, from the importation of a fresh strain of the infection by a soldier, suffering from a surgical complaint, admitted to the St. Elmo Hospital from a transport vessel. Before it was recognised that he was also suffering from influenza, and before he could be isolated, he had infected other patients to the number of eight, as well as five of the staff. He had broncho-pneumonia, and 13 of the 33 others he had infected developed pneumonia, including one of the medical attendants. Three of the cases terminated fatally, including one of the orderlies. An outbreak occurred at the Polverista Barracks among the 1st Garrison Battalion of the Northumberland Fusiliers, 600 in number. The first case was reported on August 20th, and up to the end of that month 102 men were attacked. During September, to the 23rd, about 10 cases were reported daily, and altogether, up to November 30th, a total of 427 cases came under observation in this Battalion; there were 12 deaths out of 22 who developed septic pneumonia. The men were generally of a low category, being mostly above the usual military age. Their work was monotonous and arduous, including the duty of guarding the prisoners of war, among whom 551 cases of influenza were reported, 28 developing pneumonia, of whom 15 died. The Polverista Barracks were old and very badly ventilated, and this probably helped in spreading the infection.

As regards the question of second attacks, Major Bruce states that relapses in the second week of the illness were fairly common, but authenticated attacks some time after the

first illness were uncommon. So far as has been ascertained, out of over 4,000 cases only 12 have had two attacks at an interval of over six weeks, and of these only two came from units which had influenza in its mild form five months ago. With due reservations, Major Bruce says: "On the whole, "one attack of influenza, even though of the mildest "character, has protected the individual, with few excep- "tions, during a period in many cases amounting to five "months."

The measures employed by the military authorities in Malta to control the epidemic included early notification of cases, isolation of those attacked, special attention being directed to pneumonia cases. Picture palaces and theatres were put out of bounds during the worst part of the epidemic, and military schools were closed for a period of five weeks. Troops disembarking were medically examined and kept under surveillance for four days, so as to detect early cases of influenza. Prophylactic vaccines were not used.

Influenza at the Royal Naval Hospital, Malta.

The following table is taken from a report by Surgeon-Captain G. T. Broatch, R.N., presented to the Lieutenant-General of Malta, dated 11th January 1919 :—

Cases treated at the Malta Royal Naval Hospital.

1918.	Influenza.		Broncho-Pneumonia.	
	Cases.	Deaths.	Cases.	Deaths.
January	5	—	—	—
February	1	—	—	—
March	5	—	—	—
April	5	—	1	—
May	156	—	5	—
June	167	—	2	1
July	44	—	3	1
August	74	—	—	—
September	480	—	3	1
October	289	—	45	27
November	146	—	11	4
December	126	—	22	9
Total	1,498	—	92	43

Prior to May, only sporadic cases of influenza were admitted to the Royal Naval Hospital, but from May onwards influenza

appeared in epidemic form among the ship's companies of the war vessels working to the westward and calling at Bizerta (Tunis), where the infection had already appeared among men of the French Navy. Shortly afterwards cases occurred in vessels that had called at Gibraltar, and a little later influenza appeared on a British warship, the "*Isonzo*," running to Taranto, the Italian Naval Base, where the infection had probably been received.

There was a great increase in the number of cases during September, in which month 480 attacks occurred; this coincided with a widespread infection among the dockyard employees who were working ashore or engaged in repairing ships. Of the 289 cases in October, about half the number came from H.M.S. "*Cæsar*," which had recently arrived at Malta from Bermuda, on board which vessel, at one time or another, no fewer than 400 attacks were reported, many of them being treated on board or in temporary sick quarters on shore, apart from the Royal Naval Hospital.

Most of the cases in November and December came from ships engaged in patrol or escort duties in the Eastern Mediterranean.

During the last three months of 1918 recovery was retarded by the unfavourable weather conditions, namely, cold winds and damp.

On the whole, the epidemic was not of a virulent kind; 1,498 cases were mild, and in 92 there was broncho-pneumonia, 78 of which occurred during the last three months of the year. Surgeon-Captain Broatch believes that two distinct infections were present (1) an influenzal infection of a mild type, and (2) a pneumonococcal infection of a more virulent kind. In practically all of the fatal cases a postmortem examination was made, and confluent broncho-pneumonia was invariably present. Cultures from the lung substance, from the bronchial secretions, from the spleen or from the heart blood, showed the presence of the pneumococcus in pure cultures in almost all cases, the exceptions being cases where staphylococcus aureus, streptococcus longus, microcatarrhalis, and a bacillus indistinguishable from B. Pfeiffer, were found in mixed culture with the pneumococcus and pneumobacillus of Friedlander. When malaria co-existed with influenza, it was regarded as a most unfavourable factor and indicative of a bad prognosis.

In March 1919 a statement appeared in the press that a recrudescence of the influenza epidemic had occurred in Malta on a somewhat serious scale, and that this return of the disease had been traced to the carnival festivities, which had led to overcrowding in public places of assembly or entertainment. From reports recently received, there occurred 1,135 influenza

cases in Malta during April 1919, but no other details have come to hand.

Summarising the incidence of influenza and its mortality in Malta and Gozo during 1918, there appear to have been a total of 17,473 cases and 783 deaths, of which 9,652 cases and 588 deaths occurred among the civil population of Malta; 2,131 attacks, of which 85 were fatal, among the inhabitants of Gozo; 4,192 cases among the population under military command, with 67 deaths; and in the Royal Naval Hospital, 1,498 cases and 43 deaths. Reckoned on the total figures, the case mortality rate of influenza in Malta in 1918 was 4·5 per cent., the highest fatality, 6 per cent., being among the civil population, and the lowest, 1·6 per cent., among the military.

Switzerland.

Influenza is one of the diseases of which the notification is made compulsory by law in Switzerland, and the annual reports of the Swiss Central Health Department show that, in 1914, 75 such cases were notified, 228 in 1915, 229 in 1916, and 205 in 1917. It is believed in Switzerland that in 1918 a new strain of infection of influenza had been imported, quite distinct from the endemic form previously reported. Usually this endemic form of influenza manifests itself in January, February, and March of each year, and practically no cases are reported during the summer months. What happened in July 1918 was therefore quite unusual. Sporadic cases of influenza were notified during the first half of 1918, the total number for that period being 34, 2 of which were reported in January, 4 in February, 6 in March, 13 in April, 7 in May, and 6 in June. But in July the number suddenly rose to 53,688, followed by 41,626 in August. The disease continued to be epidemic to the end of the year, the highest number of notifications being received in October, namely, 263,399, the total for the six months being 664,420. The prevalence persisted during the first half of 1919, but in gradually decreasing amount, the notified cases in January being 36,019, and by April the number had fallen to 7,825. The mortality statistics of Switzerland for the year 1918 have not yet been published, but a statement has appeared in the press that the deaths from influenza in Switzerland in 1918 numbered 21,848. Calculating the fatality of the disease on these figures and the 664,463 notifications given above, the case mortality rate is only 3·3 per cent. There were two separate and distinct epidemic waves of influenza in Switzerland during 1918, the first with its crest in July, and the second with its height in October. The following table gives the number of cases reported in Switzerland during each of the last six months of 1918.

Influenza in Switzerland in 1918.

	Notified Cases.		Notified Cases.
July - - -	53,688	October - -	263,399
August - - -	41,626	November -	159,413
September - -	41,672	December -	104,622
3rd quarter of 1918 -	136,986	4th quarter of 1918 -	527,434

The corresponding figures for the first three months of 1919 were: January 36,019, February 22,730, and March 16,160; total for the first quarter of 1919, 74,909.

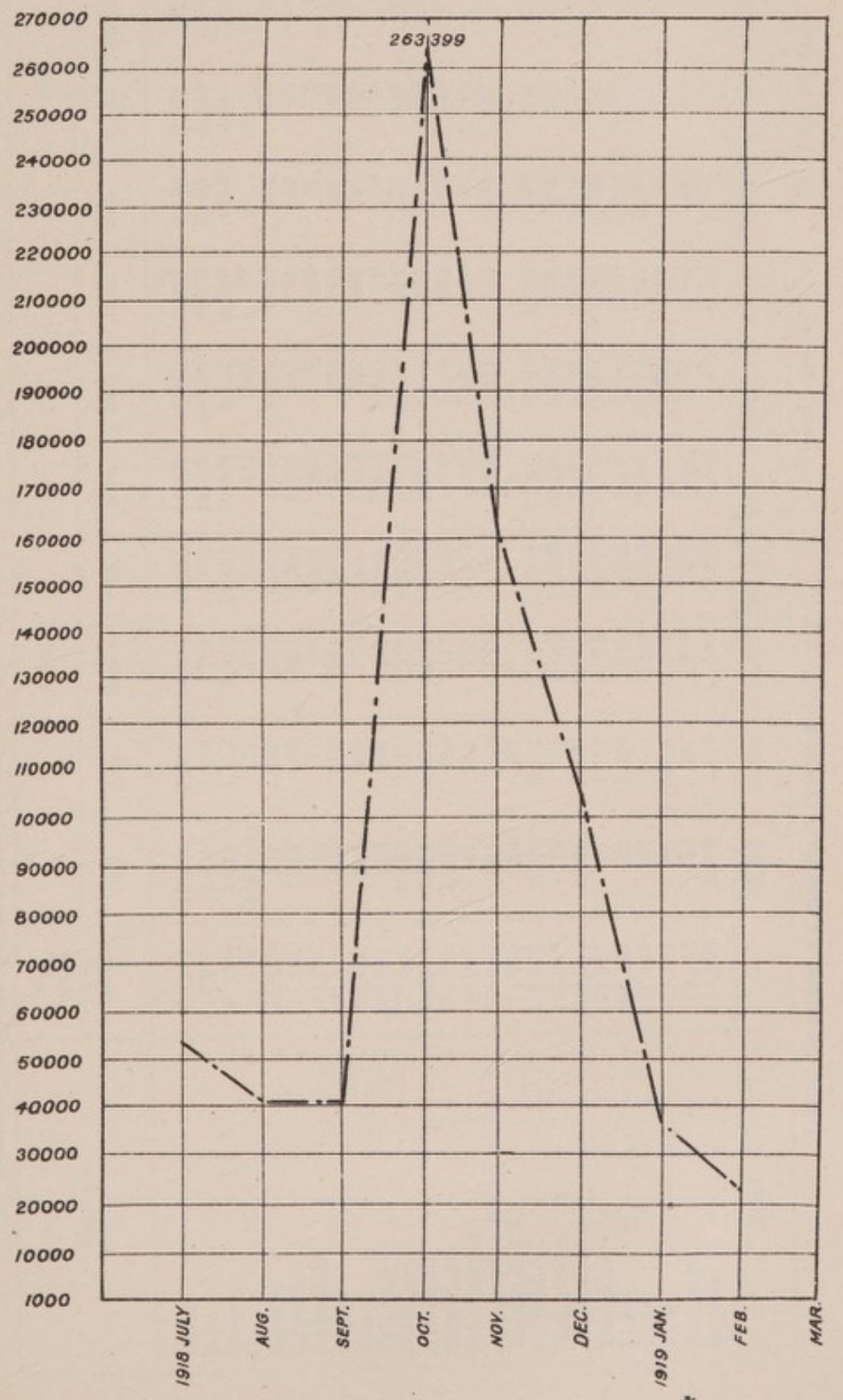
By the end of March 1919 the epidemic had practically ceased; the notified cases in April were 7,825, in May 1,041, and in June only 388.

In the following table is shown the distribution of the influenza cases in the various Swiss Cantons from July 1918 to 30th June 1919:—

To face p. 250.

SWITZERLAND.

INFLUENZA.
MONTHLY INCIDENCE.
NOTIFIED CASES.



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Cases of Influenza notified in the Swiss Cantons from July 1st, 1918, to June 30th, 1919.

Cantons.	Population, 1910.	1918.						1919.					
		July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	April.	May.	June.
Zürich	503,915	13,451	7,031	4,134	41,355	17,240	12,390	2,988	2,162	1,878	802	85	80
Bern	645,877	14,530	8,879	1,704	22,730	29,362	23,388	9,670	6,039	3,555	1,636	249	64
Luzern	167,223	1,213	1,713	3,320	14,421	10,804	6,923	3,263	947	2,091	1,226	169	8
Uri	22,113	Epidemic	112	326	1,570	544	119	79	—	—	—	—	—
Schwyz	58,428	534	2,092	518	6,061	2,925	2,011	496	204	133	85	8	5
Ofwalden	17,161	185	165	224	2,305	2,041	482	61	139	76	5	—	—
Nidwalden	13,738	40	71	38	1,334	367	208	37	89	5	2	1	—
Glarus	33,316	106	684	851	1,880	1,450	935	240	331	64	61	18	3
Zug	28,156	234	550	776	2,537	1,654	832	307	311	194	11	2	2
Fribourg	139,654	899	559	1,445	7,201	6,815	2,019	79	19	18	5	4	3
Solothurn	117,040	5,460	543	1,252	8,756	5,406	2,638	1,512	1,033	624	323	27	—
Baselstadt	135,918	2,672	1,477	1,545	15,031	4,652	4,804	1,849	1,590	1,410	564	188	192
Basselland	76,488	1,306	420	1,117	6,534	4,302	1,846	855	407	327	71	23	4
Schaffhausen	46,097	41	712	773	3,913	2,012	1,681	400	21	18	—	—	—
Appenzell-A-Rh.	76,488	828	953	776	4,184	1,730	1,349	403	348	234	18	3	—
Appanzell-I-Rh.	14,659	50	282	—	—	—	—	—	—	—	—	—	—
St. Gallen	302,896	3,106	6,403	7,300	26,949	12,831	8,137	2,018	763	399	110	5	2
Graubünden	117,069	12	774	2,185	14,320	10,556	4,546	1,505	407	644	852	3	—
Aargau	230,634	3,001	2,384	2,741	22,183	9,803	6,724	2,853	2,100	1,810	1,167	60	—
Thurgau	134,917	737	1,157	2,099	10,106	5,734	3,354	882	787	293	97	22	3
Ticino	156,166	435	1,858	848	3,900	6,236	3,291	1,474	2,144	1,691	564	90	11
Vaud	317,457	106	—	5,239	25,319	13,541	10,055	1,629	1,501	680	118	62	—
Valais	128,381	480	Epidemic	174	3,117	2,404	1 831	670	175	135	21	—	—
Neuchatel	133,061	432	237	1,704	7,786	3,449	3,750	1,493	256	116	56	4	—
Geneva	154,906	3,830	2,570	583	9,907	3,555	1,309	1,256	957	365	31	18	11
Total	—	53,688	41,626	41,672	263,399	159,413	104,622*	36,019	22,730†	16,160‡	7,825	1,041	388

* Total for second six months of 1918 = 664,420 cases. † Later, 22,739. ‡ Later, 16,200.

The total deaths from influenza reported in Switzerland during 1918 amounted to 21,846 ; but the deaths, if any, caused by the disease during the first half of 1919 have not yet been published.

The following table gives for each of the Swiss Cantons the number of notified cases of influenza during 1918 ; also the number of deaths and the Death rate per 1,000 from the disease in each canton :—

Canton.	Influenza.		Case Mortality Rate per Cent.	Influenza Death Rate per 1,000 Population.
	Cases.	Deaths.		
Zürich - - - -	95,601	2,370	2·47	4·24
Bern - - - -	100,596	4,383	4·35	6·32
Luzern - - - -	38,394	954	2·48	5·52
Uri - - - - -	2,681	182	6·79	7·46
Schwyz - - - -	14,131	301	2·13	4·85
Obwalden - - - -	5,402	144	2·66	7·87
Nidwalden - - -	2,058	86	4·18	6·07
Glarus - - - -	5,916	176	2·97	5·10
Zug - - - - -	6,583	194	2·94	6·36
Freibourg - - - -	18,938	842	4·44	5·76
Solothurn - - - -	24,055	838	3·48	6·55
Baselstadt - - -	30,181	745	2·46	5·20
Baselland - - - -	15,525	430	2·77	5·22
Schaffhausen - - -	9,131	255	2·79	4·74
Appenzell-A-Rh. - -	9,820	239	2·43	3·92
Appenzell-I-Rh. - -	332	78	23·55	5·13
St. Gallen - - - -	64,726	1,436	2·22	4·65
Graubünden - - -	32,393	781	2·41	6·41
Aargau - - - -	46,836	1,158	2·47	4·75
Thurgau - - - -	23,187	606	2·61	4·42
Ticino - - - -	16,568	925	5·58	5·69
Vaud - - - -	54,260	1,928	3·55	5·70
Valais - - - -	8,009	998	12·46	7·12
Neuchatel - - - -	17,358	842	4·85	6·23
Geneva - - - -	21,754	955	4·39	5·53
Total -	664,435*	21,846†	3·29	5·46

* A later report gives the total number of reported influenza cases in Switzerland during 1918 as 664,420.

† Another table published in the Swiss Official Bulletin for November 29th, 1919, gives the total deaths due to the epidemic as 21,689, of which 18,349 died from pneumonia complications.

Of the 21,846 deaths, 12,913 were males and 8,933 females ; the fatal incidence on males was, therefore, much greater than on females. Among the males about one third of the deaths occurred in the age group 20 to 50 years ; and for the female sex this proportion was about two thirds.

In an official report, published in the *Bulletin des Schweizerischen Gesundheitsamtes*, for August 9th, 1919, it is remarked that the mortality in the age group 20 to 50 years was without

doubt a consequence of the mobilisation; the death rate was one and a half times greater in the male sex than in the female (6·56 per 1,000 in males, against 4·4 in females). Under 15 years of age the influenza mortality was lower; and the proportion reversed, the death rate being 2 per 1,000 in females, against 1·7 per 1,000 in males. The death rate from the epidemic disease for the whole of Switzerland in 1918 was 5·46 per 1,000. If the estimate of 2,000,000 of attacks already mentioned be accepted, then the case mortality rate for Switzerland was only 1·1 per cent. (or 1 death in 91 cases), but as shown in the table above, if only notified cases are accepted then the fatality rate was 3·29 per cent.

The largest numbers of cases reported during the second half of 1918 in the separate cantons were 100,593 in Bern, 95,601 in Zürich, 64,726 in St. Gallen, and 54,260 in Vaud. But the number of cases which occurred during the epidemic is not represented by the notification figures. It is asserted that for every case notified there were two not notified, so many persons were not medically attended. It is estimated that up to the end of 1918 about 2,000,000 of people in Switzerland were attacked by the epidemic malady, or about half of the population of the country.* During the first half of 1919 about 500,000 persons were attacked by the epidemic; in July there were 239 cases, in August 119, and in September 183. In October the number rose to 536.

According to the *Bulletin du Service Swisse de l'Hygiene publique* for 13th July 1918 the so-called Spanish "grippe" began to be prevalent in Switzerland towards the end of June, though failure at first to recognise the epidemic nature of the malady probably delayed its notification. It is now generally held that the disease, epidemic in 1918, was identical in its character with that which swept over the world in 1889–94. The first wave in 1918 was of a mild nature. Some medical authorities have made the suggestion that this epidemic represented only a revival in virulence of the infection which had been showing itself in Switzerland and other countries since 1889, and was not due to a fresh importation of infection. The Swiss Army was seriously affected by the epidemic in July 1918. It was stated in the press that on the evening of 15th July the number of sick in the Army was 6,892, of which 95 per cent. were due to influenza. From 15th July to 26th July the total number of influenza cases in the Swiss Army was 11,550, of which 305 were fatal, and by the end of the year the total number of fatal military cases had risen to 500. It was found, as a matter of experience, that soldiers ill of influenza could not stand removal to any great distance. It was said that the spread of influenza among the troops on the

* Bericht des Schweizerischen Gesundheitsamtes über seine Geschäftsführung im Jahre 1918.

Swiss frontier was greatly assisted by the fact that the soldiers were lodged in dirty and overcrowded billets. Medical men formed a relatively high percentage of the victims of epidemic influenza in Switzerland, especially among the young practitioners attached to the hospitals, or those attached to military units. The summer of 1918 in Switzerland was hot and dry, and this is said to have been favourable for the development of the epidemic. The epidemic attained its height in the eastern cantons from one to two weeks later than in the western cantons. and from this it is suggested that the infection entered Switzerland from the west, and not from the east. In the more virulent wave of influenza that began in October, pneumonia became a frequent complication, and in many cases empyema developed. The disease was exceptionally fatal among pregnant women.

It has been suggested that a fresh strain of the influenza infection was introduced, in the autumn of 1918, by the prisoners of war from Germany, brought into Switzerland to be exchanged.

Some of the Swiss medical experts were inclined from their experience to infer that an attack of epidemic influenza conferred some immunity upon the individual against subsequent attacks. Some said that the whole epidemiology of influenza speaks for at least a partial immunisation by the pandemic, and the growing up of a new and susceptible generation. The occurrence of cases of catarrhal fever, mistakenly termed influenza (or grippe), has helped to prevent the due realisation of the actual immunity caused by an attack of true influenza.

As regards prophylactic measures, Sahli, of Bern (in the *Correspondence Blatt für Schweizer Aerzte* for 15th February 1919) states that there were so many objections to the employment of masks that their use was not regarded as practicable. None of the commercial masks tested were found impervious to germs. He suggests that it would be better if the patient wore the mask to prevent his infection reaching others; but it is admitted that this might not be without harm to the patient, whose respiration, already hampered by the nature of the illness, would be further obstructed by the mask. Medical men are particularly liable to contract the influenza infection while auscultating the patient; it is therefore necessary that they should be protected either by a mask or a temporary screen (a towel) held between them and the patient. The Government issued a decree to the canton authorities granting financial help in the carrying out of measures for combating influenza, *e.g.*, for the provision of temporary hospitals, the employment of nurses, and the payment of fees to medical men for notification of influenza cases. Later, another decree was issued authorising payment of money to persons deprived of their means of existence by the closing of establishments and businesses in which they had been employed, when such closing

had been ordered for the purpose of preventing the spread of influenza.

About the commencement of the Swiss epidemic of 1918 the American Red Cross voted a sum of 100,000 dollars to provide better accommodation for sick soldiers, a large hotel being secured for convalescents at Interlaken. In some districts the canton authorities forbade the holding of meetings or entertainments, and the visits of friends and relations to hospital patients were stopped for a time until the epidemic had declined.

Italy.

At the time of writing no official statistics concerning the incidence and mortality from the recent epidemic of influenza in Italy have come to hand, so that what is known has mostly been gathered from lay and medical journals. As in most other European countries there were three waves of influenza in Italy. The first began at the end of April 1918, and reached its height in the last week of May and first week of June, from which time the incidence declined, and by July the disease had almost disappeared. This first wave was characterised by the mildness of the disease and the comparatively small mortality that resulted. Many of those attacked during this period suffered only slightly, and numbers of them did not seek medical assistance. In September the second wave began, and this time the cases assumed a graver character, the infection spreading to nearly every one of the 69 provinces of Italy. It was especially severe in the province of Palermo, appearing in military camps as well as among the civil population. The part of Italy which suffered most was the middle portion of the kingdom and Sicily. In Palermo, during the week ended 25th September there were 962 deaths attributed to the disease, and it is said that in the month of September about 150,000 persons were attacked. In the battle ground of the Piava, influenza became epidemic among the troops shortly before the military operations began in the latter part of October and added somewhat to the difficulties of the advance against the Austrians. Among the Italian authorities there was a natural tendency to conceal the ravages made by the epidemic in various provinces, on the plea that knowledge of what was regarded as bad news would prove alarming to the people generally and render them more susceptible to the prevalent infection. It was also asserted that the effects of the epidemic had been far more serious than had been allowed to appear in the public press. In Rome, it is said, during the height of the second wave, the mortality reached a figure exceeding 200 a day. In the province of Bari upwards of 7,000 deaths from the epidemic were reported. A deputy in December asserted in the Italian Parliament that the epidemic in Italy had carried off about 800,000 persons, but probably this was an exaggeration. This statement was repeated in

1919 in the *Seculo*, of Milan, which added that this number was 60 per cent. higher than the total Italian mortality in the war. It was also mentioned as an example of the ravages of the epidemic that in one village which had lost 62 men in the war, nearly 150 persons died of epidemic influenza in the late autumn of 1918. In Leghorn, with a population of 106,000, in a period of five weeks, there were 200 deaths from influenza. At the beginning of 1919 a third epidemic wave developed, but no details of this have as yet come to hand.

There was a noticeable mortality among medical men attending influenza patients in Italy, and among these victims were some British medical officers attached to the troops. Dr. Morelli, Professor of Medicine in the University of Pisa, died in October as the result of infection contracted in the course of a bacteriological research on influenza.

The subject of immunity conferred by an attack of influenza against subsequent attacks was much discussed in the Italian medical journals. Dr. Vincenzo stated that at his hospital at Spezzia, in which a large number of soldiers suffering from influenza were treated during the first epidemic wave, all his staff contracted the disease, whereas in the more severe prevalence of September those of his staff who remained, and who had suffered in the outbreak of May, entirely escaped. In Dr. Vincenzo's view, in influenza, as in scarlatina and measles, one attack usually confers immunity. He does not, however, express any opinion as to the duration of this immunity of influenza.

Dr. Sacoma testifies to the immunity enjoyed in the winter epidemic by persons who had suffered in the summer outbreak. Dr. Artom asserts that none of his patients attacked by influenza in May or June suffered again from the disease in September or October, although exposed to the infection; even abortive attacks in May and June seemed to confer immunity. He says that this immunity is not established till after the lapse of from 20 to 30 days from the initial attack.

As regards quinine prophylaxis of influenza, some observations were made in hospitals where malaria patients were specially treated.

Dr. Betti relates that, among 1,000 soldiers, in a hospital near Lake Como for malaria, undergoing intensive quinine treatment, only five of them contracted influenza, and that in a mild form. The majority of these soldiers were in a bad state of health, and, therefore, presumably more likely to fall victims to a current infection. Dr. Roccavilla, who was in charge of a military hospital for malaria at Ravenna, relates a similar experience. Out of 1,200 soldiers suffering from malaria and receiving daily doses of quinine only 9 or 0·75 per cent. contracted influenza, which, however, was of a mild character. But he states that small doses of quinine, *i.e.*, 3 to 7 grains daily afford no protection against influenza. On the other hand,

Dr. Colale asserts that out of 118 malarial cases under his care in a military hospital, 115 contracted influenza, 50 of them developing broncho-pneumonia, of which 42 of them died. All of the 118 men in his hospital were undergoing intensive quinine treatment for malaria, and, therefore, if that drug is to be regarded as a prophylactic for influenza, these men should not have contracted that disease. Dr. Sacoma states that the administration of quinine under rigorous supervision among troops for malaria did not, in his experience, prevent an outbreak of influenza among them or reduce the mortality from the disease.

The use of masks was advised by the military medical authorities to doctors and nurses while at work in wards where influenza cases were being treated. During the more serious outbreak in September and October patients suffering from pulmonary complications were isolated in hospitals by themselves, apart from the less serious cases. In the larger towns cinemas and other places of entertainment were closed on the ground that they acted as dangerous diffusion centres for the influenza infection. There was a great scarcity of nurses as well as of medical men, owing to so many being engaged on active service, and in many instances the available hospital accommodation was inadequate.

The course of the influenza epidemic of 1918 in Italy was outlined in the *Policlinico* (Vol. XXV., fasc. 43), by Professor Lutrano, as follows:—The disease appeared first about the end of spring. From the end of April until June it was present in many places in a mild and often unrecognised form. During April numerous cases were treated at the Collegio Principio di Napoli at Assise. In May the malady spread seriously among the employees of the steel works and other establishments in Terni. It spread also in Domodossola, especially in the prisons, and at Spezzia among the troops and workers in the arsenal, as also among the civil population of the provinces of Modena, Plaisance, Verona, and Pisa. In June it was present in all the communes of the province of Bari and at Taranto. Cases of influenza were also reported among the Italian soldiers at the front, mostly in mild form. This constituted the first phase of the epidemic. The beginning of the second phase was observed in Calabria, where there was noted a tendency of the disease to spread in a graver form than before. It then appeared at Rosarno in the province of Reggio Calabria, extending later to the province of Catanzaro. The infection then appeared in Messina, attacking a large proportion of the population. After this, other provinces were invaded, including Aquila, Parma, Cosenza, Caserta, and Chieti; also at Turin, Alexandria, in Liguria, and Palermo. During September influenza continued to spread until almost every part of the kingdom had been affected. Campania, Genoa, Naples, and Avellino were gravely attacked, and in somewhat less degree Benevento and Salerno. The malady spread over

the island of Sicily, affecting all its provinces. It appeared in a severe form in the provinces of Foggia and Bari. The declining incidence of the epidemic was first observed in Southern Italy, though in Northern Italy the disease continued at the same time to show itself in a graver form in Latium, Abruzzi, Piedmont, and Lombardy, and in a rather milder type in Liguria, Emilia, Tuscany, and the Marches. The cases in Venetia were nearly all of a sporadic nature.

In the city of Bologna with a population of over 170,000 and situated 82 miles from Florence, influenza was very prevalent during the last three months of 1918 during which period about a thousand people died from the epidemic disease. Professor N. Samaga in the *Bull. delli Scienzes Med.* for February 1919, states that in the military hospital, to which he was attached, 4,190 soldiers and Government officials were admitted during October and November; of these 554 or 13 per cent. died. He draws attention to the frequency of empyema as a complication of influenza in the autumn epidemic of 1918 at Bologna.

The bacillus of Pfeiffer was isolated from cases by Professor Di Vestea of Pisa, Professor Canalis of Genoa, and Professor de Giaxa of Naples. At Palermo a Scientific Commission nominated by the Minister of the Interior, isolated this same bacillus in some cases. From this and other evidence it was generally accepted in Italy that the disease which had been so widely epidemic in 1918 was true influenza and nothing else.

Baccarini believes that influenza is a polybacterial epidemic, no one germ being responsible for it. The organisms which cause the disease have had their virulence suddenly enhanced by some mysterious influence "electric, atmospheric, or telluric, "some sudden modification of the physical and chemical am- "biant in which mankind lives." Besides bacteria, the pathogenic factor is probably a complex of many minute causes, most of which are beyond our ken.*

Albania.

There has not been much information obtainable as to the influenza epidemic which occurred during 1918 in Albania, but from French sources a report has been issued respecting an outbreak of the disease at Koritza, or Gortcha, where during May, June, and July a large number of cases occurred. Many of the cases were mild, running a short course. A second wave was reported in September and October, attacking many of the troops in that region. The severe cases were treated in the French divisional ambulance, and 433 were studied clinically and bacteriologically; they were divided into four classes, namely, (1) those with acute generalised bronchitis; (2) those with congestion of one or both lungs; (3) those with broncho-

* *Journal of the American Medical Association*, June 1919.

pneumonia in multiple foci, and (4) those with "spleno-pneumonia."

Of a total of 1,188 cases treated in the French divisional ambulance at Koritza during September and October, 280, or 24 per cent. died.*

Greece.

Influenza appeared suddenly in Greece during May 1918, but the cases at first were mild and the illness soon passed off. The infection was also present in May and June in the Allied army in the Balkans, and as it is well known that "three days fever" is endemic in the country, and that the phlebotomus insect is also present in many localities, it was assumed that the current malady was phlebotomus fever. But it soon became apparent that this view was incorrect, and that the disease was epidemic influenza.

In the French army in Macedonia the epidemic of May and June was said to have been characterised by its sudden appearance, its rapid extension, its extremely infectious nature and the predominance of the thoracic symptoms. Bacteriological examination showed the presence of non-specific organisms, streptococcus, pneumoccoccus, and M. catarrhalis.

In September there was a second wave of influenza in Greece, but of a far more serious kind, many of the cases being complicated with broncho-pneumonia. It is stated that about 50 per cent. of these complicated cases terminated fatally. So rapidly fatal were some of the cases that the people believed that the prevailing malady was plague or cholera, and at one time great alarm seized upon the inhabitants. The second phase of the epidemic continued until December. In a report by Dr. T. G. Filtzos, inspector for the American Consulate General, dated January 16th, 1919, it is stated that the general mortality in Athens and the Piræus was greatly increased. In Athens the disease became prevalent in August, and out of 9,613 deaths from all causes 108 were certified as due to influenza. In Athens and the Piræus the deaths from all causes and from influenza, September to December 1918, were as follows:—

1918.	Death from all Causes.	Deaths from Influenza.
September	771	115
October	1,430	605
November	1,515	670
December	771	229

The age group most affected was that from 20 to 45 years. The injection of a solution of corrosive sublimate was tried to

* *Bulletin de l'Académie de Médecine*, 21st January 1919.

stop the course of the illness, but was soon abandoned owing to the bad results obtained. Later colloidal metals such as colloidal silver or electrargol, injected subcutaneously or intravenously, were tried with apparently good results. Treatment by vaccines, however, was not attempted at all.

Among the general measures used to control the spread of the disease were the closure of public and private schools, and of theatres and cinemas. Instructions were issued urging the people to avoid crowded places and tramcars, and to take to bed as soon as any of the initial symptoms of influenza appeared.

At Patras, the presence of a malignant form of influenza was reported towards the end of October, and during the week ended 3rd November, 214 deaths were recorded from the epidemic. At Salonica and at Kalamata influenza was also prevalent from September to December. During that period 1,164 deaths from influenza occurred at Salonica.

Among the medical men who died of the disease at Salonica were several British Army medical officers; and, at Corfu, Dr. Tribondeau, medical officer of the French Naval Hospital, died there from influenza contracted in the discharge of his duties.

Serbia.

Owing to the disturbed state of Serbia during 1918 little direct information has come to hand respecting the epidemic of influenza, which swept over that country during the year. From accounts published in the press it appears that the disease was widely prevalent in the last quarter of 1918 and that "thousands had succumbed to it." The sufferings of the Serbians were greatly increased by the fact that the Germans and Austrians in their retreat had carried away, or completely destroyed beds and bedding belonging to the people; the enemy had also pillaged the hospitals, so that neither beds, sheets, nor blankets were left in any of these institutions. For the same reason medicines of all kinds were wanting, and it was found impossible to establish in the Serbian towns any hospital, even of the most modest dimensions, for the reception of influenza cases. The Croats hearing of the sufferings of the Serbians from the epidemic sent to Belgrade, towards the end of the year, 32 medical men, 10 medical students, and a number of nurses and hospital attendants; and further offers were made by some sympathetic Croatian towns, including Zagrob and Ossiek, to provide "complete" beds for those suffering from the epidemic; the two towns just mentioned offered each to send immediately 2,000 beds with bedding, and these, at the time the press report was published, were being despatched as quickly as possible to the stricken Serbians.

Bulgaria.

No reports have been received from Bulgaria respecting influenza, and the only information as to the epidemic in that country during 1918 is contained in a brief paragraph in the *Times* of 8th November, which states that the Bulgarian army had suffered considerably from the prevailing epidemic of influenza. The collapse of the Balkan campaign in the autumn of 1918 and the subsequent political disorganisation that followed the surrender of Bulgaria in the field, no doubt seriously interfered with the preparation and publication of any official statistics concerning the recent influenza epidemic among the civil and military populations of that country.

Cyprus.

An influenza epidemic appeared in the island of Cyprus in the early autumn of 1918. The total number of influenza cases treated in hospitals and dispensaries during the year was 4,693, in a population of 306,997, but the number of deaths has not as yet been published. Influenza, however, seems to have been present in the island during 1917, for 3,144 cases were treated during that year in the hospitals and dispensaries.

Austria.

Owing to the conditions brought about by the war, the transmission of reports from Austria to this country was stopped, and consequently there is a lack of detailed information respecting the waves of influenza which occurred in that country during 1918. From foreign sources, and chiefly from reports in the journals of neutral countries some facts have been gathered, but these relate mostly to the incidence of the disease in Vienna. Towards the end of 1917 a "grippal," or influenza outbreak, occurred in Vienna and spread through Lower Austria, but whether it was the same disease as that which became epidemic during 1918 is not known. It is said that although search was made Pfeiffer's bacillus was not discovered in the cases investigated during the latter part of 1917. The first epidemic wave in 1918 appears to have been recognised in that city about the beginning of July, when there was a marked increase in the number of deaths ascribed to influenza and pneumonia. The average weekly number of such deaths prior to July had been from 40 to 50, the deaths from all causes being from 700 to 800 a week in a population of 2,200,000. In the second week of July the deaths from influenza and pneumonia rose to 123, and from this time they decreased almost to the normal number. The first wave was

comparatively mild in character and the deaths were relatively few. But in September the second wave began in Vienna, and in the week ended 21st September the fatal cases of influenza and pneumonia numbered 121, and in the following week 227. In the week ended 5th October there were 364, and in that ended 19th October they reached 1,468, the total deaths from all causes in these last two weeks numbering respectively 1,753 and 2,607. The death-rate from all causes in Vienna reached its highest point in the week ended 19th October, when it was as high as 58·6 per 1,000. From 1st September to 19th October the deaths attributed to the epidemic were 3,125. It was calculated in Vienna that the total number of persons attacked by the influenza infection was about 180,000. A considerable proportion of the fatal cases belonged to the age group 20 to 30 years. It was stated in a medical journal that of 14,000 tramway employees in Vienna 2,500, or about 18 per cent., contracted the disease between mid-September and mid-October. The female members of the staff, conductresses and such like, were most affected, most of them being in the age group 20 to 40 years. It was remarked that "it is impossible to imagine a "larger collection of human beings in a smaller space than the "over-filled interiors of the cars in which these conductresses "were working." The male staff was much less affected, but their numbers were less owing to the younger men being away on military duty.

In consequence of the number of influenza cases in mid-October among the railway employees, the Austrian Ministry of Railways suspended entirely for three days the whole of the goods traffic. This unfortunately aggravated the troubles of the influenza-stricken city by stopping the supply of coal for about a week. The epidemic interrupted school attendances, and about one-fifth of the schools closed themselves through the absence of the scholars from the current illness. It is understood that there is no compulsory notification of influenza in Austria, so that the number of actual cases that occurred in Vienna is not known. In addition to the deaths certified as due to influenza, the fatal cases of pneumonia were three times more numerous during the height of the autumn epidemic than in normal times. Among those who were attacked by influenza in the latter part of the year were the ex-Emperor Carl and his family.

The subject of the behaviour of influenza, when it attacks malarial patients, received some attention, as it did in Italy, from the medical profession. Dr. Matko stated in a Vienna medical journal that the seriously high mortality of malarial patients who are attacked by influenza is undoubted. In a group of 67 cases attended by Dr. Leitner, 53, or 80 per cent., died. Dr. Matko mentions a group of 38 malaria cases complicated by influenza under his own care in a Vienna military

hospital; of these, 30, or 78 per cent., died, the course of the cases being precisely similar to those described by Dr. Leitner; they were characterised by heart failure and intense dyspnoea. In the malarial patients there was a high mortality in the age group 31 to 40 years. A large proportion of Dr. Matko's cases were under-nourished, weakly individuals. He attributes the unfavourable prognosis of influenza occurring in malaria cases to the fact that the functional activity of the liver and spleen, which organs play an important part in combating infection, has been injured by malaria. The heart muscle in such cases often becomes involved.

In Prague, which has a population of 376,374, influenza was epidemic from September to November 1918. There are no details available as yet regarding this outbreak except that it was at its height in mid-October, in which month at least 760 deaths were ascribed to influenza, and there were doubtless many others certified as pneumonia, broncho-pneumonia, and other respiratory diseases, but due to the prevalent influenza infection. So far as can be learned, about 1,100 deaths were attributed to influenza in Prague during the latter part of 1918, not including the deaths recorded from the complications of the disease. The general death-rate from all causes in the week ended 19th October reached the high point of 94·4 per 1,000 of the population.

There was a third wave of influenza in Austria reported about the beginning of 1919 in the public press, but details of it have not yet been received.

The above particulars are in all probability only a small fragment of the actual occurrences of influenza in Austria during the late pandemic, but the circumstances of the country were such during the year as to make any definite description of the outbreak impossible from the official point of view.*

Trieste.—Although Trieste has now ceased to belong to Austria, at the time the influenza epidemic was raging it still formed part of that now fallen Empire. It is, therefore, included for the purposes of this report under Austria. Trieste has a population of 244,655, and in 1918 the total deaths from all causes numbered 6,719, which was 1,535 more than the previous year and 2,606 more than in 1916. The fatal incidence of

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* Since the above was written it appears from an official report, "Die Änderungen der Tuberkulosehäufigkeit Österreichs durch den Krieg," by Dr. S. Rosenfeld, that the mortality of women in Vienna during the years 1914–19 was as follows:—

	Tuberculosis.	All other causes.
1914	2,635	12,588
1915	3,004	13,216
1916	3,543	13,326
1917	4,469	16,181
1918	4,729	19,006
1919	5,018	16,054

influenza became evident in September, when the deaths from all causes amounted to 503, as against 363 in August. During October the number of deaths registered from all causes rose to 1,374, and in November and December they were respectively 950 and 847. Of the total deaths (all causes) in 1918, 726 were certified from influenza, 725 from pneumonia, and 882 from "other respiratory diseases."

Although the weekly incidence of fatal influenza and pneumonia in Trieste has not been obtainable, the following figures taken from the annual report of the medical officer of health for the city show the effects of the epidemic on the monthly mortality returns :—

Deaths from all Causes in Trieste during 1918.

1918.	Deaths. All Causes.
January	506
February	396
March	414
April	367
May	363
June	327
July	309
August	363
September	503
October	1,374
November	950
December	847
Total	6,719

The appended chart, taken from the annual report of the medical officer of health for Trieste, shows the monthly mortality for the previous years and for 1918. From this it is apparent that the number of deaths in the last quarter of 1918 was far in excess of anything recorded in any of the four previous years.

Hungary.

The information available as to epidemic influenza in Hungary during 1918 is even more scanty than that for Austria. It was, however, stated in the London press early in October that over 100,000 cases of "Spanish influenza" had occurred at Budapest (population 1,031,256), the capital of the country. During the first nine months of 1918 there had been certified in Budapest, almost every week, a few deaths from "influenza"; but in October the number of such deaths increased, and the fatal prevalence of the disease persisted to the end of the year

and into the early part of 1919. The development of the epidemic can be seen in the following table, which gives the number of cases and deaths recorded from influenza from 1st September to 28th December 1918:—

The Incidence of Influenza in Budapest during the last Four Months of 1918.

1918. Week ended	Influenza Cases.	Deaths.
7th September	Not received	1
14th „	„ „	1
21st „	„ „	3
28th „	„ „	2
5th October	„ „	4
12th „	„ „	16
19th „	„ „	54
26th „	523	143
2nd November	5,577	266
9th „	5,436	481
16th „	3,533	536
23rd „	1,373	358
30th „	962	339
7th December	1,116	199
14th „	1,693	297
21st „	2,153	282
28th „	1,530	386
Total	23,896	3,368

Calculated on these figures, the case mortality rate was about 14 per cent., but the notification of the cases was incomplete, being wanting for the first seven weeks of the table, so that probably the fatality of the disease was rather less than this.

The fatality of the autumn epidemic in Budapest in 1918 reached its height in the week ended 16th November, and gradually declined until the first week in December, after which there was again an increase, and this continued till the end of the year and into the early weeks of January 1919. The number of cases for the week ended 4th January was 977, and the deaths 185, but for the week ended 11th January the cases were reduced to 451, the deaths being 224; and for the following week there were 274 cases and 200 deaths. After this the numbers rapidly decreased, and on 5th February the influenza deaths were only 28, and in the following week they had further fallen to 13. By the end of April there were only about four deaths a week from influenza in Budapest. Except that school closure was largely employed in Hungary to control the epidemic, there is little known as to the preventive measures adopted to stop the spread of influenza in that country.

Russia.

Russia is one of the countries in which influenza is endemic, and from it the recent epidemic, like that of 1889, is believed by some epidemiologists to have begun its progress across Europe. Russia forms only an intermediate area between Europe and Central China, which is said to be a home of the disease. Clemow* is of opinion that the area in which the pandemic of 1889 began its ravages lies on the borders of European and Asiatic Russia, from which influenza spread eastwards, westwards, and southwards. It has been suggested that influenza is endemic among the nomadic Kirghiz tribes inhabiting the steppes in the above-mentioned area. It is of interest to learn from Clemow that the Kirghiz themselves call influenza "the Chinese fever," although this Russian endemic area is more than 1,000 miles from the Chinese frontier. A distinguished French authority, Netter, states that remote parts of Asia and Russia are endemic centres of influenza, and that "just as cholera is endemic on the banks of the Ganges, "so is influenza in the interior of Asia, and its endemic and "epidemic domain is in the bordering countries, especially "Russia." Dr. Doty, formerly health officer for the port of New York, reported in the *New York Times* that "during an "official visit to Russia some years ago I found in the over-"crowding, poverty, and dirt a very satisfactory explanation "why the epidemic of influenza in certain sections of that "country involved over 75 per cent. of the population."

The information regarding the recent epidemic of influenza in Russia is very scanty, the revolutionary troubles and the consequent disorganisation of administrative control of epidemic disease in Russia during 1918 having prevented any official reports from reaching this country. It is, however, known that the disease was very widely prevalent throughout Russia during the fourth quarter of 1918, and very probably during the third quarter as well. In the Ukraine the infection was especially severe, and about mid-October the number of cases in Odessa (population over 500,000) was said to be 70,000. At the same time the malady was also prevalent in the city and district of Archangel, where it lingered until December. These occurrences can only be a fraction of what happened in Russia, and judging by past experience of Russian epidemics it is certain that many lives would be lost through influenza in Petrograd, Moscow, and other large cities. Owing to the Bolshevist régime no press accounts were permitted to be sent out of Russia to other Europern countries, and hence almost nothing has been learnt as to the mortality that has resulted

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* "Geography of Disease."

from the epidemic. There can be little doubt that the appalling conditions of poverty, privation, cruelty, and bloodshed in Russia during 1918, with the cessation of all sanitary administration, especially in the larger towns, the flight of thousands of terrified people from their homes, and the exposure to the rigours of winter without food or shelter, have afforded opportunities for the spread of epidemic influenza and the development of its many fatal complications among the unhappy inhabitants of Russia, the full extent of which will probably never be properly known.

Germany.

"Influenza" had been occurring in Germany prior to 1918. For example, in a recent report of the Bochum Administration of Sick Insurance it is mentioned that in that area during 1915 there were 9,117 cases of influenza, and that in 1916 the number rose to 12,788.

In a report published in the *Veröffentlichungen des Reichsgesundheitsamts* for 3rd December 1919, entitled "Ergebnisse der Todesursachenstatistik im Deutschen Reiche für die Jahre 1914, 1915, and 1916," the following figures are given :—

Deaths from Influenza in Germany.

——	Males.	Females.	Soldiers.	Total.
1914 - - - - -	2,440	2,866	10*	5,316
1915 - - - - -	2,945	3,430	33	6,408
1916 - - - - -	2,885	3,709	42	6,636

* From 1st August only.

Whether this was the same disease as that which was epidemic in 1918 is not clear. It has been stated by certain German military authorities that influenza was prevalent on the eastern war front during the summer and autumn of 1917, and that the infection had probably reached the German troops in that region from Asia through Russia. Another German theory put forward as to the origin of the 1918 epidemic was that the infection was introduced into the battlefields of Europe by Chinese labourers brought by the British Government to France for employment behind the fighting lines. The rapid spread of influenza in Germany and elsewhere was favoured by the war conditions. An instance of this was the following :— In July 1918 a German battalion, said to be the first that was attacked on the western front by influenza in the field, fought an action in that month at close quarters with an American detachment, presumably infected at the time. Other

German battalions sent to relieve the first quickly fell victims to influenza one after the other immediately following their arrival. But whatever may have been the way by which the infection reached Germany in 1918 the epidemic, once it had begun, did not spare any single part of the country. In the *Journal of the American Medical Association* for 3rd May 1919 there is a statement to the effect that from various German sources of information an estimate had been made as to the mortality of the influenza epidemic from which it appears that in Germany there had resulted approximately 400,000 deaths. The symptoms of the malady were practically the same as those observed in the pandemic of 1889. One authority states that in 1918 the main portion of the fatal cases in Germany were persons under the age of 30 years, and from this it was inferred that some amount of immunity had remained to those people who had suffered during the previous epidemic of 1889 to 1893. In another medical periodical* it was mentioned that a form of ophthalmia, believed to be caused by Pfeiffer's bacillus, had been present in the eastern war zone for more than a year, that is, before the 1918 influenza epidemic began. Dr. Ladislaus Schmidt, a military surgeon, had a number of such cases in his regiment, and on examining the sputum of the men suffering from this ophthalmia he found organisms closely resembling those found in the eye discharges. He believed that Pfeiffer's bacillus had been activated in some way, as yet unknown, and that it then attacked not only the conjunctiva but also the mucous membrane of the respiratory tract. Attention was also called by some German writers to the frequency of the occurrence of empyema during the epidemic. It is worthy of mention that before the presence of epidemic influenza was recognised in Central Europe, and as far back as 1915, pneumonia had been very prevalent in the German camps in which the Allied prisoners of war were confined.

There were two distinct waves of influenza in Germany during 1918, the first in July and the second in October. A third wave occurred in the early part of 1919, attaining its height in the second week of February, but it was not of great severity.

In Berlin cases of influenza were of frequent occurrence during July 1918, but few of them were of a serious kind. It was, however, different in October, the type of the disease then being much more severe. In that month the total deaths in Berlin from all causes in a population of over two millions amounted to 5,385, of which 2,770, or more than half, were attributed to influenza and pneumonia. In November the total mortality from all causes fell in Berlin to 3,478, but the number of these which were due to influenza and pneumonia

* *Wien. Med. Wochenschrift*, 1918, s. 1450.

has not been ascertained. Of the 2,770 deaths attributed to the epidemic in October 1,771, or 63·9 per cent., were females. Dr. Weber, commenting on the many deaths in Berlin from secondary pneumonia and other complications, attributes them to reduced resistance to the infection, due partly in his opinion to the effects of the blockade of Germany by the Allies. At present no detailed statistics of the epidemic of influenza in Berlin are available, but it is probable that they will be published at a later date.

In most of the large towns of Germany, judging from the general death-rate (all causes), the height of the epidemic was reached simultaneously in the fourth week of October. Berlin, Hamburg, Cologne, Leipzig, Dresden, Düsseldorf, Chemnitz, Stettin, Breslau, and Nürnberg all recorded the highest mortality rate in the above-mentioned week. Those rates ranged from 41·7 in Berlin to 72·5 in Breslau.

In Munich the behaviour of the disease was similar to that observed in Berlin, and many serious cases were treated in the hospitals. At Kiel, during the autumn epidemic, 573 severe cases were treated by one of the hospital physicians and 166 of them died, giving a fatality rate of 29·0 per cent.; but if the cases which were moribund on admission be deducted the rate is reduced to 18 per cent.

The majority of the above-mentioned cases in Kiel were in the age-group 20 to 40, as will be seen in the following table*:—

Age, Incidence, and Mortality of Influenza Patients admitted to the Kiel Municipal Hospital in 1918.

Age Periods.	Influenza Cases.	Deaths.	Case Mortality.
			Per cent.
0 to 9 years - - - -	18	7	38·8
10 „ 19 „ - - - -	102	24	23·5
20 „ 29 „ - - - -	253	66	26·0
30 „ 39 „ - - - -	126	40	31·7
40 „ 49 „ - - - -	32	12	37·5
50 „ 59 „ - - - -	16	5	31·2
60 „ 69 „ - - - -	9	4	44·4
70 „ 79 „ - - - -	3	2	66·7
Age unknown - - - -	14	6	42·8
Total - - -	573	166	29·0

At Frankfort-on-Main another series of 300 influenza cases were treated between June and August by a hospital physician,

* Compiled from a paper by Dr. Hoppe-Seyler in the *Deutsche Med. Wochenschrift*, No. 44, 1918.

and of these 45, or 15 per cent., died. In Frankfort, as elsewhere, the infection chiefly attacked people in the prime of life, while young children and persons over 40 years of age generally escaped. In a still further series of 73 cases treated in a Frankfort hospital the fatality rate was 11 per cent. Of this series, one-third of the cases occurred among the hospital staff or among the in-patients already under treatment for some other kind of illness, 50 per cent. of them were in the age-group 20 to 30 years, 20 per cent. between 10 and 20, 20 per cent. between 30 and 40, 4 per cent. between 50 and 60, and 2 per cent. over 60 years.

In Stettin and Swinemünde there were influenza epidemics in July and October 1918, and in February 1919 there was again a third outbreak in both places.

The influenza epidemic of 1918 affected seriously the health of the German army in the field. According to a report by Surgeon-General Schulzen, up to the beginning of October, that is, before the height of the second epidemic wave was reached, 180,000 cases had occurred among the troops, and many of the attacks were of a severe character. The number of deaths has not yet been ascertained.

Among the various German administrative districts, the population of which in 1918 suffered somewhat severely from the influenza epidemic, were Düsseldorf, Merseburg, Oppeln, and Breslau, and also the towns of Nürnberg and Hamburg.

Nürnberg. At the beginning of July 1918, influenza began to spread in Nürnberg and soon was widely diffused throughout the city. In August and September there was a considerable decrease in the number of cases notified, but in October there was a recrudescence of the epidemic, and this reached its highest point during that month, followed in November and December by a rapid decrease. Judging by the recorded weekly death rate from all causes, the epidemic was at its height in the week ended October 26th when the rate was 60·8 per 1,000 of the population, in the following week it fell to 53·1 and in the next week to 27·1 per 1,000. The number of cases "notified by medical men," month by month, in Nürnberg was as follows:—

1918.	Notified Cases.
July	3,007
August	101
September	108
October	10,495
November	985
December	433

But in addition to these cases officially notified, there were others that also came under official observation in other ways, bringing the number of known attacks in Nürnberg from July

to December, up to 21,145, of which 714, or 3·4 per cent., had a fatal termination.

Of these fatal cases 227 were males and 487 were females. The population of Nürnberg is estimated at 300,977, and in 1918, owing to the war, the females greatly outnumbered the males. In a group of cases amounting to 6,592 which were subjected to careful analysis by Dr. Federschmidt, 2,645 were males and 3,947 females. The morbidity and mortality was highest in the age-group 15 to 30 years.* Most of the fatal cases died from streptococcal pneumonia. In the age-period 20 to 30 years, nearly 5 times more women than men died of the epidemic disease. It has to be remembered that a large proportion of men in the above mentioned age-group had been called up for military service, and if they remained in Nürnberg and contracted influenza, they would appear in the military statistics and not with those of the civil population which alone are here being considered. According to Medizinalrat Dr. Federschmidt,† the maximum of morbidity during the epidemic was experienced by the age-group 15 to 30 years, and the highest mortality in the age-group 20 to 35. The explanation why influenza should expend its greatest virulence upon individuals in the height of their vigour, offered by Dr. Federschmidt is that in the age-periods in which infancy and childhood are included the anti-toxine present in the blood serum of those individuals is so strong that any germs of influenza that penetrate into the system are destroyed; and persons of ages above 35, having for the most part already suffered from an attack of influenza in 1889–90 or later, when the disease was widespread throughout Germany, were now in Dr. Federschmidt's opinion more or less immune to the disease.

Hamburg. There were two epidemics of influenza in Hamburg during 1918, the first in July and the second in October, each of which lasted about a month. The July epidemic, as elsewhere, was mild in character, but that of October was of a serious kind, being attended with severe and often fatal pulmonary complications. As evidence of this it is stated that, whereas in the period from October 1st to November 15th 1917, 115 deaths from pneumonia were recorded, during the same period of 1918 these deaths amounted to 684. In the appended Chart I. the mortality from all causes is shown week by week from May 1918 to the end of February 1919, and also the deaths from influenza and from pneumonia. These figures are taken from a report by Dr. Nocht, the medical officer of health for the City of Hamburg. This chart shows that in 4 weeks of October the deaths from all causes were respectively 304, 546, 803, and 832, while those from influenza were in the same 4 weeks, 25, 158, 356 and 407, respectively, the deaths from pneumonia being 50, 112, 149 and 139.

The age-group in Hamburg that suffered most was that from 15 to 30 years. Many of the male members of this group were

* *Münch. Med. Wochenschrift*, 1919, s. 359.

† *Muenchener Medizinische Wochenschrift*, 28th March, 1919.

away on military service, and those left behind were mostly those who were unsuited for active duty, *i.e.*, physically unfit, and therefore less able to resist infection. The epidemic caused an increase in mortality among persons suffering from tuberculosis.

The deaths from tuberculosis in Hamburg during 1914 amounted to 1,179, but in 1918 the deaths from this disease numbered 1,788, which was 53 per cent. more than the average of the six years 1908 to 1913. The population of Hamburg before the war was about 950,000, but it is estimated that in 1918 there were 156,000 fewer people living in this city than in 1913.

In the second chart a comparison is given of the monthly mortality in Hamburg in the years 1914, 1915, 1916, 1917, and 1918, as well as the average mortality for the five years period 1909–1913. It will be seen from this that in October 1918 the mortality rate from all causes rose to over 51 per 1,000 of the population, the highest previous mortality rate in any month in previous years having being 21·3 in February 1917.

The following table gives for Hamburg the weekly number of deaths from all causes, from influenza and from pneumonia, from May 1918 to the end of February 1919:—

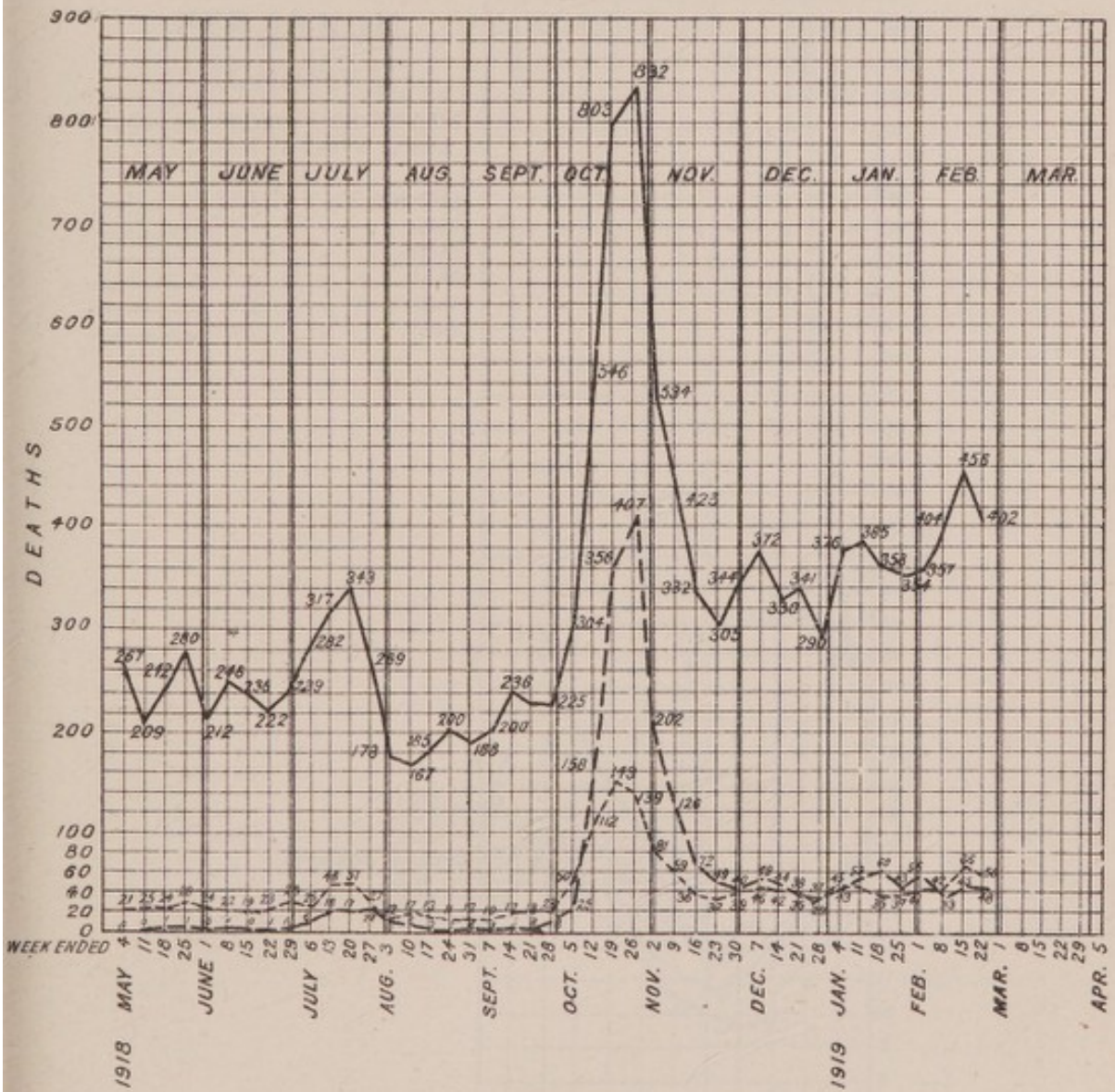
Week ended	Deaths.		
	All Causes.	Influenza.	Pneumonia.
1918.			
4th May	267	—	21
11th „	209	—	25
18th „	242	1	24
25th „	280	1	28
1st June	212	—	24
8th „	246	1	22
15th „	236	—	19
22nd „	222	1	23
29th „	239	1	28
6th July	282	6	25
13th „	317	18	45
20th „	343	17	51
27th „	269	22	27
3rd August	173	3	12
10th „	167	6	17
17th „	185	2	13
24th „	200	2	11
31st „	188	6	12
7th September	200	3	10
14th „	236	5	17
21st „	226	3	18
28th „	225	8	23
5th October	304	25	50
12th „	546	158	112
19th „	803	356	149
26th „	832	407	139
2nd November	534	202	81
9th „	423	126	59

To face p. 272.

HAMBURG.

WEEKLY DEATHS 1918-1919.

TOTAL DEATHS.
INFLUENZA.
PNEUMONIA.



H. & C. GRAHAM LTD LITHRS LONDON, S.E.

TOTAL DEATH RATE IN THE CITY OF HAMBURG FOR 1,000 INHABITANTS PER ANNUM.

(CHART 2)

ooooooo AVERAGE DEATH RATE FOR THE FIVE YEARS 1909-1913.
.—.—. DEATH RATE FOR 1913.
- - - - - - - " " " 1914
..... " " " 1915
——— " " " 1916
— — — " " " 1917
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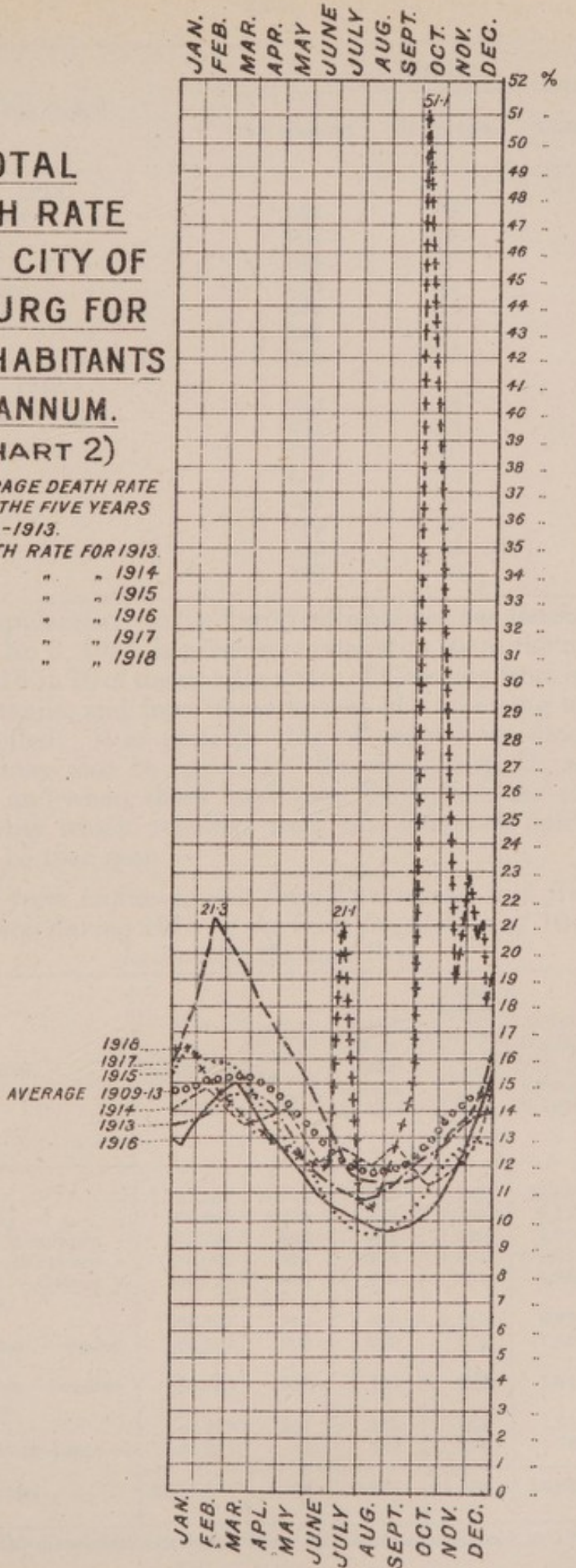

H. & C. GRAHAM LTD LITHOS LONDON, S.E.

Week ended	Deaths.		
	All Causes.	Influenza.	Pneumonia.
1918.			
16th November - - -	332	72	36
23rd „ - - -	305	49	32
30th „ - - -	344	40	39
7th December - - -	372	49	46
14th „ - - -	330	44	42
21st „ - - -	341	35	38
28th „ - - -	290	37	29
1919.			
4th January - - - -	376	43	45
11th „ - - - -	385	53	45
18th „ - - - -	358	60	35
25th „ - - - -	354	43	37
1st February - - - -	357	56	41
8th „ - - - -	404	33	42
15th „ - - - -	456	45	66
22nd „ - - - -	402	48	56

In a supplement* to the *Veröffentlichungen des Reichsgesundheitsamts*, No 6, 1920, is given some statistics as to the mortality during 1918 in 10 of the smaller states of Germany from influenza and pneumonia, and from these figures the following table has been compiled. It is possible that at some later date similar statistics may also be given for the remaining 16 states of Germany, and when these have been published the extent of the mortality which resulted from the influenza epidemic of 1918 will be then seen :—

Deaths from Influenza and from Pneumonia, and from all Causes, during 1918 *in the Civil Population of* 10 *of the smaller German States.*

States.	Civil Population 1917.	Deaths from Influenza in 1918.	Deaths from Pneumonia in 1918.	Total Deaths from Influenza and Pneumonia in 1918.	Total Deaths from all Causes in 1918.	Death Rate, all Causes, 1918.
1. Hesse - - -	1,127,305	3,825	2,025	5,850	22,035	19·5
2. Oldenburg - -	449,393	1,379	874	2,253	8,117	18·0
3. Sachsen-Meiningen -	223,727	602	624	1,226	4,974	22·2
4. Sachsen-Altenburg -	186,070	675	376	1,051	4,227	22·7
5. Sachsen – Coburg – Gotha.	230,717	540	415	955	4,836	20·9
6. Anhalt - - -	293,725	763	849	1,612	6,621	22·5
7. Schwarzen – Rudolstadt.	85,063	308	206	514	1,830	21·5
8. Schwarzen – Sondershausen.	80,533	367	153	520	1,830	22·7
9. Neuss-ä L. - -	58,298	227	126	353	1,315	22·5
10. Schaumburg-Lippe -	40,521	81	107	188	727	17·9
Total - -	2,775,352	8,767	5,755	14,522	56,512	20·4

* *Die Sterblichkeitsverhältnisse im Deutschen-Reiche* in 1918.

As regards the age and sex distribution of influenza deaths the above-named German statistical report gives the figures for 9 out of the 10 smaller states included in the preceding table (Hesse being omitted). The figures include 4,942 deaths, which were distributed as follows :—

Deaths from Influenza in 9 small German States in 1918.

Age Groups.	Males.	Females.	Total.
0 to 1 year - - - -	107	101	208
1 „ 5 years - - - -	197	226	423
5 „ 15 „ - - - -	207	286	493
15 „ 30 „ - - - -	431	880	1,311
30 „ 60 „ - - - -	632	889	1,521
60 „ 70 „ - - - -	277	296	573
70 years and over - - -	177	225	402
Not known - - - -	8	3	11
Total - - -	2,036	2,906	4,942

The deaths from all causes among males in the nine smaller states during 1918 numbered 16,177, and among females 18,279. The percentage of deaths from influenza to total deaths from all causes was 12·6 in males and 15·9 in females.

II.—Epidemic Influenza in the Western Hemisphere during 1918–19.

Canada.

During September 1918 influenza began to be epidemic in Canada, and ultimately swept over the whole Dominion, extending even to the scattered Indian tribes in the north-west, and to the Esquimaux in the remote parts of Labrador. The infection is alleged by some to have been introduced into St. John's, Quebec, from the New England states*, the first victims being soldiers. Another view is that there is an endemic region of influenza situated in north-west Canada, from which at times the infection spreads to other parts of the country in epidemic form. It cannot be said that either of these opinions is supported by enough trustworthy evidence.

Province of Quebec. — In the third week of September influenza was beginning to be prevalent in the province of Quebec, and the outbreak continued during October and November. A press report states that the cases are estimated at 530,700, of whom 13,880, or 2·62 per cent., died. The

* The New England states are Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island, situated in the eastern corner of the U.S.A.

disease is not notifiable, so that the above figures can only be regarded as an approximation to the actual number of persons attacked. Another estimate calculates that over a tenth of the population had to be absent from work owing to illness at one time or another during the period of the epidemic. In the city of Montreal the outbreak was at its height in mid-October, when the influenza deaths were as many as 200 a day. It is estimated that at least 3,000 deaths from influenza occurred in Montreal in about five weeks. The funerals became so numerous that the cemetery officials could not dispose of the corpses fast enough, and at Mount Royal Cemetery at one time hundreds of dead lay there in their coffins unburied. Theatres and entertainments, as well as other public gatherings, if of more than 25 persons, were prohibited ; all schools were closed, and office hours in business houses were greatly curtailed. The public were somewhat alarmed at the extent of the outbreak. Spitting in the streets was forbidden by law, and many persons were fined for contravention of this regulation. For a short time business operations in the city were paralysed. The churches remained closed for a period of about five weeks by order of the Board of Health. Among the military in Montreal up to 10th October 1,050 cases and 70 deaths were reported.

A recrudescence of influenza in the Province of Quebec was reported in April 1919, but details of this are not yet to hand.

Province of Ontario.—So far as can be ascertained influenza appeared in this province in the fourth week of September, and in October the disease became acutely epidemic. The deaths from influenza in each of the five months, October 1918 to February 1919, were as follows :—

	Influenza deaths.
1918, October	3,015
November	2,608
December	1,568
1919, January	1,514
Febuary	812
Total in five months	9,517

The population of the province is 2,523,000.

At the height of the epidemic in the City of Toronto the deaths from influenza numbered about 150 a day, the total up to the end of November being 2,000. Some medical men used a prophylactic vaccine prepared by the Ontario Board of Health and spoke well of the results, but the precise nature of the vaccine has not been ascertained. There was much disturbance of local business by the epidemic in Toronto and other towns. Up to the end of November no fewer than 108 medical men in Ontaria and the Western Provinces of Canada died of influenza

during the 1918 epidemic. In the large towns, hotels were turned into temporary hospitals, as the general hospitals were already filled to overflowing. Although the epidemic in the Province of Ontario showed evidence of waning in January 1919, it still was causing deaths in various towns. For example, during January, 279 influenza deaths were recorded in Toronto, 72 in Hamilton, 96 in London, and 44 in Ottawa.

Influenza became prevalent during October and November in the sparsely populated districts in the north-west part of Ontario, among the Indians. The press record instances of Indian mothers, themselves suffering from influenza, travelling many miles by canoe bringing with them to the nearest settlement their sick children for medical assistance. Medical men are comparatively few and far between in the western portion of Ontario, for instance, in one district only two doctors are available in an area of 260 miles of country. Many Indians were found dead in their camps. On Christian Island, Georgian Bay, out of a population of 275, 48 died of influenza. Sometimes the bodies of dead Indians were found in the bush, where they had died for lack of attention.

Hunters in Northern Canada spread a report that influenza was decimating big game and also smaller animals towards the end of 1918; but no satisfactory confirmation of this allegation has as yet been forthcoming. A specific statement by the *Times* correspondent at Toronto was published in the issue of 27th January to the effect that "Moose were dying from Spanish influenza," but so far there has been no corroboration of the statement. On the other hand Mr. C. Gordon Hewitt, consulting zoologist at Ottawa, stated in reply to a letter sent by the Board, dated 2nd May 1918, that nothing was known officially of the alleged epidemic among the moose and no post-mortems had been made on these animals. He, however, admitted the possibility of the occurrence of epidemics of infections of the influenza type among the native mammals especially among those which associate in bands during the winter months.

At Toronto under the Temperance Act two licensed vendors of alcoholic liquors were empowered to supply them for cure, or prevention, of influenza; and it is stated that long lines of waiting citizens testified to the number of prescriptions written by the doctors for the purpose.

Province of Manitoba.—Influenza was present in this province during October 1918. At Winnipeg, the capital of Manitoba, with a population of over 180,000, the disease was epidemic during October. There are no available reports for October, but in November it is said that 9,031 persons were attacked, and of these 526 died. During December there were 2,859 cases and 197 deaths and during the first three weeks of January 973 attacks with 101 deaths, making a total, for a period of nearly 12 weeks, of 12,863 cases and 824 deaths, giving

a case mortality rate of 6·4 per cent. During January 1919, the disease was prevalent in Northern Manitoba among the Indians. In three weeks 250 deaths were reported among them. The *Times* correspondent writes on 5th February that in one house 20 Indians were found lying on the floor ill of influenza, with four corpses among the number. Dr. O. J. Grain, Chief Medical Officer for the Federal Government of Indian Affairs reported that 750 deaths had occurred from influenza in a total Indian population of 14,179. In Winnipeg the churches were kept closed for seven weeks on account of the epidemic.

Influenza was made notifiable in Manitoba on 11th October 1918, and medical officers of health were empowered to isolate cases, to close schools, churches, theatres, and cinemas.

In the *Lancet* of 24th May 1919, Major F. T. Cadham, C.A.M.C., gives some details of the use of a vaccine in the recent epidemic of influenza at Winnipeg. He states that the disease appeared in the city during the first week of October 1918, and the height of the incidence peak was reached in the second week in November, and then the epidemic slowly, but gradually, subsided. It was considered advisable to use a vaccine, prepared from micro-organisms infecting the respiratory tract of those suffering from the disease, as a prophylactic against respiratory infection. Among the military the first inoculations were given on 20th October and a second inoculation seven days later. There were 7,600 soldiers in the district at the time and 4,842 received one inoculation and about half of that number had a second inoculation. From 1st October 1918 to 28th February 1919, 520 soldiers were admitted to hospital suffering from influenza; of these 282 had been inoculated and 238 had not been inoculated. Among the inoculated 17 developed pneumonia of whom five died. Of the five that died three had received their first inoculation on the day they were admitted to hospital. No soldier died who had been admitted subsequent to the second inoculation. Among the 238 uninoculated who were admitted to hospital 41 developed pneumonia and 17 of them died.

Report of the Winnipeg Military Hospital for Influenza Cases.

——	Admissions for Influenza.	Developed Pneumonia.	Died.
Inoculated - - - -	282	17 or 6·05 %	5 or 1·7 %
Uninoculated - - - -	238	41 or 17·1 %	17 or 7·1 %

The disease and complications were not so severe among the inoculated as among the uninoculated, and the average stay in hospital was twice as long for the uninoculated as for the inoculated.

Among the civil population the results were reported to the authorities by medical men and in reply to the question, "Do "attacks of influenza appear to be modified if contracted "subsequently to one or more injections of the vaccine?" 101 answered in the affirmative out of 108. In reply to another question, "Since influenza and pneumonia have been "fatal in such large percentage of pregnant women, has "vaccine apparently afforded protection?" 32 out of 37 practitioners answered in the affirmative. The majority of the inoculations in the civil population were given in the earlier stages of the epidemic, but no attempt was made to keep accurate statistics on this point. 28,815 persons were inoculated once, 24,184 receiving a second inoculation.

The number of uninoculated in the clientele of practitioners reporting results was 85,941.

Incidence of Influenza and Pneumonia in the Civilian Population, Inoculated and Uninoculated, of Winnipeg.

——	A.—Inoculated.			B.—Uninoculated.
	After first Inoculation.	After second Inoculation.	Total.	
	Per Cent.	Per Cent.	Per Cent.	Per Cent.
Influenza - -	2,843=9·7	2,360=9·7	5,203=9·8	21,285=24·8
Pneumonia -	177=0·65	123=0·5	308=0·57	1,869= 2·2
Deaths - -	61=0·21	24=0·09	85=0·16	563= 0·68

Major Cadham concludes that these results show that incidence of pneumonia was about four times as great, and the mortality four times as great, in the uninoculated as in the inoculated. He believes that the vaccine used as a prophylactic for the military personnel was of value. The incidence of pneumonia was less than half and the mortality rate less than one-third in the inoculated as compared with the uninoculated admitted to the special military hospital under similar conditions. The general result obtained by the civil medical practitioners appears to have been favourable.

Keewatin.—In the neighbouring province of Keewatin 12 per cent. of the Indians on the reserve are stated to have died of influenza.

British Columbia.—There is little information from British Columbia at the time of writing, except that at Vancouver

(population, 130,000) during the two weeks ended 18th January 1919, 850 cases were reported, of which 103 were fatal. During October 1918, influenza was reported to be present at Prince Rupert and Victoria, but no details were given.

Alberta and Saskatchewan.—It is known that influenza was epidemic in the latter part of 1918 in the provinces of Alberta and Saskatchewan, but no particulars as to the first-named province have been received up to the time of writing. In Saskatchewan, up to the 31st March 1919, there had been 4,322 deaths from influenza. The fatal incidence, month by month, was as follows:—

	Deaths.
1918, September	1
October	352
November	1,561
December	1,110
1919, January	638
February	340
March	320
Total in seven months	4,322

The greatest mortality was in the age-group 20 to 29 years, in which 1,360 deaths or 31·4 per cent. of the total occurred; in the age-group 30 to 39 years there were 1,180 deaths, or 27·3 per cent. of the total. In 35 instances where pregnancy was given as the primary cause, influenza was given as the secondary cause of death. In 14 other cases influenza in pregnancy was the cause given on the medical certificate, making 49 deaths of pregnant women during the epidemic. The population of the province of Saskatchewan is 675,000. Among the Indian population of Saskatchewan (numbering 7,559) in the autumn of 1918, there were 4,379 reported influenza cases, with 489 deaths; of these deaths 235 were adults and 254 children.

Nova Scotia.—During the month of October 1918, influenza was epidemic in Nova Scotia. Early in the month it appeared at Halifax and at Sydney, also at Annapolis, Port Hawkesbury, Yarmouth, and in the Louisburg district. From an incomplete return for the last quarter of 1918 it appears that upwards of 700 deaths were attributed in the province to pneumonia regarded as the result of the influenza epidemic. Other deaths not included in these figures occurred from pneumonia, but were ascribed to other causes than influenza. Almost every portion of the province was invaded by the epidemic disease. The central Board of Health for the province aided the local boards by the issue of leaflets on the prevention of the disease

for general distribution among the people, and special bulletins were supplied to medical practitioners for their information and to help them to deal with the cases. Anti-influenza vaccine was distributed to all local boards of health and to medical practitioners who asked for it. The majority of the medical men who used the vaccine testified in its favour; although its value curatively was not demonstrated, it appeared to be of use as a prophylactic.* It is expected that more precise details will be published by the Provincial Board of Health in due course.

Prince Edward Island.—Little has been heard of the influenza epidemic in Prince Edward Island except that the disease was present at Charlotte Town and Summerside in the middle of October. The public had great faith in the value of alcohol in the treatment and prevention of the disease. Two clergymen were appointed to grant prescriptions for alcoholic liquors and for the purpose the temperance laws were temporarily modified.

New Brunswick.—Early in October 1918, influenza appeared at Moncton, and a little later at Campbelltown and St. John, but so far no particulars have been supplied.

Newfoundland.

During October 1918, influenza was epidemic in St. John's (population, 35,000), the capital of the island; cases were also reported in some of the outlying districts. The prevalence of the disease continued up to January 1919. The extent of the influenza incidence has not yet been officially stated. The strip of Atlantic coast on the mainland, known as Labrador, is attached to Newfoundland, and in it influenza was epidemic at the end of 1918 and beginning of 1919 among the inhabitants, mainly Esquimaux, many hundreds of whom died of the disease. Owing to the amount of sickness among the survivors, the bodies of those who succumbed did not receive speedy burial, but were left lying about for a time; many of the corpses were, in consequence, devoured by dogs.

From Sandwich Bay it was reported that 20 per cent. of the population had died during the epidemic. At Hebron, the most northern of the Labrador villages, with a population of 220, only 70 survived, and at Okak, 50 miles south of Hebron, of 266 inhabitants, there were only 59 left, all the deaths being due to influenza and its complications. It is believed that the infection was introduced into Labrador by a sailor on board the Moravian mission steamer, "*Harmony.*" At the sealing station whole families of Esquimaux died without having had

* *Quarterly Bulletin of the Department of Health for Nova Scotia*, January 1919.

any assistance; the bodies were afterwards devoured by dogs, and the only traces left being a few skull bones. On a small island on which a family of five persons lived all were attacked by influenza and died. When help arrived, too late, the family hut was found to be surrounded by ravenous dogs endeavouring to reach the corpses inside. It was found necessary to shoot many of these animals before order was restored.*

Owing to the difficulty in the villages of disposing of the dead, the few survivors left behind being too feeble to dig graves in the frozen ground, holes were made in the ice and the dead bodies thrown in.

In the care of the sick and disposal of the dead, the Moravian missionaries displayed great courage and humanity.

The United States of America.

Before the great pandemic of 1918, influenza had been occurring in the United States from time to time in recent years in epidemic form. For example, towards the end of 1915 there was a prevalence of a disease, clinically identical with epidemic influenza, which extended practically to all parts of the States, from the Atlantic coast to that of the Pacific. The infection was very rapid in its spread, and Pfeiffer's bacillus was stated to have been isolated in a number of instances. In some districts pulmonary complications were notable in their amount, and there was a greatly increased mortality from pneumonia, which was taken as an index of the prevalence of the epidemic illness. It is noteworthy that in this outbreak the spread of the disease was so rapid that much difficulty was found in carrying on business, owing to the simultaneous attack of so many persons. In Detroit, for instance, with a population of about half a million, there were, in the month of December 1915, about 100,000 cases of the influenza-like illness; the hospitals were overcrowded, and unable to receive many seeking for admission. In some localities the schools had to close, owing to the large number of scholars absent through the epidemic malady. The city of New York was among the sufferers in 1915–16, and the department of health caused a bacteriological investigation to be made of some of the typical cases. Out of 50 patients whose attacks were diagnosed as influenza, streptococcal infection was found in 26, or 52 per cent.; the diplococcus lanceolatus in 19, or 38 per cent.; the micrococcus catarrhalis in 18, or 36 per cent.; and the influenza bacillus in 9, or 18 per cent. Other organisms were also discovered in the sputum and nasal discharges. The account of the 1915–16 outbreak reads very much like that of the 1918 epidemic, though the latter, as will be seen, was more fatal,

* *The Times*, 2nd August 1919.

and caused far more alarm in the public mind. In the New York State alone, in 1915, 1,414 deaths were attributed to influenza and 17,209 to pneumonia; while, in 1916, the influenza deaths were 2,267, and those from pneumonia (all forms), 17,314. Another example was the State of Wisconsin, where, in 1911, 360 deaths were recorded from influenza, 255 in 1912, 356 in 1913, 227 in 1914; in 1915 the number of influenza deaths rose to 490, but fell to 300 in 1916, 351 being reported in 1917. The Census Bureau of the United States gives the number of persons who died in the registration area from influenza, during 1917, as 12,974, giving a death rate of 17·2 per 100,000 of the population. The corresponding rate for 1916 was even higher, namely, 26·2 per 100,000.

Some difficulty has been encountered in the search for trustworthy figures respecting the incidence and mortality of epidemic influenza in the United States during 1918. The number of cases in many localities is not known, for the notification of the disease was not made obligatory until the height of the outbreak had almost passed, and where the cases were reported as "influenza" deaths from the epidemic illness were often certified as "influenza and pneumonia," so that it has not been possible, in many instances, to separate the pneumonia deaths from those from influenza, or to calculate the respective mortality rates. An American official statistician has summarised the difficulties as to getting correct returns about the influenza epidemic in the United States as follows:—(1) In no locality, practically, can it be assumed that complete reports of the number of cases were made; (2) Since the disease was not reported with any degree of comparable completeness in different localities, comparisons of localities as to prevalence of the disease are obviously impossible; (3) For the same reasons, statements of the incidence, according to sex, age, and other conditions with which it might be desirable to attempt correlations are not feasible except in general terms; (4) The chronology of the epidemic in, perhaps, the great majority of localities has not been recorded, and, if recorded, is subject to serious errors with respect to the beginning of the epidemic and the true height of its peak; (5) Fatality rates cannot be ascertained. With the exception of data obtained from special field studies, and from limited areas and population groups, it is necessary to fall back upon mortality reports for the main statistics of the epidemic.*

During April and May 1918 fevers of an undetermined nature were reported in several States, including Norfolk and Louisiana. An investigation of the records and reports of the

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* Extract from *Preliminary Statistics of the Influenza Epidemic*, by Edgar Sydenstricker, Public Health Statistician, U.S. Public Health Service, a paper read before the Vital Statistics Section of the American Public Health Association, at Chicago, 9th December 1918.

local medical practitioners who treated the cases led to the belief that these fevers were mainly influenza of a mild type.

Influenza appeared in the American army during February and March 1918. One of the first reports on the subject dealt with an outbreak about mid-March at Fort Oglethorpe, which is in the State of Georgia, that is to say, it lies inland in the south-eastern section of the United States. This outbreak, in point of time, appears to have coincided with the outbreak of influenza in the American Expeditionary Force, as well as in the British army in France, and in the French civil and military population. The outbreak at Fort Oglethorpe lasted three weeks, and 1,468 cases were admitted to hospital with influenza, out of a total strength of 28,586 men. There were many cases of so mild a type that they were not sent to hospital. The average incubation period was three days. Inquiry showed that previous to the outbreak there had been sporadic cases occurring for some time, and that the disease had been present in 1917. The early manifestations of epidemic influenza in North America during the early months of 1918 were comparatively mild, and added little to the death roll; but in September the disease developed more virulent characters and swept swiftly over the country, mainly from east to west, causing considerable mortality.

So far as can be ascertained at present, the first area to be affected in the second half of 1918 by epidemic influenza was the eastern portion of the State of Massachusetts and some other places on, or near, the Atlantic coast, these being attacked prior to 14th September; but it is probable that the disease was epidemic in some of these localities at the beginning of that month. During the week ended 21st September, influenza assumed epidemic proportions along a considerable part of the Atlantic coast from the southern part of the State of Maine to Virginia, as well as in some other scattered districts in the interior. In the following week (ended 28th September), the infection had extended to a number of areas adjoining the localities already attacked, and the disease had by that time attained considerable proportions in the New England States, the North Atlantic districts, the Central States, and in certain areas bordering on the Pacific Coast. By 5th October, the epidemic had become general over the whole country, except in the more isolated rural districts and in some sections of the Central States, the mountain states, and in the north-west. But by the middle of October even these localities were invaded except the most remote rural areas. The epidemic became widespread over the greater part of the United States in four or five weeks. It has been suggested that, perhaps, sources of infection already existed in some of the largest cities, or other populous centres, before the prevalence of the disease was recognised in September 1918, and that, in fact, it was spreading long before

the danger was recognised. Various estimates have been made as to the mortality caused by the influenza epidemic in the United States during 1918. One estimate published in the press gave the number of deaths as, approximately, 400,000 up to the end of November. The Special Committee on Influenza of the American Medical Association reported at a meeting at Chicago, on 8th December 1918, that, "On the basis of the best "data available, it is estimated that not less than 400,000 "deaths from the disease occurred in the United States during "the months of September, October, and November. The "major portion of the mortality occurred between the ages of "20 to 40, when human life is of the highest economic "importance." Some investigators are of opinion that a much larger number of deaths occurred up to December 1918. A compilation of the mortality from influenza and pneumonia in 118 localities is given by Dr. Sydenstricker in his paper already mentioned (*see* Table below). The death rates from the epidemic disease in these localities showed a wide variation, from 0·8 to more than 10 per 1,000. Even in the larger cities the death rate from influeuza and its complications ranged from 1 to 8 per 1,000. There appears to have been no well-defined relation between the mortality from the epidemic and the size of the town or district. Later statistics, compiled by the New York Metropolitan Life Insurance Company, show that 450,000 deaths occurred in the United States from influenza during the recent epidemic. More men died than women, and the highest mortality was among the wage-earners, especially those belonging to the poorest class.

It has already been stated that influenza had been appearing in the United States long before the present pandemic developed, but it is suggested by some authorities that a fresh strain of a more virulent form of influenza was imported into America from Europe prior to the recent epidemic, and that it was brought on board ships to the Atlantic States. There are definite statements to this effect published in the American press; for example, in June a steamship from Liverpool arrived at Philadelphia with 27 lascars and a British quartermaster suffering from pneumonia, all, it is said, in a "desperate condition" when landed, their illness being subsequently regarded as having been influenza with pneumonia complication. On July 7th the New York press stated that "Spanish influenza" had occurred on board a troopship returning from Europe to America; it had left Liverpool on June 15th; six days later six members of the crew became ill, and on the following day 15 others were attacked, but their illness was of a comparatively mild nature. During August several vessels arrived at Atlantic ports from Europe with influenza cases on board. On one of these there were 21 cases, some complicated by pneumonia. A Scandinavian steamship

arriving at New York on August 12th reported that there had been 200 cases of influenza on board during the voyage. According to the New York press, influenza had been coming to the port of New York on vessels since the early part of June. A Navy bulletin states that two steamships from Norway and a third from another Scandinavian port, not named, arrived at New York on August 14th and 15th, having on board cases of influenza. In August a steamer arrived at Newport News (Virginia) with influenza on board. There can be no doubt that these and other ships arrived at Atlantic ports of the United States bringing influenza infection from Europe and elsewhere. On the other hand, there is evidence that the influenza infection was conveyed from some ports of the United States to Europe, especially by troopships taking American reinforcements to France viâ England. For example, the S.S. "*Olympic*," with 5,194 military of all ranks on board, arrived at Southampton from a United States port, and landed 573 cases of influenza and pneumonia; within a week about 1,000 fresh cases occurred among the troops that had come by the "*Olympic*." There were about 300 deaths in connection with the outbreak among the American drafts landed from this vessel. In its next voyage, in October, the "*Olympic*" conveyed 5,430 U.S. troops from New York to Southampton, but only 34 cases of influenza were landed on arrival. On the authority of Colonel A. M. Whalet, of the U.S.A. Medical Corps, it is reported that, during the height of the American epidemic, quite a number of the convoys arriving at Liverpool were heavily affected by influenza.

Whether the American epidemic of influenza that began to attract notice in September 1918 was a recrudescence of an existing infection that had in some unknown way acquired more virulent characters or, on the other hand, was due to the importation from some foreign country of a fresh and very virulent strain of the infection, it is not at present possible to say, but, perhaps, when there has been time to collect and sift all the evidence, it may be found possible to trace the real origin of this remarkable outburst which destroyed the lives of so many of the American people in the autumn of 1918. The statement has been made in the American medical press that, in the United States, the onset of the influenza epidemic was about three weeks later than in London and Paris.

In some parts of the United States the mortality from pneumonia has been taken as an index of the ravages of the epidemic. In the following table is given the number of deaths from pneumonia reported in 10 cities from September 14th to December 28th, 1918, a period of 16 weeks. In this period are included the weeks in which the influenza prevalence began, reached its height, and subsided :—

*Deaths from Pneumonia reported in 10 large Cities of the United States in the 16 weeks period from September 8th to December 28th.**

—	Population.	Weeks ended															
		September			October				November					December			
		14.	21.	28.	5.	12.	19.	26.	2.	9.	16.	23.	30.	7.	14.	21.	28.
Baltimore - - -	594,637	7	5	19	*87*	371	793	600	204	47	20	20	21	21	24	35	24
Boston - - -	767,813	*27*	93	175	225	177	126	52	38	14	15	15	13	23	30	33	29
Chicago - - -	2,547,201	15	24	*74*	246	476	863	826	456	250	155	105	69	103	158	159	158
Cleveland - - -	692,259	5	8	10	15	22	*64*	106	153	102	82	54	52	48	74	73	69
Kansas City, Mo. -	305,816	5	—	10	11	*21*	55	75	55	35	34	26	38	48	73	43	24
Los Angeles - -	535,485	9	—	2	*14*	41	31	49	53	21	265	–?	5	9	8	11	10
Louisville - - -	240,808	3	9	4	14	77	154	157	60	37	22	20	42	28	5	38	22
New York - -	5,737,492	74	*98*	145	434	1,142	2,099	2,251	1,855	1,070	568	365	249	288	317	318	419
Philadelphia - -	1,735,514	20	32	*76*	207	938	1,644	1,182	467	–?	38	23	31	33	47	90	47
Washington, D.C. -	370,000	10	—	*17*	57	101	96	61	36	14	13	16	19	21	15	40	42

* The figures in italic indicate when the weekly mortality from pneumonia became markedly in excess of the average of previous years.

In a number of towns in the United States the deaths from influenza and from pneumonia are classed together and Dr. Sydenstricker, in his paper already quoted, gives a table which shows the number of such deaths in a series of towns of various sizes during the six weeks period in which the epidemic was at its height. The towns in the table are classed according to their population :—

Table showing the number of Deaths and the Death Rate per 1,000 of the Population, from Influenza and Pneumonia (all forms) during six epidemic weeks, compared for Groups of Cities classified according to size.

Cities Grouped according to Class.	Number of Cities Reporting.	Total Population of Cities in each Class.	Deaths from Influenza and Pneumonia (all forms) in six epidemic weeks.	
			Number.	Rate per 1,000 of the Population.
Under 25,000 inhabitants -	23	425,388	2,542	6·0
25,000 to 50,000 - - -	23	880,054	4,311	4·9
50,000 to 75,000 - - -	16	1,006,270	3,830	3·8
75,000 to 100,000 - - -	4	339,621	934	2·7
100,000 to 125,000 - -	10	1,140,118	5,696	5·0
Under 125,000 - - -	76	3,791,451	17,270	4·6
125,000 to 250,000 - -	16	2,758,926	8,956	3·2
250,000 to 375,000 - -	9	2,595,047	5,966	2·3
375,000 to 500,000 - -	8	3,419,342	12,211	3·6
Under 500,000 - - -	109	12,564,768	44,413	3·5
500,000 to 1,000,000 - -	6	4,136,867	16,520	4·0
1,000,000 and over - -	3	9,573,931	39,902	4·2

In the U.S. Public Health Reports of 29th November 1918, is given a resumé of the mortality from influenza and pneumonia in 29 large cities during a period of 8 weeks covering the epidemic. As has been stated the fatal incidence of influenza did not in point of time coincide in all these cities, but the following table shows the duration of the epidemic in each city as judged by the mortality ; and the succeeding table gives the comparison of the mortality rate in each town week by week during the epidemic period in each :—

Table showing the number of Deaths from Influenza and Pneumonia (all forms) for corresponding weeks, after the beginning of the 1918 Epidemic for large Cities of the United States.

City.	Population.	Week preceding the Epidemic.	Week of the Epidemic.							
			1st.	2nd.	3rd.	4th.	5th.	6th.	7th.	8th.
Philadelphia -	1,735,514	76	706	2,637	4,597	3,031	1,203	375	164	103
Baltimore -	594,637	19	117	563	1,537	1,073	397	147	57	36
New Orleans -	377,010	29	144	624	682	333	158	76	—	—
Washington -	370,000	—	38	181	547	606	372	164	55	42
Nashville -	118,136	5	120	193	127	54	53	15	16	—
Boston - -	767,813	46	265	775	1,216	1,027	589	126	137	76
San Francisco	471,023	19	130	552	738	414	198	90	—	—
Fall River -	129,828	9	20	97	201	192	97	40	24	14
Richmond -	158,702	4	41	131	177	128	71	28	23	13
Lowell - -	114,366	8	32	93	141	116	84	30	8	8
Cambridge -	114,293	4	105	140	115	63	21	19	5	9
New Haven -	152,275	2	15	36	77	152	183	168	82	48
Memphis -	151,877	—	80	182	166	71	29	17	18	—
Dayton - -	128,939	5	31	134	137	115	67	21	5	—
Oakland -	206,405	3	18	42	138	237	157	70	35	—
Cleveland -	692,259	40	168	453	682	524	351	240	—	—
Chicago - -	2,547,201	91	417	1,047	2,105	2,367	1,470	738	390	251
New York -	5,737,492	191	733	2,121	4,237	5,201	4,402	2,277	1,053	657
Omaha -	177,777	7	68	160	147	94	117	48	—	—
Rochester -	264,714	6	36	102	213	209	104	52	40	—
Louisville -	240,808	14	92	180	181	69	58	39	35	—
Birmingham -	189,716	5	17	61	110	133	85	46	46	44
Kansas City -	305,816	10	37	96	168	193	197	138	80	64
Denver - -	268,439	19	59	139	147	108	101	77	87	—
Columbus -	220,135	10	28	73	117	94	50	36	43	—
Atlanta - -	196,144	7	30	81	101	45	34	32	26	—
Indianapolis -	283,622	10	24	46	128	115	84	58	48	62
Milwaukee -	445,008	15	69	113	175	125	95	70	49	—
St. Louis -	768,630	—	86	156	233	257	229	228	190	—

The figures in the above table are those reported to the U.S. Public Health Service, and checked with figures published by the Bureau of the Census.

The following table gives the death rates per 100,000 inhabitants from influenza and pneumonia (all forms) for corresponding weeks after the beginning of the epidemic in 1918, for 29 large cities of the United States :—

City.	Week preceding the Epidemic.	Week of the Epidemic.							
		1st.	2nd.	3rd.	4th.	5th.	6th.	7th.	8th.
Philadelphia -	4·4	40·7	151·8	264·9	174·1	69·3	21·6	9·5	5·9
Baltimore -	3·2	19·7	94·6	258·7	180·5	66·8	24·7	8·6	6·1
New Orleans -	7·7	38·2	165·5	180·8	88·4	41·9	20·2	—	—
Washington -	—	10·3	48·9	147·9	163·8	100·5	41·6	14·9	11·4
Nashville -	4·2	109·2	163·4	107·6	45·7	44·9	12·7	13·6	—

City.	Week preceding the Epidemic.	Week of the Epidemic.							
		1st.	2nd.	3rd.	4th.	5th.	6th.	7th.	8th.
Boston - -	6·0	34·5	101·0	158·4	133·9	76·7	16·4	17·9	9·9
San Francisco -	4·0	27·6	117·2	156·6	87·9	42·0	19·1	—	—
Fall River -	6·9	15·4	74·7	154·8	147·9	74·8	30·8	18·5	10·8
Richmond -	2·5	25·8	82·6	111·5	80·7	44·7	17·6	14·5	8·2
Lowell - -	7·0	28·0	81·3	123·3	101·4	73·4	26·2	7·0	7·0
Cambridge -	3·5	91·9	122·5	100·6	55·1	18·4	16·6	4·4	7·9
New Haven -	1·3	9·9	23·6	50·6	99·8	120·2	110·3	53·8	31·5
Memphis -	—	52·6	119·8	109·4	46·7	19·1	11·2	11·9	—
Dayton - -	3·9	24·1	103·9	106·3	89·2	51·9	16·3	3·9	—
Oakland - -	1·5	8·7	20·7	66·8	114·7	76·0	33·9	17·0	—
Cleveland -	5·8	24·3	65·4	98·5	75·7	50·7	34·7	—	—
Chicago - -	3·6	16·4	41·2	82·7	92·9	57·7	28·9	15·3	9·9
New York -	3·3	12·8	37·0	73·9	90·8	76·8	39·7	18·4	11·5
Omaha - -	3·9	38·2	90·0	82·7	52·8	65·9	27·0	—	—
Rochester -	2·3	13·6	38·6	80·0	79·0	39·3	19·7	15·1	—
Louisville -	5·8	38·2	74·8	75·2	28·7	24·1	19·0	14·5	—
Birmingham -	2·6	9·0	32·2	58·0	70·1	44·8	24·2	24·2	23·2
Kansas City -	3·3	12·1	31·4	55·0	63·1	64·4	45·1	26·1	20·9
Denver - -	7·1	22·0	51·8	54·8	40·2	37·6	28·7	32·4	—
Columbus -	4·5	12·7	33·2	53·1	42·7	22·7	16·4	19·5	—
Atlanta - -	3·6	15·3	41·3	51·5	23·0	17·3	16·3	13·3	—
Indianapolis -	3·5	8·5	16·2	45·1	40·6	29·6	20·4	11·9	21·8
Milwaukee -	3·4	15·5	25·4	39·3	28·1	21·3	15·7	11·0	—
St. Louis - -	—	11·2	20·3	30·3	33·5	29·8	29·7	24·7	—

The rise and fall of the influenza epidemic of 1918 can be studied, perhaps, with advantage, in the camps of the troops under training in the United States. These camps were distributed over the whole country, and the men were under medical observation all the time. There had been outbreaks of influenza in these camps before the outburst of September. According to Colonel Victor C. Vaughan, U.S. Medical Corps, "influenza struck our camps in February 1918, but the "type was mild, and it left but little mark upon the mortality "returns." The occurrence of influenza on ships in August had drawn the attention of the authorities to the probable importation of the disease from the ports to inland districts, and watch was held, particularly on the ports of New York and Newport News. But the disease appeared at the port of Boston first, and was conveyed thence to Camp Devens, only a few miles distant from that city. From Camp Devens it spread rapidly over the adjoining districts, and after that became widely diffused throughout the States.

In an article published in the Journal of the American Medical Association for December 7th, 1918, Major George A. Soper, of the U.S. Medical Service, gives an account of the behaviour of the disease in 38 camps distributed over the various States. He says that the disease appeared suddenly and "ran a meteoric career" in the camps; he comes to the conclusion that its appearance and course among the civil population was somewhat similar to that observed among the

military. In the appended table he shows the dates when marked increases occurred in the deaths from pneumonia in various cities and camps in different parts of the country. He infers that the increased incidence of pneumonia in the principal cities of the States preceded that observed in the Army camps, and therefore the latter derived their infection from their immediate environment and not from one another. The table shows that the States of Massachusetts, New York, New Jersey, Maryland, and Virginia were attacked first and the Western districts near the Pacific coast last.

For Table *see* p. 291.

An epidemic of influenza and pneumonia occurred among the military at Fort Riley, Kansas, beginning on 15th September and lasting till 1st November 1918, during which time out of a total average strength of 63,374 there occurred 15,170 cases of influenza, 2,624 of whom developed pneumonia and 941, or 35·8 per cent. died. The incidence of pneumonia among the influenza cases was 17·2 per cent. After the post mortem examination of 55 fatal pneumonia cases where cultures from the lungs, pleural fluids, heart's blood, sinuses, mastoids, and other tissues were made, the pneumococcus was discovered in 56·1 per cent. and streptococcus hæmolyticus in 41·1 per cent. In addition the latter organism was found in 41·5 per cent. of 65 empyemas that resulted from the epidemic disease. Among 300 nasopharyngeal cultures from influenza patients on human blood agar the influenza bacillus was the predominating organism in 35·7 per cent. In sputum cultures from 928 pneumonia patients the B. influenzæ occurred singly or in combination in 18·7 per cent., and among 77 fatal pneumonia cases B. influenzæ was found in the sputum cultures in 5·2 per cent. All the deaths that occurred during this influenza epidemic were due to pneumonia or its complications. In 41 necropsies meningitis of sub-acute basilar type, associated with œdema, was a striking feature in 58·5 per cent., while brain œdema without evident meningitis was present in 21·8 per cent. Otitis media and mastoiditis existed in 41·5 per cent., while sphenoiditis was seen in 68·3 per cent., and ethmoiditis in 31·7 per cent. The above facts are taken from a report by Major Willard J. Stone and Captain W. Swift of the Medical Corps of the United States Army published in the journal of the American Medical Association of 15th February 1919. These observers state that B. influenzæ had not been frequently found in the sputum or throat secretions of patients in the locality during the year preceding the influenza outbreak at Fort Riley.

An outbreak at Camp Devens was reported on 7th September, and reached its height on 20th September, on which day 1,543 fresh cases were reported. Meanwhile pneumonia became a frequent and fatal accompaniment of the epidemic. From the

Table showing when the marked increases in Deaths from Pneumonia occurred in the Principal Cities and Army Camps of the U.S.A. during the period 8th September to 25th October, inclusive.

Week ending	Atlantic Coast.		North Central.		South-eastern.		South-western.		Pacific.	
	Cities.	Camps.	Cities.	Camps.	Cities.	Camps.	Cities.	Camps.	Cities.	Camps.
14th September	Boston	—	—	—	—	—	—	—	—	—
21st ,,	Lowell	Devens	—	—	—	—	—	—	—	—
21st ,,	New York	Dix	—	—	—	—	—	—	—	—
28th ,,	Cambridge	Lee	Pittsburg	—	—	—	—	—	—	—
28th ,,	Worcester	Upton	Chicago	—	—	—	—	—	—	—
28th ,,	Jersey City	Syracuse	Minneapolis	—	—	—	—	—	—	—
28th ,,	Philadelphia	Humphreys	—	—	—	—	—	—	—	—
28th ,,	Providence	—	—	—	—	—	—	—	—	—
28th ,,	Washington	—	—	—	—	—	—	—	—	—
5th October -	Baltimore	Meade	Cincinnati	Custer	Birmingham	Beauregard	Denver	Pike	Los Angeles	—
5th ,, -	Buffalo	Hoboken	Indianapolis	Funston	—	Sevier	—	—	—	—
5th ,, -	Fall River	Eustis	—	Grant	—	Logan	—	—	—	—
5th ,, -	Newark	Newport News	—	Sherman	—	Hancock	—	—	—	—
5th ,, -	Richmond	—	—	Taylor	—	Gordon	—	—	—	—
5th ,, -	Syracuse	—	—	—	—	—	—	—	—	—
12th ,, -	Albany	—	Kansas City	Dodge	Atlanta	Greene	—	Travis	Oakland	—
12th ,, -	Rochester	—	Louisville	—	New Orleans	McClellan	—	Fowie	Seattle	—
12th ,, -	—	—	Milwaukee	—	—	Wadsworth	—	—	—	—
12th ,, -	—	—	Omaha	—	—	Jackson	—	—	—	—
12th ,, -	—	—	St. Paul	—	—	Johnston	—	—	—	—
12th ,, -	—	—	St. Louis	—	—	Greenleaf	—	—	—	—
19th ,, -	New Haven	—	Cleveland	—	—	Sheridan	—	MacArthur	San Francisco	Fremont
19th ,, -	—	—	Columbus	—	—	—	—	Cody	Spokane	Lewis
19th ,, -	—	—	Dayton	—	—	—	—	—	Portland	—
19th ,, -	—	—	Toledo	—	—	—	—	—	—	—

records collected the camp outbreak could be divided into four parts, as may be seen in the next table. The rise of the epidemic occupied a period of about eight days, the peak two days, the rapid decline eight days, and the slow subsidence of the outbreak 19 days. Half of the deaths, and nearly three-quarters of the pneumonia, occurred within a period of less than three weeks.

*The Rise and Fall of the Epidemic at Camp Devens.**

1918.	Duration in Days.	Influenza Cases.	Pneumonia Cases.	Deaths.
Rise, 2nd to 10th September	8	3,283	43	16
Peak, 20th to 31st September.	2	2,722	205	43
Rapid decline, 21st to 29th September.	8	3,141	1,495	298
Slow decline, 30th September to 18th October.	19	571	571	310
Total - -	37	9,717	2,314	667

An epidemic of influenza occurred at the end of September and first half of October 1918 at Camp Sherman, Ohio, and was reported in the *Journal of the American Medical Association* for 16th November 1918 by three military medical officers. At the time when this outbreak began influenza was epidemic in the New England states, and, although prior to this many cases of coryza and bronchitis had been occurring in the camp, they were not at first regarded as due to the influenza infection. The total daily number of soldiers in Camp Sherman at the end of September was 33,044, and of these, during a period of about three weeks, which ended 11th October, 10,979 suffered from an attack of influenza. Of the 10,979 influenza cases 2,001, or 18·2 per cent. developed pneumonia, and 842 died, giving the high case mortality rate, for the pneumonia attacks, of 42 per cent., or, if the whole number of influenza attacks be included, the fatality rate would be 7·6 per cent. It is worthy of mention that of the men in camp 15,493, or 46·8 per cent., had been there only a month or less. This group of men furnished 2,944, or 69 per cent., of the first 4,269 cases which occurred in Camp Sherman. The incidence of the disease appeared to decrease with the length of residence in camp. In this outbreak the influenza bacillus was not demonstrated, the pneumoccocus

* *See* Major George A. Soper's report in the *Journal of the American Medical Association* for 7th December 1919.

being the predominating organism. The hæmolytic streptococcus also occurred in 47 per cent. of the sputum cases examined bacteriologically.

The number of reported cases of influenza and pneumonia in the military camps and barracks of the army in training in the United States began to be published in the third week of September, and the numbers, week by week to the beginning of 1919, are given in the table appended :—

Reported Cases of Influenza among U.S. Troops in Camps and Barracks during latter part of 1918 *and beginning of* 1919.

Week ended	Influenza Cases.	Pneumonia Cases.	Death Rate from all Disease in the U.S. Army per 1,000.
20th September 1918	10,094	758	4·4
27th „	37,493	4,313	34·4
4th October	88,478	8,655	81·2
11th „	90,393	17,882	206·4
18th „	43,779	11,013	190·1
25th „	19,996	2,454	92·7
1st November	16,516	3,004	43·1
8th „	6,887	2,392	33·0
15th „	4,485	1,335	19·7
22nd „	3,936	1,079	13·7
29th „	3,000	1,136	13·2
6th December	4,056	1,120	11·7
13th „	3,630	1,061	14·9
20th „	1,957	814	13·7
27th „	1,262	539	12·1
3rd January 1919	1,191	518	10·5
10th „	1,888	567	14·4
17th „	1,897	702	16·7
24th „	1,442	611	16·0
31st „	939	394	12·0

The average strength of the army in training in the United States was about 1,500,000.

The military camps in the United States were divided into four groups, the first region being that on the Atlantic coast, the second the south-eastern areas, the third the north central, and the fourth the south-western. The subjoined table gives the total number of cases of influenza and pneumonia and deaths from both diseases combined from the beginning of the epidemic to the end of October in the 38 largest camps, along with the percentage of troops attacked by influenza and pneumonia, the percentage of pneumonia to influenza, the percentage

of deaths from pneumonia, and the duration of the epidemic in days for each camp :—

Cases and Deaths from Influenza and Pneumonia in 38 *of the large Camps of the Army in the United States during the period from* 12*th September to* 31*st October* 1918, *inclusive.*

Camps.	Cases of		Deaths from Influenza and Pneumonia.	Per Cent. attacked by Influenza.	Per Cent. attacked by Pneumonia.	Percentage of Deaths from Pneumonia.	Duration of Epidemic in Days.
	Influenza.	Pneumonia.					
Group I.							
Cody	2,337	252	46	49·8	10·8	18·2	28
Beauregard	5,252	1,007	422	39·6	19·2	41·9	36
Wadsworth	5,505	357	60	38·6	6·5	16·9	20
Bowie	4,052	110	104	38·1	2·9	89·8	32
Hoboken	13,563	2,280	794	30·5	16·9	34·9	42
Dixens	13,398	2,288	794	30·1	17·1	34·9	49
Group II.							
Dodge	9,398	1,847	570	29·0	19·7	30·8	32
Custer	11,626	2,437	669	29·0	20·9	27·8	31
MacArthur	6,010	852	188	28·1	14·1	20·4	24
Meade	11,449	2,013	796	27·8	25·3	27·4	41
Pike	13,273	1,379	455	26·7	10·3	32·7	37
Grant	10,717	2,335	1,068	25·8	21·8	45·7	39
Greene	4,200	626	258	25·8	14·9	41·8	30
Funston	13,526	2,328	888	24·9	17·2	37·4	41
Forrest	2,307	33	22	24·9	1·1	86·6	25
Travis	8,470	1,742	168	24·7	20·5	9·0	30
Logan	3,137	393	16	24·6	12·4	3·8	41
Lee	11,298	1,919	672	22·9	17·0	34·6	43
Hancock	7,715	1,209	462	22·2	15·8	37·4	31
Sheridan	4,758	521	132	20·4	10·3	24·6	28
Greenleaf	4,747	343	263	20·3	7·2	75·2	20
Group III.							
Taylor	11,587	2,800	830	19·2	24·2	29·0	39
Dix	9,283	1,673	829	19·0	17·9	49·8	44
Jackson	7,500	1,114	362	18·9	14·9	31·3	43
Shelby	1,761	94	19	18·8	5·3	24·8	28
Syracuse	2,031	401	164	18·4	19·8	40·6	42
Sevier	4,526	896	319	16·3	19·7	35·7	38
McClellan	4,718	9,993	218	16·1	20·2	21·3	28
Eustis	1,745	67	10	14·8	4·0	16·2	36
Newport News	3,897	601	195	14·2	15·5	30·5	34
Upton	5,090	974	343	13·9	19·2	34·9	48
Sherman	4,789	1,717	1,058	13·5	35·7	61·3	32
Kearney	2,450	186	37	13·5	7·9	19·8	34
Gordon	4,155	626	192	11·3	14·8	30·4	42
Johnston	2,117	383	161	11·1	18·2	40·9	30
Group IV.							
Fremont	2,437	392	132	9·8	16·1	31·2	23
Lewis	3,141	994	148	9·7	31·6	14·8	33
Wheeler	70	361	61	0·8	516·0	17·8	—

This table is taken from a paper published in the *Journal of the American Medical Association* for 7th December 1918 by

Major George A Soper, Medical Corps, United States Army, entitled, "The Pandemic in the Army Camps." From the Division of Infectious Diseases and Laboratories, Medical Department, U.S. Army.

In the seven weeks represented by the table, 306,719 cases of influenza and 48,079 of pneumonia were reported in the army in the United States, the deaths registered from influenza being 725, and those from pneumonia being 18,701.

Of 31 camps attacked by influenza between 12th September and 30th September, 18 were barrack camps, 11 were tent camps, and 2 were camps at port of embarkation. As the tents afforded a greater separation of the men than the barracks did, it is of interest to note the incidence of influenza in the two groups. There was little difference in the rate of spread of the infection in tent camps and barracks, but the percentage of cases which developed pneumonia was greater in the barracks than in the tents, as may be seen in the tabular statement* below :—

	Barrack Camps.	Tent Camps.
Camps attacked, 12th to 30th September -	20	19
Combined strength - - - - -	748,632	313,362
Influenza cases - - - - - -	158,104	69,761
Pneumonia - - - - - - -	30,099	9,740
Per cent. attacks by influenza - - - -	21·1	22·2
" " " and pneumonia -	19·0	13·9
Case mortality of pneumonia - - - -	35·0	30·1

In Camp Logan the medical officers reported an increase in the number of cases of appendicitis during the course of the influenza epidemic. From 13th September to 31st December 92 cases were operated upon, the number of troops in camp at the time being about 10,000. A close relation between the two diseases is suggested.

It is worth mentioning here that, in connection with an outbreak of influenza on the ships belonging to the recruiting service of the Shipping Board of East Boston, a large number of the patients were accommodated in a tent hospital on shore. Cold and wet weather prevailed at first, and on this account the cases were confined to their tents; but as soon as the weather permitted the patients were removed from their tents to the open air, a sufficient amount of warmth being maintained by hot bottles or heated bricks. The results were gratifying, for nearly every patient thus treated had a lower temperature at night than in the morning, and expressed himself as feeling better and more comfortable. The benefits of fresh air and sunshine in the treatment of influenza and pneumonia were thus demonstrated. Of 351 cases treated in this way there

* Taken from Major Soper's report.

were only 35 deaths. It has to be remembered that most of the cases brought to the tent-hospital were of a serious kind, and in the circumstances the mortality may be regarded as having been small. Very few of the nurses or attendants contracted this infection.*

Dr. V. Heiser, in a paper on "Barrack Life and Respiratory Diseases,"† discussed the latter in connection with the recent influenza epidemic in America, and arrived at the following conclusions: The American experience since the war began has shown that soldiers who live under barrack conditions have a high mortality, particularly from respiratory diseases. The mortality rate among the Students Army Training Corps in the recent outbreak of influenza was higher than among students who lived under pre-war conditions. Civilians throughout the country had a much lower mortality than soldiers in cantonments or students in barracks, at colleges and universities. Other conditions being equal, the mortality was higher among groups of men who slept many in a room; but there have been factors responsible other than overcrowding during sleep, *e.g.*, infection through closer contact at meals, infected mess kit, and hand infections. The high infectivity of respiratory diseases at the present time justifies the requirement that men should live under barrack conditions only in cases of extreme emergency. Patients suffering from different respiratory infections should not be quartered in the same room or ward, even when there is the highest standard of ventilation; but if emergency conditions render such a course necessary, patients and attendants should, where practicable, wear approved masks or be otherwise protected. Finally, Dr. Heiser says: "Sanitary art has not "arrived at a point at which it can adequately safeguard the "lives of men against respiratory diseases who live under "barrack conditions."

It is freely admitted that influenza was spread, and increased in virulence in transports, and other shipping, conveying men and material to and from Europe. The conditions on shipboard were sometimes almost intolerable, and added to this were exposure, poor ventilation, and particularly close contact of individuals in cramped spaces. Under these conditions bronchopneumonia was common and often yielded a case mortality of 50 per cent.

But with trained and seasoned troops in the field with outdoor military life and work neither too hard nor too anxious, with adequate clothing, warm food, and some kind of dry shelter in which to sleep, influenza gave but a little temporary inconvenience, and deaths were comparatively few. Early recognition and removal to hospital prevented complications. If patients were segregated in cubicles and masks were worn, the risks of cross-infection would be greatly diminished.

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* *American Journal of Public Health*, October 1918.

† See *Journal of the American Medical Association* for 7th December 1919.

Observations made by competent experts in military camps in the United States during the recent influenza epidemics tend to show that the disease must to some extent be spread by infected drinking vessels. In certain camps with a combined total population of 66,000 inquiry showed that the incidence of influenzal-pneumonia and its mortality were distinctly affected when all the mess equipment, after use, was immersed in boiling water; the influenza rate was 12 times less in battalions where this precaution was used than in others where the old method of washing the vessels in lukewarm water was followed.

As regards the point whether one attack of epidemic influenza confers protection against subsequent attack, the following facts are of interest. At Camp Funstan, in the State of Kansas, three separate waves of influenza were noted during 1918. The first occurred in March, the second in April, and the third in May. Each wave appeared to spend itself quickly having, as it seemed, used up in each instance all the available susceptible material. In each of the three separate outbreaks the attacks occurred mainly in the newly drafted men who had been brought into the camp a short time before the outbreak began. Corresponding to each influenza wave there was an increased incidence of pneumonia and bronchitis, the cases occurring in the same drafts as the influenza. The men who had been in the camp for some months were attacked in the first outbreak, whereas in the second, and again in the third, the attacks were chiefly in the new drafts that had arrived respectively in the camp shortly before each of the two latter outbreaks began.*

It is a disputed point whether any of the lower animals suffer from epidemic influenza when the disease is prevalent in man. There is very little definite evidence on the subject, and reported instances in the present writer's experience have hitherto generally been found not to bear investigation; but in the *Journal of the American Medical Association* for 12th April 1919 there is a statement to the effect that at Camp Cody, soon after influenza had become epidemic, the guinea-pigs kept for investigation purposes at the camp laboratory began to show an unusual mortality, the evidence of pneumonia in the fatal cases being unmistakable. Cultures from the pleuritic exudation and from the lung tissue disclosed the presence of pneumococci, streptococci, and B. influenzæ, that is precisely similar results as those found in fatal human cases during the influenza epidemic. Thirty guinea-pigs died within a period of three weeks. A fresh batch of 50 guinea-pigs were obtained from El Paso, Texas. All arrived well, save four which had died on the journey. The remaining 46 were lodged in a previously unused apartment. They remained in good health for a fortnight, when one of them died. This was the com-

* *Journal of the American Medical Association*, 11th January 1919.

mencement of a second guinea-pig epidemic, precisely similar to the first; in the course of a few weeks the whole of the 46 guinea-pigs died, presumably of epidemic influenza, notwithstanding the segregation of the sick animals.

In the public press statements appeared that an epidemic of influenza had broken out in the Yellowstone National Park among the buffalo herds. Inquiry made officially resulted in a report that locally nothing was known of it. There had, however, been two epidemics of hemorrhagic septicæmia among the buffalo herds affecting them unfavourably.

Influenza in the City of Boston.

As has been already mentioned, Boston was one of the first cities in the United States to be attacked by epidemic influenza during the autumn of 1918. But the disease was present in the city during the first half of the year, 40 deaths being certified from this cause, namely, 4 in January, 6 in February, 18 in March, 10 in April, 1 in May, and 1 in June. There were no deaths in July or August. In the second week of September 19 deaths were ascribed to influenza, and from that date to the end of the year fatal cases continued to be recorded, the total number during the four months period being 3,852, with an additional 1,103 from pneumonia (all forms), making a combined total of 4,955 deaths believed to have been due to the epidemic. In the first half of 1918 the fatal cases of pneumonia registered in the city amounted to 1,133. In July there were 63 deaths from pneumonia and 33 in August, but in September there were 464. In the subjoined table are given the number of cases of lobar pneumonia and influenza reported in each of the last four months of 1918 and in the first month of 1919, together with the number of deaths certified from all forms of pneumonia and from influenza:—

City of Boston: Influenza and Pneumonia Cases and Deaths from September 1918 *to January* 1919 *inclusive.*

	Notified Cases.		Certified Deaths.		Total Deaths from Pneumonia and Influenza.
	Lobar Pneumonia.	Influenza.	Pneumonia, all Forms.	Influenza.	
1918.					
September - - -	498	*	464	1,355	1,819
October - - - -	485	3,271	461	2,024	2,485
November - - - -	108	595	83	145	228
December - - - -	269	5,723	173	446	619
1919.					
January - - - -	365	9,589	154	591	745
Total - -	1,725	19,178	1,335	4,561	5,896

* Influenza became notifiable on 4th October 1918.

The population of Boston is 767,813.

An examination of the weekly returns of sickness and death in the city shows that in December there was a second wave of the epidemic which became obvious in the third week of that month, but it appears not to have been of so virulent a nature as that which began in September.

The course of the two waves of influenza which were separated by an interval of about six or seven weeks can be seen in the next table, which gives the reported cases and deaths from pneumonia and influenza, week by week, from 1st September 1918 to 25th January 1919, a period of 21 weeks :—

The Incidence of Pneumonia and Influenza, and Deaths therefrom, in the City of Boston from the Beginning of the Autumn Epidemic of 1918.

Week ended	Cases reported.		Deaths certified.		Total Deaths from Pneumonia and Influenza.
	Lobar Pneumonia.	Influenza.	Pneumonia, all Forms	Influenza	
1918.					
7th September	3	—	8	—	8
14th „	26	—	27	19	46
21st „	121	—	93	172	265
28th „	278	—	175	600	775
5th October	236	115*	225	989	1,214
12th „	122	1,520	177	850	1,027
19th „	105	1,188	126	463	589
26th „	43	304	52	174	226
2nd November	51	200	38	99	137
9th „	23	72	21	55	76
16th „	19	71	20	27	47
23rd „	29	216	21	33	54
30th „	28	181	13	34	47
7th December	32	255	23	36	59
14th „	57	506	30	47	77
21st „	78	1,046	33	91	129
28th „	72	2,363	29	163	192
1919.					
4th January	83	3,558	43	188	231
11th „	98	1,626	38	183	221
18th „	83	1,127	29	116	145
25th „	84	931	33	108	141

* Influenza made notifiable on 4th October 1918.

During February and March 1919 the deaths from influenza and pneumonia gradually decreased.

Influenza in the State of New York.

The estimated population of the State of New York in 1918 was 10,681,667, of which 5,872,667 were referred to the City of New York.

Influenza has appeared annually in the death returns of this State for the last 12 years, but in no year has the mortality from the disease ever reached anything like the height that it did in 1918. The following table gives the number of deaths certified in the State of New York from influenza and pneumonia (all forms) for each of the 12 years 1907 to 1918, together with the death-rate for each of those diseases per 100,000 of the population :—

Table showing the Number of Deaths certified from Influenza and from Pneumonia, together with the Death Rates from these Diseases from 1907 *to* 1918 *in the State of New York.*

Year.	Influenza Deaths.	Influenza Death-Rates per 100,000.	Deaths from Pneumonia, all Forms.	Pneumonia Death-Rates per 100,000.	Including Broncho-Pneumonia.		Lobar and Undefined Pneumonia.	
1907	2,386	27·8	18,104	210·7	These forms of pneumonia were not distinguished till 1914.			
1908	1,595	18·2	14,852	169·2				
1909	1,122	12·5	16,597	185·1				
1910	1,452	15·9	17,115	187·0				
1911	1,736	18·6	16,460	176·2		Rates per 100,000.		Rates per 100,000.
1912	1,057	11·1	16,537	173·4				
1913	1,384	14·2	16,530	169·9				
1914	910	9·2	15,510	156·4	6,720	67·7	8,790	88·7
1915	1,414	14·0	17,209	170·2	7,208	71·2	10,001	99·0
1916	2,267	22·0	17,314	168·1	6,942	67·4	10,372	100·7
1917	1,694	16·1	18,673	178 0	6,604	63·0	12,069	115·0
1918	26,676	249·7	35,050	328·1	10,571	99·0	24,479	229·2

Prior to 1906 influenza did not appear under a separate heading in the mortality statistics of the State, but it was estimated that in an epidemic of the disease which occurred in 1899–1901, 11,500 deaths resulted, and in another epidemic in this State in 1903–4, at least 10,000 persons died of influenza and its complications. Influenza was present in New York State and in the City of New York in the early months of 1918, especially during the months of March and April, 268 deaths from influenza (and 3,308 from pneumonia) being registered in March, and 216 (and 2,844) in April. During May, June, July, and August, the fatal cases of these diseases were comparatively few, but in September there was a considerable increase, the epidemic reaching its height in October, continuing in reduced amount during November and December. The fact that an influenza epidemic was developing was recognised by the State Board of Health during the latter part of September. The fatal

incidence of epidemic influenza and pneumonia in the State of New York, and in the City of New York, month by month, during 1918, is shown in the next table :—

The Bulk of these Figures are taken from the New York State Official Bulletin for November 1918.

Table showing the Number of Deaths from Influenza and Pneumonia in the State of New York and in the City of New York, Month by Month, during 1918 *and the First Half of* 1919.

	New York State (including the City).		New York City.	
	Deaths from		Deaths from	
	Influenza.	Pneumonia.	Influenza.	Pneumonia.
1918.				
January	148	2,316	72	1,472
February	139	2,129	52	1,304
March	268	3,308	133	2,296
April	216	2,844	106	1,747
May	31	1,416	36	855
June	27	713	7	445
July	20	601	6	354
August	10	487	5	313
September	272	913	114	470
October	17,690	13,556	8,385	7,137
November	5,306	4,105	2,729	2,669
December	2,499	2,652	916	1,519
1919.				
January	3,405	3,771	1,967	2,562
February	1,933	2,845	1,311	2,055
March	1,382	2,821	875	1,818
April	560	1,903	321	1,471
May	307	1,185	148	726
June	83	563	38	351

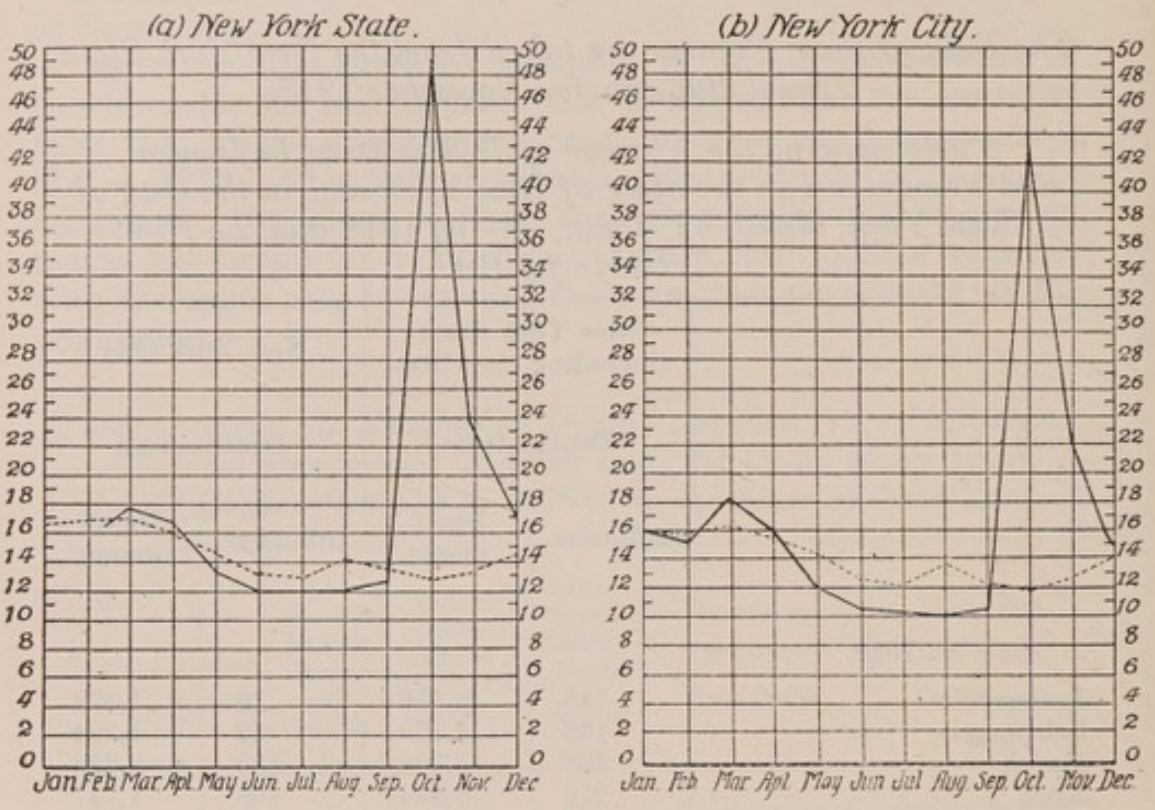
The notification of influenza was made obligatory in this State on 11th October 1918 and came into force on 14th October.

The influence of the epidemic upon the general death rate (1) of the State, and (2) of the City of New York is shown in the appended curves which are taken from the Official Bulletin of the New York State for January 1919.

The effect of the influenza epidemic upon the general death rate in (*a*) the State of New York and (*b*) New York City is shown for 1918 in the appended charts.

General Death Rate 1918 (*Months*) *compared with the Mean of* 1913–17 *in New York State and in New York City.*

——— = 1918. ..... = 1913-17.



The *New York State Medical Bulletin* for 1st August 1919 states that from July 1918 to June 1919 it is estimated the cumulative excess of mortality in the State, directly chargeable to the influenza epidemic was nearly 52,000 deaths, not including others which have resulted later from damage done to the system during the course of the epidemic.

The New York State Influenza Commission made a special study of the incidence of influenza during 1918 as regards age and sex in two urban districts within the state, namely, Oswego and Watertown, each of which has a population of about 26,000. It was not possible to obtain details for the whole of these populations, but in Oswego the facts concerning 2,259 families comprising 12,952 individuals were carefully inquired into, and in Watertown the facts as regards 5,068 families including 20,473 individuals were closely investigated. It may be mentioned that near Oswego there were situated army barracks which received many cases of influenza that occurred among the military. The survey in Oswego was begun in November and completed by the middle of December; and that in Watertown, begun on 19th December, was completed on 7th January. It was found that neither sex nor any age group was disproportionately represented, allowance being made for male adults absent on war service. The following figures represent the results obtained by the intensive study of the influenza cases in Oswego:—

*The Incidence of Influenza and Pneumonia (all Forms) among an enumerated Population in Oswego.**

Age Groups.	Males.				Females.				Both Sexes.			
	Population.	Cases.	Deaths.	Fatality Rates.	Population.	Cases.	Deaths.	Fatality Rates.	Population.	Cases.	Deaths.	Fatality Rates.
				Per cent.				Per cent.				Per cent.
All ages	6,118	2,897	65	2·2	6,834	3,197	81	2·5	12,952	6,094	146	2·4
Under 5 years	781	378	14	3·7	747	383	14	3·7	1,528	761	28	3·7
5–14 years	1,233	736	3	0·4	1,273	724	6	0·8	2,506	1,460	9	0·6
15–19 „	524	309	5	1·6	595	359	8	2·2	1,119	668	13	2·0
20–39 „	1,770	933	33	3·6	2,239	1,143	47	4·1	4,009	2,076	80	3·9
40–59 „	1,385	467	7	1·5	1,468	492	4	0·8	2,853	959	11	1·2
60 and over	425	74	3	4·1	519	96	2	2·1	937	170	5	2·9

*The next Table gives the same Details for the enumerated Population of the Town of Watertown.**

All ages	9,626	2,626	87	3·3	10,847	3,139	95	3·0	20,473	5,765	180	3·1
Under 5 years	1,084	332	10	3·0	1,114	347	15	4·3	2,198	679	25	2·7
5–14 years	1,542	577	7	1·2	1,733	660	10	1·5	3,275	1,237	17	1·4
15–19 „	781	241	7	3·0	913	325	3	0·9	1,694	566	10	1·8
20–39 „	2,043	936	53	5·7	4,011	1,242	44	3·6	7,054	2,178	97	4·5
40–59 „	2,481	476	6	1·3	2,249	475	20	4·2	4,730	951	26	2·7
60 and over	695	64	4	6·3	827	90	1	1·1	1,522	154	5	3·3

* These figures are taken from a Report by Gladdon W. Daker in the *New York State Official Bulletin of the Department of Health* for 1st May 1919.

It is believed that the results in the enumerated populations may be taken as typical of the whole.

Up to the beginning of December it was estimated that 257,758 persons in New York State, not including New York City, had been attacked by influenza since September.

In addition to making influenza a notifiable disease, a force of doctors and nurses was organised for despatch to stricken communities in the State, and soon 126 nurses and 37 medical men were at work in the field furnishing and assisting in organising relief for the sick and unattended under the direction of the Department of Public Health of the State. Temporary hospitals were opened in many places where local hospitals either did not exist or where their accommodation was overtaxed. The preparation of food and its transport by motor cars was arranged to supply invalid households unable to cook for themselves.

An emergency housekeeping service was organised, and the care of children whose parents were ill was undertaken. These and other useful functions were taken over by the Red Cross chapters, branches, and auxiliaries in many parts of the State. A commission for the study of the causes, prevention, and treatment of influenza was appointed by the Governor of the State, of which Dr. Herman Biggs, Commissioner of the New York State Department of Health, was chairman, who in the early part of October issued the following poster :—

"*Epidemic Influenza.*

"Help to protect yourself and your town from a serious epidemic.

"It is unlawful to cough or sneeze without turning the face away from others and covering the nose and mouth with a handkerchief, or to spit on the floor of any public place or public conveyance, or on the sidewalk.

"A violation of this regulation is a misdemeanor, punishable by a fine or imprisonment, or both.

"Epidemic influenza is conveyed solely from the discharges from the nose and throat.

"Help to enforce the law. Save yourself and others.

"Herman M. Biggs, M.D.,
"State Commissioner of Health."

Copies could be obtained in reasonable quantities on application to the State Department of Health, Albany.

As regards the advisability of school closure to limit the spread of epidemic influenza, Dr. R. S. Cleveland, of New York City, has stated that the most important part of disease control is the public school system. Seven-tenths of the school children in New York City come from tenement houses, the poorest homes on earth. "It is better that these children should be permitted to go to school when the schools are in good sanitary

state, and are under strict regulation of the every-day system of disease control, than to allow them to linger in the school yards or in the basements." It is, he says, better for those children to be attending school than to be at home under unpleasant and unhygienic surroundings.

If any child shows symptoms or signs indicating possible influenza the child is put in a room by himself until examined by a nurse or a doctor to discover whether he has influenza. If found to be suffering from the disease the child is sent home under care of the board of health, whose medical officer or nurse finds out whether the family has a medical attendant, and if so whether isolation of the case is feasible, to protect other members of the family; if not, the child is removed to hospital. It is far better to care for children in this way than to turn them loose on the streets to play where they like.

On 10th January 1919 the Public Health Council added epidemic influenza to the list of diseases requiring exclusion from schools and gatherings, exclusion being for such time and under such conditions as may be permitted by the local health authorities.

Respecting the closing temporarily of moving-picture houses, theatres, and other crowded places of assembly, the line taken by the State Department of Health was that though sometimes this closure was imperative it should usually be determined by the outlook of the epidemic, and by the attention which the managers and frequenters of these places of entertainment paid to the requirements of health of the public; for example, by free ventilation and perflation, cleanliness, &c. Daily medical inspection of employees in factories, large stores, offices, &c., was recommended, the thermometer being used in every suspicious case. Maintenance of a high standard of health was urged through fresh air, avoidance of dust, suitable food, adequate clothing, proper exercise, and "temperance in all things."

At the time of writing the Influenza Commission appointed by the Governor of the State of New York had not reported, but investigations were being carried out in various directions. Ultimately, it is expected, valuable results will be obtained for further guidance in future influenza epidemics on such points as the bacteriology, protective vaccines, probable point or points of entry of the infection, mode of spread, types of the disease, effects of age, sex, nationality, morbidity and mortality of the malady, use of masks, &c.

Influenza in the City of New York.

For 20 years or more before the beginning of the recent epidemic deaths from influenza had been annually recorded in the city of New York. The following table gives the number

of deaths attributed year by year to influenza from 1899 to 1917 :—

——	1899.	1900.	1901.	1902.	1903.	1904.	1905.
New York City. Influenza deaths - -	461	612	856	157	418	501	311

——	1906.	1907.	1908.	1909.	1910.	1911.
New York City. Influenza deaths - -	241	714	403	335	336	486

——	1912.	1913.	1914.	1915.	1916.	1917.
New York City. Influenza deaths - -	269	350	336	526	853	657

The disease, therefore, might be regarded as having been endemic in the city, but whether, again, this was the same malady as that which was epidemic in the latter part of 1918 it is not possible to say. It is, however, noteworthy that the deaths attributed to influenza were above the average in the years 1915, 1916, and 1917.

The New York epidemic of 1918 began approximately about 12th September, and, judged by the mortality, reached its highest point about 22nd October, the total deaths recorded in 1918 from influenza amounting to 12,560, and there were in addition 20,628 others from pneumonia. Of these numbers 12,030 from influenza and 11,325 from pneumonia occurred in the months of October, November, and December. A later report states that from 14th September 1918 to 10th March 1919 the deaths from influenza reached a total of 15,000, those from broncho-pneumonia 6,600, and from lobar pneumonia 10,775, making a grand total of 32,375 deaths from these causes during the above-named epidemic period.*

During the epidemic a bacteriological examination of 145 cases in the Presbyterian Hospital, New York, enabled study to be made by naso-pharyngeal cultures and blood cultures; sputum was examined for pneumococcal types, as also other material obtained post mortem. Pfeiffer's bacillus was found to predominate in the naso-pharyngeal cultures, pneumococcus III. in the blood cultures, and pneumococcus IV. in sputum and

* *Monthly Bulletin* of the Department of Health, City of New York, for March 1919.

lung cultures. The percentage of naso-pharyngeal cultures containing B. influenzæ was somewhat higher among the uncomplicated cases than among those in which pneumonia had supervened. At one stage of the investigation Pfeiffer's bacillus was found in 75 per cent. of the naso-pharyngeal cultures. The pneumococcus IV. strains isolated have been found to belong to various groups. The blood of influenza convalescents has been found to contain agglutinins for strains of influenza bacilli isolated during the recent epidemic.*

A report furnished to the Department of Health by Dr. Anna W. Williams, Assistant Director of the Bureau of Laboratories, gives the following conclusions on the bacteriology of influenza :—

"1. The evidence in favour of the influenza bacillus being the initiating cause of the present pandemic of influenza is strong, but does not constitute proof.

"2. No evidence as strong has been brought forward in favour of any other cause.

"3. Whether or not the B. influenzæ is the initiating cause, it has given evidence that it has a marked pathogenic action, and the study of a vaccine to prevent this action should be continued until we are at least sure that it has no marked effect."

Of 33,188 deaths in the City of New York during 1918 from influenza, lobar pneumonia, and broncho-pneumonia, the age and sex distribution was as follows :—

	Influenza.		Lobar Pneumonia.		Broncho-pneumonia.	
	Males.	Females.	Males.	Females.	Males.	Females.
Under 5 years -	744	752	1,126	975	1,916	1,631
5 to 9 „ -	180	257	152	196	106	126
10 „ 14 „ -	124	209	136	178	38	70
15 „ 19 „ -	359	351	314	280	78	72
20 „ 24 „ -	698	984	601	694	195	182
25 „ 29 „ -	1,235	1,390	950	927	262	295
30 „ 34 „ -	1,078	961	943	697	254	187
35 „ 39 „ -	628	504	778	421	164	126
40 „ 44 „ -	361	268	531	270	80	79
45 „ 49 „ -	246	201	465	223	101	62
50 „ 54 „ -	166	153	397	216	62	55
55 „ 59 „ -	112	104	324	204	82	77
60 „ 64 „ -	87	89	273	193	73	86
65 „ 69 „ -	53	83	224	211	67	86
70 „ 74 „ -	45	41	173	169	61	82
75 „ 79 „ -	16	38	103	114	49	63
80 and upwards -	11	32	77	106	37	83
Total - -	6,143	6,417	7,567	6,074	3,625	3,362

* Paper by Miriam Olmstead in the *Proceedings of the Society for Experimental Biology and Medicine*, 20th November 1918.

The progress of the influenza epidemic in New York City, week by week, is shown in the appended table:—

Week ended	Influenza.		Pneumonia, all Forms.		Total Deaths all causes.	General Death Rate.
	Cases.	Deaths.	Cases.	Deaths.		
1918						
28th September -	1,304	42	274	145	1,210	10·7
5th October -	7,200	299	966	424	1,793	15·9
12th ,, -	21,060	979	1,928	1,142	3,308	29·3
19th ,, -	30,341	2,218	3,508	2,009	5,666	50·3
26th ,, -	22,353	2,971	5,153	2,251	6,783	60·2
2nd November -	26,574	2,547	4,640	1,855	5,819	51·7
9th ,, -	6,057	—	1,576	1,074	3,566	31·6
16th ,, -	2,317	—	716	568	2,282	20·2
23rd ,, -	1,247	—	300	365	1,797	15·9
30th ,, -	841	175	280	249	1,577	14·0
7th December -	824	158	264	228	1,596	14·1
14th ,, -	1,119	160	380	318	1,606	14·7
21st ,, -	2,049	339	485	419	1,707	15·1
28th ,, -	1,687	259	396	386	1,803	16·0
1919.						
4th January -	2,341	307	619	434	1,978	17·8
11th ,, -	2,525	351	652	519	2,092	18·1
18th ,, -	3,310	428	844	575	2,431	21·1
25th ,, -	3,988	540	917	653	2,537	22·3
1st February -	3,067	538	981	623	2,448	21·2
8th ,, -	1,755	392	665	501	2 209	19·1
15th ,, -	1,438	300	526	486	2,090	18·1
22nd ,, -	1,488	267	480	521	2,129	18·4
1st March -	1,451	325	504	339	2,157	18·7
8th ,, -	1,132	255	448	592	2,084	18·1
15th ,, -	956	237	414	425	1,954	16·9
22nd ,, -	724	164	369	380	1,815	15·7
29th ,, -	628	162	329	354	1,751	15·2

During April, May, and June 1919, the number of cases and deaths from influenza and pneumonia in New York City continued to decline, and up to the end of 1919 there had been no marked recurrence of the epidemic.

There was a general impression in official circles in New York that vaccines, so far, had not proved helpful in preventing the disease.

The overcrowding of tramcars and suburban railway carriages was believed to have acted as a means of spreading influenza in New York. In order to reduce this overcrowding of public conveyances, an arrangement was made whereby the directors of different places of business, employing a large number of workers, opened and closed their establishments at different times, thus reducing the congestion of cars and railway carriages in the mornings and evenings.

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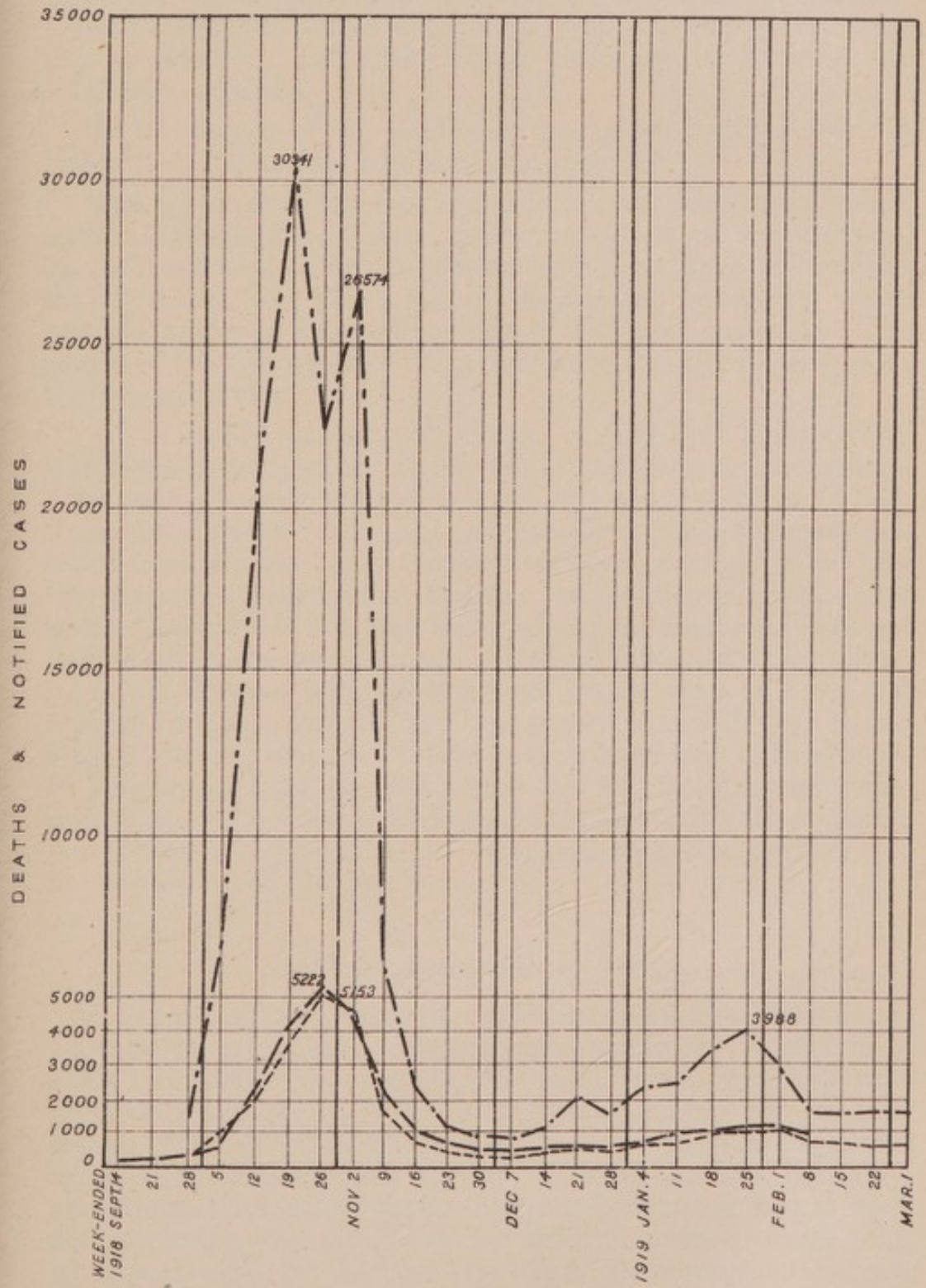
NEW-YORK.

NOTIFIED CASES OF INFLUENZA.
NOTIFIED CASES OF PNEUMONIA.
DEATHS FROM INFLUENZA AND PNEUMONIA (ALL FORMS) COMBINED.



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One of the features of the 1918 epidemic of influenza in the city was the high mortality at all the age groups under 45 years of age. The number of influenza and pneumonia deaths occurring during the 28th year of life were 1,013 (to 23rd November), 1,817 in the 29th year, and 816 in the 30th year. From 21 to 31 years of age there were 3,530 deaths among males in the above period, giving a yearly death rate of 41·5 per 1,000 of those living in New York at this age group. From the beginning of the epidemic about 15th September till 11th October, the males were considerably more affected than the females, but after 12th October the disparity between the sexes was equalised.

At the height of the epidemic, that is, on 20th October, no fewer than 809 deaths were recorded that day.

The epidemic in the city began in mid-September and lasted until mid-November. After an interval of about seven weeks, a second wave of influenza passed over New York and raised the mortality, but the peak of the second prevalence never reached anything like the height of the first outbreak. The course of the two waves of the influenza epidemic can be seen in the appended chart.

Up to mid-November 202 deaths occurred in childbed, in which the primary cause was given as influenza or pneumonia. A statement became current that the Chinese population of New York had suffered much less in proportion from influenza than other nationalities. At a Christian Science meeting in the city the statement was repeated and the explanation was given that as the Chinese did not read the newspapers they could not get frightened at the depredations caused by influenza, their escape from the disease being due to their placid state of mind and absence of fear. But the city Department of Health then issued a report stating that the Chinese in New York had suffered more in proportion than the Americans, for the mortality rate from influenza and pneumonia among the Chinese during the epidemic period was 9·5 per 1,000, as compared with 3·4 per 1,000 of the population of the city.

Epidemic Influenza in Chicago.

"Influenza" as a cause of death had been appearing in the Chicago official records for some years before the pandemic of 1918. In 1913, for example, 85 deaths from influenza were registered, and 117 in 1914; in 1915 the number rose to 281, and in 1916 still further increased to 404. In 1917 the deaths ascribed to influenza fell to 201. During January, February, and March 1918, a few deaths, ranging from two to nine a week and amounting to a total of 67 for the first quarter of the year, were registered in the city. In April, there was an increase

though not to any great extent, in the number of deaths from influenza and pneumonia in Chicago, 80 fatal influenza cases being registered, and 732 from pneumonia. This may be taken as evidence that a mild wave of the disease lasting about four weeks passed over the city during April. January to April is the usual period for pneumonia prevalence year by year in Chicago and other parts of North America, as also at any other time when the weather is cold and wet, and when houses are shut up to exclude the fresh air. The following table gives the number of deaths from influenza and pneumonia during the 13 weeks' period from 3rd March to 1st June. The height of the peak both for influenza and pneumonia occurred during the fortnight ended 13th April.

Dr. Edwin R. Le Count of Chicago has stated publicly that in the first week of April 1918 he made several post-mortems, in which he found the lungs full of hemorrhages. The possibility of these being due to a new disease was considered, but he says it was not till the autumn that the true nature of the disease was recognised, when similar conditions were found in fatal cases during the epidemic of influenza.

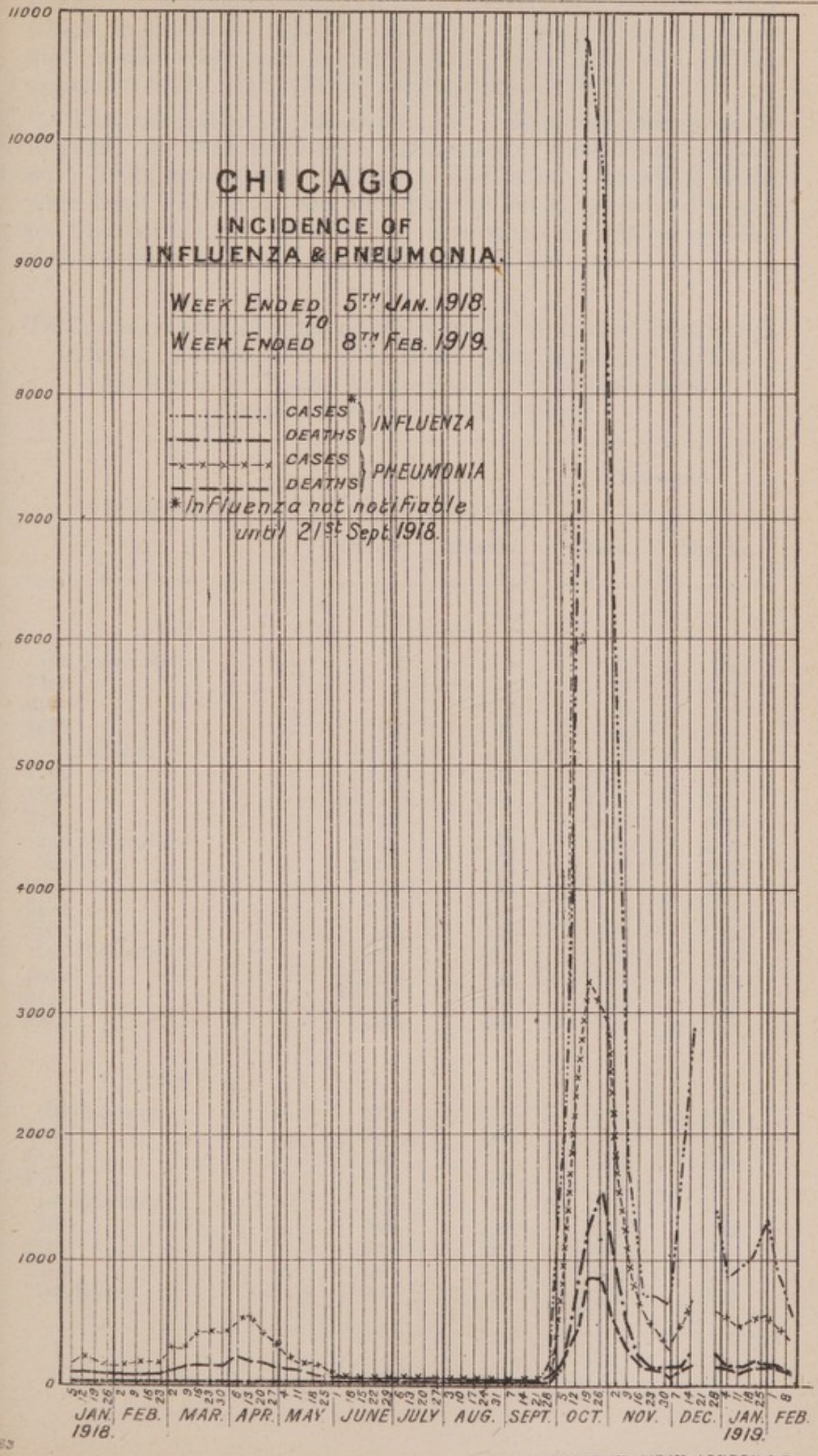
Table showing the Deaths Registered in Chicago from Influenza and Pneumonia, Week by Week, during the Months of March, April, and May 1918.

1918. Week ended	Deaths from	
	Influenza.	Pneumonia.
9th March	8	145
16th „	3	143
23rd „	6	162
30th „	8	158
6th April	24	218
13th „	23	193
20th „	20	185
27th „	13	136
4th May	—	108
11th „	9	104
18th „	6	73
25th „	1	55
1st June	2	37

The deaths from influenza in May amounted to 18, and in June to 5. In July a single fatal case was registered, with 5 in August. During September 61 influenza deaths occurred, the majority of them during the last 10 days of the month. The second wave of epidemic influenza in Chicago beginning towards

To face p. 310.

INFLUENZA & PNEUMONIA IN CHICAGO DURING 1918-1919.



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the end of September, reached its height on 19th October, after which it gradually subsided, though not altogether, for cases and deaths continued to be reported, as will be seen in the appended table, well into the early months of 1919.

The notification of pneumonia was in force in Chicago prior to 1918. In 1916 there were 9,303 cases of pneumonia notified, with 3,883 deaths, and in 1917, 11,864 attacks of which 5,018 were fatal. Influenza was added to the list of notifiable diseases on 21st September 1918 at the time when it was recognised that a serious epidemic was developing in the city.

Table showing the Number of Cases of Influenza and Pneumonia notified in Chicago, Week by Week, from September 1918 *to* 1st *March* 1919, *and the Deaths Certified from these Diseases during the same Period.*

Week ended	Influenza.		Pneumonia.	
	Cases.	Deaths.	Cases.	Deaths.
1918.				
7th September	1	1	26	17
14th „	1	1	27	15
21st „	7	1	34	24
28th „	193	17	153	74
5th October	2,210	171	988	246
12th „	7,743	571	2,327	476
19th „	10,791	1,242	3,275	863
26th „	9,747	1,541	2,968	826
2nd November	4,943	1,014	1,975	456
9th „	1,559	488	926	250
16th „	714	235	602	155
23rd „	728	146	462	130
30th „	641	148	264	69
7th December	1,716	159	487	103
14th „	2,869	269	672	158
21st „	2,722	337	916	159
28th „	1,360	281	567	158
1919.				
4th January	868	179	547	142
11th „	926	166	473	103
18th „	1,042	190	515	138
25th „	1,330	183	551	157
1st February	866	152	461	125
8th „	543	102	363	92
15th „	554	100	402	135
22nd „	498	102	482	131
1st March	480	92	451	138

There was no recurrence of the epidemic during the rest of 1919. For the last six months of the year the weekly number of influenza deaths averaged only three.

According to Dr. J. Dill Robertson, Commissioner of Health for Chicago, September 1918 was an unusually cold month and decidedly favourable for the occurrence of pneumonia. Thousands of occupiers, with coal supplies in their cellars, were unwilling to start warming their houses so early in the season, feeling it to be their duty to save the fuel as far as was possible. In this way thousands of Chicago's citizens and their families shivered in discomfort during September, but endured it from patriotic motives to help to win the war. It is probable that this discomfort may have predisposed individuals to fall victims to the influenza infection whenever they were exposed to it.

When the epidemic was assuming alarming proportions the majority of the Chicago hospitals set aside practically their whole establishments for the reception of influenza and pneumonia patients, the only surgical admissions being emergency cases. The U.S. Public Health Service sent a number of their assistant surgeons, who, among other duties, made surveys of factories and visited the homes of the sick reported to be suffering from influenza. The epidemic was locally compared to a prairie fire which, if left alone, burns itself out when there is no more material to consume. There is no doubt that the preventive measures carried out in Chicago helped, to some extent, to protect the human consumable material threatened by the epidemic. Complete reporting of the cases could not be enforced, as most of the medical men were overworked. The deaths occurred mostly in the age-groups from 20 to 40 years. Among the measures carried out to stop the spread of the infection was an effort by the police department to initiate an anti-spitting campaign. Warnings were issued to the citizens to keep their dwellings well warmed, and landlords failing to supply heating were reported to the police. Placards were posted up pointing out the dangers arising from sneezing, coughing, and spitting in cars and elevated railways. All street cars and elevated railways were required to be cleaned and disinfected at least once daily, windows to be kept open if weather permitted. The schools were strictly ventilated, and the parents of scholars were urged to have their children warmly clothed. Any of the pupils displaying any signs of illness were sent home at once. Attention was called to the advantage of using masks, especially in hospitals.

It had been the custom in Chicago before the epidemic to isolate cases of pneumonia, and this was continued during the epidemic, and applied as far as practicable to influenza, too. The visits of friends and relations to patients in the hospital

were stopped. The assistance of the public press in the dissemination of information regarding the disease and its prevention was secured. The police were requested to arrest "open" coughers and sneezers in the street or open places. Home nursing was provided by the Visiting Nurses' Association. The proper ventilation of churches was required. Public dancing halls were closed, public funerals were forbidden, and the attendance at funerals was limited to 10 persons. Theatres, skating rinks, picture palaces, night schools, and "lodge halls" were closed. The hours of beginning and leaving work at different factories and other places of business were regulated so as to prevent overcrowding of public conveyances at certain times in the mornings and evenings. The manufacture of a prophylactic vaccine under the supervision of a commission was begun in the city laboratory about the middle of October, the time when the epidemic had already reached its height. The vaccine in question appears to have been that recommended by Dr. E. C. Rosenow. At the end of October, the ban upon musical and other entertainments in restaurants was removed, and theatres were allowed to open again after they had been thoroughly renovated. "Two-minute health talks" were given at each performance. Early in November, public dancing halls, skating rinks, and "lodge halls" were allowed to re-open after inspection and approval by the health department. It will be seen from the table on page 311 that there was an increase in the number of influenza cases and deaths at the beginning of December, but whether this was due to the re-opening of places of amusement or to other causes, the information available does not afford grounds on which to form an opinion.

As has been stated already, the schools were not closed during the epidemic, the Commissioner of Health for Chicago being convinced that a better knowledge of existing conditions could be had when the schools were made a source of information as to sickness among the children and their families. It would, he says, be possible to supervise the children and keep many of them under better conditions during the school hours than if they were allowed to stay at home and run free in streets and alleys, or play on premises where sick persons were living.

In Chicago, as elsewhere, the serious effects of the influenza epidemic on pregnant women, especially when complicated by pneumonia, was noted. For instance, in one of the hospitals of the district, of 101 cases where pregnant women developed pneumonia during the epidemic, 52 died, giving a case mortality of 51·4 per cent., as compared with 719 deaths in 2,154 cases of pneumonia in non-pregnant women, giving a case mortality rate of 33 per cent.; 73 per cent. of the pregnant women died within 48 hours of admission to hospital. It is, therefore, concluded that the influenza death rate is materially higher in pregnant women than in those who are not pregnant. If abortion or miscarriage follow upon the influenza attack, the chances of recovery are very small. If, however, the attack

does not result in premature emptying of the uterus, there is a better prospect of recovery.

In this connection, the experience of two obstetricians, Professor Paul Titus and Dr. J. M. Jamison, in the Western Pennsylvania Hospital, Pittsburg, is of interest. These observers made a careful study of the effects of influenza in 50 pregnant women admitted for treatment during the autumn epidemic.* The total cases of influenza treated at this hospital was 950, in whom the case mortality was 22·3 per cent. But of the 50 pregnant women, 31, or 62 per cent., died. Of the 31 fatal pregnant cases, 14 died undelivered, as compared with 17 who either aborted or had premature labour; 13 of the pregnancies were of 5 months' duration or under, while 37 had lasted from 6 to 9 months. Of 10 of the infants born alive, one died. The ages of the 50 pregnant women ranged from 17 to 37 years. The conclusions drawn from this study were that the mortality from epidemic influenza in pregnant women is much greater than in non-pregnant women; the mortality, though high, (48·2 per cent.) without interruption of the pregnancy, was greatly increased by abortion, miscarriage, or premature labour, being then 80·9 per cent.

In July 1919, the Mayor of Chicago issued a proclamation to the people of the city, in which he reminded them that in September 1918 the epidemic of influenza had reached Chicago; that, although the suffering was "terrific" and the loss of life great, yet, thanks to the efficiency of the Health Department, Chicago's record was the best of the American large cities. So severe was this outbreak that it had taxed the nurses of the city far beyond their ability to care for the afflicted, thousands of persons being unable to obtain nursing service of any kind. The proclamation went on to remind the citizens that when, in 1889, influenza visited Chicago it remained an unwelcome visitor for more than 3 years, recurring each year after the initial outbreak. The Commissioner of Health of Chicago and other experts believed that the disease would behave as it did 30 years ago, and return in the autumn and winter of 1919. In preparation for this, it had been arranged to institute an eight weeks' free course of training for mothers, wives, and sisters, of Chicago, who desired to become proficient in home training. A Board of Directors had been appointed for this Nursing Institution, which was to be known as the "Chicago Training School for Home and Public Health Nursing." The first course was to begin on 4th August 1919. The mayor, in concluding his proclamation, added: "As we know, the lack of nurses during "the outbreak of influenza last year cost the people much "suffering and the city many lives, I, therefore, urge the "mothers, wives, and sisters to enrol for nursing service by "joining either the day or the evening classes of the institution, "so that they may be prepared not only to care for influenza

* *Journal of the American Medical Association*, 7th June 1919.

" patients, but be versed in the general home care and nursing " of those sick with any of the communicable diseases."

It may be added that up to the end of 1919, no fewer than 2,100 women had undergone an intensive course of instruction, lasting eight weeks, in the Chicago Training School for Home and Public Health Nursing, and were, therefore, regarded as being competent to lend assistance in home nursing in the event of a recurrence of the influenza epidemic.

In the *Times* of 22nd January 1920 there appeared a communication from its New York correspondent, dated 21st January, to the effect that influenza had re-appeared in epidemic form in the United States, and that it was " at its worst " in Chicago, where, in six days, 5,000 persons were attacked, and 36 died. Some theatres had been closed by the city authorities, and some others were under notice to improve their ventilation forthwith.

The District of Columbia.

In the District of Columbia lies the City of Washington, the capital of the United States. The district has an area of 60 square miles and an estimated population of 526,000, of which about one-third are coloured persons. During February and March 1918 there was a prevalence of influenza in the district, but it was of a comparatively mild type, few deaths occurring. After an interval of about five months a more severe wave of influenza swept over the District of Columbia, attaining its height in the week ended 19th October, when 527 fatal cases of influenza were certified, with 97 of pneumonia, the total number of deaths from all causes being 775, giving an annual death rate of 101·8 per 1,000 of the population, the highest rate ever recorded within the memory of the officials of the health department of the district. This prevalence lasted five or six weeks.

A third wave began in the third week of December, and lasted till the third week of January 1919, but it was much milder in character than the second wave.

Influenza was not notifiable in the District of Columbia prior to the 1918 epidemics, but in previous years deaths continued to be certified from this cause, as may be seen in the following table :—

Deaths Certified from Influenza and Pneumonia in the District of Columbia during the Seven Years 1911 *to* 1917 *inclusive.*

——	1911.	1912.	1913.	1914.	1915.	1916.	1917.
District of Columbia.							
Deaths from influenza -	67	62	49	65	114	112	106
„ „ pneumonia	588	525	484	468	671	604	636

In the year 1918 the influenza deaths amounted to 1,944, and those from pneumonia 3,318.

From the above table it appears that in 1915 there was a marked increase in the number of deaths certified from influenza and from pneumonia, and the number of deaths from these diseases remained above the average during 1916 and 1917.

The appended table gives for the District of Columbia, week by week, the number of deaths certified from influenza and pneumonia, and from all causes, together with the general death rate from all causes, for each week of the year 1918 and the first three months of 1919 :—

Deaths from Influenza, Pneumonia, and from all Causes, together with the General Death Rate Week by Week in the District of Columbia during 1918 *and First Quarter of* 1919.

Week ended	Deaths from Influenza.	Deaths from Pneumonia.	Deaths from all Causes.	Death Rate from all Causes.
1918.				
5th January	—	26	151	19·3
12th „	3	26	148	19·4
19th „	—	31	156	20·5
26th „	—	31	150	19·7
2nd February	9	39	161	21·1
9th „	4	43	161	21·1
16th „	2	30	150	19·7
23rd „	3	21	146	19·2
2nd March	—	28	140	18·4
9th „	2	23	147	19·3
16th „	3	34	177	23·2
23rd „	—	49	206	27·1
30th „	5	45	186	24·4
6th April	4	24	145	19·0
13th „	—	28	156	20·5
20th „	—	29	179	23·5
27th „	—	12	150	19·7
4th May	—	19	161	21·1
11th „	—	15	144	18·9
18th „	—	9	107	14·1
25th „	—	20	110	14·4
1st June	—	11	111	14·6
8th „	—	11	119	15·6
15th „	—	4	108	14·2
22nd „	—	2	129	17·0
29th „	—	7	114	15·0
6th July	—	3	115	15·1
13th „	—	5	129	16·9
20th „	—	7	135	17·7
27th „	—	4	148	19·4
3rd August	—	7	128	16·9
10th „	—	5	171	22·5
17th „	—	2	104	13·7
24th „	—	4	88	11·6
31st „	—	5	95	12·5

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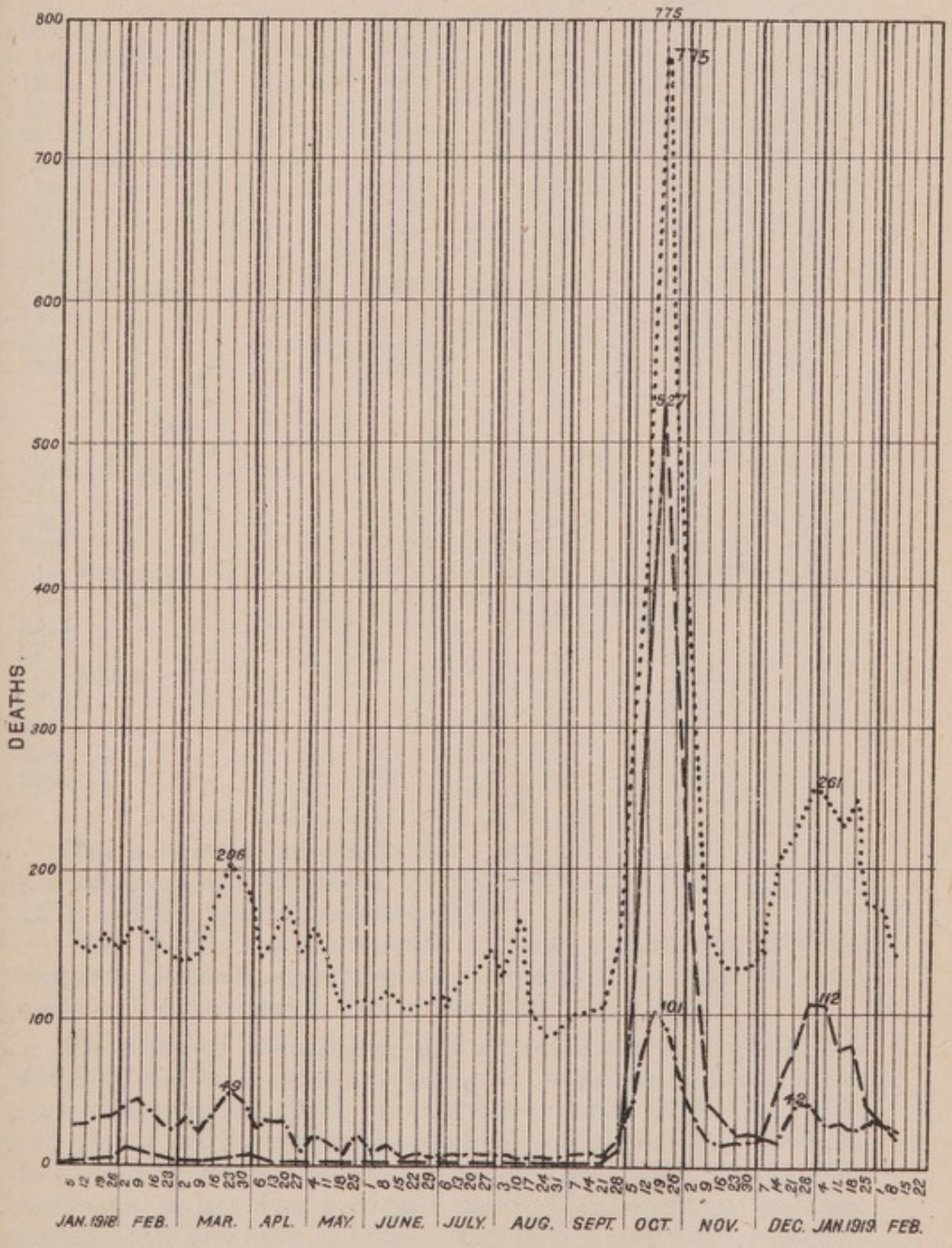
DISTRICT OF COLUMBIA.

DEATHS FROM INFLUENZA, PNEUMONIA AND ALL CAUSES DURING 1918 AND BEGINNING 1919

— — — INFLUENZA.

—.—.— PNEUMONIA.

..... ALL CAUSES.



H. & C. GRAHAM LTD LITHRS LONDON, S.E.

Week ended	Deaths from Influenza.	Deaths from Pneumonia.	Deaths from all Causes.	Death Rate from all Causes.
1918.				
7th September	—	7	103	13·5
14th „	—	8	105	13·8
21st „	1	8	108	14·2
28th „	12	17	142	18·6
5th October	116	57	306	40·2
12th „	387	101	514	80·6
19th „	527	97	775	101·8
26th „	326	61	505	65·3
2nd November	145	36	302	39·7
9th „	41	16	161	21·1
16th „	29	13	141	18·5
23rd „	22	16	139	18·3
30th „	23	19	143	18·8
7th December	20	21	149	19·6
14th „	58	15	213	28·0
21st „	80	40	231	30·3
28th „	112	42	261	34·2
1919.				
4th January	110	29	256	31·9
11th „	80	29	239	29·8
18th „	83	24	252	31·4
25th „	42	31	182	22·7
1st February	31	29	179	22·3
8th „	20	22	150	18·6
15th „	11	29	149	18 6
22nd „	—	21	141	17·6
1st March	8	27	163	20·3
8th „	6	32	156	19·4
15th „	11	15	140	17·4
22nd „	3	23	152	18·9
29th „	4	26	114	14·2

The annexed chart shows the mortality curves of influenza, pneumonia, and "all causes" in the District of Columbia during 1918 and the first two months of 1919.

The deaths from influenza in the district from March onwards became comparatively trivial, and only 10 were registered from June to December 1919.

The City of Washington has a population of a little over 400,000, and in it influenza began to be epidemic towards the end of September 1918. In the week ended 28th September the deaths attributed to the epidemic numbered 34; the wave lasted about six weeks, during which time 1,889 deaths were attributed to it. This epidemic reached its height in the week ended 19th October, in which 622 fatal cases were reported. After this, deaths continued to be reported during the next five weeks, *i.e.*, from 2nd November to 7th December, but not in

large numbers, until the second week in December when another wave of influenza appeared lasting six weeks, during which time 715 more deaths were registered from influenza and pneumonia. From 8th September 1918 to 8th February 1919 the deaths attributed to the epidemic of influenza amounted to 2,994.

It is not possible, considering the space that is available, to attempt to deal in detail with each state or large town in the United States as regards the incidence of epidemic influenza in them during 1918, but the particulars already given may be taken generally as representing what occurred elsewhere. In the following table is given the number of deaths due to influenza and pneumonia week by week in 46 large American cities from 8th September 1918 to 15th March 1919, a period of 27 weeks. The table also, in addition to the names of the cities, gives the State in which they are situated and their estimated population. In these 46 cities the deaths due to the epidemic during the above-mentioned period amounted to more than 142,631 (*see* pp. 319–320).

It has been suggested that the figures in the table do not actually represent all the fatal results of the influenza epidemic, for many other deaths occurred in persons suffering from chronic maladies, such as Bright's disease or cardiac affections, the fatal termination of which was undoubtedly hastened by the advent of influenza.

The incidence of fatal influenza and pneumonia during the epidemic was high among medical men and other attendants on the sick. For example, in the Weekly Journal of the American Medical Association for 26th October, notes are given of the deaths of 44 medical men that had occurred from the epidemic since the previous week's issue; and in the number of the same Journal for 4th January 1919, a summary is given of the deaths, from all causes, of medical men in the United States and Canada during 1918; of the total such deaths amounting altogether to 2,616, 428 were attributed to influenza and pneumonia, and 375 others to pneumonia alone. During 1919, the deaths reported among medical men in the United States and Canada from "influenzal pneumonia" numbered 313, out of a total of 2,105 from all causes.

The experience of the city of San Francisco in the matter of masks as a preventive of epidemic influenza is interesting though by no means conclusive. A "mask ordinance" was passed on 24th October 1918, and on the 25th, 90 per cent. of the population were using masks. The ordinance remained in force until 21st November. The epidemic in San Francisco reached its height during the week ended 2nd November when the fatal cases amounted to 738 falling in the following week to 414, and the week after that to 198. In the week ended 23rd November the deaths from influenza and pneumonia had still further fallen to 90 and in the following week to 56. In

Deaths Registered as due to Influenza and Pneumonia (all forms) combined, in certain Cities of the United States, from 8th September 1918 to 15th March 1919 inclusive, by Weeks.

(Figures taken from the *Weekly Health Index*, issued by the Bureau of the Census.)

City.	Population 1st July 1918 Estimated.	1918. Week ended															1919. Week ended											Total in 27 Weeks.
		September.			October.				November.					December.				January.				February.				March.			
		14th	21st	28th	5th	12th	19th	26th	2nd	9th	16th	23rd	30th	7th	14th	21st	28th	4th	11th	18th	25th	1st	8th	15th	22nd	1st	8th	15th	
Albany, N.Y.	112,565	—	—	—	—	45	110	186	155	52	20	4	14	7	11	11	13	12	12	8	11	18	15	16	10	12	11	12	765
Atlanta, Ga.	201,752	—	—	—	—	30	81	101	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	212
Baltimore, Md.	599,653	—	—	—	117	563	1,357	1,073	397	147	51	—	40	58	68	74	57	48	75	83	150	138	126	117	90	66	51	61	5,907
Birmingham, Ala.	197,670	2	2	5	16	61	110	133	85	46	46	44	72	90	—	129	53	36	44	52	41	29	21	28	—	—	—	—	1,145
Boston, Mass.	785,245	46	265	775	1,214	1,027	589	226	137	76	47	54	54	63	83	132	201	244	227	188	153	110	89	71	?	70	69	45	6,225
Buffalo, N.Y.	473,229	—	—	—	48	180	531	725	455	168	80	34	36	30	64	62	68	48	—	90	123	90	75	35	34	44	20	23	3,063
Cambridge, Mass.	111,432	—	—	105	140	115	63	21	19	5	9	9	7	6	14	17	26	39	22	20	16	13	10	3	6	2	4	4	695
Chicago, Ill.	2,596,681	16	24	91	417	1,047	2,105	2,367	1,470	738	390	251	217	262	418	496	439	321	269	328	734	277	194	235	233	230	213	232	14,014
Cincinnati, Ohio	418,022	—	—	—	18	67	192	281	248	163	97	105	94	149	208	163	83	51	18	18	26	23	39	37	78	90	107	101	2,456
Cleveland, Ohio	810,306	—	—	—	18	40	158	453	682	524	351	240	197	192	226	241	186	132	94	92	92	108	100	80	?	94	131	132	4,563
Columbus, Ohio	225,296	—	—	—	—	28	73	117	94	50	36	43	64	98	83	59	21	15	14	10	20	19	11	15	20	27	27	60	1,004
Dayton, Ohio	130,655	—	—	—	—	31	134	137	115	67	21	—	13	16	33	41	21	12	12	14	9	11	8	—	11	16	13	—	735
Denver, Colo.	268,439	—	—	—	—	59	139	147	108	101	77	108	132	184	201	163	86	65	47	35	—	—	—	—	—	—	—	—	1,652
Fall River, Mass.	128,392	—	9	20	97	201	192	97	40	24	14	10	7	17	5	14	18	10	18	16	14	17	17	15	—	13	12	14	911
Grand Rapids, Mich.	135,450	—	—	—	—	-	11	22	18	13	29	22	18	30	51	38	23	18	8	8	—	—	—	—	—	—	—	—	309
Indianapolis, Ind.	289,577	—	—	—	—	46	128	115	84	58	48	62	100	72	66	43	48	34	40	25	—	—	—	—	—	—	—	—	969
Jersey City, N.J.	318,770	—	—	—	66	231	—	425	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	722
Kansas City, Mo.	313,785	—	—	—	37	96	168	193	197	138	80	64	97	178	248	171	83	49	50	68	45	58	40	51	46	55	47	43	2,302
Los Angeles, Cal.	568,495	—	—	—	—	69	131	293	382	309	300	196	167	125	134	141	117	99	151	178	177	104	47	21	8	14	8	13	3,184
Louisville, Ky.	242,707	—	—	—	—	92	180	181	69	58	39	35	62	55	91	55	37	22	20	21	30	20	19	19	37	34	88	112	1,376
Lowell, Mass.	109,081	—	—	32	93	141	116	84	30	8	8	11	2	10	10	8	—	13	—	20	26	11	—	18	4	13	9	5	672
Memphis, Tenn.	154,759	—	—	—	—	80	182	166	71	—	17	18	—	—	27	29	—	20	—	—	47	30	60	19	20	15	9	14	824
Milwaukee, Wis.	453,481	—	—	—	—	69	113	175	125	95	70	49	88	—	182	166	105	65	—	—	—	—	—	—	—	—	—	—	1,302
Minneapolis, Minn.	383,442	—	—	—	—	48	99	150	120	95	93	51	45	69	71	96	68	37	45	24	—	—	—	—	—	—	—	—	1,111
Nashville, Tenn.	119,215	—	—	—	—	129	193	127	54	53	15	16	23	26	28	29	22	20	17	21	21	17	15	16	23	19	18	11	913
Newark, N.J.	428,684	—	6	8	53	189	396	431	376	177	111	70	56	57	79	74	70	72	66	57	?	50	45	32	46	54	38	45	2,658
New Haven, Conn.	154,865	—	—	15	36	77	152	183	168	82	48	20	24	32	30	52	36	40	38	27	26	—	12	11	—	—	12	—	1,121
New Orleans, La.	382,273	—	—	—	29	144	624	682	333	158	76	37	43	42	68	69	56	94	141	202	201	125	58	49	44	30	27	30	3,362
New York, N.Y.	3,215,879	—	106	191	733	2,121	4,227	5,222	4,402	2,277	1,053	657	424	446	477	534	678	753	870	998	1,193	1,212	893	786	788	904	747	695	33,387
Oakland, Cal.	214,206	—	—	—	—	18	42	138	237	157	70	38	12	16	19	33	40	66	92	111	—	—	—	—	—	—	—	—	1,089
Omaha, Nebr.	180,264	—	—	—	—	68	160	147	94	—	48	38	34	92	155	101	57	25	25	17	—	—	—	—	—	—	—	—	1,061
Philadelphia, Pa.	1,761,371	—	—	—	706	2,635	4,597	3,021	1,203	375	164	103	93	102	105	143	127	142	194	229	259	308	262	232	231	207	183	164	15,785
Pittsburgh, Pa.	593,303	—	17	34	69	114	389	576	630	798	532	385	297	200	202	144	127	99	103	111	145	174	145	163	137	134	116	118	5,959
Portland, Oreg.	308,399	—	—	—	—	-	41	94	157	156	87	85	38	72	67	69	46	55	101	123	122	50	15	10	12	7	8	9	1,424
Providence, R.I.	263,613	—	—	—	99	186	255	218	135	65	36	33	23	34	39	45	64	47	59	62	61	35	30	28	14	21	36	22	1,647
Richmond, Va.	160,719	4	3	4	41	131	197	128	71	28	23	13	24	28	56	51	—	50	26	34	30	23	11	9	—	—	19	10	1,014
Rochester, N.Y.	264,856	—	—	—	—	36	102	213	209	104	52	40	37	49	65	87	73	59	26	17	21	12	16	16	—	10	19	15	1,278
St. Louis, Mo.	779,951	—	—	—	—	86	186	233	257	229	228	190	235	375	469	293	129	67	83	75	71	75	53	64	?	81	93	119	3,691
St. Paul, Minn.	257,699	—	—	—	—	61	75	57	102	109	135	88	69	68	65	64	41	39	25	14	—	—	—	—	—	—	—	—	1,012
San Francisco, Cal.	478,530	—	—	—	—	—	130	552	738	414	198	90	56	50	71	137	178	194	290	310	149	59	41	20	18	21	22	17	3,755
Seattle, Wash.	366,445	—	—	—	—	75	108	160	109	85	69	34	47	77	117	103	78	55	70	57	57	27	40	21	12	12	20	8	1,441
Spokane, Wash	157,656	—	—	—	—	4	19	—	19	71	46	30	34	54	76	24	24	10	—	17	—	—	—	—	—	—	—	—	428
Syracuse, N.Y.	161,404	—	—	38	139	219	253	140	68	28	23	7	7	—	9	11	8	8	13	4	14	18	10	10	—	—	16	9	1,052
Toledo, Ohio	262,234	—	—	—	—	9	49	138	115	86	46	42	28	36	49	84	53	19	15	19	20	16	6	11	21	14	23	28	927
Washington, D.C.	401,681	—	—	34	173	488	622	389	181	55	42	37	42	41	86	120	154	139	109	107	73	60	42	40	28	35	38	34	3,169
Worcester, Mass.	173,650	—	17	101	129	230	160	89	59	—	13	29	20	11	9	34	32	40	36	44	22	23	21	23	—	8	20	—	1,240
Total	—	68	449	1,453	4,558	11,386	19,939	20,806	14,818	8,442	5,038	3,492	3,192	3,619	4,635	4,650	3,846	3,483	3,565	3,924	4,199	3,360	2,586	2,321	1,971	2,352	2,274	2,205	142,631

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the middle of December there was a recrudescence of the epidemic which continued for about 6 weeks reaching its height in the week ended 18th January, during which 310 fatal cases were recorded. Dr. W. C. Hassler, the city health officer, proposed to enforce again the mask ordinance in December, but a very strong opposition developed. The Christian Scientists attending the supervisors' meeting strongly opposed the proposal, business men were also against the measure and the public generally said they did not want it. Appeals were issued by the Mayor, the Chamber of Commerce and by the Naval and Military authorities urging the people to wear masks voluntarily, but these appeals had very little effect, only about 10 per cent. of the public complying with the request. Among the local medical men there was great divergence of opinion as to the utility of the mask. The mask recommended by the authorities consisted of four folds of gauze covering the mouth and nose; but many of the people used masks made of two layers of butter-cloth, which were sold at 10 cents a piece. From the evidence gathered from various sources, it cannot be said that the efficacy of masks was demonstrated at San Francisco; they came into use at a time when the epidemic was almost at its height, and there is no proof that their use was the cause of the decline of the epidemic that subsequently occurred.

In a comparatively recent paper by Dr. W. H. Kellogg, Executive Officer California State Board of Health, and Miss Grace MacMillan, Bacteriologist in the State Hygienic Laboratory, published in the *American Journal of Public Health* for January 1920, the results of a number of experiments with masks are given. But the final conclusion of these two observers is that "masks have not been demonstrated to have " a degree of efficiency that would warrant their compulsory " application for the checking of epidemics."

Influenza among the American Indians.

During the period from 1st October 1918 to 31st March 1919, out of a total Indian population of 304,854, there were reported 73,651 cases of influenza with 6,270 deaths, giving a fatality rate of 8·5 per cent. The reporting of the attacks is probably incomplete but the figures given above show that the epidemic was severe among the American Indians. The mortality varied in different localities especially being high among the Indians of the Mountain States (Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah and Wyoming). O a total Indian population in these Mountain States of 91,475, the attacks numbered 32,285 and of these 3,553 or 11 per cent. died. The highest mortality occurred among the Indians in Colorado, Utah and New Mexico. These figures are taken from a statement furnished to the U.S. Public Health Service

by the Office of Indian Affairs, Department of the Interior.* A second wave of influenza developed among the Indian population in April 1919.

The following table gives the incidence of influenza and the mortality from it among the Indian population of the various States, from October 1918 to June 1919 :—

Cases of Influenza and Deaths among Indians in United States from 1st October 1918 to 30th June 1919.

State.	Population (Indians).	Influenza.		Case Mortality, Per Cent.
		Cases.	Deaths.	
Arizona	45,707	17,237	1,948	11·3
California	16,416	4,398	256	5·8
Colorado	1,222	490	59	12·0
Florida	585	66	10	15·1
Idaho	4,208	650	75	11·5
Iowa	356	125	9	7·2
Kansas	2,275	860	20	2·3
Michigan	1,097	50	2	4·0
Minnesota	12,003	2,052	122	5·9
Mississippi	1,253	649	61	9·4
Montana	12,079	2,132	139	6·5
Nebraska	2,834	861	60	6·9
Nevada	11,190	3,594	271	7·5
New Mexico	22,005	10,971	1,245	11·3
New York	5,982	800	80	10·0
North Carolina	2,343	781	37	4·7
North Dakota	9,216	2,349	120	5·1
Oklahoma	118,227	15,227	861	5·7
Oregon	4,355	1,097	94	8·6
South Dakota	23,890	8,559	755	8·8
Utah	1,704	482	77	15·9
Washington	10,315	2,021	172	8·5
Wisconsin	9,696	2,710	158	5·8
Wyoming	1,696	16	1	6·3
Total	320,654	78,177	6,632	8·5

* From the U.S.A. Public Health Reports for 17th October 1919.

Alaska.—During May 1919 a very serious epidemic of influenza was reported in Alaska, nearly the entire population of some localities being stricken at one and the same time. The situation in these places was said to have been desperate. Little medical or nursing attendance was available and to meet the emergency the Government of the United States dispatched from San Francisco on 4th June a steam vessel laden with medical supplies, doctors and nurses for Alaska, to succour the

* *U.S. Public Health Reports*, 9th May 1919.

suffering inhabitants of that region. The vessel was instructed to call at Port Townsend (Washington State) to take up more doctors and nurses.

The following table, compiled from the figures published in the U.S. Public Health Reports of August 15th, 1919, and giving the data furnished to the Central Health Authority by the medical officers of 37 States, shows the number of officially reported deaths from influenza and from pneumonia (all forms) in each state, together with the death rate from each of these diseases per thousand of the population and from the two combined :—

Influenza and Pneumonia Deaths in 33 States during 1918.

States.	Population July 1918.	Influenza Deaths.	Pneumonia Deaths.	Total Deaths from Influenza and Pneumonia.	Influenza Death Rate per 1,000 Population.	Pneumonia Death Rate per 1,000 Population.	Combined Death Rate from Influenza and Pneumonia per 1,000 Population.
Alabama	2,395,270	5,446	5,689	11,135	2·3	2·4	4·6
Arizona	272,034	577	2,226	2,803	2·1	8·2	10·3
Arkansas	1,792,965	3,029	2,399	5,428	1·7	1·3	3·0
California	3,119,412	13,268	5,268	18,536	4·3	1·7	5·9
Connecticut	1,286,268	7,418	3,204	10,622	5·8	2·5	8·3
Delaware	216,941	426	1,443	1,869	2·0	6·7	8·6
District of Columbia	401,681	2,028	1,298	3,326	5·0	3·2	8·3
Florida	938,877	3,113	1,675	4,788	3·3	1·8	5·1
Illinois	6,317,734	17,879	14,445	32,324	2·8	2·3	5·0
Indiana	2,854,167	6,219	5,609	11,828	2·2	2·0	4·1
Iowa	2,224,771	6,260	3,818	10,078	2·8	1·7	4·5
Kansas	1,874,195	2,639	6,052	8,691	1·4	3·2	4·6
Kentucky	2,408,547	9,181	5,302	14,483	3·8	2·2	6·0
Louisiana	1,884,778	5,925	4,026	9,951	3·1	2·1	5·3
Maine	782,191	2,546	1,972	4,518	3·3	2·5	5·8
Maryland	1,384,539	5,244	6,311	11,555	3·8	4·6	8·3
Massachusetts	3,832,790	11,100	19,354	30,454*	2·9	2·4	5.3
Michigan	3,133,678	6,797	?	6,797†	2·2	—	—
Minnesota	2,345,287	7,182	1,955	9,137	3·1	0·8	4·0
Mississippi	2,001,466	?	2,873	2,873‡	—	1·4	—
Missouri	3,448,498	9,677	7,337	17,014	2·8	2·1	4·9
Nebraska	1,296,877	1,014	4,169	5,183	0·8	3·2	4·0
New York	10,646,989	26,676	35,050	61,726	2·5	3·3	5·8
North Dakota	791,437	326	2,355	2,681	0·4	3·0	3·4
Ohio	5,273,814	14,988	13,042	28,030	2·8	2·5	5·3
Oklahoma	2,377,629	4,047	2,446	6,493	1·7	1·0	2·7
Oregon	888,243	1,315	1,192	2,507	1·5	1·3	2·8
Pennsylvania	8,798,067	50,951	28,056	79,007	5·8	3·2	9·0
Rhode Island	637,415	2,306	1,639	3,945	2·6	2·6	6·2
S. Carolina	1,660,934	1,710	6,667	8,377	1·0	4·0	5·0
S. Dakota	735,434	1,847	544	2,391	2·5	0·7	3·3
Texas	4,601,279	11,854	7,636	19,490	2·6	1·7	4·2
Utah	453,648	1,459	796	2,255	3·2	1·8	5·0
Vermont	366,192	1,728	433	2,161	4·7	1·2	5·9
Washington	1,660,578	4,154	1,628	5,782	2·5	1·0	3·5
Wisconsin	2,553,983	4,339	6,129	10,468	1·7	2·4	4·1
Wyoming	190,380	169	819	988	0·9	4·3	5·2

* Lobar pneumonia only. † Influenza only. ‡ Pneumonia only.

The States for which no figures are available are North Carolina, Virginia, West Virginia, Colorado, Georgia, Idaho, Montana, Nevada, New Hampshire, New Jersey, New Mexico and Tennesee.

Human Experiments in the Transmission of Influenza.

At Boston and at San Francisco extensive attempts were made to transmit influenza experimentally.* Sixty-eight men from the Naval Detention Camp at Deer Island volunteered for the experiment at Boston. Most of these volunteers gave no history of previous illness suggestive of an attack of influenza. Inoculations were made of pure cultures of the influenza bacillus, of secretions from the upper air passages of persons in the early stage of influenza; and suspensions of freshly isolated bacilli were introduced within the nasal cavity. Both filtered and unfiltered secretions from the air passage of typical cases of influenza in the active stage of the disease were inoculated by means of spraying and swabbing of the nose and throat. Volunteers were also placed in close proximity, for a few minutes, with each of 10 selected influenza patients, who were instructed to cough directly into the faces of the volunteers. Filtered secretions and the blood of influenza patients were injected subcutaneously into another group. In San Francisco the volunteers had not been previously exposed to influenza during the present epidemic, but they had been vaccinated with large doses of a mixed vaccine of influenza baccilli, pneumococci, and hemolytic streptococci. As a result of previous experiments, it was believed, however, that this vaccine was of no use in protecting against influenza. In this series of experiment suspensions of influenza baccilli were introduced into the nose. In one group the suspension was first filtered through a Berkefeld candle. Filtered, as well as unfiltered, emulsions of respiratory secretions from active influenza cases were introduced into the nose. Filtered emulsions were dropped into the eye and injected subcutaneously. Blood taken during the acute stages of influenza was injected subcutaneously. Not one of the volunteers in either of these two series of experiments developed influenza; three developed acute tonsilitis, and in two, cultures gave almost pure growths of hemolytic streptococci. It is somewhat surprising that there should have been failure in these attempts to transmit influenza, in view of the positive results published by Nicolle and Bailly.

Animal Experiments.

A preliminary report on "An Acute Respiratory Disease in "Monkeys produced by Inoculation with Bacillus Influenzæ"

* *Journal of the American Medical Association*, January 25th, 1919.

was published by Dr. Francis G. Blake and Dr. Russell L. Cecil, of the Bacteriological Laboratories, Army Medical School, Washington, in the *Journal of the American Medical Association*, of January 17, 1920. Their conclusions were that B. influenzæ can initiate in monkeys an acute infection of the upper respiratory tract which may be complicated by acute sinusitis, tracheobronchitis, and broncho-pneumonia; this disease appears to be identical with influenza in man. B. influenzæ, when injected intratracheally, will produce in monkeys a tracheobronchitis and broncho-pneumonia, the pathology of which appears to be essentially identical with that which has been ascribed to pure influenza bacillus infection of the lungs in man. In view of these facts, and the constant association of B. influenzæ with early uncomplicated cases of influenza, the above-named observers think that it is reasonable to infer that B. influenzæ is the specific cause of influenza.

Institutional Influenza.

An instance of an institutional outbreak of influenza is the following, which was reported in the *Public Health News* for June 1919, published by the Medical Department of the State of New Jersey. The outbreak in question appeared at the end of September 1918 in New Jersey State Village for Epileptics. The total population of the institution, including staff, amounted to 900. Up to November 27th, when the outbreak terminated, 422 inmates were attacked, of whom 67, or 15·8 per cent., died. Of the 67 deaths 34 were due to lobar pneumonia, 23 to broncho-pneumonia, 7 to myocarditis, 1 to pulmonary gangrene, 1 to meningitis, and 1 to exhaustion following upon an epileptic seizure.

The age incidence of the cases was as follows :—

Age Periods.	Number of Inmates.	Influenza.		Case Mortality per Cent.
		Cases.	Deaths.	
Under 5 years - - -	4	1	—	—
5 to 9 - - - -	27	16	2	12·5
10 to 14 - - - -	70	51	4	7·8
15 to 19 - - - -	89	65	9	13·8
20 to 24 - - - -	128	88	15	17·0
25 to 29 - - - -	108	63	11	17·4
30 to 34 - - - -	113	53	10	18·8
35 to 39 - - - -	101	40	6	15·0
40 to 49 - - - -	131	31	8	25·8
50 to 59 - - - -	86	13	1	7·6
60 and over - - -	43	1	1	—
Total - -	900	422	67	15·8

The sex distribution was as follows :—

——	Cases.	Deaths.	Case Mortality per Cent.
Males - - - - -	234	57	24·3
Females - - - - -	188	10	5·3
Total - - -	422	67	15·9

There is therefore a very marked incidence, and especially fatality, in the males as compared with the females, since the population of the institution contained 443 males and 457 females.

The most susceptible age for attack appeared to be from 10 to 20 years. A vaccine prepared from Pfeiffer's bacillus produced no immunity against the type of influenza that prevailed in this institution in 1918.

General Preventive Measures in dealing with the American Epidemic.

In a report presented in December 1918 by a committee on influenza, appointed by the American Public Health Association, it is stated that it has not yet been satisfactorily established whether the current influenza epidemic is identically the same disease as that which had been previously occurring in the United States and generally spoken of as "influenza." There are as yet no known laboratory methods by which an attack of "influenza" can be differentiated from an ordinary cold, or bronchitis, or other inflammation of the mucous membranes of the nose, pharynx, or throat. Epidemic influenza, this committee states, is believed to be due to "an undetermined "organism which causes an infection which lowers the resistance "of the body as a whole, and of the respiratory organs in particular." This allows of the invasion of other pathogenic micro-organisms. The most important complicating infections are due to the "influenza bacillus," different strains of pneumococci, and different varieties of streptococci, though some careful observers regard these organisms as the primary cause of influenza. In each case of influenza one or several of these micro-organisms may be present. It is not certain whether previous epidemics of so-called influenza were due to the same infection as that of 1918.

It is the opinion of this committee that epidemic influenza is spread solely through the discharges from the nose and throat of infected persons finding their way into the nose and throat of susceptible persons. This being assumed, it is hardly necessary to wait for definite discoveries by bacteriologists as to

the true cause of the disease, and action in the following directions is indicated, namely :—

> "I.—Break the channels of communication by which the infective agent passes from one person to another ;
> "II.—Render persons exposed to infection immune, or at least resistant, by the use of vaccines ; and
> "III.—Increase the natural resistance of persons exposed to the disease by augmented healthfulness."

As regards I., this can be done by preventing droplet infection, by controlling spitting and the infection of the hands, as also by removing the danger arising from contaminated drinking vessels ; also by supervision of food and drink, though the danger from these does not appear to be great.

Among the chief preventive measures recommended by the committee are compulsory notification, and the searching out of unreported cases by lay or professional assistants. Education of the public as to the disease, its modes of spread, and the means to prevent it ; the closure of places of public assembly is recommended, but schools require special consideration ; the services in churches should be reduced to the lowest possible number ; theatres, picture palaces, and the like, should be dealt with from the point of view of efficiency of their ventilation and general sanitation. Public funerals should be forbidden. The wearing of masks should be compulsory in hospitals, also for barbers and dentists during epidemic times.

The action taken by the United States Public Health Service when the epidemic was beginning to develop deserves special mention. This central health authority received at the beginning of the epidemic urgent calls from many localities for help, asking especially that medical and nursing reliefs should be despatched immediately to the stricken communities. To these all available regular medical officers of the service were sent at once, but the number fell far short of what was urgently needed, and unfortunately at the time the central authority had no nurses for such emergency service. In these circumstances the Surgeon-General called upon the Volunteer Medical Service Corps, the Red Cross, as well as the medical and nursing professions generally, and others, to render assistance in dealing with the influenza epidemic. It may be mentioned here that before the epidemic began a considerable number of medical practitioners and trained nurses had volunteered for military service, and that in consequence some localities had experienced great difficulty in obtaining adequate medical and nursing assistance in the earlier stages of the epidemic. These difficulties became more and more acute as the epidemic progressed. Meanwhile an appeal was made to Congress for a special appropriation to meet the expenditure arising from the emergency,

and to supply funds for controlling the epidemic. The necessary funds were promptly voted by Congress, a million dollars being appropriated on 1st October 1918 to cover the expenses up to June 1919 of combating and suppressing influenza, and to aid State and local boards of health, including pay and allowances of medical and sanitary personnel, medical and hospital supplies, printing, clerical services, transportation of freight, and all other expenses that might be necessary. Congress also authorised and directed respectively the Secretary of War, the Secretary of the Navy, and the Secretary of the Treasury, to utilise jointly the personnel and facilities of the Medical Departments of the Army and the Navy, as well as of the Public Health Service, in combating the current epidemic.

In response to the appeal for doctors by the Public Health Service, the Volunteer Medical Service compiled a list of 1,000 names classified by states. Appointments were then offered by telegraph to these doctors, and within 48 hours groups of medical men were on their way to some of the worst stricken communities in New England, where at the time the epidemic was raging very seriously. Soon after other medical units were despatched to New Jersey, New York, North Carolina, and to Arizona.

There was more difficulty in meeting the demand for nurses. Already most of the available trained nurses were hard at work. A few nurses and trained attendants were, however, secured by the American Red Cross and sent to localities that were in most urgent need of them. The use of intelligent volunteer female workers under trained supervision was considered, and in a number of communities the organisation of this group of nursing volunteers did a good deal to relieve the situation.

The Public Health Service at the outset made it perfectly clear that there was no desire to supersede the state and local boards of health, but to aid them in their difficult work. An officer of the medical staff of the Public Health Service was detailed to each state, in order to secure the best organisation and co-ordination of the health activities of the service.

As the epidemic extended and demands for help made on the Public Health Service increased great assistance in securing more medical men was given by the American Medical Association, and by the medical journals and newspapers, which published appeals to the doctors to volunteer for service in connection with the epidemic. By 22nd October over 600 medical men, as well as a number of other workers, nurses, clerks, &c., were on duty in various states. In some localities the few medical men in practice had been attacked by influenza, leaving their patients unattended and therefore in urgent need of medical help from without.

The general view of protection against influenza for individuals is summed up in characteristic fashion by the Publicity Officer of the North Carolina State Board of Health. After calling attention of the public to the fact that the methods advised by irresponsible persons for preventing influenza are no value (such, for instance, as carrying "buckeyes" in the right hand breeches pocket, or assafœtida bags worn about the neck) he goes on to remark :—

> "There are several institutions in North Carolina in which not a case of influenza occurred, and still the disease was epidemic on every side. The students in these institutions did not wear sulphur in their shoes, assafœtida in their bosoms, cucumbers on their ankles, or potatoes in their pockets. What they did was to use separate towels, dishes, and drinking cups, and keep their mouths covered when coughing and sneezing. They stayed away from public gatherings of all sorts, but were permitted on the streets, even when ambulances were frequently passing with patients for the influenza emergency hospitals. These students lived a normal life, happy and jubilant. They did not breathe filtered air, nor drink concoctions of native herbs, but used common sense."*

In February 1919 the Surgeon-General of the United States Army, the Surgeon-General of the United States Navy, the Surgeon-General of the Public Health Service, and the Director of the Census, designated officers from their respective departments to form a joint committee on influenza to study the recent epidemic, and to arrange the data regarding the disease collected by the different departments of the Government. The following members were nominated, namely—Dr. William H. Davis (chairman) and Mr. C. Sloane, representing the Bureau of the Census, Dr. Wade H. Frost and Mr. Edgar Sydenstricker, representing the Public Health Service, Lieut.-Commander J. R. Phelps and Surgeon Carrol Fox, representing the Navy, and Colonels D. C. Howard, F. F. Russell, and A. G. Love, representing the Army.

Statistical studies were commenced in 1919 by the Public Health Service concerning epidemic influenza in the United States during 1918. These studies included five main divisions, namely (1) special surveys to determine the incidence, rate, type, duration, and fatality of the disease in representative areas in different sections of the country in relation to colour, age, sex, domestic housing, and time of occurrence ; (2) detailed analyses of morbidity records available through state health departments to show chiefly the chronology, sex, and age distribution of reported cases ; (3) preliminary analyses of records of mortality

* Quoted by the *Wisconsin State Board of Health Bulletin*, Vol III. No. 4, 1918.

of the epidemic in a few states with reference to deaths in various civil sub-divisions by colour, sex, and age distribution, in successive periods of the epidemic; (4) a special inquiry into the nature and scope of preventive and relief measures adopted in larger cities to combat the epidemic; (5) in addition certain compilations and analyses are being made from published data, including current mortality statistics, from certain foreign countries, and as to mortality from influenza and pneumonia during previous years in the United States.

Dr. G. W. McCoy, Director of the Hygienic Laboratory of the United States Public Health Service, has summed up the case of prophylactic vaccination against influenza as follows:—

> "The general impression gained from uncontrolled use of vaccines is that they are of value in the prevention of influenza, but in every case in which vaccines have been tried under perfectly controlled conditions they have failed to influence in a definite manner either the morbidity or the mortality."*

Mexico.

Epidemic influenza is stated to have appeared in Mexico during the first week in October 1918, and to have spread rapidly throughout the whole republic. The central tableland seems to have suffered much, including the City of Mexico (population 475,000) the capital of the republic. During October the average daily number of deaths rose from 75 in normal times to 230. The disease is said to have appeared in various forms, namely (1) the hemorrhagic; (2) the nervous; and (3) the gastro-intestinal, and was frequently complicated by broncho-pneumonia.

It was observed that young adults were most affected and that persons who had previously had the disease in a severe form in 1890 escaped in 1918. A report by a foreign representative at Mexico City early in November stated that the outbreak had been particularly virulent in that town, observing that "no doubt the Mexicans are exceptionally easy victims on "account of the conditions brought about by the revolution. "There are, moreover, no proper organised medical arrange- "ments and no funds available to meet the emergency. It is "reported that in some of the towns in Mexico there is at "present no means for burying the dead. In Mexico City the "death rate—believed already to be the highest in any town of "the world—is thought to have doubled in the last two or "three weeks. Many members of Congress have already died." During October the number of deaths from influenza in Mexico City was officially reported to be 438 and 544 from pneumonia (all forms); and in November there were 1,402 deaths from

* *Journal of the American Medical Association*, 9th August 1919.

influenza and 1,162 from pneumonia. In December the figures were 65 from influenza and 261 from pneumonia; in January 1919, the numbers further declined, there being only 11 deaths from influenza and 231 from pneumonia, the numbers for February being respectively 7 and 211.

At Puebla, the third city in Mexico (with a population of about 100,000) and situated 116 miles by rail from the City of Mexico, the deaths numbered, at the height of the epidemic in October, about 300 a day. At Piedras Negras, 152 deaths were reported in two weeks, and many cases occurred at Nuevo Leon and Tamanlipas, all on the Texan border. In the State of Chiapas, at the town of Tapachula, during the two weeks ended 17th November, 500 fatal cases of influenza were reported. During the month of October 467 deaths from influenza occurred at the Mexican seaport of Tampico (population 20,000). At the port of Vera Cruz, about half of the crew of an Argentine war vessel were attacked by influenza early in October and the disease was present at this port up to the end of January 1919. From a ship that arrived at Tampico on 18th October from a United States port, six of the crew were landed suffering from influenza. The disease was epidemic in October in the coal mining districts near Sabina, in the province of Coahuila causing an average of 10 deaths daily. The epidemic gave rise to considerable ravages in the States of Senora and Sinaloa on the Pacific coast, but no figures as to those have been obtained.

Towards the end of January 1919 the epidemic showed a general decline. The total deaths caused by influenza in Mexico during the period from October 1918 to January 1919 have been variously estimated; one estimate published in the Press gave the total number of fatal cases as, approximately, 432,000, while another raised the number to half-a-million in round numbers. At the City of Tapachula 3,000 deaths occurred in a population of 30,000; and in the State of Chiapas it is said that a tenth of the population died from influenza and its results during the epidemic. A statement published at Mexico City on 2nd January 1919 gives the results of the epidemic in 25 states and the Federal District. This shows that the highest mortality occurred in the State of Michoacan, namely, 48,000 deaths in a population of 991,000, and the lowest, 900 fatal cases, in Colima with a population of 77,700. No estimates have yet been made as to the influenza mortality in Campeche, Morelos, Yucatan, Lower California, or in the territory of Quintana Roo.

The latest reports issued early in January 1919 stated that the disease was still seriously epidemic in the states of Chiapas and Tabasco,* but there had been a general decline in most of the other states.

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* *United States Public Health Reports*, 7th March 1919

Central America.

Guatemala.

On 11th October 1918 a report from an American source stated that influenza was present in Guatemala City (population about 80,000) and that it was also present in November at the port of Livingstone. An official report by the British Minister at Guatemala dated 30th December gave some particulars of the outbreak in Guatemala City, and in the country generall The epidemic of influenza was marked by the frequency of pneumonic complications, it spared no locality, whether on high or low ground, on the coast or on the elevated tablelands. Its intensity in the capital was at its height in December, and many of the poorer classes died of the disease. The daily mortality varied from 50 to 100, while the normal daily mortality was only eight to nine. The better classes did not succumb in such numbers as the poor, but many were attacked and some died, among the latter being two members of the diplomatic corps, the representatives of Mexico and Costa Rica, both comparatively young men. Among the measures enforced to prevent the spread of the disease was a regulation making it a punishable offence to appear in public without wearing a linen mask. Business was adversely affected in Guatemala by the epidemic, except as regards druggists and undertakers. The epidemic developed at an unfortunate time just as the coffee berry picking was beginning ; the Indian labourers succumbed in great numbers to the infection, the remainder fleeing to their homes, leaving the coffee berries to deteriorate on the trees, and causing a loss of the crop estimated at from 20 to 30 per cent. of the whole for the year. A report published in the United States Public Health Reports of 26th December 1919 stated that 325,220 cases of influenza occurred in this republic during 1918 and that 43,733 or 13·4 per cent. died.

British Honduras.

In October 1918 epidemic influenza was present in Belize (population about 10,500), the capital of British Honduras, and in the surrounding country. The type was mild and the mortality low, but notwithstanding this the outbreak greatly disorganised local business ; schools, churches, and places of amusement had to be closed ; and meetings with more than 10 persons present were prohibited. A later report stated that the epidemic had been far worse in the outside districts than in Belize itself. In view of the fact that the disease had been ravaging New Orleans and Mobile, the ports in the United States nearest to the Colony, and as it had also been similarly present in ports of Central America, it was regarded by the British officials that Honduras had on the whole escaped comparatively lightly. A report by Dr. T. Gann, the principal medical officer at Belize, dated 27th December, stated that the infection had been brought into the colony by labourers of the United Fruit Company arriving at Belize from Puerto Barrios

where the disease was then raging. The following figures as to the incidence of influenza in Belize are given by Dr. Gann :—

———	Cases.	Deaths.
From 11th to 23rd October 1918 - - -	142	7
„ 24th to 30th October 1918 - - -	1,045	
„ 31st October to 6th November 1918 -	833	24
„ 7th to 13th November 1918 - - -	260	42
„ 14th to 20th November 1918 - - -	91	15
„ 21st to 10th December - - -	74	12
Total - - - - -	2,445	100

The case mortality reckoned on these figures was 4 per cent. In addition to the cases reported above, 118 others were treated in the hospital, of whom 102 were admitted ill of influenza, while 16 acquired the disease in hospital while under treatment for other maladies. At the quarantine station and at the barracks, 119 cases were treated with only one fatality; and in Belize prison, where 74 of the inmates suffered from the disease, there were no deaths. According to Dr. Gann, the grand total of cases in the town from 11th October to 10th December was 2,556. A large proportion of the people in Belize are very poor and very ignorant. They seldom, even in serious cases, called in a medical man during the epidemic. The mortality was largely due to broncho-pneumonia and other complications or sequelæ. Quarantine was put in force, and "may have helped "to some extent in restricting the spread of the disease." Dr. Gann concludes his report with the following remarks regarding quarantine: "It is doubtful whether, even in the "case of a comparatively isolated place such as Belize, even the "strictest quarantine, amounting to actual segregation from "other ports, would be effective in keeping out such an "extremely contagious disease as influenza in pandemic form."

The British Honduras authorities passed an ordinance, on 1st November, "to facilitate the prevention of the disease "known as 'Spanish influenza,' which is hereby declared to be "an infectious disease within the meaning of the law relating to "Public Health (1914 Revision). The Central Board of Health "may, if they think fit, dispense with the notification of Spanish "influenza (by order published in the *Gazette*) either entirely, "or modify it, as may be specified in the Order." In the Ordinance the Central Board of Health was also empowered to prohibit or restrict the opening of schools, churches, places of amusement, and the holding of public meetings. Power was also obtained to apply quarantine to vessels bringing foul bills of health from ports where "Spanish influenza" existed. In

the case of a vessel so infected, it might be dealt with as follows :—

(1) The sick to be isolated.
(2) Other persons to be subjected to detention for four days from the time of landing.
(3) The vessel to be fumigated.
(4) Persons employed in discharging the vessel or landing cargo to be subject to detention on board the vessel for four days from the day of exposure to the infection.

In the case of vessels not infected, but coming from a port that is infected by influenza :—

(1) All persons may be detained for four days from the date of departure from the infected port.
(2) The vessel may be fumigated.

Though the Bill authorising these quarantine arrangements was passed by the Legislative Council on 26th October, the provisions, if duly enforced, seem to have had little effect in stopping the spread of influenza in British Honduras, for the disease was rampant during November in various parts of the colony, though in a comparatively mild form.

THE REPUBLIC OF HONDURAS.

In mid-September 1918, influenza was stated to have been present at Amapala, a small seaport town on the Pacific coast of Honduras, but no details were given.

On 7th October, influenza was epidemic at Tegucigalpa, population 23,000, the capital of the republic, many cases being reported, and a number of relapses being observed.

During November the disease was reported to be epidemic at the town of San Pedro Sula, which is situated on the railway line that runs from the interior to Puerto Cortez on the Caribbean coast, but no figures have been published as to the number of cases or deaths.

SALVADOR.

Epidemic influenza was present in the Central American Republic of Salvador in the early part of September 1918, and during that month and in October about 20,000 persons are said to have been attacked by the disease in the city of San Salvador (population about 60,000), the capital of the republic, but no report as to the mortality caused by the epidemic in the city or other districts has been received.

COSTA RICA.

On 12th October 1918, influenza was reported to be prevalent at Limon (population, 6,000), the chief Atlantic port of the Republic of Costa Rica, but no details were given.

COLOMBIA.

The only information received as to the concurrence of epidemic influenza in the Republic of Colombia, is that about 20th October 1918, the disease was present in a mild form at Barranquilla (population, 40,000), the chief port of the republic; and at Cartagena (population about 30,000), one of the principal cities of Colombia, where, in addition to affecting the civil population, it caused an outbreak among the troops in the local barracks. During October, also, influenza was epidemic at Bogota (population, 150,000), the capital of the republic, where up to 26th October, about 100 deaths had resulted. No other details have come to hand.

SOUTH AMERICA.

PERU.

The information received respecting the influenza epidemic in Peru has been of a very fragmentary kind. It is, however, known that an outbreak of the disease occurred in October 1918 in the mining regions of Marococha, Smelter, and Cerco de Pasco. The epidemic became so general that the mining and smelting works had to be closed. Although some 5,000 persons were attacked, the case mortality was only 0·75 per cent. Later, the larger cities in Peru were invaded, more particularly Lima and Callao. Towards the end of 1918, it was stated that in many cities there was a great prevalence of a disease of a catarrhal character lasting three or four days, probably influenza, and that death only occurred in cases with complications. Schools were closed and leaflets were distributed among the people giving instructions how to avoid the disease. At Lima the epidemic, which began in October, became more virulent in November, and by the end of December a total of 600 deaths had been attributed to the epidemic. Notification of the disease was not enforced. The schools were closed for a time. It is thought that the celebrations of the Armistice in November led to a further outbreak of the disease in more virulent form. The Peruvian Academy of Medicine appointed a committee to investigate the prevalent disease, but their report has not yet been received.

CHILI.

Very little has been heard from Chili as to the influenza epidemic there, beyond the fact recorded in the press that a serious outbreak had occurred during the autumn of 1918 in the republic. The Argentine press referred, in January 1919, to this outbreak, stating that nowhere in South America had the influenza epidemic created such ravages as at Santiago de Chili, but so far no official report is available. The British Vice-Consul at Punta Arenas (Chili) reported that an epidemic of "grippe" broke out there in the second week of September

1918, and as the epidemic seemed to be severe the governor ordered the closing of all schools, cinemas, &c. In the four weeks ended 4th October, 54 deaths were certified from "grippe." By 10th October the epidemic had subsided. There was a recrudescence of influenza at the end of August 1919 generally throughout Chili, and it invaded nearly every city. A Santiago newspaper stated that some 1,500 cases of influenza and typhus had proved fatal in a period of 20 days. The *Times*, of 25th August 1919, stated that a quarter of the whole population of Valparaiso was suffering from influenza.

It is probable that the Chilian Government will publish an official report on the influenza epidemic in the republic, giving details of the incidence and mortality in the various towns and districts of Chili, but up to the time of writing no report has been received.

THE ARGENTINE.

Early in October 1918 an epidemic of pneumonia was reported at the town of Jujuy, with a population of over 8,000, the capital of the province of that name, which is the most northern of the Argentine provinces. The case mortality rate was 30 per cent. A number of the cases occurred among the soldiers of the garrison. It has not been definitely stated whether this was in reality the result of epidemic influenza, but it is not unlikely. A commission of the Departamento Nacional de Higiene was sent to the spot to make inquiries, under the leadership of Professor Kraus, but the report has not yet been received.

An epidemic of influenza commenced in the middle of October in the city of Buenos Aires, the capital of the Argentine, with a population of over a million, and spread swiftly through the city until about two-thirds of the entire population had been attacked. Fortunately, the majority of the cases were at first of a mild type, but during the last 10 days of October the deaths attributed to the epidemic amounted to 1,477, and during the first five days of November 405 more fatal cases were recorded, making 1,882 influenza deaths in 15 days. Bacteriological examinations made at the Hospital Muniz showed the presence of Pfeiffer's bacillus in the patients' sputum in 60 per cent. of the specially grave cases. Pneumococci or streptococci were discovered in a little over 50 per cent. In the fatal cases, Pfeiffer's bacillus was found in 50 per cent., sometimes in pure culture, but more frequently associated with the pneumococcus or other organisms. To control the epidemic in Buenos Aires, persons affected by the disease were isolated. Race meetings, theatres, schools, and churches were closed for the time being throughout the republic. A sanitary cordon was placed on the Chilian frontier to prevent the influx of infected persons from Chili, where the influenza epidemic was said to have been of a specially fatal kind. The influenza infection,

notwithstanding the precautions taken, spread all over the Argentine Republic. In the interior of the country there was a recrudescence of the epidemic in the spring of 1919, and to assist in combating the malady, doctors and nurses were sent by the Government to various places, including Salta, Jujuy, Catamarca, La Rioja, the Province of Corrientes and the National Territories of the Chaco, Formosa, and Missiones. A recent message from La Banda states that epidemic influenza was attaining alarming proportions at that place.* In one of the concentration zones for naval recruits, located in the harbour of Buenos Aires, 200 cases and six deaths occurred in the course of a few days.

URUGUAY.

A widespread epidemic of influenza was reported to have prevailed during the last two weeks of October, and the early part of November 1918, at Monte Video, the capital of Uruguay, with a population of about 300,000, and situated on the north shore of the estuary of La Plata River. In the department of Monte Video, with a population of 378,993, there were 130,000 cases and about 130 deaths. The type of the disease was comparatively mild. It was also reported that concurrently with the prevalence of influenza at Monte Video, many cases of the same kind had occurred in the interior of the Uruguayan Republic, but no details were given. Six deaths from "grippe," or influenza, appeared in the mortality statistics of Monte Video for the year 1917, but whether these were due to the same disease as was epidemic in 1918 it is at present impossible to say.

In the *Times* of 26th July 1919, it is reported that influenza had again re-appeared at Monte Video. The medical authorities had addressed a series of queries to the practitioners of Uruguay with a view of obtaining precise information as to the incidence of epidemic influenza throughout the country, and a report was in due course to be published giving full details.

PARAGUAY.

Influenza became epidemic in Paraguay during November 1918, but little information has been made public regarding the incidence of the disease except in Asuncion, the capital of the republic. About the middle of November influenza began to be epidemic in Asuncion which has a population of about 80,000. During the four weeks' period ended 13th December, a total of 1,064 deaths from the prevalent malady was recorded. By 18th December the epidemic was subsiding, but no later figures are available. Some of the cases at the commencement of the epidemic were so rapidly fatal that they were regarded as probably of the nature of plague, and one medical man, Dr. Lopez Moreira, so notified them to the authorities. But he

* *Buenos Aires Herald*, 9th May 1919.

himself contracted the malady and could no longer attend to his cases which were taken over by another practitioner who certified that the patients notified as ill of plague were not suffering from that disease, but from epidemic influenza. During the early part of December, the influenza deaths in Asuncion numbered about 40 a day, but later in the month the daily number of these deaths fell to 24. A press report, however, stated that all the deaths due to the epidemic were not recorded. Theatres and cinemas were closed, and owing to the inroads of the malady upon the staff, only half of the usual number of city tramcars were running. At one time the postal arrangements were almost brought to a standstill through the absence from duty of officials occasioned by the epidemic. Owing to the inability of the medical practitioners to overtake the work imposed upon them, the Diplomatic Corps held a meeting and offered to bring doctors and nurses from other countries to relieve the situation, but this offer was declined by the authorities of the city. Various philanthropic societies including the Cruz Blanc came to the rescue; a house to house visitation of the poorer quarters of the town was carried out. The people were instructed to hang out a white flag if they needed a doctor, medicine or help of any kind. Milk was distributed gratuitously among the sick poor. Notwithstanding these and other measures there is said to have been a large amount of suffering and privation among the poor people. The Argentine Society of "Socorros Mutuos y Beneficiencia" sent 10,000 Argentine dollars for the benefit of the necessitous sick.*

BRAZIL.

Rio de Janeiro.—Early in October 1918 influenza began to be epidemic in Rio de Janeiro and spread rapidly over the whole city, attacking, it was said, more than half of the total inhabitants. The precise source from which Rio de Janeiro was infected has not yet been stated. In the early days of the epidemic, influenza was known to have caused the death of 20 of the crew of a United States ship of war lying in the harbour. Although it was at first announced by the authorities that the type of the epidemic malady in the city was mild, many deaths occurred and a panic developed among the inhabitants. The sanitary administration broke down, business was at a standstill, the food supplies ran short, and what articles were available were sold at such exorbitant prices as to place them beyond the reach of the poorer classes. Many shops were closed and the streets were almost deserted. Restaurants and places of amusement also closed their doors. Owing to the amount of illness among the men, the railway services were greatly reduced, and communication by telegraph or telephone was almost suspended for the same reason, the operators being

* *Buenos Aires Herald*, 13th December 1919.

largely on the sick list. At some of the public relief stations no medical stores were available and medical assistance in many instances could not be obtained. The druggists raised their prices enormously, making such drugs as quinine quite unobtainable by the lower classes. Some of the newspapers added to the panic by printing alarming accounts of the ravages of the epidemic, and attacked with bitterness the sanitary authorities of the city, an attitude that led to the resignation of the Director of Public Health. Many corpses could not obtain burial, and it was stated that "at more than one cemetery over 100 coffins were lying awaiting burial." Considerable difficulties were encountered in getting means of transport to convey the sick to the hospitals, and this led to great delay in securing treatment for the patients. It is estimated that from 600,000 to 700,000 of the inhabitants were attacked by influenza in a period of some six weeks, out of a population of about 915,000; the mortality caused by the epidemic in the city is given approximately as 15,000. But the official mortality returns for October and November 1918 give smaller figures, namely deaths from all causes in October 11,291, of which 8,876 are ascribed to influenza, and in November 5,705 deaths from all causes, 3,287 being attributed to influenza. It is possible that other deaths due to the epidemic may have been classed under the heading of "Respiratory or other Diseases." Later, no doubt, a full statistical account of the epidemic will be officially published. So far there is no information as to any recrudescence of influenza in Rio de Janeiro after its subsidence in November 1918. A later report by Dr. Torres states that during 1918 in Rio de Janeiro, the deaths attributed to influenza numbered 12,720; in 1915 there were 584 deaths from influenza, 426 in 1916, and 411 in 1917. The deaths from all causes in 1917 and 1918 were respectively 21,502 and 34,894.

The course of the epidemic of influenza in Rio de Janeiro during 1918 and in the first half of 1919, is shown month by month in the following table:—

—	Deaths from Influenza.	—	Deaths from Influenza.	—	Deaths from Influenza.
1918.		1918.		1919.	
January -	31	July - -	37	January -	87
February -	32	August -	40	February -	50
March - -	34	September -	48	March - -	48
April - -	33	October -	8,817	April - -	47
May - -	41	November -	3,287	May - -	55
June - -	36	December -	284	June - -	45
Total in 1918 - - -			12,720	1st half of 1919 -	332

Of the total 12,720 influenza deaths 6,844 were in males and 5,876 in females.

As regards the age distribution of the fatal influenza cases, the following table gives the numbers in each age group :—

Age Incidence of Fatal Influenza in Rio de Janeiro during 1918.

Age Group.	Males.	Females.	Total.
Under 1 year - - - -	526	543	1,069
1 to 2 years - - - -	523	431	954
2 „ 3 „ - - - -	334	346	680
3 „ 4 „ - - - -	232	242	474
4 „ 5 „ - - - -	174	198	372
5 „ 10 „ - - - -	349	415	764
10 „ 15 „ - - - -	146	218	364
15 „ 20 „ - - - -	354	399	753
20 „ 30 „ - - - -	1,670	1,337	3,007
30 „ 40 „ - - - -	1,302	779	2,081
40 „ 50 „ - - - -	607	398	1,005
50 „ 60 „ - - - -	279	202	481
60 „ 70 „ - - - -	127	148	275
70 „ 80 „ - - - -	62	89	151
80 „ 90 „ - - - -	14	53	67
90 „ 100 „ - - - -	5	16	21
Over 100 „ - - - -	2	2	4
Age unknown - - - -	138	60	198
Total - - -	6,844	5,876	12,720

The deaths from pulmonary tuberculosis in Rio de Janeiro during 1918 numbered over 700 more than in the previous year.

A later report states that there were 703 deaths from influenza in Rio de Janeiro during 1919.

The State of São Paulo (population, 4,420,229).—In October 1918 influenza was present in the State of São Paulo, but the epidemic prevalence appears to have reached its height in November. The capital of the State is the city of São Paulo with a population of 470,872. The number of attacks in this city has not so far been obtained, but there were 5,331 fatal cases, namely, 319 in October, 4,580 in November, and 432 in December. A recent official report gives the total deaths from influenza during 1918 in the State of São Paulo as 12,386 out of a total of 36,654 from all causes. The influenza deaths in October were 873, in November 8,251, and in December 3,298.

Santos (population, 96,050) is a port in the State of São Paulo. Influenza appeared at Santos, according to press reports, in June 1918, but does not seem to have been of a severe type. A second outbreak occurred in October and November. The

influenza deaths in October were 369; in November 463 persons were reported to have died of influenza in Santos and only 18 in December, making a total to 31st December 1918 of 850. Other towns attacked by influenza in the State of São Paulo in 1918 were Campinas (population, 105,160), where 209 influenza deaths were certified, and Ribeiro Presto (population, 52,839), where, in these three months, 205 deaths occurred from the epidemic malady.

The epidemic continued, though in a less virulent form, during the first half of 1919, 566 deaths being attributed to it in the chief towns of the São Paulo State, 207 of which were referred to the capital, 171 to Campinas, 107 to Ribeiro Presto, 25 to Santos, 25 to São Carlos, 24 to Botucatu, and 13 to Guaratingueta. In August the disease was said to be increasing again, but there was no epidemic up to the end of the year.

Bahia (population over 230,000) is the capital of the State of that name, and is a port of some importance in Brazil. According to reports published in the press, influenza was occurring in Bahia during September and October 1918, the type of the disease being mild and the mortality small. During these two months it is estimated that 5,000 persons in the town were attacked. Of the total deaths, 1,172, from all causes during September and October, 217 were attributed to influenza, and in November and December 167, making a total of 384 influenza deaths during 1918 in Bahia.

Influenza had appeared in the mortality returns of Bahia in previous years, namely, 16 in 1914, 10 in 1915, 28 in 1916, and 17 in 1917. Outside the city, except that influenza was stated to be occurring in the State of Bahia in September and October, there is no other information available as to the 1918 epidemic in the State of Bahia.

Pernambuco.—Information from an official source stated that an epidemic of "Spanish influenza" had broken out early in October 1918 at the port of Pernambuco (which has a population of over 150,000). Pernambuco is a busy Brazilian port on the easternmost point of the coast of the republic. Dr. de Freitas, Director of Health, reported on 25th November 1918 that the mortality rate per 1,000 of the male population caused by the epidemic was 9·6 per 1,000, and of the females 7·9. Pernambuco suffered from previous epidemics in 1889 and 1894. It is interesting to know that influenza appeared on the island of Noronha, 350 kilometres from the Brazilian coast simultaneously with Pernambuco, although there was said to have been no intercourse at the time between the two places.

Para is a port in North Brazil on the River Para, 70 miles from the sea, and has a population of upwards of 200,000. It was infected by influenza in the latter part of 1918. The infection was widespread, but the mortality was comparatively small. During December 7,100 cases were reported with

103 deaths, and 40 others from pneumonia. Beyond these meagre details no other information has been received.

It is very probable that the reports of outbreaks in the above-mentioned places do not by any means cover the whole of the incidence of epidemic influenza in Brazil during the latter part of 1918, but they comprise all that have come to hand from various sources at the time of writing.

Dutch Guiana.

Influenza was reported to be present in September 1918, becoming epidemic at the end of November and throughout December, at Paramaribo with a population of 37,000, a port at the mouth of the River Surinam, and the capital of Dutch Guiana. Reports received in January 1919 stated that the disease was still present in Paramaribo, and during that month 48 deaths from influenza were reported with 116 in February. In March the influenza deaths numbered only 7, and from that month up to the end of September no further fatal cases were reported. In October (1919) a single death occurred from influenza in Paramaribo and 4 more in November, but none in December.

British Guiana.

Influenza manifested itself in epidemic form in British Guiana at Georgetown, the capital of the colony, during November 1918. Details of the outbreak have not yet been published, but Dr. E. G. Rose, the Government bacteriologist, in a paper contributed to the *Lancet* of 15th March 1919, mentions that "the disease has taken a fearful toll, more "particularly of the lower classes of the community, namely, "the poor East Indian and black, generally already debilitated "by the ravages of chronic malaria or filarial infection." Death in the vast majority of cases was due to a rapidly fatal broncho-pneumonia. Nearly 1,000 persons were prophylactically inoculated with a vaccine containing the bacillus of influenza, pneumococcus, streptococcus, and staphylococcus, strains isolated during the outbreak being used. Reactions appear, as a rule, to have been mild or almost absent. Dr. Rose speaks guardedly about the vaccine, and observes that "it is yet too early to judge of its efficacy."

Venezuela.

There is at present no documentary evidence to show that influenza occurred in Venezuela during 1918 prior to the month of October, when His Majesty's Minister at Caracas reported that the disease had appeared at La Guaira about the middle of that month, and that it was spreading rapidly throughout

the whole republic. At La Guiara (population about 16,000), which is the port of Caracas and distant from it about 6 miles, influenza during a period of six weeks attacked about 50 per cent. of the population. So many people were ill at the same time that it was found necessary to close business establishments. There is a strong opinion that influenza was introduced into La Guaira from the United States. There is a statement also that a vessel, the S.S. "Venezuela," arrived at La Guaira on 25th October from a port in Trinidad having influenza cases on board. Official reports, however, mention the beginning of the epidemic at La Guaira as having been about 22nd October, that is, before the arrival of the S.S. "Venezuela." The actual number of deaths caused in La Guaira by the epidemic has not been reported.

Caracas, the capital of the country, has a population of over 80,000, and in it influenza became epidemic towards the end of October; up to 30th November 1,745 persons had died of the disease. The height of the epidemic was reached in the period from 1st to 5th November, at which time there were over 100 deaths a day. Nearly all the industrial establishments were closed, the tramways almost ceased to run, and all churches, theatres, and places of entertainment were closed by order of the police. There was great distress among the lower classes, and a lack of medical comforts and medicines for the sick. Many doctors were attacked by the epidemic disease, and difficulty was experienced by the authorities in providing medical attendance for the sick in the lower quarters of the city. Similarly many chemists, being ill, had to close their shops, and this added to the shortage of medicines. A volunteer ambulance service for removing the unattended sick to hospital organised by the students was greatly hampered by the fact that many members of its staff contracted the influenza infection and could no longer assist.

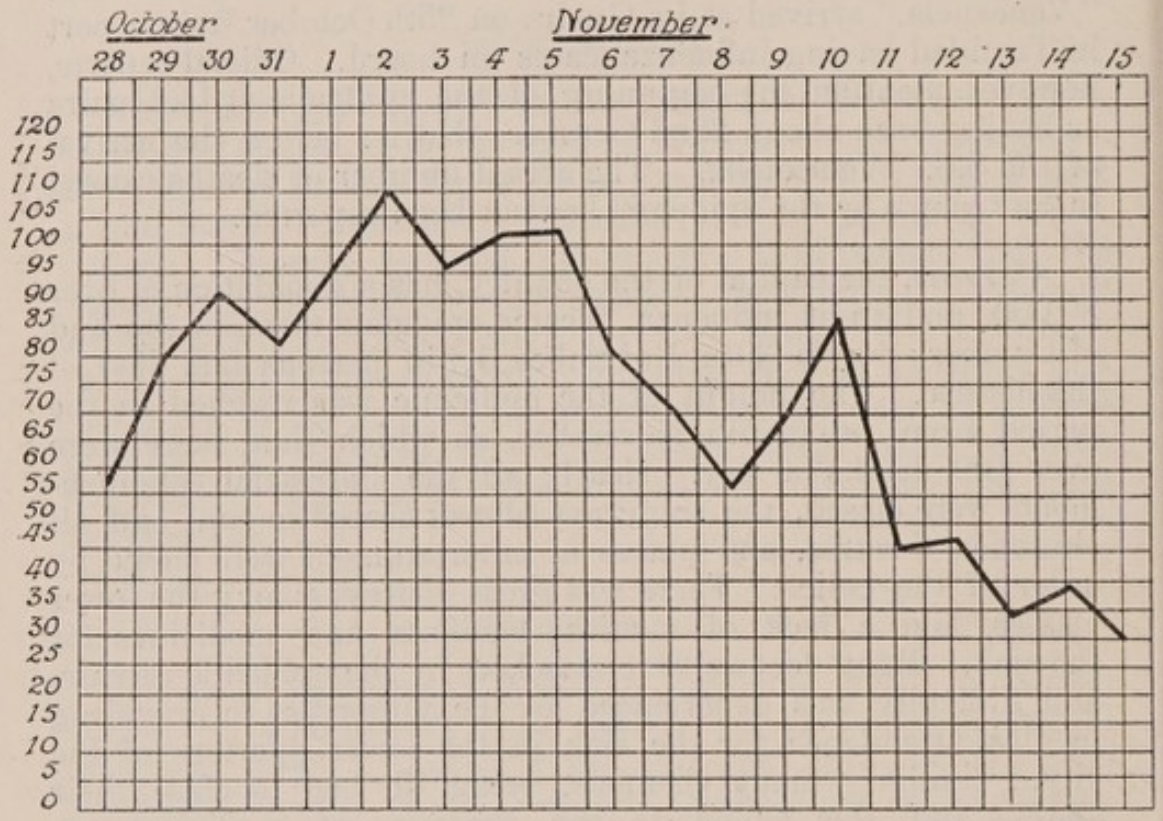
The mortality in Caracas during the epidemic was greatest among children up to 10 years of age and among adults between 20 and 30. It is estimated that 75 per cent. of the population of Caracas contracted the disease.

Writing in the *Gaceta Medica de Caracas* for 15th January 1919, Dr. Risquez remarks that he was greatly impressed by the number of cases of relapse, the symptoms returning after three to five days of apparent recovery. These relapses, he states, occurred among those who had got up from their beds and contracted chills. He never noticed any pulmonary complications among those patients who kept to their beds four or five days after defervescence. From 80 to 90 per cent. of the total cases in Caracas ran a mild course of about three days' illness. The warding off of complications was the chief duty of the medical attendant. In his opinion an attack of epidemic

influenza seems to confer some immunity against subsequent attacks.

The appended diagram of the mortality from influenza in Caracas from 28th October to 15th November was published by *La Officina de Sanidad Nacional* :—

Curve of Influenza Mortality in Caracas.



The influenza epidemic spread with extraordinary rapidity throughout Venezuela from La Guaira and Caracas, and the local outbreaks at Puerto Cabello, Valencia, Maracaybo, Carupano, Ciudad Bolivar and Goro, were especially severe. Puerto Cabello is a seaport town of about 14,000 inhabitants in the province of Carabobo, situated 78 miles due west of the port of La Guaira. During the six weeks epidemic period in October and November there were over 500 deaths attributed to influenza in Puerto Cabello, or more than 3 per cent. of the population. Valencia is a town of about 40,000 inhabitants, the capital of the state of Carabobo, and 34 miles from Puerto Cabello which is its port and with which it is connected by rail. In Valencia it is estimated that 10,000 of the inhabitants were attacked by influenza in November, and during that month there were 568 deaths recorded from the disease in the town.

Unfortunately up to the present no statistics are available as regards epidemic influenza in Venezuela as a whole.

The population of the Republic of Venezuela is about 3,000,000.

The West Indies.

Cuba.—"Influenza" as a cause of death has been present for some time in the mortality statistics of Cuba. For instance, in 1914, 199 deaths were certified from this cause at Habana, 64 at Pinar del Rio (about 160 miles from Habana), 58 at Santa Clara, 59 at Matanzas, 16 at Oriente, and 8 at Camaguey, making a total of 404 influenza deaths at these six places.* During that year the total deaths from pneumonia in the same areas numbered 425. The annual death returns for Cuba in 1915 have not been obtained, but in 1916 in the city of Habana there were 93 deaths reported from "Influenza." The statistics for 1917 have not been received.

On 5th October 1918, the S.S. "Alfonso" from ports of the United States arrived at Habana (population 300,000) with a large number of the crew suffering from influenza, and there had been 19 deaths from this cause during the voyage. Another ship from the United States, the S.S. "Adonis" arrived at Cienfuegos, a Cuban port (population about 30,000) 140 miles by rail from Habana, with influenza on board. A report dated 9th October states that influenza was then occurring at Camaguey where already about 2,000 cases had been reported, and that from five to ten deaths occurred daily from the disease. Another outbreak was noted at Nuevitas, about 40 miles from Camaguey. It may be mentioned, as showing the frequency of importation of the disease by shipping into Cuba, that in the week ended 12th October, 10 cases were landed at Habana from vessels that had arrived from ports of the United States. On 15th October influenza was stated to be generally epidemic in the island of Cuba. From 21st to 31st October, 1,746 cases were reported at Habana, and 42 in its suburb Regla. Four cases of influenza were landed at Habana on 19th October from the S.S. "Excelsior" from New Orleans, and two more on 20th October, while on 22nd October, six cases were reported on the S.S. "Morro Caste." In consequence of the frequency with which influenza was being brought to Cuba by vessels, the Cuban Quarantine service prescribed the following measures on 20th October to be enforced at all Cuban ports on arrivals from foreign ports, and on vessels engaged in coastwise or interior navigation, viz.:—

(1) The sick to be landed and sent to hospital if one is available, and if no hospital exists the sick to be treated on board the ship.
(2) The quarters that have been occupied by the patients to be fumigated, and any other parts of the vessel that may be considered infected.
(3) The clothing and bedding of the sick to be fumigated.

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* *Boletin oficial de la Secretaria de Sanidad y Beneficiencia.*

(4) Convalescents to be detained on board as long as coughing continues.
(5) All vessels in the port to be inspected at frequent intervals (for the discovery of cases).

Between 28th and 30th October three other vessels were found to have influenza on board. Between 1st and 10th November 976 cases of influenza were notified in Habana, and from 11th to 20th November, 952 other cases were reported. The majority were of a mild type, and the case mortality was only a little over 1 per cent. Up to mid-November it was estimated that 4,000 cases had occurred at Camaguey and 3,000 at Santiago de Cuba. At a medical conference held at Habana Dr. L. Ortega condemned the use of vaccines as a routine measure, and Dr. L. Plasencia claimed to have discovered a new micro-organism in the sputum of influenza patients, regarding it as the specific microbe of the disease. He had experimented with it on monkeys, and had produced in these animals the characteristic symptoms of epidemic influenza. In the early part of 1919 cases of influenza continued to be notified in Habana and in its suburb Regla. From 11th January to 10th February, 476 cases had come under medical observation, and of these 84 had died, or 17·6 per cent.

Jamaica.—Epidemic influenza is alleged to have been brought to Jamaica by ships from American ports in September 1918, and up to the end of the year about 4,000 deaths had resulted from the disease. Business at such places as Port Antonio and Montego Bay was brought for a time to a full stop owing to the prevalence of the malady during October. The deaths were chiefly due to pneumonia. In some districts the disease displayed great virulence. At Kingston (population over 60,000) there were six deaths certified from influenza in October, 182 in November, and 124 in December. The general death rate, which was 23·7 in September and 33·9 in October, rose to 89·7 in November, and fell to 54·3 in December. Many deaths resulted from pneumonia, and some of the victims were prominent persons in the island. No official report has yet been published regarding the influenza epidemic in Kingston, so far at least as is known at the time of writing.

Porto Rico.—During September 1918 two vessels infected by influenza arrived at the port of San Juan, the capital of Porto Rico (population of over 50,000). The first of these was the S.S. "Brazos," from New York, which arrived at San Juan on 18th September, eight cases being reported among the crew. The second vessel was the S.S. "Benevente," from New York; when this ship reached San Juan there were 48 influenza cases on board. In consequence of these occurrences the boarding officers of the Porto Rico Quarantine Service were instructed to board all vessels arriving at ports of the island from the

United States and to report all cases of influenza discovered on board. In the early part of December, influenza was said to be very widely prevalent throughout Porto Rico (population over 1,000,000) but by the end of the month the malady had subsided in the large towns, and was at that date confined to some of the outlying rural districts. No statistical reports have been received from Porto Rico, so that it is not possible to give approximate figures respecting the incidence and mortality of influenza in the island during the last four months of 1918.

San Domingo. — Owing to the danger from ship-borne infection of influenza the Government of San Domingo (which is a state forming the eastern part of the island of Hayti, with a population of over 100,000), ordered, in November 1918, that all imported cases arriving at Dominican ports should, in future, be placed in quarantine and cared for at the cost of the owners of the vessel, or of the maritime company that had conveyed the affected persons to these ports. About the middle of December a severe epidemic of influenza was reported at La Plata, San Domingo, (population 15,000), and about the same time the disease was said to be present in various parts of the republic, the spread having been very rapid. The statistics of this epidemic are fragmentary, but it is stated that in the week ending 31st December, 4,521 cases and 78 deaths from influenza were reported in the Dominican Republic. Of these, 2,417 cases (and 31 deaths) were referred to La Plata, and 1,065 (28) to San Domingo, the capital (population about 20,000). During the four weeks period ended 25th January 1919, 1,101 cases and 92 deaths were recorded from influenza in this republic.

Guadaloupe. — Epidemic influenza was reported during August 1918 as being present in the island of Guadaloupe, one of the French Lesser Antilles 77 miles distant from Martinique, and an epidemic occurred at Basse Terre (population 9,000) the capital of the island, but no details have so far been published.

Trinidad.—During 1917, 14 deaths were registered from influenza in Trinidad. Very little information is available as yet regarding the epidemic of the disease in 1918. All that is at present known is that the malady in a mild form was prevalent in the island in the latter part of the year.

The Bermudas.

On 25th September 1918 an epidemic of influenza was reported in the Bermudas, which are situated in mid-Atlantic, but no information has been received as to the extent of the outbreak nor as to its source of origin.

The Virgin Islands.

During the last four months of 1918 about 300 cases of influenza were reported in the Virgin Islands. The schools,

churches, and other places of general assembly were closed during the height of this prevalence.

The population of the British islands of the group is about 5,000 and about 40,000 under the Danish and United States Governments.

The Azores or Western Islands.

The Azores.—This group of islands situated in mid-Atlantic, is regarded as a Province of Portugal and not as a colony; the population is about 250,000. On 16th September 1918 the S.S. "Shemsei" arrived at Ponta Delgada, a town on the island of Sao Miguel, with a population of about 18,000. All of the crew were suffering from an illness believed to be influenza and six deaths had occurred. The port from which this vessel came has not been stated. On 28th October the British Consul reported that an epidemic of influenza was then raging at Ponta Delgada, some of the cases taking the form of pneumonia, others developing meningitis. During the week ending 2nd November 120 deaths from influenza were recorded in the town.

On 28th December the S.S. "Aikoku" arrived at Ponta Delgada with nearly the entire crew suffering from influenza, but from what port it had come has not been reported.

On 26th April 1919 it was reported that influenza was still present on the island of Sao Miguel, which is the largest and most important of the Azores group.

CHAPTER II.

The General Statistics of Influenza in Australasia and Parts of Africa and Asia,

BY

Lieut.-Col. S. P. James, M.D.

AUSTRALASIA.

Australia and Tasmania.

Two subjects are of outstanding interest in the history of epidemic influenza in Australia; it was the only country which attempted to secure immunity by establishing the principles of maritime and land quarantine in a strict manner, and it was the only country which escaped, for at least some months, the terrifying type of influenza which, from October to November 1918, raged elsewhere almost throughout the world. The immunity, however, did not persist; it lasted until January 1919, but from that month onwards, at different times in different parts of the country, the presence of a virulent form of influenza with pneumonic complications became manifest. The experience of Tasmania was quite similar.

There has been much painstaking inquiry into the various factors which possibly or probably might be responsible for the temporary immunity and for the subsequent epidemic invasion, but no completely satisfactory explanation has yet emerged.

The subject is complicated by the knowledge that there was an unusual prevalence of influenza in various parts of Australia during 1918, before there was any risk of epidemic infection from other countries. The statistics show that the local type of influenza began in July and continued in a quietly progressive form during October, November and December. It is stated, however, "as an established fact" that, whatever influenza incidence obtained during those months, the disease was not to be considered as in any way comparable epidemiologically with the disastrous pandemic form. A chief difference was that although the increased prevalence applied to the statistics of influenza, it did not apply to the statistics of acute bronchitis, broncho-pneumonia, and pneumonia, which were

not widely prevalent as causes of death. The difference is shown in the following statement :—

———	New S. Wales.	Victoria.	Queensland.	S. Australia.	W. Australia.	Tasmania.
Influenza figures, 1917.	3·0	4·3	3·7	2·5	3·2	1·5
Influenza figures, 1918.	17·5	15·3	19·8	10·2	22·8	19·3
Pneumonia figures, 1917.	60·1	66·1	40·9	46·2	48·4	50·2
Pneumonia figures, 1918.	67·4	70·7	62·9	50·3	48·8	65·3

Evidence of the presence of this local variety of influenza in New South Wales in July 1918 is provided in the following history of the voyage of the transport "Borda," which left Sydney on the 18th July and arrived at Durban on the 19th August :—"During "the first few days after embarkation a number of men on "board suffered from colds and tonsilitis, the daily sick parades "numbering from 4 to 6 per cent. of the total number of "troops. On the 6th of August, when nine days out from "Fremantle, the sick parades became gradually double their "size from an outbreak of a form of influenza. The outbreak "commenced to abate about the 15th August, but according "to official advices received from South Africa two cases of "pneumonia and nine of influenza were landed on arrival."

By September 1918 the outbreak of what was regarded as the local type of influenza in Sydney had become very extensive, unofficial estimates stating that 30 per cent of the population of the city and suburbs suffered from the disease. Also at Lithgow (New South Wales) a very virulent form of influenza complicated with pneumonia was prevalent during September ; and at Lockhart (in the southern portion of New South Wales) a similar form of a fatal type was prevalent during October. In the hospital at Melbourne in November there were about 30 cases of a form of influenza "sufficiently severe in type to excite "comment."

These occurrences were known, but it was held that they represented nothing which could be likened in any way to the extraordinarily severe type of influenza which attacked South Africa and New Zealand.

The application of maritime quarantine from the 17th of October 1918 was an endeavour to prevent the entry of that type of influenza. During the seven months from October 1918 to April 1919 the quarantine service dealt with 149 uninfected vessels and 174 infected vessels, with a total personnel of 81,510, including 1,102 actual patients. In some of the vessels detained in quarantine serious epidemics of the pandemic type of influenza

occurred, but it is definitely stated that no evidence was at any time obtained of an escape of infection by a demonstrable chain from these persons or ships in quarantine to the shore population. For this and other reasons it was concluded that from October to December the measures of maritime quarantine which were taken had the effect of holding at the sea frontiers an intensely virulent and infective form of influenza which during those months was causing disastrous epidemics in New Zealand and South Africa.

In the meantime, however, what was regarded as the ordinary type of influenza continued. It has already been mentioned that in Melbourne during November this type included some cases which were "sufficiently severe as to excite comment."

In the Australia reports at present available, the next reference to the incidence of influenza in Melbourne relates to the 9th January 1919 on which date, it is said, the first cases of the epidemic form of the disease occurred. As regards these cases, however, the official report by Dr. J. H. L. Cumpston, Director of Quarantine, stated that "they excited no comment "and were not known to the State Health Authorities until "some days afterwards, when the occurrence of further cases "attracted attention." It seems probable that for some time it was doubtful whether the cases which occurred early in January in Melbourne were of the type already present in the country or were a manifestation of the pandemic type. By 20th January, however, it was determined "that there had "occurred in Melbourne some 50 to 100 cases of a disease, "which appeared, from the accounts received, to resemble the "severe form of influenza." Immediately following these cases a more or less extensive epidemic developed in Victoria, followed by epidemics in South Australia, New South Wales, Queensland, and Western Australia. The available statistics of deaths from influenza during these epidemics in the different states are as follows :—

Influenza in Australia.

Table showing Number of Deaths from Influenza so far as these are at present available.

Week ending	Victoria.	New South Wales.	South Australia.	Queensland.	Western Australia.
	Whole State.	Whole State.	Whole State.	Metropolitan Area only.	Whole State.
1919.					
January 3 -	—	—	—	—	—
„ 10 -	2	—	—	—	—
„ 17 -	3	2	—	—	—
„ 24 -	6	—	—	—	—
„ 31 -	45	—	—	—	—

Week ending	Victoria.	New South Wales.	South Australia.	Queensland.	Western Australia.
	Whole State.	Whole State.	Whole State.	Metropolitan Area only.	Whole State.
1919.					
7 February -	122	—	—	—	—
14 „ -	164	13	2	—	—
21 „ -	115	—	—	3	—
28 „ -	88	—	—	1	—
7 March -	69	6	1	—	—
14 „ -	59	5	—	1	—
21 „ -	33	11	—	1	—
28 „ -	36	53	2	—	—
4 April - -	61	152	12	—	—
11 „ - -	122	250	7	—	—
18 „ - -	187	285	11	—	—
25 „ - -	227	268	6	—	—
2 May - -	265	283	9	1	—
9 „ - -	235	217	22	6	—
16 „ - -	177	161	32	20	—
23 „ - -	107	99	29	38	—
30 „ - -	87	91	41	68	—
6 June - -	107	82	22	68	3
13 „ - -	89	118	52	29	4
20 „ - -	75	319	20	No return	7
27 „ - -	58	634	31	15	11
4 July - -	60	677	10	6	4
11 „ - -	85	612	—	6	2
18 „ - -	120	540	8	1	13
25 „ - -	135	343	9	2	5
1 August -	162	231	8	3	30
8 „ -	155	117	15	2	26
15 „ -	76	120	18	—	41
22 „ -	41	56	11	1	36
29 „ -	24	47	48	2	34
5 September -	16	52	22	2	25
12 „ -	9	25	15	3	28
19 „ -	5	20	14	2	15
26 „ -	2	20	16	3	21
3 October -	1	23	11	2	14
10 „ -	Nil	17	9	3	7
17 „ -	„	8	5	1	5
24 „ -	„	11	6	Nil	6
31 „ -	„	2	5	„	3
7 November -	„	2	4	„	1
14 „ -	„	Nil	2	„	Nil
21 „ -	„	„	1	„	„
28 „ -	„	„	2	„	„
5 December -	„	„	2	„	„

On different dates from the 27th January, New South Wales, Victoria, and other States attempted to limit the spread of this epidemic by measures of land quarantine designed to check public travel as far as was practicable. But people crossed

prohibited borders in large numbers at places where there was no supervision ; other evasions of the restrictions were numerous ; and many additional difficulties were encountered. Ultimately the control of border traffic proved to be impracticable. Moreover, it seemed clear that, as the epidemic became well established in South Australia, New South Wales, and Queensland, at comparatively short intervals after its appearance in Victoria, the land quarantine measures had failed in their primary purpose of protecting the states which imposed the restrictions.

It has already been mentioned that there is as yet no completely satisfactory explanation of the remarkable differences between the recorded experience of epidemic influenza during 1918 and 1919 in Australia and in other countries. In January 1919, when it had been definitely decided that cases of epidemic influenza were present in Melbourne, it was uncertain whether these cases were due to either :—

(*a*) the escape of infection from the maritime quarantine defence ; or

(*b*) the continuity of the epidemic which was prevalent in Australia during the later months of 1918.

It appears that expert local officers who have studied the subject at first hand with great care and attention to detail are not prepared at present to favour one of these epidemiological explanations rather than the other. Dr. J. H. L. Cumpston, Director of Quarantine in the Commonwealth, writing on the 25th August 1919, in connection with the causes of the epidemic invasion, made the following remarks : "Although the utmost "care was taken, by both land and sea quarantine measures to "prevent the introduction of infection into Western Australia, "influenza with pneumonic developments definitely appeared "in early January. Similarly in Tasmania, which is an "isolated island, all shipping was carefully and strictly "quarantined between January and August 1919, but in the "third week of August cases of pneumonia definitely made "their appearance. There are two possible alternatives as "hypothetical explanations—either a disease which was introduced and was prevalent in Australia in August—September "1918 (corresponding with English May—June outbreak) "remained dormant until it developed an added virulence "under the influence of some unknown factor ; or there is some "phase of this virus which escapes all quarantine measures, "and which must lie dormant for months at a time until some, "as yet unknown, factor stirs it into activity. The quarantine "measures imposed by State Governments were rigidly and "consistently carried out from January to August, but, nevertheless, the epidemics occurred in the different States at the "times indicated. The explanation of this has completely "puzzled me."

New Zealand.

Influenza occurs annually in New Zealand, and in common with the rest of the civilised world, the Dominion suffered very severely from the pandemic of 1918. As in other countries the disease appeared in two distinct waves, the first having its maximum about August or September, the second developing during the first fortnight of November. The mortality in the first wave, although higher than is usual in New Zealand, was not alarming, but the virulence of the second wave was far in excess of anything which had previously been experienced in connection with influenza in the country. The period of greatest intensity of this wave almost coincided in Great Britain and in New Zealand.

Prior to November, 1918, the disease was not notifiable among the civil population, but the statistics from military camps give a fair indication of the incidence throughout the country. The following were the admissions to hospital and the deaths in the larger training camps during 1918.

—	Jan.	Feb.	Mar.	Apr.	May	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Cases -	22	30	16	13	32	16	145	571	1,216	1,126	4,369	15
Deaths	—	—	—	—	—	—	—	2	—	2	280	

From these and other military statistics it was concluded that the primary wave began in July—about two months later than in Great Britain—and reached its crest in September, waning slightly in October. The second wave rose very suddenly during the last few days of October, reached its maximum on the 9th of November and died away by the 18th. In the camps of some native troops, however, the second wave appears to have shown itself somewhat earlier and to have reached its maximum about the end of October.

For the civil population the following deaths, based on the Registrar General's returns, were recorded :—

Deaths from Influenza among Europeans.

Jan. 1918.	Feb. 1918.	Mar. 1918.	Apr. 1918.	May 1918.	June 1918.	July 1918.	Aug. 1918.
2	1	1	—	3	5	8	7

Sept. 1918.	Oct. 1918.	Nov. 1918.	Dec. 1918.	Jan. 1919.	Feb. 1919.	Mar. 1919.	Apr. 1919.
11	50	3,294	2,177	86	18	11	4

Deaths from Respiratory Diseases among Natives (Maories).

Jan. 1918.	Feb. 1918.	Mar. 1918.	Apr. 1918.	May 1918.	June 1918.	July 1918.	Aug. 1918.	Sept. 1918.	Oct. 1918.	Nov. 1918.	Dec. 1918.
3	3	5	4	7	6	7	8	29	10	200	950

More detailed statistics available from certain areas showed that there was a very steady increase in mortality from the first week of October 1918, that the epidemic death-rate was established in the week ending 4th November, that it rose rapidly during the first three weeks of that month, reaching its peak on the 25th, and that it then declined as rapidly as it had risen.

The following general information is summarised from a special report by Dr. Makgill, District Health Officer, Auckland, and from the report of the "Influenza Epidemic Commission" appointed by the Governor-General of New Zealand on the 28th January 1919.

Incidence.—The statistics from camps indicated that between 30 and 40 per cent. suffered in the first wave and about 50 per cent. in the second wave. About 10 per cent. of the cases developed pneumonic complications. It was considered probable that as regards the whole population of the Dominion about 40 per cent. were attacked during the second wave.

Mortality.—Among Europeans the deaths recorded as being due to influenza in 1918 numbered 5,559 (about 5 per 1,000 of the population), of which all except 38 occurred during the second wave of the epidemic. There was also from July onwards an abnormal increase in the death rate from all catarrhal diseases other than influenza, and this rise was peculiar to 1918, for, although there is usually a seasonal rise in the winter months, the rate in most years falls as the Spring advances. Among Natives it was estimated that in 1918 between the 1st October and the 31st December, 1,130 deaths (22·6 per 1,000 of the population) resulted from the disease.

Case Fatality.—In the military camps during the second wave about 4·5 per cent. of those attacked died, but there are no figures indicating the fatality among the general population.

Influence of Age, Sex, and Race.—In New Zealand, as in other countries, the death rates according to age were not a true indication of the case incidence. Of the total deaths about half were among persons between the ages of 25 and 40, but only 24 per cent. of the population are in that age-group. In the Featherstone Training Camp an analysis of over 2,000 hospital cases showed that although men of from 20 to 25 yielded a high proportion of cases, the death-rate among such

cases was proportionately less than among those aged between 25 and 40. The younger men were perhaps equally susceptible to influenza, but were not equally liable to pneumonic complications, and when such complications developed, they were better able to combat the effects. Thus among the more serious pneumonic cases the fatality among the younger men was 57 per cent., but among the older 72 per cent. The death-rate among males was 6·5 per 1,000 as compared with 3·5 among females, but among children under 15 years of age the proportion of deaths was slightly higher for females than for males, and a similar tendency towards equal figures for both sexes was apparent in the statistics of deaths of persons above 50 years of age.

Native races (Maoris) were more susceptible to attack than Europeans and the disease was more fatal among them.

Incubation Period.—The evidence available pointed to an incubation period which was not more than 48 hours and in some cases was less than 36 hours.

Period of Infectivity.—It is said that there was evidence that the cases were very infective in their earliest stages—in some instances even before the symptoms were such as to enable a diagnosis to be made. There was some evidence, also, that infectivity ceased within a short period. No case of infection was recorded as having occurred in the temporary convalescent hospitals to which patients were sent as a rule about the seventh or eighth day of their illness.

Views regarding the Nature and Spread of the Epidemic.—Owing to unavoidable depletion of staff few bacteriological observations could be made, but such results as were obtained did not differ from those reported in other countries, and, in view of thorough clinical and epidemiological enquiries, it was concluded that the epidemic disease was practically identical in form, incidence, and character, with the pandemic influenza which devastated Europe and America. Detailed enquiry was made into the causes of origin and spread of the virulent form of the epidemic. On the one hand the finding that a recent attack of the type of influenza prevailing during the first wave conferred a certain degree of immunity against the type prevailing in the second wave was held to establish the bacteriological identity of the two outbreaks, and for this and other reasons it appeared as if the various factors concerned were already present in the Dominion during the primary wave, and that the second wave was but the manifestation of a more virulent activity of one or all these factors. On the other hand there were grounds for believing that the second outbreak could be accounted for only by the seaborne introduction from outside the Dominion of some new factors of which a "new infective element" was the most important. The latter was

the view arrived at by the Official Influenza Epidemic Commission, their conclusions on this subject and on the causes of spread being stated (in effect) as follows:—

(*a*) The cause of the introduction of the recent epidemic of influenza into New Zealand was the conveyance by sea of the infective element of the "epidemic influenza" lately prevalent in Europe, Great Britain, South Africa and America.

(*b*) The extension of the epidemic from its first appearance in Auckland was largely the result of a general disregard of precautionary measures in the initial stages, due to want of knowledge regarding the nature of the disease. The infection was largely spread by the congregation of large crowds of people in the various centres in connection with the Armistice celebrations, race meetings, &c., and the fact that no restriction was placed upon the movements of the people in travelling, even when they had individually been in contact with infected persons.

(*c*) The mode of introduction of the epidemic is not capable of absolute demonstration, but the evidence raises a very strong presumption that a substantial factor in the introduction of the epidemic was the arrival in Auckland on the 12th October of the R.M.S. "Niagara" with patients infected with the epidemic disease. The evidence, however, does not exclude the possibility of other sources such as the presence of infection from other vessels arriving at the same time or shortly before the arrival of the "Niagara."

The difficulty of arriving at a final conclusion on the above subject will be apparent from the following paragraph in the special report by Dr. Makgill, District Health Officer, Auckland: "we learn also from the newspaper reports of that period that "an epidemic of influenza of virulent type was present in Auck- "land early in October. Thus, in the *New Zealand Herald* of "the 9th and 10th October, special articles appeared describing "the epidemic as of widespread proportions, and as being more "virulent than previously, and generally emphasizing the "serious position of affairs. It is interesting to note that this "occurred prior to the arrival of the R.M.S. "Niagara" which "is popularly supposed to have brought the new type of "infection."

Measures.—At the commencement of the epidemic no fewer than 228 doctors were absent from New Zealand on military duty, and of those whose services were available many contracted the disease. In the larger towns this shortage of medical aid was compensated for to some extent by adopting a "block system," under which each doctor had his own special

district, which was not overlapped by any other. In each district a citizens' committee was set up with the duties of providing food and attendance to stricken households, of summoning medical assistance where necessary, and of arranging for the transport of serious cases to hospital. In some districts these committees undertook the equipment and management of temporary hospitals, and for this purpose various bodies, such as the St. John Ambulance Brigade and Association, the Red Cross Society, and the Women's National Reserve, worked in conjunction with them. Schools, halls, and other public buildings were opened as emergency hospitals to accommodate the large number of serious cases for which there was no room in the existing hospital; the milder cases were, as far as possible, nursed in their own homes.

Among the administrative measures adopted by the Public Health Department were the gazetting of influenza as a dangerous infectious disease, the prohibition of "tangis," the prohibition of the sale of alcohol except on a medical prescription, and the enforcement of burial within 48 hours of death. Pamphlets of advice were freely circulated and a trial was made by the Department, as a prophylactic measure, of the inhalation treatment with 2 per cent. solutions of zinc sulphate atomised by means of steam under pressure. Chambers for this treatment were established at railway stations, wharves, and in all centres of population. The experience of over two years in the military camps in New Zealand was wholly favourable to the use of weak solutions of zinc sulphate as an inhalation for checking epidemics of measles, cerebro-spinal fever, and diphtheria, but as regards influenza it was said that, on account of the rapidity of invasion, unless the treatment was repeated very frequently and soon after exposure to infection, its value was less. When applied to the general public it was doubtful whether the procedure was useful, and the chambers at the railway stations and wharves were associated with harmful crowding. In regard to the use of masks there was little or no experience in New Zealand, and experience with inoculation was practically confined to troops. Quarantine in connection with shipping was not adopted.

Fiji.

In a report dated the 18th January 1919, the Chief Medical Officer of Fiji states that it is a matter of uncertainty, or even impossibility, to define the manner in which the epidemic of influenza first gained admission to the Colony, especially in view that (1) there was undoubtedly influenza (which in some cases was of a severe type and complicated by pneumonia) in widely separated districts of Fiji for some months before the epidemic; (2) all incoming vessels arrived with clean bills of health until the disease was raging all through the Colony.

In Suva there were some cases of influenza of moderate severity during October, and reports were received of localised outbreaks in Taveuni, Labasa, Navua, and Kadavu, but the main epidemic in Fiji burst out about the middle of November. The earliest fatal cases in Suva occurred on 17th November, and thereafter the reports of burials for influenza fatalities on certain dates were as follows :—

November.							
18	19	20	21	22	25	27	30
2	2	1	1	3	5	9	16

December.										
1	2	3	4	6	7	9	12	15	19	20
20	26	27	30	31	25	20	14	6	4	0

It was not found possible to keep records of the number of persons attacked, but the Chief Medical Officer considered that it would not be an exaggerated estimate to say that 85 to 90 per cent. of the whole population of Suva and district were affected at some time or another during the course of the epidemic.

The epidemic, once firmly established at Suva, spread with alarming rapidity, both to neighbouring districts and to the districts of Rewa, Ba, Lautoka, Nadi, and Nadrogi, in Viti Leva; also to Levuka in the island of Ovalau, to Labasa and Savu Savu in Vunna Levu, and to the island of Taviuni.

In a despatch, dated the 16th April 1919, the following statistics of mortality based on the final district reports of the epidemic are given :—

	Population on 31st December 1917.	Number of Deaths.	Percentage.
Europeans - - - -	4,824	69	1·41
Half-Castes - - - -	2,756	76	2·75
Indians - - - - -	61,153	2,553	4·17
Fijians - - - - -	91,013	5,154	5·66
Others - - - - -	4,226	293	6·93
Totals - - -	163,972	8,145	4·96

The decline of the epidemic was said to be rapid from the middle of December. By the 24th of that month all four temporary hospitals which had been established in Suva were

closed, and after the 28th no more notifications of genuine cases were received.

Measures.—Owing to the absence of many officers on War service, to local shipping difficulties, and to the dislocation of the steamship service from Australia and New Zealand, the Colony was ill-prepared to deal with so serious an epidemic.

In November the disease was made notifiable under the Public Health Ordinance of 1911, and circulars were sent to all medical officers, native medical practitioners, and native officials, calling attention to the epidemic, suggesting methods of treatment, advising that food be stored in all villages, and authorising the use of plantation hospitals for the treatment of cases. Temporary hospitals were equipped and maintained in Suva, Levuka, and other centres. Depôts were also established from which soup, arrowroot, sago, and other food and medical comforts were distributed to the homes of natives who were too ill to help themselves. In this work the medical officers and other officials in the Colony were assisted by a large number of voluntary workers. A relief party of 4 medical officers, 1 lady doctor, 4 nurses, 3 senior students, and 24 orderlies was sent by the New Zealand Government to assist in dealing with the epidemic, and arrived at Suva on the 17th December. The complete cessation of steamship communication with Sydney prevented the arrival of a relief party which had been asked for from Australia, but the services of two medical officers and some medical orderlies, who were members of the relief party sent by Australia to deal with the epidemic in Samoa, were obtained.

The expenditure incurred in relief measures in connection with the epidemic was estimated to be approximately 10,000*l.*

The Samoa Islands.

A Commission appointed by the Governor-General of New Zealand to inquire into the circumstances and causes of the introduction of epidemic influenza into the Islands of Western Samoa reported that in their opinion there was no doubt whatever that epidemic pneumonic influenza was introduced into Western Samoa by the S.S. "Talune" on the 7th November 1918. This ship left Auckland (where influenza was seriously prevalent) on the 30th October, and influenza broke out among the passengers and crew during the voyage to Samoa. Within seven days after her arrival, pneumonic influenza was epidemic in Upolu. It spread with great rapidity throughout this island and later throughout Savaii, the other island of Western Samoa. It was calculated that up to the 31st December 1918, out of a population of 30,738 in Western Samoa, 7,542 persons had died either directly from influenza or in consequence of its prevalence.

The Commission reported that American Samoa (Pago Pago) had entirely escaped the ravages of influenza, and it appears, from their report, that from the 20th November, to avoid the risk of introduction from Apia, the United States Governor at Pago Pago imposed on all ships arriving at this port five days absolute quarantine before discharging or taking on board any mail or cargo.

Polynesia.

Dr. Cumpston, Director of Quarantine for the Commonwealth of Australia, has drawn attention to the relative incidence of influenza in certain of the Pacific Islands. He notes that the Tonga and Samoan Islands are based upon New Zealand, from which they receive all supplies. Both these groups of islands were very heavily infected and suffered a high mortality.

On the other hand, the Gilbert and Ellice Islands, New Hebrides, Norfolk Island, Solomon Islands, British and German New Guinea and New Caledonia all are based on Australia, with which alone they have communication. This group of islands entirely escaped influenza infection, a result which is ascribed by the French Authorities and by the local British Administration to the strict outward quarantine precautions which were taken by the Australian Quarantine Service in respect of all vessels leaving Australia for these island groups.

AFRICA.

Union of South Africa.

On the 3rd of December 1918, the Governor-General of the Union of South Africa appointed a Commission "to enquire into matters concerning the influenza epidemic in the Union." The Commission held sittings at Pretoria, Johannesburg, Durban, Petermaritzburg, East London, Bloemfontein, Kimberley, and Cape Town; they heard in all 192 witnesses and received a number of statements from individuals who did not appear in person.

The following summary is based on the information given in the Commission's Report which was presented to the Governor-General on the 8th February 1919.

Type of the Disease.—The Commission distinguish in their report between the disease ordinarily called "Influenza" and the disease called "Epidemic Influenza." In their opinion the latter disease, as it appeared in the Union, was identical with the epidemic disease which in October and November 1918 was prevalent in practically every country of the world.

Origin and Spread.—For some months prior to the outbreak of "Epidemic Influenza" in the Union the disease ordinarily described as "Influenza" had been more than usually prevalent in the larger centres of population, and ships had been arriving at Durban and Cape Town with many cases of that illness on board. The cases were in such numbers as to make the Port Health Officers feel that there was a certain abnormality, but they differed in no way from ordinary catarrhal colds which are commonly called influenza, and no special measures seemed to be indicated or were taken. On the other hand, the pandemic disease was prevalent in Europe, Sierra Leone, and other localities which were ports of departure or call of ships arriving in the Union, and although it was impossible to determine the exact manner in which introductions of the epidemic disease from outside operated, and "impossible to fix on any route or vehicle," the Commission, on the evidence adduced before it, were of opinion that infection from the outside was a deciding factor in precipitating the epidemic. The infection may have been introduced by both the western route from Europe and Sierra Leone and the eastern route from countries lying east and north of South Africa.

The earliest outbreak of epidemic influenza in the Union occurred in the vicinity of the Harbour area, Durban, on the 14th September 1918. Thence, it spread to the central Rand area, where numerous cases were observed on or about the 18th September, chiefly among natives working in the mines. Several thousand natives were attacked within a few days, but the mortality among them was comparatively low. On the 23rd September, cases of the epidemic disease appeared at Kimberley, and on the same date the disease was officially reported among the Nigerian troops and in the South African Labour Corps at Cape town. In a short time the disease appeared in several other places and within two or three weeks became pandemic.

Incidence and Mortality.—The Commission found it impossible to obtain statistical information which could be relied upon in regard to the attack rate of the disease, but according to the returns and estimates furnished by magistrates and local authorities the total number of cases of pandemic influenza which occurred in the four provinces of the Union between the 1st August and the 30th November 1918 was 2,616,805, of which 454,653 cases were in Europeans. On these figures it would appear that about 32 per cent. of the European inhabitants of the Union and about 46 per cent. of the native and other non-European inhabitants were attacked during the four months referred to. The mortality records are said to be more accurate. The total number of deaths reported from the disease between the 1st August and the 30th November was 139,471, of which 11,726 deaths were of Europeans. These figures indicate that about 8 per thousand of the European

inhabitants and about 27 per thousand of the non-European inhabitants died from the disease during the period. In proportion to population the disease caused fewer deaths in Natal than in any of the other provinces, although, according to the information available, the rate of incidence in all provinces was about the same. No satisfactory explanation of this lesser fatality in Natal was apparent.

A tabular statement of the incidence and mortality statistics collected by the Commission is shown on the next page.

Age and Racial Incidence.—Persons in the third and fourth decades of life were said to be particularly susceptible to attack and the fatality rate was greater at those age groups. Children and old persons seemed to be partially immune.

The attack rate, the fatality rate, and the death rate in proportion to population, were all greatest among natives and other non-European races; and Europeans born in the country seemed to be more susceptible than European immigrants.

Mode of Spread. — The Commission found no reason to question the view that influenza is spread by contact only; and the general trend of evidence was to the effect that spread was facilitated and accelerated by the railways. The disease had a tendency to run its epidemic course within a period of from three to four weeks.

Measures.—On 9th October, the Union Government through their Health Department circularised all Magistrates and Local Authorities urging that everything reasonable should be done to combat the epidemic and stating that the Government would bear half the cost. Generally speaking the measures taken by Local Authorities consisted in providing medical relief, nursing relief, food and other supplies, arrangements for burial of the dead, improvising hospitals and transport of the sick. This work was usually done through voluntarily organised committees headed by the Mayors, Magistrates, or other prominent citizens. As a rule supplies were adequate, but in many cases there was some shortage of petrol and drugs. Certain of the municipalities, notably Bloemfontein, adopted a method of requisitioning essential supplies. Up to the 30th November, 104 medical men, 227 nurses and 163 medical students, hospital orderlies and inoculators, were engaged by the Union Health Department, and placed on duty in various localities.

From about 7th October a polyvalent vaccine prepared at the Government Laboratory, Cape Town, and at the South African Institute for Medical Research, Johannesburg, was available for general use. The opinion was generally held that the vaccine when properly used caused no ill effects. A number of medical men thought that the vaccine "did a certain amount of good," but this opinion was not supported by statistical evidence.

Union of South Africa.

Statistics of Epidemic Influenza and its Complications.

Table of Cases and Deaths, 1st August to 30th November 1918.

(Compiled from Returns and Estimates furnished by Magistrates and Local Authorities.

Provinces.	Population.			Cases.			Incidence Per Cent.			Deaths.			Death Rate Per Cent. of Persons Attacked.			Death Rate per 1,000 of Population.		
	European (1918)*.	Other than European (1911).	Total.	European.	Other than European.	Total.	European.	Other than European.	Total.	European.	Other than European.	Total.	European.	Other than European.	Total.	European.	Other than European	Total.
Cape -	617,131	1,982,588	2,599,719	192,007	1,009,223	1,201,230	31·11	50·90	46·20	5,855	81,253	87,108	3·04	8·05	7·25	9·48	40·98	33·50
Transvaal	498,413	1,265,650	1,764,063	140,639	491,448	632,087	28·31	38·82	35·83	3,267	25,397	28,664	2·32	5·16	4·53	6·55	20·06	16·24
Orange Free State.	181,613	352,985	534,598	79,532	150,492	230,024	43·79	42·63	43·02	2,242	7,495	9,737	2·81	4·98	4·23	12·34	21·23	18·21
Natal -	120,903	1,095,929	1,216,832	42,475	510 989	553,464	35·13	46·62	45·48	362	13,600	13,962	·85	2·66	2·52	2·99	12·40	11·47
Union -	1,418,060	4,697,152	6,115,212	454,653	2,162,152	2,616,805	32·06	46·03	42·79	11,726	127,745	139,471	2·57	5·90	5·32	8·26	27·19	22·80

* Preliminary figures. Final figures 1918 Census not yet available.

On the 14th October epidemic influenza was proclaimed a contagious or infectious disease in terms of the various Acts applicable to the several provinces. This gave to the Local Authorities "power compulsorily to remove to hospital or other "place of isolation cases suffering, or suspected to be suffering, "from the disease, or any person exposed to infection, or to keep "them under observation or surveillance."

On the 30th October and 5th November, Government Notices gave Local Authorities power to close theatres, bioscopes, &c. It was said that this power was used by a number of Local Authorities.

On the 30th November the following instructions were issued to Port Health Officers introducing a modified form of quarantine in respect of the disease.

To The Port Health Officer:
Durban, Port St. John's, East London, Knysna, Port Elizabeth, Mossel Bay, Simonstown, Cape Town and Port Nolloth.

Epidemic Influenza—Port Health Measures.

The following measures in respect of epidemic influenza should be taken in the case of vessels arriving at your port:—

(A) *Uninfected Vessels* (*i.e.*, those which the Port Health Officer, after due enquiry, is satisfied have no cases of influenza on board and have been free from Influenza during the current voyage or within the four weeks preceding arrival):—

(1) Full pratique to be given.
(2) Master and all on board to be warned of the danger of influenza infection on shore and urged to restrict intercourse between the vessel and the shore as much as possible.
(3) A supply of influenza vaccine to be furnished to the master or ship's surgeon if desired by them.

(B) *Infected Vessels* (*i.e.*, those having cases of influenza on arrival or having had cases during the current voyage or within the four weeks preceding arrival):—

(1) "Restricted" pratique to be given and only such intercourse allowed with the shore as may be necessary for off-loading, loading, coaling, &c., the Port Health Officer being authorised to relax or modify the restrictions at his discretion to meet any special circumstance.
(2) All on board to be medically examined by the Port Health Officer.
(3) Cases or suspected cases of influenza amongst the crew or in-transit passengers either to be landed for treatment in an isolation hospital on shore or else isolated

on board to the satisfaction of the Port Health Officer.

(4) Cases or suspected cases of influenza in passengers for the port to be landed and removed with their baggage and personal effects to an isolation hospital.

(5) Infected cabins or other accommodation, together with their contents which have been exposed to infection, to be fumigated with burning sulphur or formalin, or treated with some other suitable disinfectant.

(6) Healthy persons to be landed and allowed to proceed to their destination, the local authority at their place of destination being notified on the usual form that they may have been recently exposed to influenza infection.

(7) A supply of influenza vaccine to be furnished to the master or ship's surgeon if desired by them.

Where cases or suspected cases of influenza are landed for isolation, or where disinfection is carried out, the usual procedure should be followed as regards obtaining guarantees from the master or agent of the vessel for the payment of expenses

The master of every vessel bound for Australia should be informed as to proclaimed first ports of entry (namely, Fremantle, Adelaide, Melbourne, Sydney, and Brisbane) for vessels from South African ports, *vide* my telegram No. 14,485 of 15th October 1918.*

Where precautions have been taken in respect of any vessel the master should be furnished before sailing with a certificate as to the precautions taken and the reasons therefor for production at his next port of call.

(Sgd.) F. Arnold,
M.O.H. for the Union.

The Commission, while holding the view that maritime quarantine must be resorted to in preventing the entry of certain epidemic diseases into the Union, state that at present it is entirely uncertain whether such quarantine can be justifiably and usefully employed in a disease of the nature of epidemic influenza. The weight of expert evidence before the Commission was so decidedly against the effectiveness of maritime quarantine in a disease of this nature, that, although giving

* The following is a copy of Telegram No. 14485 above referred to:—
" Wire name of every vessel leaving your port for Australia since 1st Sep-
" tember. In conjunction with Customs inform master every vessel leaving
" for Australia he can only enter Australia at Fremantle, Adelaide, Mel-
" bourne, Sydney, and Brisbane. Any vessel from South Africa entering
" any other Australian port, except owing to stress weather or emergency,
" will be quarantined and ordered to one of the above ports also fined heavy
" penalty "

full credit to the experience of Australia, the Commission were not prepared to state that the measure should have been enforced in the Union of South Africa, nor were they of opinion that the timely enforcement of maritime quarantine would have prevented an outbreak in the Union.

Basutoland.

The mortality from influenza was heavy both among the European and native inhabitants of the territory. The disease was very prevalent during the months of October and November 1918. Births and deaths are not registered in Basutoland; accurate statistics of mortality caused by the outbreak are therefore unobtainable. Information gleaned from the observations of the medical staff, from the district officials, from the native chiefs, and from local statistics supplied by missionaries and traders indicate that the total deaths may be "safely estimated at 15,000," and that probably 75 per cent. of the total population were affected.

Swaziland.

The influenza epidemic of 1918 affected every part of Swaziland, but appears to have been of a less severe type than in most parts of South Africa. Relief measures were appreciated, and the natives submitted freely to inoculation and to the administration of the drugs supplied. The deaths among the native population numbered about 1,250, which is slightly more than 1 per cent. of the total population.

Bechuanaland Protectorate.

Influenza broke out in epidemic form in the Southern Protectorate in October 1918. It spread rapidly, and eventually affected the whole territory except Western Kalahari and the Ngami littoral. The mortality among the native population from influenza and its complications is estimated as having been between 4 and 5 per cent.

Southern Rhodesia.

In a report by the Medical Director, Southern Rhodesia, it is stated that epidemic influenza first gained access to the country along the railway line from the South, the first case in epidemic form occurring among the railway staff at Bulawayo on the 9th October 1918. Within a few days the disease broke out with extraordinary virulence at Que Que Umoma and Salisbury. Spreading up and down the railway lines from those places it rapidly extended until practically every district was more or less affected, "the extent of the infection being

" governed by the density of the population in any particular " centre and the mode of communication with other affected " places."

Incidence among Europeans.—It was found impossible to estimate " anything like accurately " the total number of cases which occurred. The European population was estimated to be 36,953, and among them 352 deaths from epidemic influenza were recorded between October and December 1918. Most of the deaths were in Salisbury and Bulawayo, and about 63 per cent. were between the ages of 25 and 45.

Incidence among Natives.—Among Natives the disease was most severe in the large towns and on the mines. Native miners suffered more severely than any other class of the population, and labourers on farms and those living in their own Kraals suffered least. Among Natives on the mines the deaths recorded as due to influenza (or pneumonia directly attributable to influenza) totalled 3,006, which is about 97 per thousand of the number employed. The mortality in proportion to the labouring population was highest on mines in the Gwelo, Victoria and Bulawayo districts.

The indigenous native population of the territory is estimated at 770,000 and the number of deaths recorded between October and December was 19,603. From the Kraals, however, no returns of births or deaths are received. It was said that the proportion of deaths among women and children in the native territory was higher than among natives working in towns and on mines, partly, perhaps, because they were more concentrated in the Kraals and refused to scatter and live out in the bush.

Measures.—Municipalities and Village Management Boards, being the local authorities under the Public Health Act, were, with the assistance of the Government, the administrative bodies concerned with the disease in the areas under their control. It is said that, without exception, they rose to the occasion and displayed great zeal, energy, and efficiency in coping with the problem. In Bulawayo and Salisbury, where the disease was most widespread, the Mayors and Town Councils formed Committees, called for volunteers, and undertook generally the entire control of the transport and care of the sick, the feeding of the public and the distribution of food stuffs. Schools, churches and places of entertainment were closed throughout the country, emergency hospitals and soup kitchens were established, and arrangements for house to house visitation and home attendance by lay nurses were organised. In native reserves the administration of relief for the sick was in the hands of the Chief Native Commissioner, helped by the police, and by missionaries and other volunteers. Native chiefs were urged to use their influence to prevent people from visiting industrial centres, from coming into

contact with those who were fleeing from infected places and from making inter-Kraal visits. All natives were advised to adopt life in the open air.

Inoculation stations were opened at which district surgeons, veterinary surgeons, dispensers and others who were specially instructed in the technique were in attendance. The vaccine used was a mixed vaccine obtained from the laboratories at the Cape and elsewhere in the Union of South Africa, and altogether about 84,000 doses were issued. It was thought that although inoculation did not arrest the progress of the epidemic, it did much towards reducing the mortality from serious complications.

Nyasaland.

The history of the invasion of Nyasaland by influenza as gathered from a despatch by the Acting Governor, dated 27th February 1919, and from a report by the Port Medical Officer dated 14th February 1919 is somewhat as follows :—

Intelligence of the outbreak of influenza in the Union reached Nyasaland at the beginning of September 1918 ; it was then considered to be of a comparatively mild type, but attended with extreme lassitude and prostration and a low mortality.

In October the character of the disease altered ; the cases were more severe and the mortality increased, while the disease rapidly spread over the continent.

On the 18th October cases were reported from Salisbury in South Rhodesia, and on the 20th the disease appeared at Beira. The Zambesi at Chindio was reached by 27th October, some cases having been previously discovered in the river boats.

Following the Shire Highland Railway the disease first appeared in Nyasaland at Port Herald on 5th November 1918. Limbe and Blantyre were reached on 9th November and by the 18th Zomba was involved.

Fort Johnston remained unaffected until 3rd December, when cases were reported from this station and also from Mangoche and the Bar.

New Lanjenburg and Kyambila notified the presence of the disease on 5th December and Mwaya on the 6th. " From this " date the disease made its appearance in various parts of " Nyasaland, precise dates and returns, however, being difficult " to obtain."

The whole of Nyasaland was affected by the scourge, which reached its maximum virulence in December, and declined steadily during January 1919. The presence and passage of troops, who suffered severely, undoubtedly helped to spread the

disease. The following statistics are taken from the report of the Port Medical Officer :—

Europeans.			Indians.			Natives.		
Cases.	Deaths.	Rate.	Cases.	Deaths.	Rate.	Cases.	Deaths.	Rate.
		Per cent.			Per cent.			Per cent.
1,418	67	4·7	129	19	14·7	14,327	1,683	11·7

In his covering despatch the Acting Governor says that "extremely drastic precautions were taken from the first to "prevent the disease, if possible, from entering the Pro- "tectorate. . . ."

It is pointed out also that the statistics above include only patients admitted to various Government hospitals and "are "far from being an exhaustive record of the epidemic, since "scores of thousands of cases have occurred in outlying native "villages. . . ."

It was apparently difficult to do very much for the native population, to whom the disease was a complete novelty, but through the local headman instructions concerning the nature of the disease and the precautions to be taken were promulgated by the British officials throughout the Protectorate.

A good deal of tact seems to have been displayed in reassuring natives who considered the closing of churches and schools to mean that the Government "had permanently for- bidden divine worship and education." By many of the less civilised the disease was attributed to witchcraft, and "in the "remoter villages there was at first a tendency to resort to the "ordeal by poison."

The civil medical staff was aided by the military medical establishment, but, even so, great difficulties were encountered. Two medical officers and a nurse as well as 11 non-com- missioned officers of the South African Medical Corps died on duty.

Uganda.

Cases of epidemic influenza at Entebbe were reported during the last week of October 1918, and a few days later at Kampala and Jinja. The epidemic spread throughout the protectorate. At Entebbe the wave reached its height during the third week of November, and then declined rapidly, no fresh cases being reported after the middle of December. Most other stations were free by the end of December.

The statistics available represent only a very small proportion of the sickness which occurred. Up to the end of December the cases treated by Government medical officers at Entebbe, Kampala, Jinja, Nasindi, Masaka, Mbarara, Mbale, Soroti, Butiaba, and Kelle numbered 4,663, and among them 184 deaths occurred. Native inspectors in the Mbale district reported 10,587 cases, with 436 deaths, and the Native chiefs in the Lira district reported that 75 per cent. of the population had been attacked, with over 5,000 deaths. Among 104 Europeans attacked no death occurred.

Early in 1919 the Governor of the Protectorate caused inquiries to be made into the number of deaths which had occurred among natives up to the end of February. The returns received showed 15,268 deaths, of which 10,000 were in the Eastern province, 2,036 in the Northern, 960 in the Western, and 2,272 in Buganda.

Zanzibar.

Influenza was prevalent in Zanzibar from October to December 1918. Cases had already occurred in British East Africa in October, following the arrival in Mombasa of two ships from Bombay, on each of which deaths from influenza had occurred. On 15th November the deaths in Zanzibar numbered 33. All public meetings, processions, and entertainments were prohibited, schools were closed, and church services reduced to a minimum. Energetic relief organisations were soon at work. The total number of deaths in both the islands was 1,446 (population 196,733).

Somaliland.

In a report dated the 11th January 1919, the Senior Medical Officer, Somaliland Protectorate, expressed the opinion that in all probability the disease was introduced into Berbera (where the earliest cases occurred) by a steamer from Aden. The first few cases in October were among Somalis, and for a few days were not recognised to be influenza. Early in November the disease became epidemic in Berbera, and from there it spread in all directions along the caravan routes and by dhows and other vessels to practically every part of the occupied country. French Somaliland and Abyssinia were also centres from which there was a general spread of the epidemic. By the end of December the epidemic had practically ceased in most parts of the country.

Incidence and Mortality.—From the few statistics which it was possible to collect it was estimated that over 50 per cent. of the Somali population were attacked, and that of those who suffered 5 per cent. died. On the whole it was thought that

the mortality was not so high as in similar epidemics in other parts of the world. Most of the deaths were due to pneumonia, "but a few died from sheer exhaustion and some apparently from fright."

Several Europeans contracted the disease, but there was no death among them.

Among 800 Indian troops in the country about 500 (62 per cent.) were attacked, and there were 53 deaths (10·7 per cent.).

Among 550 Somali troops there were 336 cases with 15 deaths, and among 131 foot and mounted police at Berbera there were 46 cases with 3 deaths.

Egypt.

Influenza as a cause of death does not appear in the annual return of births, deaths, and infectious diseases of the Egyptian Government.

Cases of the disease were notified at Alexandria in May 1918, at Port Said in June, at Cairo in July, and in the province of Dakahlie in August.

The mortality figures for 1918 show that the disease was relatively fatal during the three last months of the year, months of high influenza mortality in neighbouring countries. Thus the number of deaths of "non-notifiable infectious disease" rose from 111 in October to 856 in November, and to 1,568 in December (annual total of 3,049). A similar rise in mortality is seen in connection with pneumonia, October 640, November 1,489, December 1,735 (annual total 6,125). Bronchitis, October 759, November 1,647, December 1,807 (annual total 10,051); and "other diseases of the respiratory system," October 79, November 209, December 169 (annual total 981). From all causes combined the mortality during the last two months of the year amounted to one quarter of the total year's death roll.

Tunisia and Algeria.

As in Egypt two "waves" of influenza swept over Tunisia and Algeria; the first appeared at the end of May and was very mild; the second, from September to December, caused a large number of deaths.

Senegal (French).

As was to be expected, the first mention of influenza in the French Protectorate of Senegal, relates to Dakar. On the 14th September, the British Consul wrote, "Serious epidemic "Spanish influenza appeared Dakar; has been brought by "vessel coming from Sierra Leone. Exact number of cases "not ascertained yet, no deaths reported."

(On the 21st September, H.B.M. Consul at St. Vincent, alludes to this and says, "epidemic influenza is devastating Dakar . . . .")

Telegraphing again on the 18th, the British Consul at Dakar states that "large numbers of cases of influenza continue to "occur at Dakar, resulting in about 30 deaths per day."

During the period, September 19–29, 1918, there were 221 fatal cases of influenza reported at Dakar, of these 22 occurred among Europeans, 30 among South Americans, 169 among natives. Dakar is the most important sea town in French West Africa, and at this busy port conditions of living approximate more closely to European standards perhaps than in any other sea port in West Africa.

Of late years, it has been a port of call for British passenger ships, and in the excellent harbour, vessels are moored alongside wharves, a rare experience on the West Coast of Africa.

Cape Verde Islands.

His Britannic Majesty's Consul at St. Vincent in a despatch to the Foreign Office, dated 21st September 1918, reported that epidemic influenza was "devastating Dakar, Sierra Leone, and "the Gambia, and has made its appearance in the harbour of "St. Vincent."

The Brazilian destroyer "Pianby" arrived from Dakar on 11th September with 22 cases on board; the number of cases increased daily until there remained only three officers and five men unaffected. One officer and one man died in hospital ashore, while there was another fatal case aboard. On the 18th of September the British steamer "Matra" arrived from Dakar and the next day the master reported 27 cases (all Lascars). In all 41 patients were removed to the lazaretto.

In a later despatch, dated 28th October 1918, His Britannic Majesty's Consul states that, at the time of writing, the epidemic of influenza had begun to abate in St. Vincent, though the disease had appeared in other islands of the group. According to the latest report then available there were about 9,000 cases with 300 deaths in St. Vincent and among the fatal cases were two British.

When it first appeared the disease seems to have been mild in character, but it spread rapidly, affected all classes, and became more virulent towards the end of October. Labour conditions were very adversely affected.

St. Vincent is a port of call for the Castle liners and other steamers bound for South Africa; there is also a good deal of maritime intercourse with the coast of West Africa.

Gambia.

No accurate information as regards births or deaths for this Protectorate is available. The influenza epidemic was serious; it commenced in September 1918 and lasted about four weeks, with the result that 6 Europeans and 317 natives died in Bathurst (population 7,700 in 1911). It is estimated that there were 7,800 deaths in the Protectorate from influenza; the population of the Protectorate in 1911 was 138,401.

Sierra Leone.

The first indication of an outbreak of influenza in this Colony was given during the week ending 24th August 1918, when an unusual prevalence of catarrhal conditions of the nose and throat was noted at Freetown. A day or two later two deaths of natives from bronchial pneumonia occurred, and on the 27th August the Serra Leone Coaling Company reported that of a staff of 600 native porters, 500 had failed to put in an appearance. Shortly afterwards labour became almost unobtainable and public offices and work places were practically closed. The week ending 31st August was the most critical period of the epidemic; the disease was spreading very rapidly, and was marked by dangerous broncho-pneumonic and cardiac complications. Both Europeans and natives were affected, and five of the medical staff were attacked. During the following week the mortality increased considerably; there were 28 deaths on the 1st; 34 on the 2nd; 74 on the 6th; and 72 on the 8th September. Drs. Allan and Young, on whose report these notes are based, state that the course of the epidemic was so rapid that the disease had a firm hold before prophylactic measures could be efficiently applied. During the second week of September the mortality began to decline steadily. So far as could be ascertained the disease accounted for about 1,000 deaths in Freetown alone and deaths were numerous in up-country districts. No reliable data were available for an estimate of the age and sex incidence. The measures of relief undertaken included house to house inspections, the distribution of medicines and food, and the opening of an auxiliary hospital; much assistance was given by voluntary workers

Liberia.

Early in October, 1918 the Government of Liberia circulated among the various West African Colonies copies of a Proclamation in which it was advised that from October 4th no person would be allowed to land at Liberian ports on account of the serious epidemics of influenza raging elsewhere in Africa but which had not, up till then, reached Liberia.

The Republic did not remain free, however, and the disease first appeared as an epidemic, in the interior, in the early part of November, travelling thence to the coast and reaching Monrovia, the capital, about November 15th.

The visitation is said by the Acting Consul-General to have been severe and in Monrovia there were some 50 deaths; the absence of any organised medical service, however, accounts for the difficulty in obtaining any reliable figures. Travelling facilities also in Liberia are few and far between, and the dwellers on the coast do not in any great numbers venture inland. The fact that the disease was brought to the coast from the Hinterland is interesting in this connection and may also be related to the closing of the Port of Monrovia. The quarantine restrictions were raised as from 2nd January 1919.

Ashanti.

The epidemic first appeared in Coomassie on 23rd September 1918, reached its height in October and declined during the following month. The disease spread from Coomassie to all villages in the vicinity, and also along the Ejura road towards the Northern Territory. In Coomassie the deaths from influenza of 381 natives, 5 Syrians, and 2 Europeans were registered. It is reported that these figures give an inaccurate idea of the mortality caused, as by far the greater proportion of bodies were taken out of the town for burial and so remained unregistered. The approximate number of deaths due to influenza is estimated as:—Central Province, 4,500; Western Province, 1,500; Northern Province, 500; Southern Province, 2,500; total, 9,000.

Gold Coast.

The Influenza figures for 1918 and the two previous years on the Gold Coast are:—

1916.	1917.	1918.
27	280	7,756

As might be expected, the disease first appeared in the littoral towns, and the earliest sign of its appearance seems to have been at Cape Coast, where the mail officer became ill, after boarding the S.S. "Shonga" on 31st August 1918. Cases were then reported from (1) Seecondee, 5th September; (2) Saltpond, 21st September; (3) Winnebah, 24th September; Accra, early in September; (4) Axim, 25th September.

From the Coast the disease travelled rapidly along the lines of communication and reached Ashanti late in September. The Hinterland of the Northern territories, to which access is less easy, was invaded last, cases were not being reported until the beginning of October.

In Accra, the capital of the Colony, the Senior Medical Officer reported :—" The pandemic had already assumed con-
" siderable proportions among the natives of Accra and
" Christianburg before it affected any member of the European
" Community. The first of the white population to suffer
" were those whose duties brought them into daily and close
" contact with the natives. The earliest case notified was the
" police magistrate, 18th September 1918, then followed in
" rapid succession members of various firms, officials of the
" police office, customs and police. On the 30th September,
" when the pandemic appeared to be at its height, 17 fresh
" cases were reported, and by the end of the month the total
" had reached just over 60. From then on, there was a steady
" decline until the 10th October when the last case was
" recorded."

Amongst 82 Europeans attacked in Accra there were eight deaths.

The Medical Officer of Health for Seecondee, who was able to make a census of a controlled portion of the population in this port, considered that the mortality rate was about 4·55 per cent.; the figures forming the base for the calculation include 2,599 natives employed in various Government Departments together with 80 Europeans.

In the colony, as a whole, the Senior Sanitary Officer thinks that probably 4 per cent. of the population died during September and October; the case mortality was higher both in the coast towns and in the northern territories than in the forest belt of Ashanti.

The pandemic created much consternation among the natives, one of whom wrote :—

" The fell disease on every side
Stalks forth with tainted breath,
And pestilence with rapid stride,
Bestrews the land with death."

The symptoms usually present were: headache, pains in the back and limbs, sore throat and pain in the line of the trachea. There were also infected conjunctivæ, slight coughs and a temperature ranging between 100–102. The commonest complication was broncho-pneumonia, but lobar pneumonia and pleurisy with and without effusion were by no means rare. Miscarriage among native women was a common occurrence.

Nigeria.

The epidemic appeared in Nigeria towards the end of September 1918, was very severe in October, and declined abruptly in November. It was imported into Lagos by sea from the Gold Coast and soon spread all over the country. Calabar was the last place to be infected owing to the infrequency of the shipping service; the epidemic was at its height in Calabar when it had all but died out in Lagos.

In the majority of cases, here as elsewhere, respiratory complications were much in evidence; a gastro-intestinal type of the disease was also noted. All the deaths among Europeans were due to broncho-pneumonia.

From statistics of prisons, police force, West African Frontier Force, and Government employees, a case incidence of 50 per cent. with a mortality of 5 per cent. "would probably be a low estimate." It is estimated from the register of deaths that 1·5 per cent. of the population of Lagos died of influenza.

Europeans who came under treatment numbered 418 of whom 15 (3·5 per cent.) died. There were 5,887 natives treated as in-patients for influenza of whom 11 per cent. succumbed.

Belgian Congo.

The acting British Consul at Boma, in a despatch to the Foreign Office dated 17th December 1918, wrote:—

> "Epidemic of Spanish influenza has broken out at Boma, Matadi, and Kinshasa, and has been spreading to the enterior of the Colony, and, (to that) of French Equatorial Africa, both from Boma and Elizabethville. It first appeared at Boma and Matadi about middle of November, and was introduced by passenger steamers from Europe. Up to the middle of December, there have been 119 deaths at Boma, (Europeans, 9; natives, 110). The epidemic seems abating."

Among the precautions taken it is mentioned that five days quarantine was imposed on each steamer, after the last case on board had been cured. No one was allowed to travel by steamer or train without a medical certificate, and quinine as a propylactic was advised.

There seems to have been a dearth of medical men in the Colony during the time the epidemic was in progress.

St. Helena.

St. Helena is one of the few places that appears to have escaped any serious epidemic of influenza in 1918. In that year the total civilian death rate for the island was only 9·85 per thousand, the lowest rate reported since 1909.

Mauritius.

Mauritius, also, appears to have escaped the almost universal epidemic of 1918. The death rate for the island in that year was only 1·1 per thousand above the mean rate of the previous quinquennium. The bronchitis and pneumonia deaths formed 5·8 and 5·6 per cent. of the total mortality, as compared with

5·0 and 5·8 during the previous five years. But the island did not long enjoy immunity. During May and June 1919, a very severe epidemic was experienced. The epidemic curve reached its height at the end of May and was at a high level throughout June. Thereafter a rapid decline in incidence was experienced, but deaths from influenza and chest complaints were in marked excess of the normal until the end of October when normal rates of mortality once more obtained.

The following table sets forth the number of deaths that were ascribed to influenza (including deaths from bronchitis, pneumonia and broncho-pneumonia) between May and October in half-monthly periods :—

1919.			1919.		
May	1–15	259	Aug	1–15	146
,,	16–31	5,108	,,	16–31	130
June	1–15	4,299	Sept.	1–15	124
,,	16–30	1,154	,,	16–30	148
July	1–15	299	Oct.	1–15	126
,,	16–31	195	,,	16–31	97 (practically normal rate).

ASIA.

Cyprus.

Towards the end of 1918 Cyprus suffered from an epidemic of influenza. Accurate statistics are not obtainable, but it is known that several thousand cases of influenza were treated in hospitals and dispensaries, and, as no other outbreak of serious infectious disease was noted during 1918 it appears safe to assume that influenza was chiefly instrumental in the increase of the death rate from 18·8 per thousand in 1917 to 26·1 per thousand in 1918. The estimated population of the island was 306,997.

Persia.

The following notes are extracted from a report by Dr. A. R. Neligan, Physician to His Majesty's Legation, Tehran.

When influenza appeared in Persia during 1918 it found the population already much weakened by two previous epidemics of typhus and relapsing fever which ravaged the country in the winter of 1917–1918 and the spring of 1918 respectively. Moreover, the years 1916 and 1917 had been unusually dry, so that crops failed and there was a food shortage.

The epidemic is said to have invaded Persia at several points.

North from Russia viâ—

(1) Askhabad, whence it gained Meshed (August 3) and from there spread west along the Tehran road and south to Birjand (August 4) and Seislan (September 2), and possibly Yezd (October 3).
(2) Enzeli (August 4) from Baku.
(3) Tabriz (September 2) from the Caucasus *viâ* the Julfa Railway from Tiflis.

South from India viâ—

(1) Bunder Abbas (October 1).
(2) Bushire (September 4), thence to Shiraz (October 3) and Kerman (November 2).
(3) Mohammerah (October 1) *vi* Basra. Thence to Ahwaz, Shuster, and Dizful.

West from Mesopotamia.—Two "waves" appeared in Mesopotamia: one began in June and lasted till August; the second in September and lasted till November. The primary wave followed the Baghdad–Kermanshah road along which there is heavy motor traffic into the interior. Kermanshah was affected in August and was followed successively by Hamadan, Kazvin, and Tehran. The disease travelled more quickly along motor routes than along caravan ways. From Tehran the disease travelled down the great South road reaching Isfahan in the centre of Persia in the third week of October and probably going on to Yezd.

Mortality.—There are no exact figures regarding the population of Persia so that incidence and mortality statistics cannot be quoted. It was found, however, that the mortality was much higher among country people than among urban dwellers; it was very heavy among malarious subjects; it was relatively much less among Europeans than among Persians and was particularly heavy among Indians.

Incubation Period.—Generally 24 to 48 hours. "In one case at Kerman it was definitely established at six hours."

Symptoms.—Similar to those observed elsewhere. At Kerman, however, most of the cases presented curious oral symptoms, consisting of "a tongue coated with a thick brown fur showing "cleaner edges and tip. The soft palate was invariably covered "with a bluish white mycelial growth which might be thick or "thin."

At Bushire several varieties of skin eruptions were noticed.

Complications. — Thoracic complications — broncho-pneumonia, lobar pneumonia and empyema—were the most common and the most frequent cause of death. Many cases of pulmonary tuberculosis, which is a common disease in Persia, rapidly went from bad to worse after influenza.

Intestinal complications, especially enteritis with blood and mucus in their stools, were very common.

Treatment.—At Kerman a vaccine was prepared and was said to have done good in four out of the five cases in which it was used—otherwise salicylates and aspirin were most extensively used.

Notes concerning the Epidemic at various Places in Persia.

Kermanshah (40,000).—Practically the whole native population attacked; mortality in towns, 1 per cent.; in country districts up to 20 per cent.

Hamadan (30,000). — Disease widespread, causing some 1,000 deaths. In one series the case mortality was 10 per cent., all due to broncho-pneumonia. Nasal douching with eusol much lauded in this district.

Kazvin (20,000). — Epidemic began on 16th September among troops that had come from Hamadan. Purpura and jaundice were observed in this case and epistaxis was common.

Tehran (250,000).—Epidemic broke out on or about 22nd September 1918 with extreme suddenness.

There was a high west wind at the time and this was thought to be the carrying agent so that the affliction was called "the disease of the wind." Disease was probably introduced by travellers from Kazvin, though the generalisation of the epidemic in the city took place within a day or two of this high wind; native doctors, too, noted that all who were out of doors during the boisterous weather invariably fell ill.

The death-rate among the poor was very high and a nominal estimate of the total mortality for three months is 2,000. Fresh air appeared to have no effect as a preventive, and out of 80 British motor transport men who had been living in the open practically all succumbed to the infection. The country people were particularly hard hit again.

Ifahan (80,000).—The disease here was not of a severe type. Deaths about 300.

Meshed-i-Sar.—This port and the Province of Mazanderan in general appear to have been invaded by way of the interior and not from Russia.

The epidemic was very widespread, and the old and young suffered severely.

At Meshed-i-Sar a case mortality of 10 per cent. was reported.

Enzeli (10,000).—Infection brought from Baku at end of August. Type mild; mortality about 2 per cent. Armenian refugees from Russia suffered severely.

Meshed (100,000).—Approximate number of cases was 70,000, of deaths 3,500. Case mortality in city, 5 per cent., in villages, 7 per cent. Outbreak coincided with violent gale of wind. The whole province of Khorassan was attacked, and from Meshed the disease travelled South to Seistan.

Tabriz (200,000).—Mild type of disease affecting half the population; the whole province of Azerbaijan was affected.

Seistan.—There were three distinct epidemics:—

(1) Infection from Meshed by way of Birjund (12,000 cases, with 100 deaths) to Nasratabad—the capital—(7,000 cases and 120 deaths). Whole province affected.

(2) Local outbreak at Kwast, probably introduced from India. In 139 cases among troops there were 40 deaths, all due to pneumonia.

(3) *Dehaneh Baghi.*—Severe local epidemic in January 1919. Infection probably brought by nomads from the Sarhad.

Bushire (30,000).—Disease introduced by ship from India. Type severe; 15,000 cases, with some 1,500 deaths. "From "this port the infection spread to Skiraz (150 miles away), "without losing any of its severity on the way, to cause, in the "Province of Fars, the most severe outbreak suffered by any "Persian Province. On its way up country it passed through "Kazerun, from which town also comes the story of a preliminary "gale, with hail."

The inhabitants took flight, and it is said that the local doctors treated their patients with prescriptions handed through barely opened doors.

Shiraz (50,000).—"The disease was of a very severe type. There were at a low estimate 2,000 deaths. The whole province was almost suddenly disorganised and a heavy sick rate among medical personnel, transport workers and telegraph and postal official added to the difficulties of rendering aid where it was wanted. The death rate in a Persian force was 10 per cent., in an Indian force also 10 per cent. A post of Indian and Persian troops of 416 men lost 31 per cent. of its strength. Another post lost 72 per cent. of its Indian garrison; these men had suffered most severely from malaria.

The disease spread to the whole province and was particularly severe round Shiraz. Young adults suffered most, and men more than women. An outdoor life did not prevent people contracting the disease, but cases treated in tents or the open air did best. The disease worked through a town, village or lines in 24–28 hours.

Kerman (40,000).—Here the epidemic was also severe and came from Shoraz *viâ* Saidabad. There were 4,000 deaths: the

case mortality in the town being 10 per cent. (7 per cent. hospital cases) and 30 per cent. to 40 per cent. in villages.

Yazd (4,000).—Disease probably introduced from Isfahan. There were 250 deaths; in some villages 25 per cent. of the population were reported to have died.

Mohammerah.—Infection from India *viâ* Basra. Outbreak severe; 6,000 cases, with about 250 deaths.

Ahwaz, Dizful and Shuster.—No severe epidemic. Two separate outbreaks: one in August among civilians and the other in November (severe) among a British force at Ahwaz.

Bunder Abbas.—Disease introduced by ship from India. Type mild: but disease lasted three months.

Afghanistan.

Information regarding influenza in Afghanistan is necessarily incomplete, but two facts of interest stand out prominently. First, Afghanistan suffered from a widespread epidemic comparable in severity to that which afflicted northern India; second, the epidemic in Afghanistan synchronised with the outbreak in northern India, the two reaching their height almost simultaneously.

No information is forthcoming regarding the source from which Afghanistan became infected.

The disease was first noted in Kabul in the first half of October and during the ensuing fortnight a large proportion of the population fell ill. A rough estimate puts the proportion affected at upwards of 80 per cent. At first the disease does not appear to have been attended by a high case mortality rate, but in the third week of October the mortality in Kabul began to increase in an alarming manner, between 60 and 80 deaths occurring daily in and around the town. During the last 10 days of October the mortality in the city alone is estimated to have been 100 a day, and the vast majority of people attacked are said to have succumbed. All the distressing features of the outbreak that were observed in India appear also to have been experienced in and around Kabul. The epidemic continued unabated till the close of the first week of November; thereafter a more or less rapid decline was experienced. The large proportion of the population attacked would appear to have determined the short duration of the epidemic, as was the case in the severely affected tracts of India. The mortality in and around Kabul attributable to influenza during the four weeks ending November 8, 1918, is roughly estimated at 10,000.

Though no definite information is forthcoming, reports state that the disease was widely prevalent throughout the whole of Afghanistan.

India.

Reports received indicate that no part of the world suffered as severely as did India during the latter half of 1918. A preliminary report by the Sanitary Commissioner with the Government of India estimated that up to the end of November 1918 no fewer than five million deaths were attributable to the influenza pandemic in British India alone (population, 238,000,000). In the native states influenza deaths certainly exceeded one million in the same period. A later report states that between 1st June and 31st December 1918, the total mortality in British India exceeded the quinquennial mean mortality for the same period by approximately seven millions. The great majority of this excess mortality occurred in the last four months of the year. There was no abnormal prevalence of other communicable disease, so it is justifiable to accept this figure as a rough approximation of the influenza death roll. In view of the conditions pertaining in India, anything other than approximations of this nature is impossible to obtain.

The presence of an epidemic disease of an unusual type was first noted in Bombay City in June 1918. Towards the end of that month the widespread prevalence of a mild influenza-like disease occasioned a shortage of labour in the offices, mills, banks, &c., of that city. Sporadic cases of a similar nature were observed in Calcutta in the middle of June, and in Karachi and Madras towards the end of the month.

The mild nature and the low case mortality rate of the early cases made it impossible to fix a date for the commencement of the epidemic in India and extremely difficult to determine its source of origin. There is evidence of a debatable kind that influenza was present in the Thana district of the Bombay Presidency in the early spring months of 1918. Some of the earliest cases recognised in Bombay occurred in a transport that arrived at the end of May; these cases only occurred after the vessel had entered dock, and it is by no means certain that infection was not acquired in Bombay city. There had been no untoward sickness on board this vessel during her voyage to Bombay.

During July and August influenza became widespread throughout India; everywhere it was of a mild form and did not produce any appreciable increase in the mortality rates. In the middle of September, however, the mortality in Bombay city began to rise in an alarming manner, and increased day by day till the 6th October, on which day 768 deaths were recorded. This second virulent epidemic (which very few parts of the country escaped entirely) occurred somewhat later in other parts of India. The total mortality in India in the month of October is without parallel in the history of disease.

Below is given a table from the preliminary report of the Sanitary Commissioner with the Government of India which, though incomplete, indicates the varying severity of the disease during the four or five months prior to 30th November 1918 in the different administrations of British India arranged in order of severity.

It is noteworthy that the first seven provinces on the list are situated in the western or central parts of India in comparison with which the eastern provinces suffered but little. Considerations of climate or mode of living appear insufficient to explain the very unequal incidence of influenza mortality, though, generally speaking, the sea coast areas suffered less than inland districts. The incidence of the disease on the inland areas of Bengal, Bihar, and Burma, however, compared favourably with that of the littoral.

In the first epidemic wave, June–August, the mortality was greater in towns than in rural areas; in the second virulent epidemic, September–December, the mortality rates in rural areas far exceeded that of the towns. How far the lower urban mortality can be ascribed to relatively efficient health organization, and medical and other assistance, is difficult to say: it is possible that these were factors of no small importance.

Provinces.	Population Census 1911.	Estimated Total Deaths from Influenza up to 30th November 1918.	Proportion of Deaths from Influenza per 1,000 Inhabitants.
Ajmer-Mewara	501,395	33,407	66·6
Central Provinces and Berar	13,916,308	790,820	56·8
Delhi	416,656	23,175	55·6
Bombay	19,587,383	900,000	45·9
Punjab	19,337,146	816,317	42·2
North West Frontier Provinces	2,041,077	82,000	40·0
United Provinces	46,820,506	1,072,671	22·9
Coorg	174,976	3,382	19·0
Madras	40,005,735	509,667	12·7
Assam	6,051,507	69,113	11·4
Bihar and Orissa	34,489,846	359,482	10·3
Burmah	9,855,853	60,000	6·0
Bengal	45,329,247	213,098	4·7
Total for British India	238,527,635	4,933,132	20·7

The second wave of influenza had not exhausted itself by the end of 1918, and there is evidence that in certain parts of the country the disease persisted throughout 1919. In Rangoon, Calcutta, and Bombay, deaths diagnosed as influenza were registered in each week of the year, and there was definite evidence of two distinct waves of enhanced virulence. Neither, however, was so widespread or of such an explosive character

as was the second wave of 1918. In Rangoon, the first wave began in the first week of February and reached its maximum in the third week. The prevalence declined gradually, the whole period covered by the wave being 16 weeks. The second wave began at the end of May or early in June, and caused its maximum mortality during the week ending 12th July. This wave lasted 14 to 16 weeks. In Bombay the first wave reached its height between the middle and end of May, but in the second wave, which commenced in the middle of July, the mortality fluctuated from week to week without showing a definite peak. In Madras, the second epidemic was widespread. In Calcutta there was a slight wave between the 20th March and 12th April, and a second wave commenced about the middle of July. Other parts of India suffered in like manner, but in comparison with the incidence in 1918, the outbreaks for the most part were relatively mild, and showed less tendency to widespread dissemination of infection.

Measures.—The epidemic of 1918 struck India at a time when she was least prepared to cope with a calamity of such magnitude. War demands had depleted the personnel, and many of the staff available were incapacitated by the disease. The effects of the almost total failure of the monsoon practically throughout the country were still more serious.

In all the larger towns where severe epidemics occurred numerous additional dispensaries were opened and numerous agencies were employed for the free distribution of drugs and milk; in certain towns municipal grain shops were opened which supplied grain below the market rate. But in remote villages it was almost impossible to do anything appreciable to alleviate suffering. In certain provinces travelling dispensaries did a large amount of useful work, and in most provinces the whole of the vaccination staff were engaged in measures of relief. Circulars and pamphlets were widely distributed and endeavours were made by all administrations to instruct the people as to measures of prevention and measures to be adopted when attacked.

Immunity and use of Vaccine.—In a report on the prevalence of the disease during 1919 it was stated that no figures were available to show how far an attack in 1918 had protected individuals from infection in 1919, but that, speaking generally, the areas which suffered most severely in 1918 escaped lightly. Early in the second virulent wave of 1918 the preparation of a vaccine was begun locally and it was largely used. During 1919 four laboratories were engaged in the manufacture of a mixed vaccine, about 1½ million cubic centimetres of which were issued. In addition large quantities were received from England for use by military units. It was said that individual medical officers spoke highly of the results, but that no statistical evidence of the preventive value of the vaccine was available.

Straits Settlements.

No detailed reports are yet to hand regarding the incidence of influenza in the Straits Settlements in 1918, though the disease was apparently widespread. Of the 36,294 deaths from all causes reported during the year only 3,500 were ascribed to influenza. The estimated population of the colony was 809,869.

In November and December the disease was present in Christmas Island.

In Labuan influenza was largely responsible for the productiod of a higher death rate than any reported since 1911.

The Phillipine Islands.

During the spring and summer of 1918 influenza was widespread in these islands, but caused very few deaths; during the autumn the pandemic fatal type of the disease became very severe, especially between the middle of October and the end of November, and is said to have caused 85,000 deaths.

China.

On 27th July 1918 it was reported that about half the population of the city of Chungking, in the province of Sze-chuen, were affected with epidemic influenza, and, although no statistical reports are available, there is reason to believe that from that month onwards the disease was widespread and severe. In September it was prevalent at Hankow, and in October Canton, Chefoo, Hankow, Hong Kong, Changsha (province of Hu-nen), and Shanghai were among localities from which reports were received that the disease was widespread among natives and the foreign population. Towards the close of the year many villages suffered a very high mortality from pulmonary complications of the disease.

In Hong Kong the deaths from influenza reported during 1918 were 405, and from pneumonia 2,251, as compared respectively with *nil* and 1,532 in 1917.

In Weihaiwei influenza was epidemic during October and November 1918 throughout the territory. Registration of deaths is not compulsory, and a large proportion of deaths go unrecorded; 900 deaths are, however, reported as having been caused by the epidemic (population 147,177).

Japan.

Between 1st October 1918 and 30th April 1919, 250,333 fatal cases of influenza were reported in Japan.

PART III.

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* A Medical Officer of the Ministry.

I.

Report on Incidence of Influenza in the University and Borough of Cambridge, and in the Friends' School, Saffron Walden.

By

S. Monckton Copeman, M.D., F.R.S.

In connection with the work of the Influenza Committee, I received instructions to undertake inquiry into the incidence of influenza during the period 1918–19, as it had affected the university as compared with the borough of Cambridge. The university population appeared to offer specially favourable material for such investigation, owing to the fact that it consists in large measure of young adults on whom elsewhere the incidence of influenza had been specially marked during the past year. Moreover, in addition to the graduate and undergraduate population, there are, at present, 400 junior naval officers, exclusive of staff officers and batmen, who went into residence at Cambridge for a period of six months from January 31st, 1919, rooms being allocated to them in certain of the colleges, more particularly Trinity, Caius, King's, Magdalene, Pembroke, and Peterhouse. At the time of my visit (March 1919), 40 military officers attending a course of instruction were also in residence at Sidney Sussex, while prior to the end of December, and for at least two years previously, there had also been at least 2,000 military cadets in residence, the length of stay of each individual cadet having been about 6 months.

As regards the undergraduate population, it is to be noted that, during the course of the war, the numbers became greatly depleted; and it was not until after the signing of the Armistice, and, for the most part only, from the commencement of the Lent term in January 1919, that the numbers again became markedly augmented. For purposes of comparison, a table is given showing the number of undergraduates on the books of the different colleges for the Lent term in the years 1914 and 1919 respectively. From the figures supplied officially by the authorities of the respective colleges—incomplete, however, as regards naval officers—it will be seen that, notwithstanding the recent expansion, the undergraduate population of the university for the Lent term of the present year was about 1,500, as compared with more than double that number for the corresponding term of 1914 :—

NUMBER OF UNDERGRADUATES ON THE BOOKS OF THE FOLLOWING COLLEGES.

Lent Term.

	1914.		1919.	
	Living In.	Living Out.	Living In.	Living Out.
1. Christ's	93	53	73	54
2. Sidney Sussex	50	60	63*	45
3. Jesus	90	104	43	9
4. Magdalene	54	48	53†	4
5. St. John's	250	30	103	17
6. Trinity	278	287	294‡	119
7. Caius	133	150	83	59
8. Trinity Hall	64	54	28	8

* Including 40 officers
† Including 18 naval officers.
‡ Including 80 naval officers.

	1914.		1919.	
	Living In.	Living Out.	Living In.	Living Out.
9. Clare - - - - -	45	149	45	23
10. King's - - - - -	110	47	171*	13
11. St. Catharine's - - - -	36	76	27	16
12. Corpus Christi - - - -	65	6	28	—
13. Selwyn - - - - -	100	1	31	1
14. Queen's - - - - -	99	80	63	14
15. Pembroke - - - - -	103	165	90†	31
16. Peterhouse - - - -	54	28	62‡	5
17. Non-Collegiate - - - -	8	100	9	90
18. Emmanuel - - - -	100	130	80	52
19. Downing - - - - -	33	59	22	38
Totals - - - -	1,765	1,647	1,368	598

* Including 23 naval officers and 90 nurses.
† Including 38 naval officers.
‡ Including 30 naval officers.

So that although, as stated above, the population has increased with unexpected rapidity since the cessation of hostilities, and although an unusual proportion of the total are shown to be "living in," the reception, in addition, of about 400 naval officers cannot be said to have given rise to over-crowding in the colleges.

My first step on arrival at Cambridge was to call on the Vice-Chancellor (Dr. A. E. Shipley, F.R.S.) in order to obtain his official co-operation in the investigation. At his request I subsequently wrote him a letter, in which was set out, as briefly as possible, the lines on which it was proposed to carry out the investigation. Printed copies of this letter, together with a covering minute from the Vice-Chancellor, in which he expressed the hope that Tutors of colleges would impress on all students the importance of replying to enquiries as fully, accurately, and promptly as possible, were subsequently distributed, together with a printed form of questions, to every resident member of the University. The collection and distribution of the schedule was most kindly undertaken by the Tutors of the various colleges, on each of whom I made a personal call in order to explain fully the nature of the proposed enquiry, and to ensure, so far as possible, smooth working of the arrangements. But, owing to the fact that the term was nearly at an end, a certain amount of difficulty was experienced in some instances, especially at Trinity College, in the distribution and collection of the printed forms. Eventually, however, a total of 1,766 of these forms duly filled in, were returned, replies to the questions on which have since been collated and summarised for the purpose of this report.

Through the good offices of the Vice-Chancellor and of Professor Buckland, a room in the Law Schools was placed at my disposal for office purposes; and two part-time clerks were engaged to deal with the distribution and collection of the printed forms, and to assist generally in the statistical portion of the enquiry.

Dr. Laird, the Medical Officer of Health for the Borough of Cambridge, readily afforded me all possible assistance, providing me with a copy of his report on the autumn outbreak of influenza at Cambridge, and undertaking to arrange with his Health Visitors, for a detailed house-to-house enquiry in certain selected areas of the town which was commenced shortly after my arrival in Cambridge.

I also conferred with Captain Fullerton, R.N., D.S.O.; in command of the Naval Contingent, and Surgeon-Commander Shewell, Senior Medical Officer, who had previously been notified by the Medical Department of the Navy of my proposed investigation, and had been directed to afford me all possible information with regard to cases of influenza among the naval officers and men at Cambridge. After discussion of the general scope of the investigation, Commander Fullerton undertook to arrange for the distribution of the printed form of enquiry to all the officers under his command, and also for their collection and return to me at the Law Schools when completed. And later on, at my request, he also issued a circular letter to all officers who, either at the time, or pveviously, had been under treatment for influenza at the First Eastern General Hospital or in their rooms, requiring information as to whether immediately before coming up to Cambridge, they had been "on leave," and if so, where, and for how long, or whether they had come direct from their ship or depôt. The reason for the issue of this circular was that a rumour had obtained currency to the effect that the original infection had been conveyed by one or more officers coming direct to Cambridge from a ship or depôt in which the disease was prevalent at the time. It may at once be stated, however, that in no single instance did the replies received afford evidence that this had been the case.

Incidence on the Naval Contingent at Cambridge.

As the outcome of an arrangement between the Admiralty and the university authorities 400 junior naval officers, accompanied by a few senior officers in charge of the contingent, went up to Cambridge for a special course of study on 31st January 1919, travelling up from London by a special train on the afternoon of that day. On arrival at Cambridge they were met and distributed in batches to various colleges. Within a few days of their arrival cases of influenza broke out among the contingent, which, as they occurred, were transferred to the First Eastern General Hospital, the first admission being on Saturday, 1st February; the numbers subsequently admitted being as follows:—

3rd	February	12	cases admitted.
4th	,,	19	,, ,,
5th	,,	11	,, ,,
6th	,,	15	,, ,,
7th	,,	3	,, ,,
8th	,,	2	,, ,,
9th	,,	—	
10th	,,	1	case admitted.

while two cases were re-admitted on the 15th and 16th February respectively. Although a few comparatively mild cases occurred during the following week or so, the outbreak, which was short and sharp, had practically come to an end by 12th February. At the time of the outbreak among the naval contingent, there was practically no influenza either among the undergraduates or in the town, so that it appears probable that the infection must have been contracted by one or more of the members of the contingent before their arrival in Cambridge.

The statement got abroad, at the time, that they had travelled down from town in a corridor train, and that so the infection from one source or another might have spread to others in the course of the journey prior to their distribution among the different colleges. This, however, on inquiry was found not to be the case. As previously stated, a further suggestion also gained currency that certain of these officers had come direct from ships on which influenza was present at the time. As the outcome of examination of returns called for from all these officers it was found that in every instance the officers had had a period of leave, the greater number of whom spent a part of it in London prior to the journey to Cambridge.

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ON HIS MAJESTY'S SERVICE.

INFLUENZA INVESTIGATION.

It is requested that this form may be filled in as accurately and concisely as possible.

College .....

Name ..... Age .....

Were you in Residence :
- (*a*) during Long Vacation, 1918
- (*b*) during October Term, 1918
- (*c*) during Lent Term, 1919

Have you suffered from Influenza :
- (*a*) Prior to the year 1918
  - If so, on how many occasions.....
  - State whether attacks mild or severe .....
  - Were you attended by a doctor? .....
- (*b*) During the period July to September, 1918 .....
  - If so, give date of attack.....
  - State whether attack mild or severe .....
  - Were you attended by a doctor? .....
- (*c*) During the period October to December, 1918 .....
  - If so, give date attack .....
  - State whether attack mild or severe .. ..
  - Were you attended by a doctor? .....
- (*d*) During January—March of present year .....
  - If so, give date of attack .....
  - State whether attack mild or severe .....
  - Were you attended by a doctor? .....

In connection with attacks of Influenza, have you suffered from any complications such as :
- Pneumonia,
- Bronchitis,
- Any other complication? .....

If so, give date of attack thus complicated:—

If attacked by Influenza, were you treated—
- (*a*) in Hospital (give date) .....
- (*b*) in College rooms do. ....
- (*c*) in Private house do. .....
- (*d*) in University Lodgings .....

If attacked by Influenza within 3 or 4 days after coming into residence in 1918 or 1919, state address for previous week .....

If attacked by Influenza within past 12 months :—
- Give date of exposure to infection, if known.....
- Give date of attack .....

Have you reason to believe that anyone has caught the infection from you, if so—
- Give date of exposure to infection.....
- Give date of attack .....

Do you suffer from common colds, if so—
- How often do you get one? .....
- How often have you had one during the last 9 months?.....
- When did you last have one?.....

This form will be called for within the next few days.

O 10699—1 &c

Further inquiry, however, elucidated the fact that one of the officers, Lieutenant O——, Trinity Hall, had been more or less ill for three days beforehand, and, according to his own statement, "felt rotten the whole journey up." It further appears, however, that he lost the special train and came down by a later one by himself. All the same, it is quite probable that within a few hours of his arrival he had come into contact with a number of his brother officers and so quite conceivably may have originated the outbreak. It has been further suggested with great probability that these naval officers were specially susceptible to the disease owing to their not having been attacked by the epidemic in the previous autumn and summer. The weather also had been specially cold about the date of their arrival, which, in conjunction with the shortage of fuel, and the insufficient number of blankets issued to them, may have contributed to make them more than ordinarily susceptible to infection.

Of naval officers and residents, I found that 60 had already been admitted to the hospital at the time of my visit to Cambridge. In none of these cases, however, were the symptoms of a specially serious nature in the first instance, and, among those attacked by influenza only, there were no fatalities.

Unfortunately, however, among those admitted to the First Eastern General Hospital with this disease nine subsequently developed cerebro-spinal fever, of whom seven died, the disease having been contracted by them in a very virulent form Dr. Laird, the medical officer of health for the borough, of whom I inquired as to the possibility of infection from previous cases of this disease, informed me that there had been no case of cerebro-spinal fever among the civilian population of Cambridge for some months past, and at first it seemed almost impossible to trace the origin of the infection.

As the outcome of conference at the First Eastern General Hospital with the Commandant, Colonel Griffiths, and Captain Shaw, R.A.M.C., it appears not improbable that this outbreak of cerebro-spinal fever originated from another officer, Paymaster-Commander W——, R.N., who had been in Cambridge on special service during the month of January 1919, during which he was attacked by an illness, the precise nature of which could not be diagnosed at the time. As the result of further inquiries it appears that he arrived in Cambridge on the 18th January 1919, having previously been staying at three different addresses in London between 23rd December 1918 and 18th January 1919. At the time of the commencement of his illness he was staying in rooms at Market Street, Cambridge, from which he was admitted to the First Eastern General Hospital on 30th January 1919, as probably suffering from malaria. No malaria parasites, however, were found as the result of an examination of his blood. A rash similar to that of scarlet fever developed, in consequence of which Dr. Laird was asked to see the patient in consultation, when, in view of the general drowsiness of the patient and of the indefinite mental symptoms, he expressed the opinion that the patient might be suffering from encephalitis lethargica. There was also pneumonia of both lungs, and "slight Kernig" was noted. The case ended fatally, but no post-mortem was obtainable. The certificate of death gave pneumonia as the cause, but in the light of subsequent events it is probable that this case must be considered as one of cerebro-spinal fever. Captain Shaw laid special stress on the conjunction of high temperature (105°) with an extremely low pulse (60 per minute) as evidence of the true nature of the disease.

Mrs. W—— was also communicated with, from whom we learnt that, so far as she was aware, her husband did not come into contact with any case of infectious disease prior to leaving London, but he had complained of feeling chilled as the result of a motor car journey from Cambridge to London and back, on duty, on Saturday, 25th, and Sunday, 26th January. The first of the naval officers was not admitted to hospital until a week subsequent to the death of Commander W——, although all these cases were in the first instance admitted to the same ward (Ward 14) as that in

which he had been. It would now appear that the missing link has been found in the person of a cadet, S——, who at the time Commander W—— was brought in was sufficiently well to help in attending on him and other of the more helpless patients in the ward. On a swab being taken from his throat it was found to give a positive result as regards the specific organism of cerebro-spinal fever, although he did not at that time nor has he since developed the disease.

That the naval contingent as a whole had not been exposed to contact with the disease outside the hospital is proved by the fact that, shortly after the appearance of the first cases, 100 of the naval officers in Cambridge were selected at random from the various colleges, and were found on examination of their throats to be entirely free from the specific organism; the only carriers discovered having been contacts of the actual cases. It is interesting that one of these carriers, Lieutenant A——, subsequently developed the disease.

At a later date, about the middle of March, two further naval officers, suffering from cerebro-spinal disease, were admitted to the First Eastern General Hospital; in whose cases it would appear, however, that the infection had been contracted during a period of leave in London.

As regards influenza among the naval contingent, a second slight outbreak occurred towards the end of March which apparently originated among the officers in residence at Emmanuel College.

Age Incidence.

The naval officers brought with them about 100 men of the Royal Marine Light Infantry as batmen. These, although somewhat crowded together in billets and living in altogether less satisfactory hygienic surroundings than the officers, escaped infection of influenza almost entirely, only two of them having been known to have been affected.

The following figures shown in tabular form are interesting:—

———	Naval Officers.	R.M.L.I.
Average age - - - - - -	20·5 years	30 years.
Percentage of influenza - - - -	15·8 per cent.	2 per cent.

Incidence on Cadet Battalions at Cambridge.

For a period of about two years prior to December 1918 there had been, at any one time, at least 2,000 military cadets in residence at Cambridge for the purpose of military instructions, each batch spending about six months in training there. Among these cadets there was a considerable outbreak of influenza, starting in the first week in June 1918, which reached its highest intensity, as judged by the total admissions to the First Eastern General Hospital, on the 17th of that month.

The accompanying chart (No. 1) shows in graphic form the daily admissions both of these cadets and also of other units of the Army, including the Royal Air Force, both for the June and July outbreak, and also for a subsequent one, somewhat less in extent as regards the number of cases, in October and November 1918. Of the June outbreak the first five cases occurred in Downing College. The table of daily admissions to the First Eastern General Hospital shows the interesting manner in which infection subsequently broke out, after varying periods of time, in the remaining colleges.

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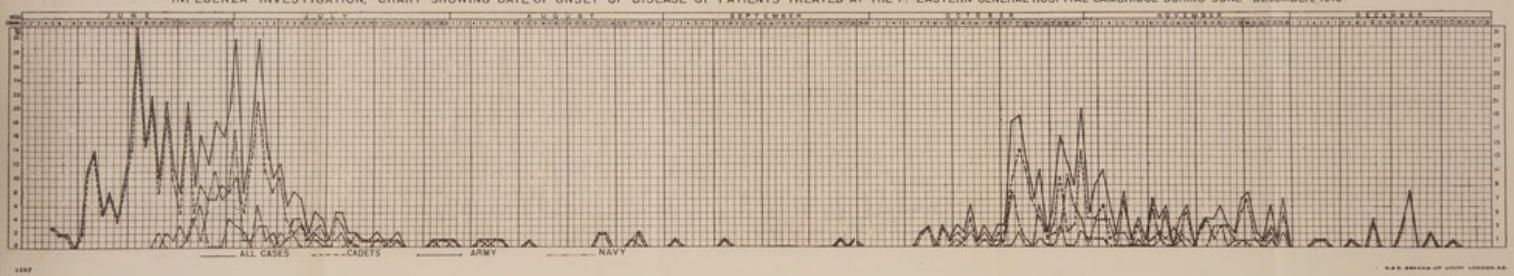
Chart. I.

INFLUENZA INVESTIGATION; CHART SHOWING DATE OF ONSET OF DISEASE OF PATIENTS TREATED AT THE 1ST EASTERN GENERAL HOSPITAL CAMBRIDGE DURING JUNE–DECEMBER, 1918.

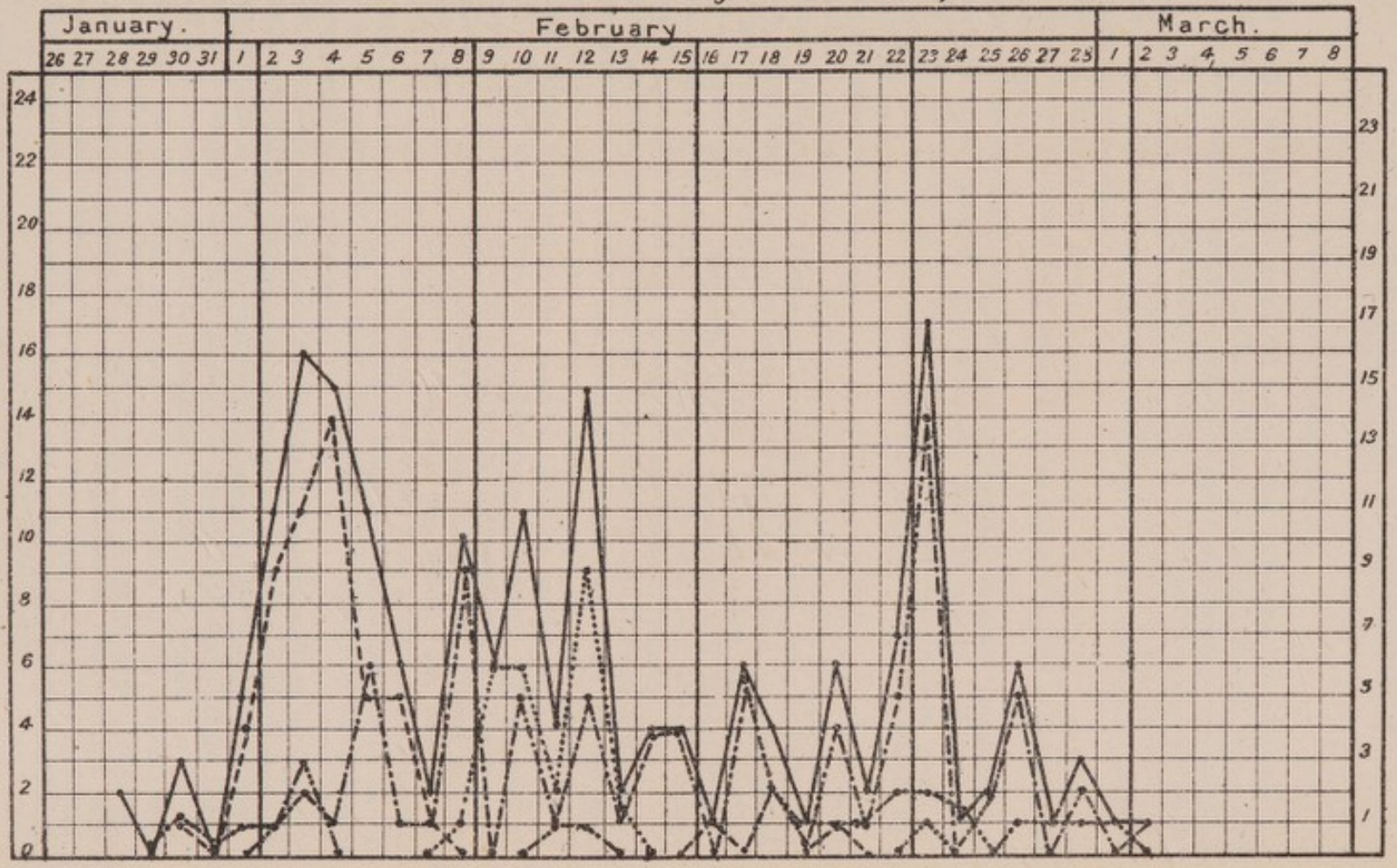


INFLUENZA CASES – 1st EASTERN GENERAL HOSPITAL. 1919.

Chart II.

185 Cases.

(Cadets no longer in Residence).



——— *All Forces & P of War.*

– – – *Naval.*

—·— *Military.*

—·—·— *R. A. F.*

……. *P. of War.*

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As a rule the attacks among these cadets were not serious in nature. Complications such as pneumonia were infrequent, but, unfortunately, there were a few fatalities, an adjutant, an N.C.O., and a cadet, all having died at the First Eastern General Hospital within two days of one another.

Influenza as affecting Resident Members of the University.

Analysis of Schedules, &c.

The only information available as to the incidence of influenza on resident members of the University has been that obtained from the replies sent in on the printed schedules, which, as previously stated, were supplied by me to the tutors of the different colleges. The printed circular which was sent with each form requested tutors to impress on all residents the importance of replying to the inquiries contained therein "as fully, accurately, and promptly as possible."

The total number of forms issued, as shown on Table A., was 2,595, of which 1,766 were returned duly entered up as regards replies to the printed questions thereon.

As was to be expected, the information supplied in some instances was considerably more detailed and complete than in others. But it was obvious that the majority of those making returns had been at pains to comply with the terms of the vice-chancellor's printed minute which was sent out with each form.

The difference between the number of forms sent out and the number returned is accounted for, in large part, by the fact that in the parcels sent out to each college more forms were included than was likely to suffice for one to be distributed to every resident, in order to provide a margin against inadvertent loss, destruction, or spoiling of forms. But, in the case of certain colleges, more particularly Trinity, Emmanuel, and Sidney Sussex, the proportion of schedules returned to those sent out, proved, unfortunately, to be much below the average for the whole University.

These schedules, after subsequent collection by the College Authorities, were returned to me at the Law Schools, where they were, in the first instance, sorted out into two classes, according as the individual whose name and college appeared at the head of the form, stated that he had, or had not, suffered at one time or another from influenza. Of the total of 1,766 replies received, 1,263 were positive, and 503 negative in this respect. Full details as regards the members of each college; and giving, in each instance, the age of the person making the return, are set out in Tables B. (1), (2), and (3). As none of the cases were admitted to hospital, either at Addenbrooke's or at the First Eastern General, no details as to symptoms or treatment, other than those furnished briefly by the patients themselves, have been forthcoming.

Table C. shows for the whole University, college by college, as also for the Naval and Military Contingents, the number of persons who have had influenza (*a*) prior to 1918, and (*b*) during the periods July to September 1918, October to December 1918, and January to March 1919, each attack being included under one or other of two headings, "severe" or "mild."

Reference to this table indicates that incidence of the disease was mainly on individuals aged 18 to 22 years. It must be noted, however, that the greater proportion of the under-graduates come within this particular age-group, although more older men have gone into residence since the cessation of hostilities than was usual prior to the outbreak of war.

The ages of all residents (graduates and under-graduates) attacked by influenza prior to 1918; and during the three outbreaks of July to September 1918, October to December 1918, and January to March 1919, are set out in detail in Table D., which also affords information in each instance as to whether the attack was severe or mild. Table D. (1) to

D. (21) give similar details for each individual college, and also for the Naval and Military Contingents.

On the whole it is obvious that the form in which the disease attacked residents of the University has been somewhat exceptionally mild, even in the October to November outbreak, as compared with the type that came under observation in other places, or among the town population of Cambridge itself. This may be due in part, at any rate, to the fact that the circumstances of the life of the average undergraduate are such as to favour escape from incidence of infectious disease, especially influenza.

Of a total number of 1,055 replies reporting attack of influenza at one or another period during the 12 months March 1918 to March 1919, 113 state that their attack was followed by various complications (*see* Table E.), of which pneumonia and bronchitis, singly or together (58 cases in all), account for by far the larger proportion.

Table F. shows the ages of persons thus attacked, and the complications from which they suffered.

As judged by examination of the Cambridge Registrar's returns, *fatalities* among the members of the University consequent on attack by influenza have been exceptionally few in number --four only among undergraduates, and three among resident graduates during the period November 1918 to March 1919. Of these, one graduate, and three undergraduates, aged 39, 32, 23, and 25 years, died in November 1918, and two graduates and one undergraduate, aged 29, 34, and 18 years respectively, in February 1919.

As regards specific *treatment* of the disease no instances have come to my knowledge in which the inoculation of special vaccines (influenza bacillus, pneumococcus, streptococcus, &c.) have been employed.

In considering the replies to the question on the printed form, a copy of which is appended, it should be borne in mind that the larger number of those who filled in the form had not been resident in Cambridge prior to the Lent term (January to March 1919), so that it is difficult to establish any satisfactory comparison between the attacks of influenza prior to and subsequent to January of the present year. Of the 1,055 individuals attacked during the period July 1918 to March 1919, 284 state that their attack was of a severe nature; whilst the remaining 771 state that in their case it had been mild. Among the total of 691 individuals attacked *prior* to 1918 the proportion of severe and mild cases was 145, and 546, respectively.

Period of Incubation.

Analysis of the replies on the forms returned from the University residents has afforded a considerable amount of information as regards the varying periods of incubation of the disease. Thus on referring to Chart 2, on which is set out in graphic form positive replies as received from the various colleges, the naval contingent, and certain military officers, it will be seen that, according to the statements received, the incubation period may vary from some time on the same day as that on which the patient was exposed to infection, to as much as 12 days or more, afterwards. Judging from the record of the replies received, the incubation period has ranged, in far the greater number of cases, at from one to four days, the second day subsequent to exposure being apparently that on which the greatest number of attacks occurred.

Of the 144 positive replies received, the incubation periods given are as follows:—

First day - - - - -	25
Second day - - - - -	33
Third day - - - - -	14
Fourth day - - - - -	19

On the other hand there are recorded as many as 14 statements as to the incubation period having extended to 12 days and even more.

At Addenbrooke's Hospital I was informed by the Resident Medical Officer of a case of leukœmia in which an attack of influenza supervened

almost exactly 48 hours after the admission to the ward of two cases of that disease.

As regards the borough of Cambridge, Dr. Laird reports that the incubation period averaged from two to three days, the exact period in one case where it was accurately known being 64 hours.

As the outcome of enquiry at the Friends' School, Saffron Walden, definite information, set out more fully in a subsequent section of this report, bearing on the incubation period was also obtained. In one of the wards of the school sanitorium, to which cases of influenza were removed, were two boys recovering from pneumonia, C—— and N——. The first cases of influenza were admitted to this ward on the afternoon of Saturday, 1st March, and both these boys subsequently developed influenza; N—— on the morning of 3rd March, and C—— on the afternoon of the same day, incubation periods of 36 and 48 hours, supposing that they become infected immediately on the admission of their comrades to the ward.

While at Saffron Walden I learnt of a series of cases at Newport, Essex, in which the date and source of infection could be fairly accurately determined This information, supplied to me by the local practitioner, Dr. Arthur Browne, is best given in the form in which he sent it to me.

"M" Family—Newport.

Wednesday, 12th February 1919.—Private M—— arrived home in the afternoon (demobilised from Germany) very ill, seen same evening by Dr. Arthur Browne. Temp. 104·2 F.

Thursday, 13th February.—His father, mother, sister, and a little niece all taken ill suddenly with the same complaint and all went to bed early.

Friday, 14th February.—The five patients seen by Dr. Browne, all in bed, except the mother who kept about until 15th February (Saturday).

Saturday, 15th February.—Pneumonia well developed in Private M——. Mrs. M—— was very deaf and Dr. Browne had to come into close contact with her face to make her hear. (She was extraordinarily deaf owing to the illness.)

Sunday, 15th February.—Mrs. M—— in bed. Dr. Browne auscultated the five patients, and was necessarily very close to all, especially in speaking to Mrs. M——. Same day he himself was seized with the complaint and went to bed about 8.30 p.m.

All the M—— family were in good health when Private M—— arrived home. Dr. Browne was suffering from an ordinary bronchial catarrh when he took ill.

Private M—— died on 23rd February, aged 27.
Mrs. M—— died on 22nd February, aged 54.
Mrs. C—— (the sister) on 25th February, aged 35.

The father, an exceedingly robust man, though very ill, recovered; as did also the little niece. Another daughter and Private M——'s wife, who nursed the invalids throughout, did *not* catch the complaint.

The deaths were all due to pneumonia, Private M—— also got acute parotitis (unilateral). The type of the disease was evidently very severe.

Dr. Browne's temperature on the second day was 104° F; on the fourth day it was normal. In spite of every care, change to sea-air, &c., he was very weak, and depressed, and shaky, as late as 8th March, and had not fully recovered until about 1st April.

From Dr. Atkinson of Saffron Walden the following statement, dealing with the question of incubation was received:—

> Writing on 10th May 1919, he says: "I was called yesterday, "Friday, 9th May, to a man suffering from influenza, who was a clerk "in an office at Chesterfield, Essex. His employer's sons stated that "they had been working in the office with him on the previous after- "noon, 8th May. On Saturday morning, 10th May, I was sent for to

" one of the sons when I found him to be suffering from the " disease."

Spread of the Disease.

This, as far as the evidence is available, would appear to be by personal contact only. Thus, reference to Table M, showing the approximate dates on which military cadets distributed among the various colleges at Cambridge were admitted to the First Eastern General Hospital suffering from attacks of influenza, indicates—on the supposition, that in each instance the period between attack by the disease and admission to hospital was approximately equal—that while the cadets at Downing College were the first to be affected, the infection subsequently spread from college to college.

In the case of the naval officers, who took the place of the military cadets at the beginning of January 1919, it appears probable that infection originated, as previously stated, with one of these officers who journeyed to Cambridge by himself, being apparently ill at the time. Although, like the cadets, distributed over the various colleges, the naval officers had ample opportunity of meeting one another, not only in their rooms but at lectures, with the result than an explosive outbreak of influenza occurred among them within a few days of their advent at the University, of so serious a character, that within about a week as many as 90 had been admitted to the First Eastern General Hospital. In this connection it may be noted that, at the time, there were practically no cases of influenza among either the university or town population.

(Borough of Cambridge.)

Reference to Chart III. and to the appended map of the borough of Cambridge on which the various sub-divisions of the town shown on the table are marked with corresponding figures, indicates as regards *deaths* (information as to case-incidence not being available), that these districts were not affected simultaneously, but that, judging from the notification of deaths received by the medical officer of health, New Town (No. 1) was the first district to be affected, a death occurring there on 1st October 1918. The next death will be seen to have been of Romsey Town (No. 2) on 13th October, followed by one in Sturton Town (No. 3) on 17th October, and one in Centre of the town (No. 4) on the following day. Three days later, on 21st October, a death occurred in the Newmarket Road (No. 5) district. Then followed deaths in each of three districts, Castle End (No. 6), New Chesterton (No. 7), and Newnham (No. 8), all on 25th October. The first death from New Cherryhinton (No. 9) occurred on 28th October, while it was not until 17th November that a death occurred in the remaining district, Old Chesterton (No. 10).

Doubtless the spread of the disease has also been influenced to some extent by other circumstances, such as over-crowding, bad ventilation, and specially close contact of persons, in schools and factories. In the case of the Friends' School, Saffron Walden, these accessory factors are likely to have operated in intensifying the effect of personal contact, the dormitories being filled with so many beds as to afford inadequate cubic space, while the arrangements for ventilation were far from satisfactory. As regards the carriage of infection in factories, Dr. Laird, Medical Officer of Health for Cambridge, informed me that, as the result of his inquiry in districts where a considerable number of young women were thus employed, he had come to the conclusion that an excessive incidence of the disease on them, as compared with the other inmates of the houses in which they were living, could only be attributed to the fact of the close contact with which they were brought with their fellow workers in the factory in which they were employed.

In considering the question of the spread of influenza, the possibility of " carriers " of the disease playing some part in the matter naturally arises, but thus far no evidence as to such a cause of infection has been capable of

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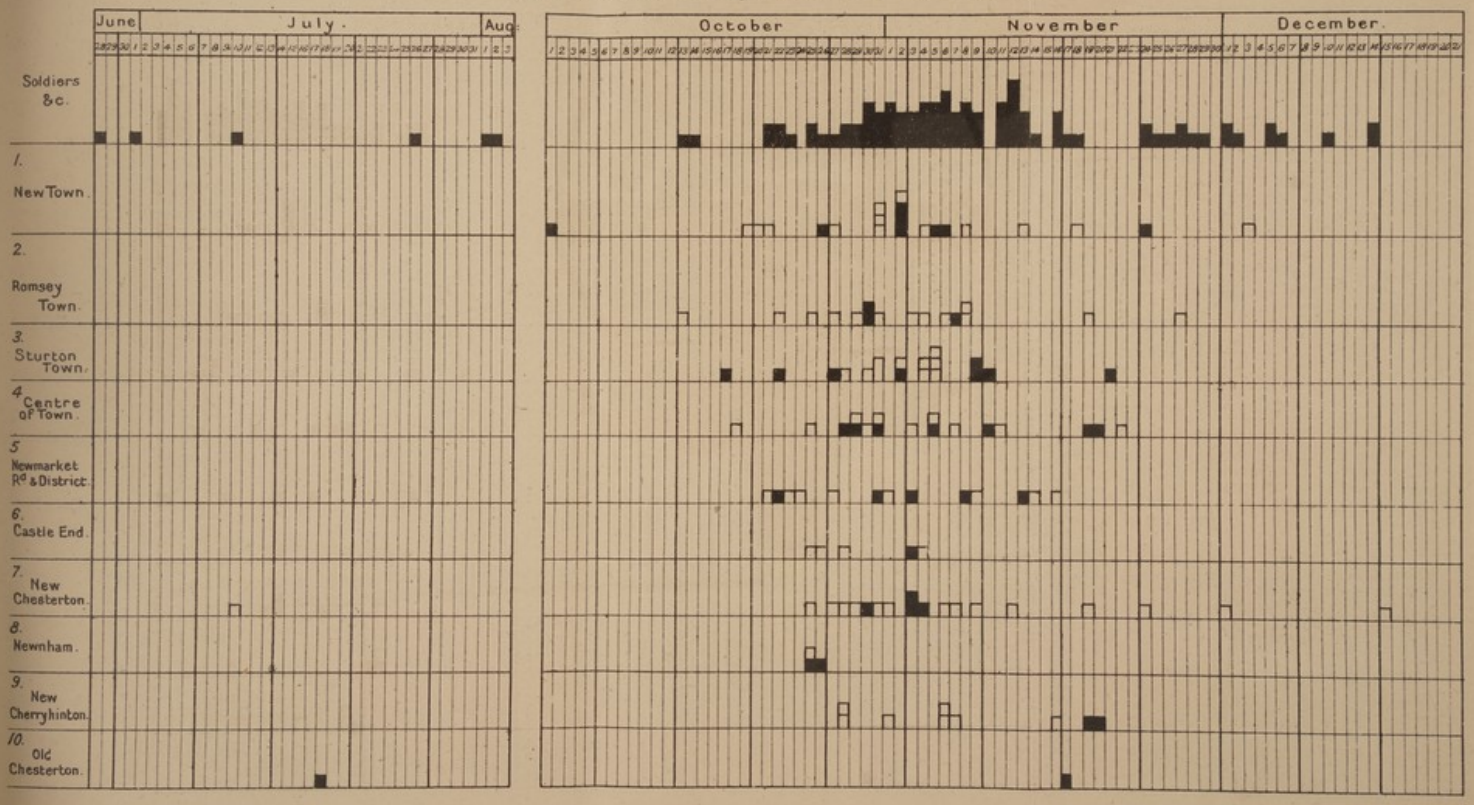
Chart III.

CAMBRIDGE INFLUENZA OUTBREAK.

June–December. 1918.

Chart showing daily number of Deaths at 1st Eastern General Hospital and in the Borough according to Districts shown on Plan



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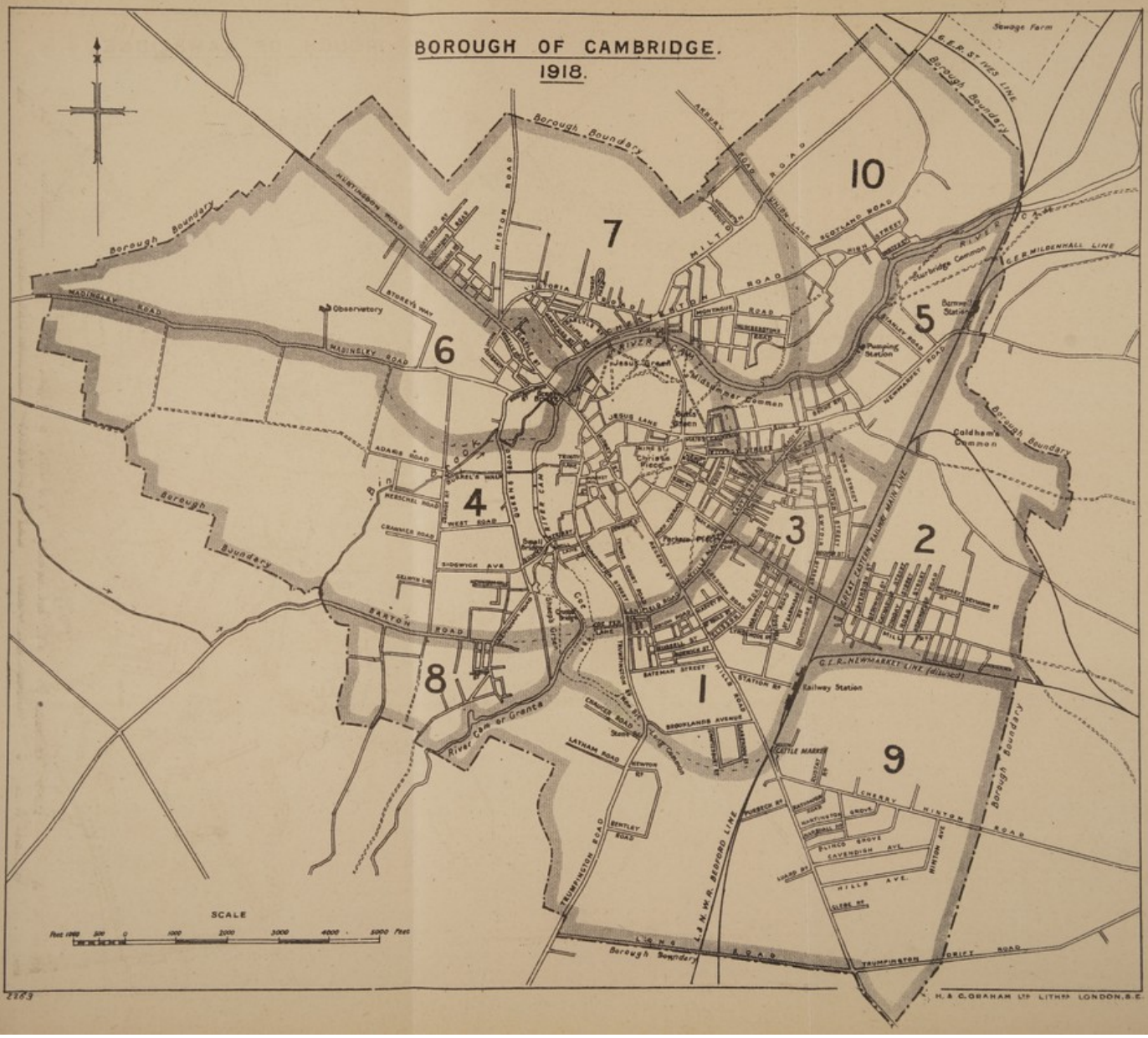
BOROUGH OF CAMBRIDGE.
1918.
Borough Boundary
Sewage Farm
G.E.R. St. IVES LINE
ARBURY ROAD
10
7
MILTON ROAD
HUNTINGDON ROAD
HISTON ROAD
SCOTLAND ROAD
RIVER
G.E.R. MILDENHALL LINE
Observatory
6
5
MADINGLEY ROAD
Pumping Station
Burrell's Walk
Borough Boundary
Coldham's Common
ADAMS ROAD
4
3
2
BARTON ROAD
G.E.R.-NEWMARKET LINE (diverted)
Railway Station
8
1
River Cam or Granta
9
LATHAM ROAD
BENTLEY ROAD
TRUMPINGTON ROAD
L.N.W.R. BEDFORD LINE
SCALE
Feet 1918
0
1000
2000
3000
4000
5000 Feet
Borough Boundary
ROAD
TRUMPINGTON
2283
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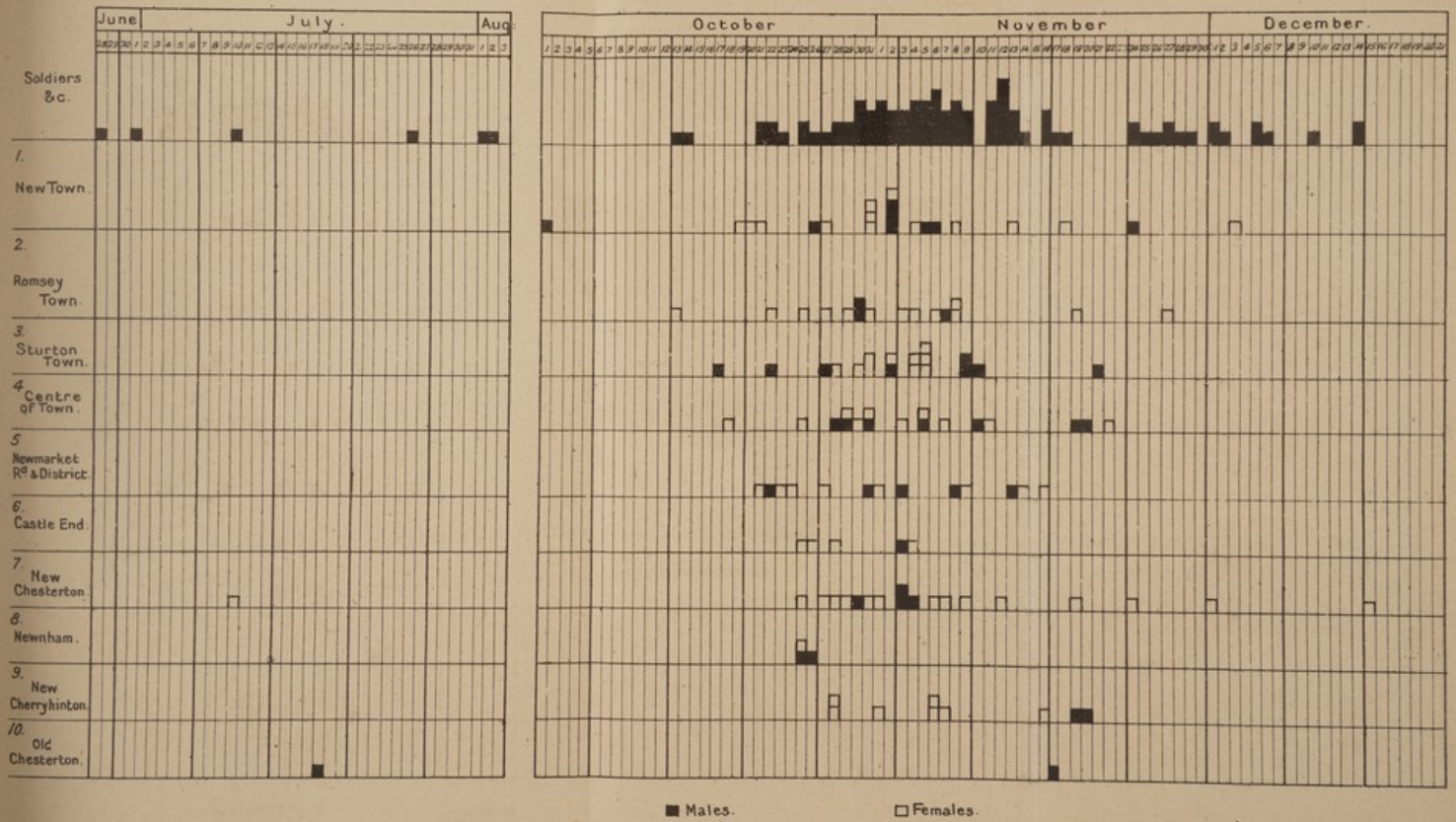
Chart III.

CAMBRIDGE INFLUENZA OUTBREAK.

June–December. 1918.

Chart showing daily number of Deaths at 1st Eastern General Hospital and in the Borough according to Districts shown on Plan



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demonstration. But although a number of instances have been recorded in which Pfeiffer's organism has been found in the sputum during the first stages of the disease, I have been unable to learn of any instance in which similar search for the presence of this organism has been carried out during or after convalescence. On the other hand, as indicated in the case of certain nurses at Addenbrooke's Hospital removed to general wards after a period of two days of normal temperature, it does not appear that this organism is capable of persisting, at any rate in virulent form, beyond the acute stage of the disorder.

Severity of Attack.

Of the three outbreaks of influenza under consideration, which occurred in June–July and October–November 1918, and in March of the present year, that occurring in October and November was for the most part considerably more severe, as indicated, for the Borough of Cambridge, by the higher temperatures recorded; the greater extent and intensity of complications observed; and the number of deaths reported. It is not possible, however, to make any similar comparison as regards the university population owing to the fact that previously to the Lent term, 1919, the number of university residents was extremely small.

In those instances where the individual has suffered from more than one attack of influenza during the 12 months April 1918 to March 1919, it would appear that, as a general rule, the attack, especially when occurring within three or four months of a previous one, has been comparatively mild, and more particularly so when the first attack was severe.

As regards the special areas in the borough investigated at my suggestion, some difference was obvious between the periods during which the greatest proportion of severe cases among males and females respectively was observed. The greater number of severe cases among males has been recorded in the period October to December 1918 and for females in January to March 1919. On the other hand, the greater proportion of mild cases among males occurred during the period July to September 1918 and in females, October to December 1918.

Complications.

In Table E. is given a list of the various complications which, as the result of the special enquiry among the university residents, are stated to have been experienced (following attacks of influenza) by the individual replying to the questions on the printed form. Special reference to these is unnecessary here, as they are set out in full, under 42 different headings, in the table referred to. Of these, as has also been the case elsewhere, in by far the greater number of cases, the complication present was either pneumonia or bronchitis, or both of these combined.

Other complications recorded in the death returns of the First Eastern General Hospital include septic pneumonia, acute spinal myelitis and paralysis of inter-costals, empyema, malignant endocarditis, and cerebro-spinal fever.

Immunity.

As an outcome of the present investigation it is obvious that one attack of influenza usually confers a certain amount of immunity against further attack by the disease although the duration of this immunity may be somewhat brief in many instances. The average duration would appear to extend from three to nine months. Dr. Laird, the Medical Officer of Health for Cambridge, informed me that he had heard of but one case of a second attack at Cambridge during the year 1918, the patient in this instance being a medical practitioner.

Analysis of the return received from the university residents, however, indicates, as shown in Table J, that certain individuals were attacked not only on a second occasion, but even for a third or a fourth time during the period 1918–19 covered by the investigation. As many as 98 instances of a second attack during this period will be found to have been recorded.

Among the boarders at King's College Choir School at Cambridge (24 in number), all of whom, as previously reported, were attacked by influenza in mild form during the outbreak of June–July 1918, not a single case occurred during the outbreak of October–November 1918, at the time when the disease was present in severe form in the borough, notwithstanding the fact that a few cases occurred among the day boys attending the school but living in the town. In the comparatively mild outbreak of March 1919 about half the boys, who had entirely escaped during October–November 1918 again suffered attack.

At Saffron Walden during the course of the outbreak in October 1918, of 30 boarders in the Grammar School there, 19 were attacked by the disease in severe form, temperatures up to 104° F. having been reported in several instances, although no case of pneumonia or other complication supervened. None of these boys had been attacked in June or July 1918, and none of them have suffered attack during the present year (1919).

Certain individuals appear to possess natural immunity against the disease as instanced by the fact that, of the 1,766 individuals who returned replies to the printed form of questions issued to the residents of the university, 503 reported that they had never suffered attack by the disease. Certain instances of this apparently natural immunity have been met with elsewhere in the course of the present investigation. As an example may be instanced the case of the small but virulent outbreak at Newport, reported to me by Dr. Browne and previously referred to. Neither the wife of the soldier by whom the infection was introduced, nor two other females of the household, as to whom no previous history of influenza could be obtained, contracted the disease although they had nursed the remaining members of their family, of whom no less than three died, during the whole of their illness.

Occasionally, however, immunity, if existent, is of so slight duration that it can hardly be said to exist at all. An instance of such a condition was reported to me by the headmaster of the Friends' School at Saffron Walden, as having been observed at another of their schools at Wigden, Cumberland. At this school, following on a small outbreak towards the end of November, a much larger one occurred, commencing on 16th January 1919, in which about 74 children were affected. Yet another outbreak occurred commencing 25th February 1919, during which 12 girls and five boys who had been attacked in the outbreak in the previous January again fell ill with the disease for the second time, in addition to a few cases among children who had not been previously affected. The period elapsing between the first and second attack varied, according to the statement of the medical officer to the school, from four to five weeks.

Old Age.

As indicated in the tabular statements appended to this report, incidence of the disease has been mainly on young adults, while older persons have to a large extent escaped attack. This has especially been obvious as regards persons of advanced age, as an instance of which may be mentioned the outcome of an inquiry at the almshouses at Saffron Walden. The inmates of these houses number 44, all of whom, with the exception of a resident nurse and two other attendants, are over 60 years of age, 24 being aged between 70 and 80 years, while eight averaged 80 years and upwards. Dr. Atkinson, the Medical Officer of Health for Saffron Walden, informed me that such of these old people as are capable of doing so, visit freely in the town, where they mingle freely with other people, and all are accustomed to be visited by their friends and relations. Notwithstanding this, not one of the inmates have suffered attack from influenza during the whole period

under investigation—April 1918 to March 1919—while Dr. Atkinson further informed me that he had no recollection of a case of influenza among the inmates at any time within the past 11 years.

Pathological Investigations.

Dr. Laird, the Medical Officer of Health for Cambridge, in a report to the Public Health Committee of the borough, on the epidemic of influenza in Cambridge in 1918 (appended to this report) gives some account of the results of pathological examinations carried out by Dr. Alden Wright at Addenbrooke's Hospital, and of the microscopical examination of material supplied by Dr. Alden Wright to Colonel Sims Woodhead, Professor of Pathology in the University of Cambridge. A similar report has been available as regards the naval and military cases from the First Eastern General Hospital. Dr. Alden Wright states that the lower portions of the lungs examined were consolidated, but in patches. A naked-eye inspection of the cut surface showed numbers of minute white points, which were evidently bronchioles filled with secretion. In a few cases infarction of the lungs was found, and two had empyema.

Professor Sims Woodhead, in his portion of the report, states that the main features of all these lungs, as seen in microscopical sections, are those characteristic of intense inflammation set up by the toxins of micro-organisms. He refers to the statement by certain other observers that these appearances do not indicate pneumonia, but he maintains that the extremely rapid and acute inflammatory process which is present must be regarded as acute pneumonia of an especially virulent form. His reason for coming to this conclusion he then proceeds to give in detail. Professor Sims Woodhead considers that catarrhal pneumonia is somewhat uncommon for the reason that when pneumonia supervenes in influenza the patient is apt to die long before the catarrhal proliferation of the epithelium in the alveoli has progressed very far, and adds that the consolidation which occurs in patches is more extensive in this form than in catarrhal pneumonia.

Bacteriology.

Apparently no systematic bacteriological work on the disease was carried out, either at Addenbrooke's Hospital or at the First Eastern General Hospital, owing, as I was informed, to the shortage of staff and consequent pressure of work. In fact, the only reference that I have found to any such examination in the case of influenza occurred in the reports of the bacteriological laboratory at the military hospital, this referring to a single examination of sputum only, which was apparently negative as regards the presence of pathogenic micro-organisms.

Addenbrooke's Hospital, Cambridge.

As the outcome of conferences with the secretary and with the resident medical officer, I learnt that no cases of influenza had been admitted from the university, as the hospital did not, at present, take in paying patients; and, as a matter of fact, very few cases of influenza had been admitted—seven cases between 1st January and 21st March 1919—and these only as cases of emergency, which had to be admitted to a general ward. A single case was admitted on 15th October 1918.

There had been, however, an outbreak of influenza among the staff extending over the period 10th October 1918 to 21st January 1919, and affecting 57 individuals, as follows:—

Resident staff	3
Nursing staff	37
Domestic staff (including kitchen maids, ward maids, and laundry maids)	17
Total	57

The resident medical officer was of opinion that the infection was contracted outside the hospital, certainly in one, and probably in other instances, seeing that the housing and feeding accommodation of the residents, nurses, and domestic staff is entirely separate, although the patient admitted on 15th October 1918 may possibly have been responsible for some extension of the disease subsequently.

The accompanying chart indicates in graphic form the incidence of the disease, week by week, during the period under consideration.

Two points of special interest coming under observation in the hospital, and bearing respectively on the incubation period of the disease, and on the period of infectivity of the patients recovering from the disease may be mentioned.

As regards the first point, a case of spleno-leukœmia, which had been in the hospital for some weeks previously, contracted influenza within 48 hours of the admission to the same ward of two patients suffering from this disease.

Duration of Infectivity.

In reference to the period beyond which a patient may be regarded as no longer capable of conveying infection the evidence is as follows:— Members of the nursing staff attacked by the disease were "warded" in the so-called "open-air colony" on the roof of the hospital. Early in December, however, the pressure on the available space was so great that it was decided to remove some of the patients to the general small wards. Ten of the nurses were thus removed as soon as their temperature had fallen to normal, and had so remained for two days. The resident medical officer informed me that this course was decided on with some trepidation, but, fortunately, no secondary cases occurred in either of the wards to which these nurses were removed.

Influenza at King's College School, Cambridge.

Learning while at Cambridge of a number of cases of influenza which had occurred at this School, I took the opportunity of calling on the Head Master in the hope that he would be able to supply me with information which might be of service in connection with the general investigation of influenza in the University and town.

The school, which had been originally founded for the purpose of housing and instructing the boys in the choir of King's College, Cambridge, in return for the musical services rendered in the College Chapel, has since been extended so as to admit of an additional number of boarders and also a number of day-boys, mostly the sons of married members of the University.

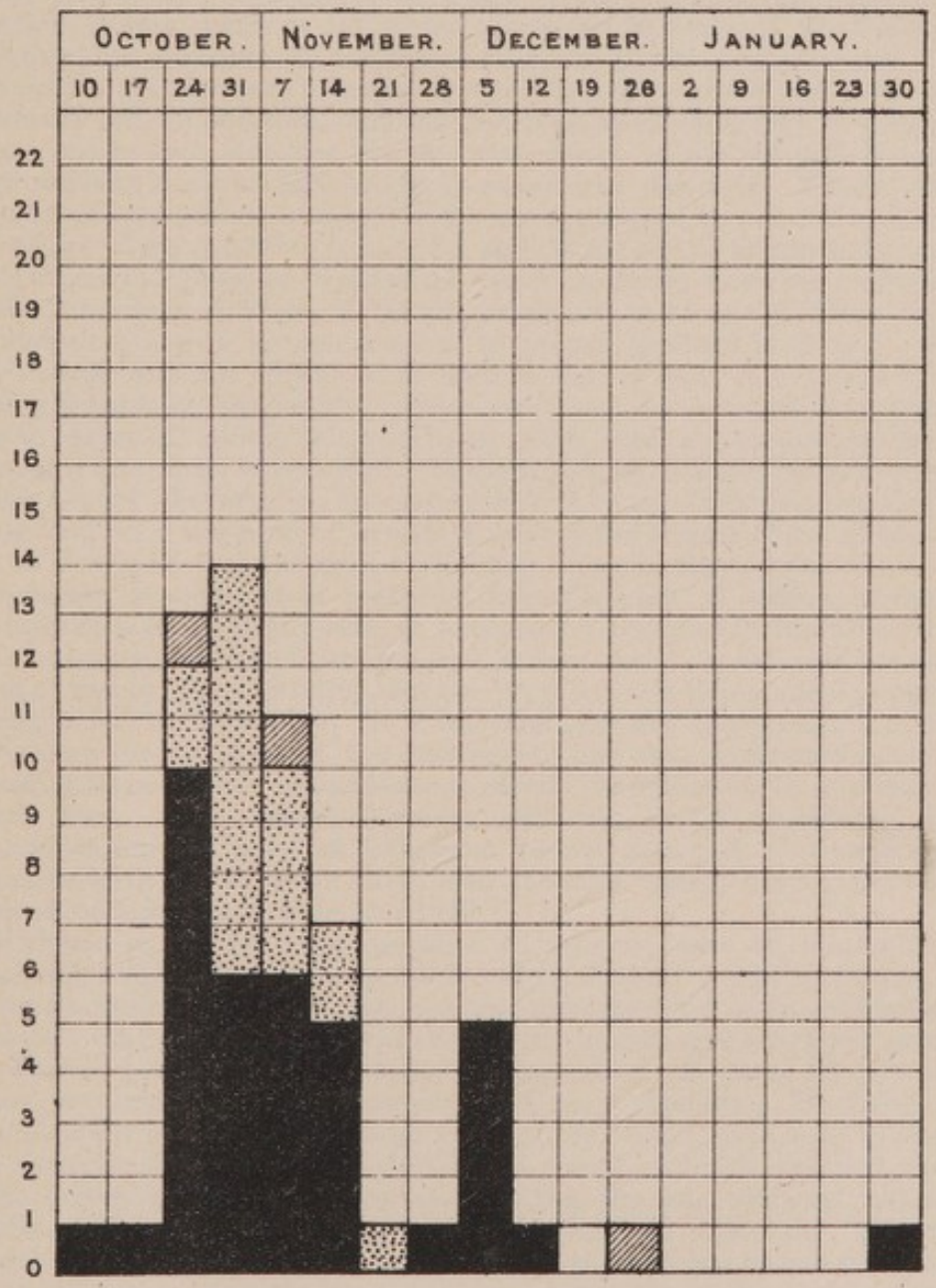
Mr. Jelf, the headmaster, told me that the present number of boarders was 24, of ages ranging from 10 to 14 years, and that it was among these, rather than among the day-boys, that the greater number of influenza cases had occurred. From a list which he gave me it would appear that everyone of the boarders was attacked by influenza in July 1918, but the cases were of comparatively so mild a character that only seven of the boys were attended by a medical man. There were no complications in connection with this attack, and the longest period that any one boy remained in bed was seven days. Of these 24 boys, 14 have been again attacked during March 1919, but with two possible exceptions their illness was of even a milder form than that of July 1918.

It is of special interest to note that not one of these boys was attacked in October or November 1918, at which time the incidence of the disease was specially great in the town; the fact that every one of the boarders had been attacked in July 1918 having apparently afforded immunity, which persisted at that period, and, judging from the smaller number attacked in March 1919, still prevailed to some extent.

To face p. 400.

Re Influenza.
Addenbrooke's Hospital.

Chart showing number of Cases of Influenza among the Hospital Staff from the Week commencing Oct: 10. 1918 to week commencing January 30. 1919.



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Owing to pressure of work at the end of term it was not possible to obtain definite information as regards the day-boys, 36 in number, but, as stated above, they had suffered attack in less degree than the boarders, this difference being not improbably accounted for by the fact that the boarders came into closer contact with one another at night time in the dormitories than in the case of the day-boys living at home with their parents.

Unfortunately, however, Mr. Jelf was unable to give any information as to the order in which the boys occupying the particular beds in the dormitory were attacked by influenza in either of the outbreaks of July 1918 or in March of the present year.

Want of Hospital Accommodation for Undergraduates of the University.

It would appear somewhat extraordinary that whereas every public school possesses a sanatorium to which cases of illness, and especially of infectious illness, occuring during term-time can be removed, the University of Cambridge possesses no accommodation for either of these cases. It is true that in the town are a couple of small hostels to which members of the University are, on occasion, removed for treatment, especially of a surgical nature, but in each instance the accommodation is limited and in certain other respects is not all that might be considered desirable. Consequently, when an undergraduate is taken ill during term-time he either has to leave the college, if his condition is not of a serious nature, or he has to be nursed in his rooms in college or in lodgings, where naturally there are no facilities for the purpose, and where, if his illness should be of an infectious nature, the undesirability of his non-removal to proper quarters is obvious. If he is in college and his illness is of such a nature that it requires skilled nursing it probably means that some other man on the same staircase will have to vacate his rooms temporarily in order that accommodation for the nurse or nurses may be provided. If the illness is such that skilled nursing is not needed the man is necessarily left very much to his own devices, seeing that his "bed-maker" is only in college for certain hours of the day, and, as a rule, has but the most elementary ideas on the subject of nursing. Fortunately, however, it is probable that this state of affairs, as regards non-infectious cases, at any rate, is likely to be remedied in the near future, since an anonymous benefactor has just announced his intention of providing a sum of money, up to 20,000*l*., for the purpose of building and equipping a nursing hostel in Cambridge. It is intended that this hostel shall be used by others than the members of the University, but that undergraduates, and especially those in poor circumstances, should be admitted for less than the normal fees. This hostel, however, will not, in all probability, have accommodation for cases of infectious disease, and this, of course, is the accommodation which would have been specially useful during the recent outbreak of influenza in 1918 and 1919.

Hitherto it has not been possible to remove cases of scarlet fever, diphtheria, and typhoid fever to the borough isolation hospital; but, as the result of a recent conference between the university and town authorities it is possible that arrangements will shortly be entered into which will provide for removal to the borough isolation hospital of all cases of infectious disease occurring among the members of the university. Dr. Laird, the medical officer of health, informed me that he would have been willing to have received cases of influenza during the recent outbreak, but that, owing to the depletion of his nursing staff during the war, this had not been possible. The suggestion has been put forward that the university authorities, possibly with the help of some portion of the recent anonymous gift, should erect a suitable hospital block (preferably on the cubicle system, so as to allow of more than one disease being admitted at the same time if necessary) in the grounds of the present town isolation hospital, and that in consideration of the payment of some annual charge to be agreed upon, which would enable the borough authorities to make some permanent

increase in their nursing staff, to have a call on the services of such staff if and when occasion should arise.

It should be mentioned that there is a fund in the hands of the university authorities known as "Crane's" Charity for the purpose of making grants "to poor scholars for their relief, for paying the physick, diet, or other "things necessary for them in their sickness." The capital sum from which these grants (about 100 guineas per annum) are made twice in the year, in May and November, was left to the university by an apothecary in Cambridge, known as John Crane, who died on the 26th May 1652, but whose benefaction, for some reason, now apparently unknown, remained inoperative until 1822, when public attention was called to the state of affairs. The Charity Commissioners in 1875 informed the trustees of the charity that out of the income the yearly sum of 50*l.* might be applied to some institution at Cambridge, or in the vicinity, for the purposes of training competent nurses to be utilised for the purpose of nursing the poor sick students of the university. Advantage does not appear to have been taken of this suggestion, and the proportion of the dividends available for the purpose is now mainly utilised for the making of grants to poor students in connection with the expenses involved in connection with treatment necessitated by accident or disease, in a nursing home or elsewhere.

I was informed by a member of the university committee recently appointed to consider the whole question of the provision of surgical and medical treatment for the undergraduates, that notices had already been sent out to representatives of all the colleges of a meeting to be held early in the following term as to the special provisions which might appear to be desirable, in view, more particularly, of the gift that had recently been announced. I learned, however, that so great a difference of opinion appeared to exist between the representatives of the various colleges with reference to questions arising in connection with the scheme, especially as to the desirability or otherwise of a form of insurance being entered into either by the colleges or the individual students, that it was somewhat doubtful what the ultimate outcome of their deliberations would be.

The expenses of surgical operations for poor students have always been paid in whole or in part by Crane's Charity, and there seems to be no doubt that some such arrangement as referred to above will be ultimately arrived at, with the town authorities, for the isolation and treatment of all cases of infectious disease.

Borough of Cambridge.

House-to-House Inquiry as to Influenza Incidence in four small selected Areas.

For the purpose of a detailed house-to-house inquiry, four distinct areas were selected, differing from one another as widely as possible in respect of house accommodation and nature of population in respect of occupation and sex-distribution. In these four areas houses to the total number of 105, with an average number of inmates ranging in different districts from 3·3 to 4·3, were visited and record made as to number of invaded houses in each area and as to distribution of occupants (418) and of cases of influenza (101), these latter being recorded also under the headings of "severe" (45) and "mild" (56). In only one of these areas were any deaths reported, three having occurred in the Cambridge Place area, in the months (one case in each) of October and November 1918 and in February 1919 respectively.

In all four areas by far the larger number of cases (69 out of 101) occurred in the period October–December 1918, during which period severity of attack was also most marked. Among females, however, the largest number of cases occurred in January–March 1919. Of the total number of inhabitants rather over 25 per cent. suffered attack, the incidence being

heaviest on females, among whom there were 70 cases, as compared with 31 males, out of a total population of 237 females and 181 males.

The age periods 5 to 15 years and 15 to 25 years were those which were most severely affected by the disease as regards the number of individuals attacked.

In none of the special areas investigated was any evidence of overcrowding obtained, the highest average number of occupants per house in any of the four areas being 4·3.

Attention was directed to possible fallacy in the statistics consequent on the recent return home of convalescent and demobilised soldiers, but, curiously enough, no instance was met with in which such recent additions to the family had come about in any of the houses visited.

Dr. Laird informed me that the disproportionate number of females attacked in the New Street area was dependent on the fact that the greater number of these women go out to work in factories and shops, where, rather than in their own houses, infection was probably contracted in most instances.

Further details of the investigation are shown in the following tables supplied by Dr. Laird :—

Influenza Investigation.

(House-to-House Inquiry in the Borough.)

Cambridge, 1918–19.

The following particulars relate to the four areas (taken together) :—

Age and Sex Distribution.

——	0–1.	1–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	
Occupants :—									
Males - - -	2	17	39	31	38	43	11	181	418
Females - - -	2	19	45	51	69	46	5	237	
Cases :—									
Males - - -	1	4	12	9	4	1	—	31	101
Females - - -	1	12	23	12	16	6	—	70	

Dates of Attack :—

June to September 1919 - - - - -	15 cases.
October to December 1918 - - - - -	69 ,,
January to March 1919 - - - - - -	17 ,,

Severity of Attack :—

Severe, 45.
Mild, 56.

Total number of houses visited - - - - -	105
,, ,, persons visited - - - - -	418
,, ,, houses with cases - - - -	36
,, ,, cases - - - - - - -	101
,, ,, deaths - - - - - - -	3*

* The three deaths occurred in Cambridge Place Area :—
Female, aged 3 years, October 1918.
Male, aged 44 years, November 1918.
Male, aged 38 years, February 1919.

De Freville Area.—Good middle-class residential area.

Age and Sex Distribution.

——	0–1.	1–5.	5–15.	15 25.	25–45.	45–65.	65 and over.	Total.	
Occupants :—									
Males - - -	1	2	10	6	9	17	4	49	123
Females - - -	—	2	20	13	20	17	2	74	
Cases :—									
Males - - -	1	—	5	3	2	1	—	12	32
Females - - -	—	2	12	2	2	2	—	20	

Dates of Attack :—

June to July - - - - - - - 10 cases.
October to December - - - - - - 22 „
January to March - - - - - - - 0 „

Severity of Attack :—

Severe, 7.
Mild, 25.

Number of houses visited - - - - - - 29
„ „ persons visited - - - - - - 123
„ „ houses with cases - - - - - - 10
„ „ cases - - - - - - - - 32
„ „ deaths - - - - - - - - Nil.

Cambridge Place Area.—Very poor property—two bedrooms ; 3*s.* to 3*s.* 6*d.* weekly.

Age and Sex Distribution.

——	0–1.	1–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	
Occupants :—									
Males - - -	1	9	16	10	13	6	4	59	129
Females - - -	1	10	11	15	22	10	1	70	
Cases :—									
Males - - -	—	4	5	2	2	—	—	13	32
Females - - -	1	6	4	3	5	—	—	19	

Dates of Attack :—

All attacks in October and November, except one in February who did not live here at the time (his family moved here on account of his death).

Severity of Attack :—

Severe, 18 (3 fatal : 1 child, 2 adults).
Mild, 14.

Total number of houses visited - - - - - 32
„ „ persons visited - - - - - 129
„ „ houses with cases - - - - 10
„ „ cases - - - - - - - 32
„ „ deaths - - - - - - 3

St. Philip's Road Area.—Good working-class neighbourhood.

Age and Sex Distribution.

——	0–1.	1–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	
Occupants :—									
Males - - -	—	1	4	5	10	19	2	35	80
Females - - -	—	2	2	14	14	12	1	45	
Cases :—									
Males - - -	—	—	1	3	—	—	—	4	17
Females - - -	—	1	1	6	3	2	—	13	

Dates of Attack :—

June to September - - - - - - - -	1
October to December - - - - - - -	12
January to March - - - - - - - -	4

Severity of Attack :—

Severe, 15 (none fatal).
Mild, 2.

Number of houses visited - - - - - -	24
,, ,, persons visited - - - - - -	80
,, ,, houses with cases - - - - - -	8
,, ,, cases - - - - - - - -	17
,, ,, deaths - - - - - - - -	Nil.

New Street Area.—Poor working-class street.

Age and Sex Distribution.

——	0–1.	1–5.	5–15.	15–25.	25–45.	45 65.	65 and over.	Total.	
Occupants :—									
Males - - -	—	5	9	8	8	7	1	38	86
Females - - -	1	5	12	9	13	7	1	48	
Cases :—									
Males - - -	—	—	1	1	—	—	—	2	20
Females - - -	—	3	6	1	6	2	—	18	

Dates of Attack :—

June to September - - - - - - - -	4
October to December - - - - - - -	11
January to March - - - - - - - -	5

Severity of Attack :—

Severe, 5 (none fatal).
Mild, 15.

Number of houses visited - - - - - -	20
,, ,, persons visited - - - - - -	86
,, ,, houses with cases - - - - - -	8
,, ,, cases - - - - - - - -	20
,, ,, deaths - - - - - - - -	Nil.

The following particulars relate to the four areas (taken together) :—

Table showing age and sex of those attacked, the nature of the attack, and the period in which the attack occurred :—

Age Periods.

Period of Attack.	0-1.		1-5.		5-15.		15-25.		25-45.		45-65.		65 and over.		Total.	
	M	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
JULY TO SEPT.																
Severe - - -	1	–	1	2	–	–	–	–	–	–	–	1	–	–	2	3
Mild - - -	–	–	4	–	1	1	–	–	1	2	1	–	–	–	7	3
OCT. TO DEC.																
Severe - - -	–	–	2	2	2	2	3	5	2	8	–	1	–	–	9	18
Mild - - -	–	–	2	6	1	19	3	4	–	3	–	4	–	–	6	36
JAN. TO MAR.																
Severe - - -	–	–	–	2	–	3	–	4	1	3	–	–	–	–	1	12
Mild - - -	–	–	–	1	–	1	2	–	–	–	–	–	–	–	2	2
TOTAL.																
Severe - - -	1	–	3	6	2	5	3	9	3	11	–	2	–	–	12	33
Mild - - -	–	–	6	7	2	21	5	4	1	5	1	4	–	–	15	41

De Freville Area.—Table showing age and sex of those attacked, the nature of the attack, and the period in which the attack occurred :—

Age Periods.

Period of Attack.	0-1.		1-5.		5-15.		15-25.		25-45.		45-65.		65 and over.		Total.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M	F.	M.	F.
JULY TO SEPT.																
Severe - - -	1	–	1	1	–	–	–	–	–	–	–	–	–	–	2	1
Mild - - -	–	–	4	–	–	–	–	–	1	1	1	–	–	–	6	1
OCT. TO DEC.																
Severe - - -	–	–	–	–	–	1	1	–	1	1	–	–	–	–	2	2
Mild - - -	–	–	–	2	–	10	2	2	–	–	–	2	–	–	2	16
JAN. TO MAR.																
Severe - - -	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Mild - - -	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
TOTAL.																
Severe - - -	1	–	1	1	–	1	1	–	1	1	–	–	–	–	4	3
Mild - - -	–	–	4	2	–	10	2	2	1	1	1	2	–	–	8	17

Cambridge Place Area.—Table showing age and sex of those attacked, the nature of the attack, and the period in which the attack occurred :—

Age Periods.

Period of Attack.	0–1.		1–5.		5–15.		15–25.		25–45.		45–65.		65 and over.		Total	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
JULY TO SEPT.																
Severe	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Mild	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
OCT. TO DEC.																
Severe	–	–	2	2	1	–	1	–	1	3	–	–	–	–	5	5
Mild	–	–	2	3	1	4	1	2	–	1	–	–	–	–	4	10
JAN. TO MAR.																
Severe	–	1	–	1	3	–	–	1	1	1	–	–	–	–	4	4
Mild	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
TOTAL.																
Severe	–	1	2	3	4	–	1	1	2	4	–	–	–	–	9	9
Mild	–	–	2	3	1	4	1	2	–	1	–	–	–	–	4	10

St. Philip's Road Area.—Table showing age and sex of those attacked the nature of the attack, and the period in which the attack occurred :—

Age Periods.

Period of Attack.	0–1.		1–5.		5–15.		15–25.		25–45.		45–65.		65 and over.		Total.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
JULY TO SEPT.																
Severe	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	1
Mild	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
OCT. TO DEC.																
Severe	–	–	–	–	1	1	2	3	–	3	–	1	–	–	3	8
Mild	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1
JAN. TO MAR.																
Severe	–	–	–	–	–	–	–	3	–	–	–	–	–	–	–	3
Mild	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	–
TOTAL.																
Severe	–	–	–	–	1	1	2	6	–	3	–	2	–	–	3	12
Mild	–	–	–	1	–	–	1	–	–	–	–	–	–	–	1	1

New Street Area.—Table showing age and sex of those attacked, the nature of the attack, and the period in which the attack occurred :—

Age Periods.

Period of Attack.	0–1.		1–5.		5–15.		15–25.		25–45.		45–65.		65 and over		Total.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
JULY TO SEPT.																
Severe	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1
Mild	–	–	–	–	1	1	–	–	–	1	–	–	–	–	1	2
OCT. TO DEC.																
Severe	–	–	–	–	–	–	–	1	–	1	–	–	–	–	–	2
Mild	–	–	–	–	–	5	–	–	–	2	–	2	–	–	–	9
JAN. TO MAR.																
Severe	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	2
Mild	–	–	–	1	–	1	1	–	–	–	–	–	–	–	1	2
TOTAL.																
Severe	–	–	–	1	–	–	–	1	–	3	–	–	–	–	–	5
Mild	–	–	–	1	1	7	1	–	–	3	–	–	–	–	2	13

OCCUPATIONS OF PERSONS ATTACKED.

De Ireville Area.

Males.		Females.	
Instrument makers	1	School teacher	1
Clerk of works	1		
Bank clerk	1		
Tailor	1		

Cambridge Place.

Males.		Females.	
Railway	1	Stetchworth dairy	1
School porter	1	Chivers' factory	2
		1st E.G.H.	1
		Home	2

St. Philip's Road.

Males.		Females.	
Boot shop assistant	1	Day girl	1
		Railway clerk	2
		Dressmaker	1

New Street.

Males.		Females.	
		Cleaner, 1st E.G.H.	1
		Cement works	1

The other persons attacked had no occupation.

Occupations of Persons in Houses Visited.

Cambridge Place Area.

Males.		Females.	
Bricklayer	1	General shop	1
Gardener	1	Stretchworth dairy	1
Labourer	6	Chivers' factory	8
Railway employee	1	1st E.G.H.	1
College servant	1		
Cement works	1		
Pumping station	1		
Painter	1		
Swiss laundry	1		
Chivers' factory	1		

De Freville Area.

Males.		Females.	
Tailor	5	Instrument maker	2
Cook	1	Typist	1
Shop manager	1	Clerk	5
Printer's manager	1	School teacher	2
Cashier	1		
Clerk	3		
Furnisher	1		
Insurance agent	1		
Accountant	1		
Newsagent	1		
Electrical engineer	1		
Grocer	1		
Clerk of works	1		
Builder	1		
Bank clerk	1		
Soldier	2		
College servant	2		
Milkman	1		
Carpenter	1		
Instrument maker	2		
Schoolmaster	1		
Tobacconist	1		

St. Philip's Road.

Males.		Females.	
Printer	1	P.O. messenger	1
Clerk	1	P.O. clerk	1
Railway employees	15	Railway clerks	2
Carpenter	1	Clerks, 1 E.G.H.	2
Bootmaker	1	Dressmaker	1
Milkman	1	Librarian	1
Bricklayer	1	Knitting factory	1
Missionary	1	Grocer's assistant	1
Farm worker	1	Day girl	1
Boot shop assistant	1		

New Street.

Males.		Females.	
Labourers	4	R.A.F.	1
Brush factory	1	Cement works	1
Dealer	1	Cleaner, 1st E.G.H.	1
Coprolite works	1	Laundry	1
Barber	1	Brush factory	1
Railway porter	1	Clerk	1
Fruiterer	1		

Influenza at the Friends' School at Saffron Walden.

While in Cambridge for the purposes of influenza investigation there during March 1919, I learnt from Sir Clifford Allbutt, Regius Professor of Physic, of an outbreak of influenza at the Friends' School, Saffron Walden. He had been called there in consultation a day or two previously, and informed me that a very severe and explosive outbreak had occurred at the school, involving 170 cases within three or four days. Under the circumstances he was of the opinion that further investigation might be worth while, as it was possible that some useful and definite information as regards incubation periods, &c., might be obtained.

Having mentioned the circumstances at the following meeting of the Influenza Committee at office, on my return to Cambridge I made an appointment with the Headmaster of the Friends' School to go over to Saffron Walden on Saturday, 29th March. Arrived there I was met by Mr. Walker, the Headmaster, Mr. Rowntree, his assistant and the only member of the establishment who had escaped influenza, and Dr. Atkinson, the medical officer to the school. All most kindly afforded me every information in their power as to the circumstances of the outbreak, Mr. Rowntree having taken much trouble to prepare beforehand for me detailed statistics and other information dealing with the incidence of the disease. I learnt from them that the school had been entirely broken up about a week before my visit, although a number of the children and staff had gone home previously, as soon as they were in a fit condition of health to do so.

Like the well known school at Bedales, the Friends' School at Saffron Walden is one in which boys and girls are mixed together in school, at meals, and at games, although, of course, having sleeping dormitories in completely separate portions of the building. At meals, indeed, the children are arranged boys and girls alternately, and this, and the common mixing in school as well as at games, probably had considerable effect upon the spread of the disease on both girls and boys of the school.

The total number of boarders at this school is 163, 94 boys and 69 girls, whilst, in addition, there are 14 day scholars, 9 boys and 5 girls. The house staff includes masters, mistresses, and a matron, and the domestic staff numbers 30, in addition to which three married masters and three gardeners live out in the town.

During the year 1918 there was no case of influenza at the school, although there was a considerable outbreak in the town of Saffron Walden during October and November of that year, during which a number of boys at the grammar school, and of the young women at the training college in the town, were attacked.

Mr. Walker attributes the entire escape of the school during October and November 1918 to the fact that he did not allow the boarders to go into the town during the whole of the term extending from 17th September to the 20th December, with the occasional exception of a boy taking a message to a shop or to the doctor's surgery. Mr. Walker did not at the time explain to me the reason of this regulation; but I subsequently learnt from Mr. Atkinson that he believed that it was made, quite at the beginning of the

term, in consequence of the occurrence of some cases of mumps, so that fortunately it was already in force at the time of the influenza outbreak at Saffron Walden.

The following term commenced on 15th January 1919, as from the commencement of which this embargo was withdrawn, although, as a matter of fact, very few of the children of the Friends' School went into the town before about mid-term.

As regards the origin of the outbreak it should be mentioned that parents of the children not infrequently go down on Saturdays and take the children out to tea, or for a walk, or possibly a motor ride, this having, as a matter of fact, taken place on Saturday, 22nd February, and on the following Saturday, 1st March. One of the visiting parents on the latter date had taken several of the children to Bishop's Stortford. These children, I understand, all went down with influenza on the following day, 2nd March.

I have not, however, been able to learn whether, in the course of their outing, they are known to have come in contact with a case or cases of the disease, either at Bishop Stortford or elsewhere. It is quite possible that these particular children may have been incubating the disease at the time, and that the infection was actually introduced into the school by a day-boy (W. ———), who is know to have been ill on Friday, 28th February, although it is possible that he might have been in an infectious state previously. He did not come to school the following day, Saturday, 29th February, and on inquiry being made, it was found that, on this date, all his family were confined to bed suffering from influenza. In this connection it is perhaps of interest that one of the first boys to develop the disease on 2nd March was a special friend of W.

The earliest cases in the school, three in number, occurred on 1st March 1919, followed by 11 other cases on the following day, after which the cases increased very rapidly as follows :—

3rd	March	27 new cases.
4th	,,	40 ,,
5th	,,	24 ,,
6th	,,	15 ,,
7th	,,	15 ,,
8th	,,	4 ,,
9th	,,	4 ,,

subsequent to which only occasional scattered cases occurred, the whole brunt of the disease therefore being confined to a period of about 10 days. The total number of individuals affected, including adults as well as boys and girls, was 182, the children in the school accounting for 143. In addition 18 of the adult staff were attacked, the remainder being accounted for by two masters and a master's wife and children living in the town, together with 4 boys and three adults who, although working at the school, sleep in the town. These latter, I understand, were among the earliest victims of the disease. Amongst the domestic staff it is of interest to note that only one of the seven maids employed at the school was attacked.

Symptoms of the Disease.

From a report supplied to me by Dr. Atkinson it appears that nearly all the individuals attacked complained of supra-orbital headache, and had acute rhinitis, although none complained of sore throat. The pharynx and uvula were found on inspection to be infected. Pains in the limbs and back were by no means universal, and, on the whole, he did not think the cases were so severe as in the epidemic which visited Saffron Walden in October and November 1918.

In uncomplicated cases the temperature stopped at 101° F., except in the case of one adult who, on the third day, had a temperature of 105° F., but which thenceforward made a gradual descent.

A noteworthy fact as regards the temperature charts is the persistence of a sub-normal temperature at the end of the disease. There was no evidence of profuse and continued perspiration, such as was characteristic of the 1918 epidemic in Saffron Walden.

Complications.

Three cases developed a scarlet rash, followed in two or three days by desquamation of the skin in large flakes. The rash was localised and somewhat suggestive of that seen in scarlet fever.

Epistaxis occurred in at least a dozen cases, in some of which it was so profuse that it could only be controlled by packing the nostril with adrenalin. One case developed otitis media with copious discharge from the ear. There were two cases, one of a boy, and one of an adult, of cardiac syncope with severe cerebral symptoms. One boy developed lobar pneumonia, and there were also three cases of catarrhal pneumonia. All these cases eventually recovered, there having been no fatal case throughout the whole of the epidemic.

Incubation Period.

Some interesting information with reference to the incubation of the disease was obtained. On Thursday, 1st March 1919, three boys were sent into the school sanitorium with what subsequently proved to be influenza. They were put into the same ward in which were already two boys who were convalescing from attacks of pneumonia (C. ——— and N. ———) from which they had been suffering for some time previous to the outbreak of influenza. On the morning of 3rd March C. ——— was found to have a temperature of 101°, which had mounted to 102·6° by the evening. By the same evening N. ——— had developed a temperature of 101°. Both of these boys subsequently developed definite symptoms of influenza. It should be mentioned that both these boys' temperature had been normal prior to the evening of 2nd March.

Again, one of the nurses especially engaged for work in the sanitorium early in the outbreak arrived at the school on 7th March, when she at once took up night duty in the wards. On the third day she was attacked with influenza, and had a temperature running up to 103° within a few hours. She had not been attending to cases of influenza prior to coming to Saffron Walden, and, so far as she is aware, had not come into contact with any case.

A further instance in which the period of incubation can be approximately determined is that of Mr. M. ———, a married master living in the town, who felt "seedy" on 2nd March, although he was able to come to school on the morning of 3rd March. But he at once had to return home to bed when he was found to have a temperature of over 100° F. On the morning of 4th March, Mrs. M. ——— was attacked, having a temperature of 100° by 1 p.m. on that day.

The case of her brother, Mr. F.———, who three days later came to look after Mr. and Mrs. M. ———, incidentally affords some evidence of immunity, as, although he remained in the house for a week, he escaped being attacked by the disease from which he had previously suffered whilst serving in the Army.

Report on Influenza Investigation, Cambridge, 1918–19.

Conclusions.

1. Of the three outbreaks of influenza during 1918–19, that occurring mainly in October and November 1918 was, as regards incidence on the Cambridge town population, the most extensive in respect of number of cases, and most severe in type.

2. Among the University population, the fatality rate at Cambridge was very low. As regards the four schools at Cambridge and Saffron Walden at which inquiry was made, it was found that, fortunately, not a single death had occurred.

3. No instance of the specially severe septic type of influenza had been reported in Cambridge itself, although at the First Eastern General Hospital there were seven deaths from influenza complicated with septic pneumonia among patients admitted from billets and camps outside the borough.

4. *Pathology.*—Reporting on the pathological appearances in sections of lung-tissue prepared by Dr. Aldren Wright from material obtained from post-mortem examinations performed at Addenbrooke's Hospital, Colonel Sims Woodhead, Professor of Pathology in the University, describes the condition present as an extremely rapid and acute inflammatory process, together with an acute pneumonia of a specially virulent form (*see* Dr. Laird's report on influenza in the borough of Cambridge in 1918).

5. No systematic bacteriological work on the disease had been carried out either in the First Eastern General Hospital or at Addenbrooke's Hospital, mainly owing, as I was informed, to the shortage of staff and the consequent pressure of work.

6. The spread of the disease appears to have taken place by personal contact alone.

7. Incidence of the disease has been mainly on young adults (15–25 years). Officers of the Naval Contingent at Cambridge (average age, 20·5 years), suffered attack to the extent of more than eight times as heavy as had their batmen (men of the R.M.L.I., average age, 30 years). Individuals of 60 years and upwards, almost without exception, escaped attack.

8. The incubation period is, in far the greater number of cases, from one to four days, with a still higher percentage between 36 and 60 hours.

9. *Onset.*—Epistaxis, occasionally with such severity as to require packing of the nostrils and the use of adrenalin, had been of frequent occurrence during the earliest stages of the disease during the outbreak of 1918–19, whereas the more classical symptom of pain in the back and limbs had been comparatively rare.

10. Of complications reported, the most frequent have been pneumonia and bronchitis. Three cases of unilateral parotitis have been reported by medical practitioners at Cambridge, at Saffron Walden, and at Newport respectively. Other complications reported in the death returns of the First Eastern General Hospital included septic pneumonia, acute spinal myelitis and paralysis of inter-costals, empyema, malignant endocarditis and cerebro-spinal fever.

11. At the Friends' School, Saffron Walden, in March 1919, a few comparatively mild cases of pneumonia occurred, while, strangely enough, at the Grammar School, in November 1918, no complication of any kind had occurred.

12. *Sequelæ.*—At the Friends' School, Saffron Walden, a number of the patients (boys and girls), during the convalescent period, ran an excessively low temperature for several days, and, in some instances, even weeks, the girls being affected in this respect to a greater extent than the boys. The headmaster's temperature is reported by Dr. Atkinson to have remained at the low level of 93° F. to 94° F. for some days after apparent recovery. A few cases of neurasthenia and one of hysterical paralysis have also been reported.

13. Immunity, of somewhat brief duration however (three to nine months), may be conferred by a previous attack of the disease. Dr. Laird

states that he heard of but one case of a second attack in Cambridge in 1918, the patient being a medical practitioner. Among the boarders of King's College Choir School at Cambridge, all of whom were attacked in mild form during the outbreak of July 1918, not a single case occurred during the outbreak of October-November 1918.

14. When a second attack of influenza has occurred within a period of about nine months, a severe attack has, for the most part, been followed by one milder in character, and *vice versâ*.

15. As regards the period subsequent to which a patient may be regarded as being no longer liable to convey infection, the experience at Addenbrooke's Hospital, where 10 nurses were returned to the general wards, after two days of normal temperature, without ill results to any of the patients in these wards, is of interest. On the other hand, Dr. Laird makes reference in his report to certain instances in which servants, convalescent from influenza but still troubled with a cough, apparently conveyed infection to the households of their employers, after returning to Cambridge from their own homes.

16. Analysis of replies received from the University residents has yielded no evidence in support of the contention, American in origin, that individuals who suffer from "chronic colds" are less liable than other people to be attacked by influenza.

TABLE A.

Showing Nnmber of Schedules distributed and returned.

College.	Approximate No. of Residents.	No. of Forms sent out.	No. of Forms returned.
1. Christ's	87	140	111
2. Sidney Sussex	108	150	80
3. Jesus	52	60	51
4. Magdalene	39	50	45
5. St. John's	120	150	122
6. Trinity	333	400	133
7. Caius	142	150	136
8. Trinity Hall	36	50	35
9. Clare	68	80	74
10. King's	71	120	73
11 St. Catharine's	43	50	34
12. Corpus Christi	28	40	31
13. Selwyn	32	50	35
14. Queen's	77	100	73
15. Pembroke	83	100	77
16. Peterhouse	37	50	39
17. Fitzwilliam Hall	99	100	45
18. Emmanuel	132	150	70
19. Downing	60	105	72
20. Naval Contingent	229	450	395
21. Sidney (Officers)	?	50	35
Totals	1,876	2,595	1,766

Table B. (1).

Influenza and Non-Influenza.

Table showing Age of Residents, &c., from whom Replies were received.

College, &c.	Total Residents.	17.	18.	19.	20.	21.	22.	23.
1. Christ's	111	5	9	13	11	12	9	7
2. Sidney Sussex	80	—	7	8	14	12	10	10
3. Jesus	51	1	12	9	7	6	5	2
4. Magdalene	45	—	3	10	5	7	2	6
5. St. John's	122	1	16	22	15	11	9	10
6. Trinity	133	—	15	21	8	19	18	20
7. Caius	136	4	27	22	14	13	14	14
8. Trinity Hall	35	1	5	5	1	1	3	5
9. Clare	74	1	15	18	7	4	9	4
10. King's	73	—	12	9	6	9	10	2
11. St. Catharine's	34	1	7	4	1	3	4	5
12. Corpus Christi	31	—	2	5	5	2	3	2
13. Selwyn	35	—	3	8	1	3	2	1
14. Queen's	73	1	10	3	11	10	6	8
15. Pembroke	77	—	20	17	5	8	6	5
16. Peterhouse	39	—	6	4	5	6	2	4
17. Non-Collegiate	45	—	3	3	2	5	3	—
18. Emmanuel	70	1	3	8	5	7	11	9
19. Downing	72	3	6	8	4	6	3	6
20. Naval Contingent	395	—	—	50	156	86	48	23
21. Sidney (Officers)	35	—	—	—	3	8	4	7
Total	1,766	19	181	247	286	238	181	150

College, &c.	Total Residents.	24	25.	26.	27.	28.	29.	30.	31.
1. Christ's	111	6	7	6	2	2	2	1	1
2. Sidney Sussex	80	1	2	2	—	2	1	—	1
3. Jesus	51	5	—	1	2	—	—	1	—
4. Magdalene	45	3	1	1	1	1	—	—	—
5. St. John's	122	5	9	3	—	1	1	1	—
6. Trinity	133	18	7	3	1	—	1	—	—
7. Caius	136	5	2	5	3	1	2	—	—
8. Trinity Hall	35	5	4	—	—	—	1	—	—
9. Clare	74	3	1	—	1	—	1	2	—
10. King's	73	3	1	3	—	1	—	1	—
11. St. Catharine's	34	5	—	—	—	2	—	1	—
12. Corpus Christi	31	3	1	3	—	—	1	—	—
13. Selwyn	35	6	1	3	1	—	—	—	—
14. Queen's	73	5	3	1	3	1	1	—	—
15. Pembroke	77	7	1	2	—	—	1	—	—
16. Peterhouse	39	3	2	—	—	—	2	1	—
17. Non-Collegiate	45	3	3	6	1	4	—	3	2
18. Emmanuel	70	7	7	3	—	—	—	—	1
19. Downing	72	9	3	3	1	3	—	—	1
20. Naval Contingent	395	11	4	3	—	2	—	—	—
21. Sidney (Officers)	35	—	4	1	1	2	1	—	1
Total	1,766	113	63	49	17	22	15	11	7

TABLE B. (1).—*continued.*

College, &c.	Total Residents.	32.	33.	34.	35.	36.	37.	38.
1. Christ's	111	2	2	—	—	2	—	—
2. Sidney Sussex	80	1	—	1	1	—	1	—
3. Jesus	51	—	—	—	—	—	—	—
4. Magdalene	45	1	—	—	—	—	—	—
5. St. John's	122	—	—	1	—	—	—	2
6. Trinity	133	—	—	—	—	—	—	—
7. Caius	136	1	—	—	—	1	1	—
8. Trinity Hall	35	1	—	—	—	1	—	—
9. Clare	74	1	—	1	—	1	—	—
10. King's	73	1	1	2	—	—	1	—
11. St. Catharine's	34	1	—	—	—	—	—	—
12. Corpus Christi	31	—	—	—	—	1	—	—
13. Selwyn	35	—	—	—	—	—	—	1
14. Queen's	73	—	1	1	2	—	1	—
15. Pembroke	77	1	1	—	—	1	—	—
16. Peterhouse	39	—	—	—	—	—	—	—
17. Non-Collegiate	45	1	—	2	1	1	—	—
18. Emmanuel	70	1	—	1	—	—	—	1
19. Downing	72	1	3	—	—	3	—	—
20. Naval Contingent	395	—	—	3	—	—	2	—
21. Sidney (Officers)	35	—	—	—	—	—	2	1
Total	1,766	13	8	12	4	11	8	5

College, &c.	Total Residents.	39.	40.	41.	42.	43.	44.	45.	46.
1. Christ's	111	1	1	—	—	3	—	—	—
2. Sidney Sussex	80	—	1	—	—	—	—	—	—
3. Jesus	51	—	—	—	—	—	—	—	—
4. Magdalene	45	—	—	—	—	1	—	—	1
5. St. John's	122	—	1	—	—	—	1	—	—
6. Trinity	133	—	—	—	1	—	—	—	—
7. Caius	136	1	—	—	—	—	—	1	—
8. Trinity Hall	35	—	1	—	—	—	—	—	—
9. Clare	74	—	—	—	—	—	—	—	—
10. King's	73	1	—	2	—	—	—	1	—
11. St. Catharine's	34	—	—	—	—	—	—	—	—
12. Corpus Christi	31	—	—	—	—	—	—	—	—
13. Selwyn	35	—	—	1	—	—	—	—	1
14. Queen's	73	—	—	1	—	—	1	—	—
15. Pembroke	77	—	—	—	—	—	—	—	—
16. Peterhouse	39	1	—	—	—	—	—	—	—
17. Non-Collegiate	45	—	—	—	1	—	—	—	1
18. Emmanuel	70	2	—	—	—	—	—	1	—
19. Downing	72	—	—	—	—	—	—	—	—
20. Naval Contingent	395	1	2	1	—	—	—	—	—
21. Sidney (Officers)	35	—	—	—	—	—	—	—	—
Total	1,766	7	6	5	2	4	2	3	3

TABLE B. (1).—*continued.*

College, &c.	Total Residents.	47.	48.	49.	50.	51.	52.	53.
1. Christ's	111	1	—	—	—	—	1	1
2. Sidney Sussex	80	—	1	1	1	—	1	—
3. Jesus	51	—	—	—	—	—	—	—
4. Magdalene	45	—	—	—	—	1	—	—
5. St. John's	122	—	1	—	—	1	1	1
6. Trinity	133	—	—	—	—	—	—	—
7. Caius	136	—	—	—	1	—	1	1
8. Trinity Hall	35	—	—	—	—	—	—	—
9. Clare	74	1	1	—	—	—	—	—
10. King's	73	—	—	—	—	—	—	—
11. St. Catharine's	34	—	—	—	—	—	—	—
12. Corpus Christi	31	—	1	—	—	—	—	—
13. Selwyn	35	—	—	1	—	—	—	—
14. Queen's	73	—	—	—	—	—	—	—
15. Pembroke	77	—	—	—	—	—	1	—
16. Peterhouse	39	—	—	1	—	—	—	—
17. Non-Collegiate	45	—	—	—	—	—	—	—
18. Emmanuel	70	1	—	—	—	—	—	—
19. Downing	72	—	1	1	—	—	—	1
20. Naval Contingent	395	2	1	—	—	—	—	—
21. Sidney (Officers)	35	—	—	—	—	—	—	—
Total	1,766	5	6	4	2	2	5	4

College, &c.	Total Residents.	54.	55.	56.	57.	58.	59.	60.	61.
1. Christ's	111	—	1	—	—	—	—	—	—
2. Sidney Sussex	80	—	—	—	—	—	—	—	—
3. Jesus	51	—	—	—	—	—	—	—	—
4. Magdalene	45	—	—	1	—	—	—	—	—
5. St. John's	122	—	2	—	—	1	—	1	1
6. Trinity	133	—	—	—	1	—	—	—	—
7. Caius	136	1	—	—	—	—	1	—	—
8. Trinity Hall	35	—	—	—	—	—	—	—	—
9. Clare	74	—	—	—	—	—	—	2	—
10. King's	73	1	1	1	—	—	—	1	—
11. St. Catharine's	34	—	—	—	—	—	—	—	—
12. Corpus Christi	31	—	—	—	—	—	—	1	—
13. Selwyn	35	—	—	—	1	—	—	1	—
14. Queen's	73	1	—	—	—	—	—	—	—
15. Pembroke	77	—	1	—	—	—	—	—	—
16. Peterhouse	39	—	—	—	1	—	1	—	—
17. Non-Collegiate	45	—	—	—	—	—	—	—	—
18. Emmanuel	70	—	—	—	—	—	—	—	—
19. Downing	72	—	2	—	—	1	—	—	—
20. Naval Contingent	395	—	—	—	—	—	—	—	—
21. Sidney (Officers)	35	—	—	—	—	—	—	—	—
Total	1,766	3	7	2	3	2	2	6	1

TABLE B. (1).—*continued.*

College, &c.	Total Residents.	62.	63.	64.	65.	66.	67.	68.
1. Christ's	111	—	1	—	2	—	—	—
2. Sidney Sussex	80	1	—	—	—	—	—	—
3. Jesus	51	—	—	—	—	—	—	—
4. Magdalene	45	—	—	—	—	—	—	—
5. St. John's	122	—	—	—	—	—	—	2
6. Trinity	133	—	—	—	—	—	—	—
7. Caius	136	—	—	—	—	—	—	—
8. Trinity Hall	35	1	—	—	—	—	—	—
9. Clare	74	—	—	—	—	—	—	—
10. King's	73	—	—	—	1	—	1	1
11. St. Catharine's	34	—	—	—	—	—	—	—
12. Corpus Christi	31	—	—	—	—	—	—	—
13. Selwyn	35	—	—	—	—	—	—	—
14. Queen's	73	1	—	—	—	—	—	—
15. Pembroke	77	—	—	—	—	—	—	—
16. Peterhonse	39	—	—	—	—	—	—	—
17. Non-Collegiate	45	—	—	—	—	—	—	—
18. Emmanuel	70	—	—	—	—	—	—	—
19. Downing	72	—	—	—	1	—	—	—
20. Naval Contingent	395	—	—	—	—	—	—	—
21. Sidney (Officers)	35	—	—	—	—	—	—	—
Total	1,766	3	1	—	4	—	1	3

College, &c.	Total Residents.	69.	70.	71.	76.	77.	78.	79.	?
1. Christ's	111	—	—	—	—	—	—	—	—
2. Sidney Sussex	80	—	—	—	—	—	—	—	—
3. Jesus	51	—	—	—	—	—	—	—	—
4. Magdalene	45	—	—	—	—	—	—	—	—
5. St. John's	122	1	—	—	—	1	—	—	—
6. Trinity	133	—	—	—	—	—	—	—	—
7. Caius	136	—	—	—	—	—	—	—	—
8. Trinity Hall	35	—	—	—	—	—	—	—	—
9. Clare	74	—	—	—	—	—	—	—	1
10. King's	73	—	—	—	—	—	—	—	—
11. St. Catharine's	34	—	—	—	—	—	—	—	—
12. Corpus Christi	31	—	—	—	—	—	—	—	1
13. Selwyn	35	—	—	—	—	—	—	—	—
14. Queen's	73	—	—	—	1	—	—	—	—
15. Pembroke	77	—	—	—	—	—	—	—	—
16. Peterhouse	39	—	—	—	—	—	—	—	—
17. Non-Collegiate	45	—	—	—	—	—	—	—	—
18. Emmanuel	70	—	—	—	—	—	—	1	—
19. Downing	72	—	—	1	—	—	1	—	—
20. Naval Contingent	395	—	—	—	—	—	—	—	—
21. Sidney (Officers)	35	—	—	—	—	—	—	—	—
Total	1,766	1	—	1	1	1	1	1	2

Table B (2).

Influenza Cases.

Table showing Age of Residents, &c., from whom replies were received.

College, &c.	Total Residents.	17.	18.	19.	20.	21.	22.	23.
1. Christ's	74	2	5	9	8	6	5	5
2. Sidney Sussex	55	—	5	4	9	10	7	5
3. Jesus	36	1	11	4	4	5	3	1
4. Magadalene	34	—	3	6	4	4	2	4
5. St. John's	82	1	11	14	10	8	5	5
6. Trinity	99	—	14	18	5	16	9	14
7. Caius	102	3	23	20	8	10	9	6
8. Trinity Hall	29	1	5	5	1	1	2	4
9. Clare	57	1	11	15	5	4	5	3
10. King's	62	—	11	8	3	7	9	2
11. St. Catharine's	21	—	4	3	1	1	4	4
12. Corpus Christi	22	—	1	4	1	2	2	2
13. Selwyn	26	—	—	6	—	3	1	—
14. Queen's	48	—	8	3	7	6	3	5
15. Pembroke	53	—	12	11	5	3	4	5
16. Peterhouse	27	—	6	3	2	4	1	1
17. Non-Collegiate	30	—	2	2	—	4	2	—
18. Emmanuel	41	1	3	6	2	5	5	6
19. Downing	48	1	4	3	3	4	1	2
20. Naval Contingent	294	—	—	43	115	62	38	13
21. Sidney (Officers)	23	—	—	—	2	4	3	5
Total	1,263	11	139	187	195	169	120	92

College, &c.	Total Residents.	24.	25.	26.	27.	28.	29.	30.	31.
1. Christ's	74	6	7	4	2	1	2	1	1
2. Sidney Sussex	55	1	1	2	—	2	1	—	—
3. Jesus	36	3	—	1	2	—	—	1	—
4. Magdalene	34	2	1	1	1	1	—	—	—
5. St. John's	82	4	8	2	—	1	1	1	—
6. Trinity	99	13	4	2	1	—	1	—	—
7. Caius	102	3	2	5	2	1	2	—	—
8. Trinity Hall	29	4	4	—	—	—	—	—	—
9. Clare	57	3	—	—	1	—	1	2	—
10. King's	62	2	1	3	—	1	—	1	—
11. St. Catharine's	21	2	—	—	—	—	—	1	—
12. Corpus Christi	22	3	—	2	—	—	1	—	—
13. Selwyn	26	5	1	3	1	—	—	—	—
14. Queen's	48	3	2	—	2	1	1	—	—
15. Pembroke	53	5	1	2	—	—	1	—	—
16. Peterhouse	27	3	2	—	—	—	1	—	—
17. Non-Collegiate	30	3	1	4	1	2	—	1	1
18. Emmanuel	41	4	2	2	—	—	—	—	—
19. Downing	48	9	1	2	1	3	—	—	—
20. Naval Contingent	294	6	2	3	—	2	—	—	—
21. Sidney (Officers)	23	—	3	—	1	2	1	—	1
Total	1,263	84	43	38	15	17	13	8	3

TABLE B (2)—*continued.*

College, &c.	Total Residents.	32.	33.	34.	35.	36.	37.	38.
1. Christ's	74	—	—	—	—	1	—	—
2. Sidney Sussex	55	1	—	1	1	—	1	—
3. Jesus	36	—	—	—	—	—	—	—
4. Magdalene	34	1	—	—	—	—	—	—
5. St. John's	82	—	—	1	—	—	—	2
6. Trinity	99	—	—	—	—	—	—	—
7. Caius	102	1	—	—	—	1	—	—
8. Trinity Hall	29	—	—	—	—	1	—	—
9. Clare	57	—	—	1	—	—	—	—
10. King's	62	1	1	2	—	—	—	—
11. St. Catharine's	21	1	—	—	—	—	—	—
12. Corpus Christi	22	—	—	—	—	1	—	—
13. Selwyn	26	—	—	—	—	—	—	1
14. Queen's	48	—	1	1	1	—	1	—
15. Pembroke	53	1	1	—	—	1	—	—
16. Peterhouse	27	—	—	—	—	—	—	—
17. Non-Collegiate	30	1	—	2	1	1	—	—
18. Emmanuel	41	1	—	1	—	—	—	1
19. Downing	48	1	3	—	—	2	—	—
20. Naval Contingent	294	—	—	3	—	—	1	—
21. Sidney (Officers)	23	—	—	—	—	—	1	—
Total	1,263	9	6	12	3	8	4	4

College, &c.	Total Residents.	39.	40.	41.	42.	43.	44.	45.	46.
1. Christ's	74	1	1	—	—	3	—	—	—
2. Sidney Sussex	55	—	—	—	—	—	—	—	—
3. Jesus	36	—	—	—	—	—	—	—	—
4. Magdalene	34	—	—	—	—	1	—	—	1
5. St. John's	82	—	—	—	—	—	1	—	—
6. Trinity	99	—	—	—	1	—	—	—	—
7. Caius	102	1	—	—	—	—	—	1	—
8. Trinity Hall	29	—	1	—	—	—	—	—	—
9. Clare	57	—	—	—	—	—	—	—	—
10. King's	62	1	—	2	—	—	—	1	—
11. St. Catharine's	21	—	—	—	—	—	—	—	—
12. Corpus Christi	22	—	—	—	—	—	—	—	—
13. Selwyn	26	—	—	1	—	—	—	—	1
14. Queen's	48	—	—	1	—	—	1	—	—
15. Pembroke	53	—	—	—	—	—	—	—	—
16. Peterhouse	27	1	—	—	—	—	—	—	—
17. Non-Collegiate	30	—	—	—	1	—	—	—	1
18. Emmanuel	41	1	—	—	—	—	—	—	—
19. Downing	48	—	—	—	—	—	—	—	—
20. Naval Contingent	294	1	2	1	—	—	—	—	—
21. Sidney (Officers)	23	—	—	—	—	—	—	—	—
Total	1,263	6	4	5	2	4	2	2	3

TABLE B (2)—*continued.*

College, &c.	Total Residents.	47.	48.	49.	50.	51.	52.	53.
1. Christ's	74	—	—	—	—	—	1	—
2. Sidney Sussex	55	—	1	1	1	—	1	—
3. Jesus	36	—	—	—	—	—	—	—
4. Magdalene	34	—	—	—	—	1	—	—
5. St. John's	82	—	—	—	—	1	—	1
6. Trinity	99	—	—	—	—	—	—	—
7. Caius	102	—	—	—	1	—	—	1
8. Trinity Hall	29	—	—	—	—	—	—	—
9. Clare	57	1	1	—	—	—	—	—
10. King's	62	—	—	—	—	—	—	—
11. St. Catharine's	21	—	—	—	—	—	—	—
12. Corpus Christi	22	—	1	—	—	—	—	—
13. Selwyn	26	—	—	1	—	—	—	—
14. Queen's	48	—	—	—	—	—	—	—
15. Pembroke	53	—	—	—	—	—	—	—
16. Peterhouse	27	—	—	1	—	—	—	—
17. Non-Collegiate	30	—	—	—	—	—	—	—
18. Emmanuel	41	1	—	—	—	—	—	—
19. Downing	48	—	1	1	—	—	—	1
20. Naval Contingent	294	1	1	—	—	—	—	—
21. Sidney (Officers)	23	—	—	—	—	—	—	—
Total	1,263	3	5	4	2	2	2	3

College, &c.	Total Residents.	54.	55.	56.	57.	58.	59.	60.	61.
1. Christ's	74	—	—	—	—	—	—	—	—
2. Sydney Sussex	55	—	—	—	—	—	—	—	—
3. Jesus	36	—	—	—	—	—	—	—	—
4. Magdalene	34	—	—	1	—	—	—	—	—
5. St. John's	82	—	2	—	—	—	—	—	—
6. Trinity	99	—	—	—	1	—	—	—	—
7. Caius	102	1	—	—	—	—	1	—	—
8. Trinity Hall	29	—	—	—	—	—	—	—	—
9. Clare	57	—	—	—	—	—	—	2	—
10. King's	62	1	1	1	—	—	—	1	—
11. St. Catharine's	21	—	—	—	—	—	—	—	—
12. Corpus Christi	22	—	—	—	—	—	—	1	—
13. Selwyn	26	—	—	—	1	—	—	1	—
14. Queen's	48	1	—	—	—	—	—	—	—
15. Pembroke	53	—	1	—	—	—	—	—	—
16. Peterhouse	27	—	—	—	1	—	1	—	—
17. Non-Collegiate	30	—	—	—	—	—	—	—	—
18. Emmanuel	41	—	—	—	—	—	—	—	—
19. Downing	48	—	2	—	—	1	—	—	—
20. Naval Contingent	294	—	—	—	—	—	—	—	—
21. Sidney (Officers)	23	—	—	—	—	—	—	—	—
Total	1,263	3	6	2	3	1	2	5	—

TABLE B (2)—*continued.*

College, &c.	Total Residents.	62.	63.	64.	65.	66.	67.	68.
1. Christ's	74	—	1	—	2	—	—	—
2. Sidney Sussex	55	—	—	—	—	—	—	—
3. Jesus	36	—	—	—	—	—	—	—
4. Magdalene	34	—	—	—	—	—	—	—
5. St. John's	82	—	—	—	—	—	—	1
6. Trinity	99	—	—	—	—	—	—	—
7. Caius	102	—	—	—	—	—	—	—
8. Trinity Hall	29	—	—	—	—	—	—	—
9. Clare	57	—	—	—	—	—	—	—
10. King's	62	—	—	—	1	—	1	—
11. St. Catharine's	21	—	—	—	—	—	—	—
12. Corpus Christi	22	—	—	—	—	—	—	—
13. Selwyn	26	—	—	—	—	—	—	—
14. Queen's	48	—	—	—	—	—	—	—
15. Pembroke	53	—	—	—	—	—	—	—
16. Peterhouse	27	—	—	—	—	—	—	—
17. Non-Collegiate	30	—	—	—	—	—	—	—
18. Emmanuel	41	—	—	—	—	—	—	—
19. Downing	48	—	—	—	—	—	—	—
20. Naval Contingent	294	—	—	—	—	—	—	—
21. Sidney (Officers)	23	—	—	—	—	—	—	—
Total	1,263	—	1	—	3	—	1	1

College, &c.	Total Residents.	69.	70.	71.	76.	77.	78.	79.	?
1. Christ's	74	—	—	—	—	—	—	—	—
2. Sidney Sussex	55	—	—	—	—	—	—	—	—
3. Jesus	36	—	—	—	—	—	—	—	—
4. Magdalene	34	—	—	—	—	—	—	—	—
5. St. John's	82	1	—	—	—	1	—	—	—
6. Trinity	99	—	—	—	—	—	—	—	—
7. Caius	102	—	—	—	—	—	—	—	—
8. Trinity Hall	29	—	—	—	—	—	—	—	—
9. Clare	57	—	—	—	—	—	—	—	1
10. King's	62	—	—	—	—	—	—	—	—
11. St. Catharine's	21	—	—	—	—	—	—	—	—
12. Corpus Christi	22	—	—	—	—	—	—	—	1
13. Selwyn	26	—	—	—	—	—	—	—	—
14. Queen's	48	—	—	—	—	—	—	—	—
15. Pembroke	53	—	—	—	—	—	—	—	—
16. Peterhouse	27	—	—	—	—	—	—	—	—
17. Non-Collegiate	30	—	—	—	—	—	—	—	—
18. Emmanuel	41	—	—	—	—	—	—	—	—
19. Downing	48	—	—	1	—	—	1	—	—
20. Naval Contingent	294	—	—	—	—	—	—	—	—
21. Sidney (Officers)	23	—	—	—	—	—	—	—	—
Total	1,263	1	—	1	—	1	1	—	2

Table B (3)

Table showing Age of Residents, &c., from whom Replies were received.

Non-Influenza.

College.	Total Residents.	17	18.	19.	20.	21.	22.	23.
1. Christ's	37	3	4	4	3	6	4	2
2. Sidney Sussex	25	—	2	4	5	2	3	5
3. Jesus	15	—	1	5	3	1	2	1
4. Magdalene	11	—	—	4	1	3	—	2
5. St. John's	40	—	5	8	5	3	4	5
6. Trinity	34	—	1	3	3	3	9	6
7. Caius	34	1	4	2	6	3	5	8
8. Trinity Hall	6	—	—	—	—	—	1	1
9. Clare	17	—	4	3	2	—	4	1
10. King's	11	—	1	1	3	2	1	—
11. St. Catharine's	13	1	3	1	—	2	—	1
12. Corpus Christi	9	—	1	1	4	—	1	—
13. Selwyn	9	—	3	2	1	—	1	1
14. Queen's	25	1	2	—	4	4	3	3
15. Pembroke	24	—	8	6	—	5	2	—
16. Peterhouse	12	—	—	1	3	2	1	3
17. Non-Collegiate	15	—	1	1	2	1	1	—
18. Emmanuel	29	—	—	2	3	2	6	3
19. Downing	24	2	2	5	1	2	2	4
20. Naval Contingent	101	—	—	7	41	24	10	10
21. Sidney (Officers)	12	—	—	—	1	4	1	2
Total	503	8	42	60	91	69	61	58

College.	Total Residents.	24.	25.	26.	27.	28.	29.	30.	31.
1. Christ's	37	—	—	2	—	1	—	—	—
2. Sidney Sussex	25	—	1	—	—	—	—	—	1
3. Jesus	15	2	—	—	—	—	—	—	—
4. Magdalene	11	1	—	—	—	—	—	—	—
5. St. John's	40	1	1	1	—	—	—	—	—
6. Trinity	34	5	3	1	—	—	—	—	—
7. Caius	34	2	—	—	1	—	—	—	—
8. Trinity Hall	6	1	—	—	—	—	1	—	—
9. Clare	17	—	1	—	—	—	—	—	—
10. King's	11	1	—	—	—	—	—	—	—
11. St. Catharine's	13	3	—	—	—	2	—	—	—
12. Corpus Christi	9	—	1	1	—	—	—	—	—
13. Selwyn	9	1	—	—	—	—	—	—	—
14. Queen's	25	2	1	1	1	—	—	—	—
15. Pembroke	24	2	—	—	—	—	—	—	—
16. Peterhouse	12	—	—	—	—	—	1	1	—
17. Non-Collegiate	15	—	2	2	—	2	—	2	1
18. Emmanuel	29	3	5	1	—	—	—	—	1
19. Downing	24	—	2	1	—	—	—	—	1
20. Naval Contingent	101	5	2	—	—	—	—	—	—
21. Sidney (Officers)	12	—	1	1	—	—	—	—	—
Total	503	29	20	11	2	5	2	3	4

TABLE B (3)—*continued.*

College.	Total Residents.	32.	33.	34.	35.	36.	37.	38.
1. Christ's	37	2	2	—	—	1	—	—
2. Sidney Sussex	25	—	—	—	—	—	—	—
3. Jesus	15	—	—	—	—	—	—	—
4. Magdalene	11	—	—	—	—	—	—	—
5. St. John's	40	—	—	—	—	—	—	—
6. Trinity	34	—	—	—	—	—	—	—
7. Caius	34	—	—	—	—	—	1	—
8. Trinity Hall	6	1	—	—	—	—	—	—
9. Clare	17	1	—	—	—	1	—	—
10. King's	11	—	—	—	—	—	1	—
11. St. Katharine's	13	—	—	—	—	—	—	—
12. Corpus Christi	9	—	—	—	—	—	—	—
13. Selwyn	9	—	—	—	—	—	—	—
14. Queen's	25	—	—	—	1	—	—	—
15. Pembroke	24	—	—	—	—	—	—	—
16. Peterhouse	12	—	—	—	—	—	—	—
17. Non-Collegiate	15	—	—	—	—	—	—	—
18. Emmanuel	29	—	—	—	—	—	—	—
19. Downing	24	—	—	—	—	1	—	—
20. Naval Contingent	101	—	—	—	—	—	1	—
21. Sidney (Officers)	12	—	—	—	—	—	1	1
Total	503	4	2	—	1	3	4	1

College.	Total Residents.	39.	40.	41.	42.	43.	44.	45.	46.
1. Christ's	37	—	—	—	—	—	—	—	—
2. Sidney Sussex	25	—	1	—	—	—	—	—	—
3. Jesus	15	—	—	—	—	—	—	—	—
4. Magdalene	11	—	—	—	—	—	—	—	—
5. St. John's	40	—	1	—	—	—	—	—	—
6. Trinity	34	—	—	—	—	—	—	—	—
7. Caius	34	—	—	—	—	—	—	—	—
8. Trinity Hall	6	—	—	—	—	—	—	—	—
9. Clare	17	—	—	—	—	—	—	—	—
10. King's	11	—	—	—	—	—	—	—	—
11. St. Catharine's	13	—	—	—	—	—	—	—	—
12. Corpus Christi	9	—	—	—	—	—	—	—	—
13. Selwyn	9	—	—	—	—	—	—	—	—
14. Queen's	25	—	—	—	—	—	—	—	—
15. Pembroke	24	—	—	—	—	—	—	—	—
16. Peterhouse	12	—	—	—	—	—	—	—	—
17. Non-Collegiate	15	—	—	—	—	—	—	—	—
18. Emmanuel	29	1	—	—	—	—	—	1	—
19. Downing	24	—	—	—	—	—	—	—	—
20. Naval Contingent	101	—	—	—	—	—	—	—	—
21. Sidney (Officers)	12	—	—	—	—	—	—	—	—
Total	503	1	2	—	—	—	—	1	—

TABLE B (3)—*continued.*

College.	Total Residents.	47.	48.	49.	50.	51.	52.	53.
1. Christ's	37	1	—	—	—	—	—	1
2. Sidney Sussex	25	—	—	—	—	—	—	—
3. Jesus	15	—	—	—	—	—	—	—
4. Magdalene	11	—	—	—	—	—	—	—
5. St. John's	40	—	1	—	—	—	1	—
6. Trinity	34	—	—	—	—	—	—	—
7. Caius	34	—	—	—	—	—	1	—
8. Trinity-Hall	6	—	—	—	—	—	—	—
9. Clare	17	—	—	—	—	—	—	—
10. King's	11	—	—	—	—	—	—	—
11. St. Catharine's	13	—	—	—	—	—	—	—
12. Corpus Christi	9	—	—	—	—	—	—	—
13. Selwyn	9	—	—	—	—	—	—	—
14. Queen's	25	—	—	—	—	—	—	—
15. Pembroke	24	—	—	—	—	—	1	—
16. Peterhouse	12	—	—	—	—	—	—	—
17. Non-Collegiate	15	—	—	—	—	—	—	—
18. Emmanuel	29	—	—	—	—	—	—	—
19. Downing	24	—	—	—	—	—	—	—
20. Naval Contingent	101	1	—	—	—	—	—	—
21. Sidney (Officers)	12	—	—	—	—	—	—	—
Total	503	2	1	—	—	—	3	1

College.	Total Residents.	54.	55.	56.	57.	58.	59.	60.	61.
1. Christ's	37	—	1	—	—	—	—	—	—
2. Sidney Sussex	25	—	—	—	—	—	—	—	—
3. Jesus	15	—	—	—	—	—	—	—	—
4. Magdalene	11	—	—	—	—	—	—	—	—
5. St. John's	40	—	—	—	—	1	—	1	1
6. Trinity	34	—	—	—	—	—	—	—	—
7. Caius	34	—	—	—	—	—	—	—	—
8. Trinity Hall	6	—	—	—	—	—	—	—	—
9. Clare	17	—	—	—	—	—	—	—	—
10. King's	11	—	—	—	—	—	—	—	—
11. St. Catharine's	13	—	—	—	—	—	—	—	—
12. Corpus Christi	9	—	—	—	—	—	—	—	—
13. Selwyn	9	—	—	—	—	—	—	—	—
14. Queen's	25	—	—	—	—	—	—	—	—
15. Pembroke	24	—	—	—	—	—	—	—	—
16. Peterhouse	12	—	—	—	—	—	—	—	—
17. Non-Collegiate	15	—	—	—	—	—	—	—	—
18. Emmanuel	29	—	—	—	—	—	—	—	—
19. Downing	24	—	—	—	—	—	—	—	—
20. Naval Contingent	101	—	—	—	—	—	—	—	—
21. Sidney (Officers)	12	—	—	—	—	—	—	—	—
Total	503	—	1	—	—	1	—	1	1

TABLE B (3)—*continued.*

College.	Total Residents.	62.	63.	64.	65.	66.	67.	68.
1. Christ's	37	—	—	—	—	—	—	—
2. Sidney Sussex	25	1	—	—	—	—	—	—
3. Jesus	15	—	—	—	—	—	—	—
4. Magdalene	11	—	—	—	—	—	—	—
5. St. John's	40	—	—	—	—	—	—	1
6. Trinity	34	—	—	—	—	—	—	—
7. Caius	34	—	—	—	—	—	—	—
8. Trinity Hall	6	1	—	—	—	—	—	—
9. Clare	17	—	—	—	—	—	—	—
10. King's	11	—	—	—	—	—	—	1
11. St. Catharine's	13	—	—	—	—	—	—	—
12. Corpus Christi	9	—	—	—	—	—	—	—
13. Selwyn	9	—	—	—	—	—	—	—
14. Queen's	25	1	—	—	—	—	—	—
15. Pembroke	24	—	—	—	—	—	—	—
16. Peterhouse	12	—	—	—	—	—	—	—
17. Non-Collegiate	15	—	—	—	—	—	—	—
18. Emmanuel	29	—	—	—	—	—	—	—
19. Downing	24	—	—	—	—	—	—	—
20. Naval Contingent	101	—	—	—	—	—	—	—
21. Sidney (Officers)	12	—	—	—	—	—	—	—
Total	503	3	—	—	—	—	—	2

College	Total Residents.	69.	70.	71.	76.	77.	78.	79.	?
1. Christ's	37	—	—	—	—	—	—	—	—
2. Sidney Sussex	25	—	—	—	—	—	—	—	—
3. Jesus	15	—	—	—	—	—	—	—	—
4. Magdalene	11	—	—	—	—	—	—	—	—
5. St. John's	40	—	—	—	—	—	—	—	—
6. Trinity	34	—	—	—	—	—	—	—	—
7. Caius	34	—	—	—	—	—	—	—	—
8. Trinity Hall	6	—	—	—	—	—	—	—	—
9. Clare	17	—	—	—	—	—	—	—	—
10. King's	11	—	—	—	—	—	—	—	—
11. St. Catharine's	13	—	—	—	—	—	—	—	—
12. Corpus Christi	9	—	—	—	—	—	—	—	—
13. Selwyn	9	—	—	—	—	—	—	—	—
14. Queen's	25	—	—	—	1	—	—	—	—
15. Pembroke	24	—	—	—	—	—	—	—	—
16. Peterhouse	12	—	—	—	—	—	—	—	—
17. Non-Collegiate	15	—	—	—	—	—	—	—	—
18. Emmanuel	29	—	—	—	—	—	—	1	—
19. Downing	24	—	—	1	—	—	—	—	—
20. Naval Contingent	101	—	—	—	—	—	—	—	—
21. Sidney (Officers)	12	—	—	—	—	—	—	—	—
Total	503	—	—	1	1	—	—	1	—

Table C.

Table showing number of persons who had Influenza prior to 1918 and during the Periods July–September, October–December 1918 and January to March 1919, with severity of attack.

College, &c.	No. of Cases.	Prior to 1918.			July to Sept.			Oct. to Dec.			Jan. to March.		
		—	S.	M.	—	S.	M.	—	S.	M.	—	S.	M.
1. Christ - -	74	47	15	32	17	5	12	24	5	19	10	4	6
2. Sidney - -	55	34	7	27	11	2	9	22	12	10	3	—	3
3. Jesus - -	36	13	3	10	9	4	5	22	8	14	—	—	—
4. Magdalene -	34	15	6	9	9	—	9	13	4	9	4	1	3
5. St. John's -	82	50	14	36	23	3	20	28	10	18	13	5	8
6. Trinity - -	99	54	11	43	45	4	41	29	8	21	10	1	9
7. Caius - -	102	58	14	44	40	4	36	30	14	16	11	2	9
8. Trinity Hall -	29	19	6	13	9	—	9	10	4	6	5	1	4
9. Clare - -	57	34	8	26	12	6	6	29	12	17	5	—	5
10. King's - -	61	46	8	38	25	6	19	22	8	14	7	2	5
11. St. Catharine's	21	12	2	10	8	1	7	11	3	8	3	2	1
12. Corpus Christi	22	11	3	8	8	—	8	7	3	4	6	1	5
13. Selwyn - -	26	21	5	16	9	2	7	10	3	7	3	1	2
14. Queen's - -	48	16	2	14	23	5	18	14	5	9	10	—	10
15. Pembroke -	53	29	2	27	19	3	16	26	8	18	5	—	5
16. Peterhouse -	27	15	3	12	9	4	5	11	4	7	2	—	2
17. Fitzwilliam Hall	30	17	3	14	11	6	5	9	2	7	8	—	8
18 Emmanuel -	41	22	1	21	19	4	15	13	4	9	4	2	2
19. Downing -	48	26	6	20	15	4	11	17	5	12	5	1	4
20. Naval Contingent.	294	139	21	118	92	18	74	92	31	61	67	18	49
21. Sidney (Officers)	23	13	5	8	10	3	7	8	5	3	4	1	3
Total - -	1,263	691	145	546	423	84	339	447	158	289	185	42	143

Table D.

Table showing Ages of Undergraduates, &c., attacked.

Ages.	Prior to 1918.			July to Sept. 1918.			Oct. to Dec. 1918.			Jan. to March 1919.			July 1918 to March 1919.		
	Total.	S.	M.	Total.	S.	M.	Total.	S.	M.	Total.	S.	M	Total.	S.	M.
17 - -	5	—	5	3	1	2	6	2	4	3	1	2	12	4	8
18 - -	61	11	50	52	13	39	65	24	41	14	3	11	131	40	91
19 - -	102	10	92	72	9	63	57	15	42	19	2	17	148	26	122
20 - -	104	18	86	58	16	42	69	23	46	33	7	25	159	46	113
21 - -	78	23	55	56	11	45	59	22	37	33	13	20	148	46	102
22 - -	60	8	52	48	8	40	46	17	29	17	2	15	111	27	84
23 - -	48	15	33	31	4	27	32	14	18	9	3	6	72	21	51
24 - -	50	13	37	33	7	26	33	11	22	11	3	8	77	21	56
25 - -	26	6	20	13	2	11	15	5	10	9	2	7	37	9	28
26 - -	18	5	13	16	4	12	15	7	8	5	1	4	36	12	24
27 - -	7	1	6	9	2	7	5	3	2	1	—	1	15	5	10
28 - -	9	2	7	7	4	3	6	2	4	2	—	2	15	6	9
29 - -	6	1	5	4	—	4	6	3	3	2	—	2	12	3	9
30 - -	3	3	—	3	—	3	3	—	3	3	1	2	9	1	8
31 - -	3	1	2	2	1	1	1	—	1	1	1	—	4	2	2
32 - -	5	1	4	3	—	3	2	1	1	2	1	1	7	2	5
33 - -	6	1	5	2	—	2	4	1	3	2	—	2	8	1	7
34 - -	9	3	6	4	1	3	5	1	4	3	—	3	12	2	10
35 - -	1	—	1	1	1	—	1	1	—	1	—	1	3	2	1
36 - -	7	2	5	1	—	1	3	2	1	1	—	1	5	2	3
37 - -	4	—	4	1	—	1	—	—	—	—	—	—	1	—	1
38 - -	3	—	3	—	—	—	—	—	—	2	—	2	2	—	2
39 - -	6	1	5	1	—	1	2	—	2	1	—	1	4	—	4
40 - -	3	1	2	1	—	1	2	1	1	2	—	2	5	1	4
41 - -	3	1	2	—	—	—	4	1	3	—	—	—	4	1	3
42 - -	2	—	2	—	—	—	—	—	—	—	—	—	—	—	—
43 - -	3	1	2	—	—	—	—	—	—	2	—	2	2	—	2
44 - -	1	—	1	1	—	1	—	—	—	—	—	—	1	—	1
45 - -	2	1	1	—	—	—	—	—	—	—	—	—	—	—	—
46 - -	3	1	2	—	—	—	—	—	—	—	—	—	—	—	—
47 - -	3	—	3	—	—	—	—	—	—	1	—	1	1	—	1
48 - -	5	3	2	—	—	—	1	—	1	—	—	—	1	—	1
49 - -	3	—	3	—	—	—	2	1	1	—	—	—	2	1	1
50 - -	2	1	1	—	—	—	—	—	—	—	—	—	—	—	—
51 - -	2	1	1	—	—	—	—	—	—	1	1	—	1	1	—
52 - -	2	1	1	—	—	—	—	—	—	—	—	—	—	—	—
53 - -	3	—	3	—	—	—	—	—	—	1	—	1	1	—	1
54 - -	3	2	1	1	—	1	—	—	—	—	—	—	1	—	1
55 - -	5	1	4	—	—	—	3	1	2	2	—	2	5	1	4
56 - -	2	—	2	—	—	—	—	—	—	1	—	1	1	—	1
57 - -	3	2	1	—	—	—	—	—	—	—	—	—	—	—	—
58 - -	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—
59 - -	2	—	2	—	—	—	—	—	—	—	—	—	—	—	—
60 - -	5	—	5	—	—	—	—	—	—	1	—	1	1	—	1
61 - -	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
62 - -	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
63 - -	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—
64 - -	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
65 - -	3	—	3	—	—	—	—	—	—	1	1	—	1	1	—
66 - -	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
67 - -	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—
68 - -	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—
69 - -	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—
70 - -	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
71 - -	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—
77 - -	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—
78 - -	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—
? -	2	—	2	—	—	—	—	—	—	—	—	—	—	—	—
Totals -	691	145	546	423	84	339	447	158	289	185	42	143	1055	284	771

Table E.

Table showing Complications and the Months in which they occurred.

Complication.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.
Pneumonia	2	1	—	10	7	—	1	—	—
Bronchitis	6	1	1	9	8	1	—	3	4
Pneumonia and bronchitis	—	—	—	3	—	1	—	1	—
Pneumonia and nasal catarrh	1	—	—	—	—	—	—	—	—
Pneumonia and defective action of heart.	—	—	—	1	—	—	—	—	—
Pneumonia and pleurisy	1	—	—	—	—	—	—	—	—
Pneumonia, bronchitis and pleurisy.	—	—	1	—	—	—	—	—	—
Bronchitis and lumbago	—	—	—	—	—	—	—	1	—
Bronchitis and asthma	—	—	—	1	—	—	—	—	—
Bronchitis and heart	—	—	—	1	—	—	—	—	—
Pleurisy	—	—	—	2	1	—	1	—	—
Asthma	—	—	—	—	—	—	1	—	—
Congestion of the lungs	—	—	—	—	—	—	—	1	—
Congestion of bronchial tubes	—	—	—	1	—	—	—	—	—
Chronic catarrh	—	—	—	—	—	—	—	1	—
Nasal catarrh	1	1	—	—	—	—	—	1	—
Lungs	—	—	—	—	1	—	—	—	—
Phthisis	1	—	—	—	—	—	—	—	—
Cough	—	—	—	1	—	—	—	—	—
Septic naso pharynx	—	—	—	—	—	—	1	—	—
Septic throat	—	—	—	1	—	—	—	—	—
Quinsey	1	—	—	1	—	—	—	—	—
Tonsillitis	—	—	—	—	1	—	—	—	—
Laryngitis	—	—	—	—	1	—	—	—	—
Pyrrhœa	—	—	—	1	—	—	—	—	—
Glands	—	—	—	—	—	—	—	1	—
Deafness	—	—	—	—	—	—	—	1	—
Inflammation of drum of ear	—	—	—	—	—	1	—	—	—
Perforation of septum	—	—	—	—	—	—	1	—	—
Neuralgia	—	—	—	2	—	—	1	—	—
Heart trouble	—	—	1	—	2	—	—	1	—
Jaundice	—	—	—	1	2	—	—	—	—
Gastritis	—	—	—	—	1	1	—	—	—
Gastric catarrh	—	—	—	—	—	—	—	2	—
Colic	—	—	—	—	—	—	1	—	—
Typhoid fever	1	—	—	—	—	—	—	—	—
Loss of all mental power	—	—	—	—	1	—	—	—	—
Loss of memory	—	—	—	—	—	—	—	1	—
Neurasthenia	1	—	—	—	—	—	—	—	—
Nervousness	—	—	—	—	1	—	—	—	—
Infantile paralysis	—	1	—	—	—	—	—	—	—
Rheumatism	—	—	—	—	1	—	—	—	—
Total	15	4	3	35	27	4	7	14	4

Table F.

Showing ages of persons whose attacks were complicated.

Complication.	Age. 17	18	19	20	21	22	23	24	25	26	27	29	30	31	32	33	34	35	36	39	40	43	54	55	65
Pneumonia	1	1	2	1	4	4	1	2	2	—	1	1	—	—	—	—	—	1	—	—	—	—	—	—	—
Bronchitis	—	1	8	6	4	3	—	3	—	1	—	—	—	1	1	—	1	—	1	—	—	1	—	1	1
Pneumonia and bronchitis	—	—	—	—	3	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Pneumonia, bronchitis, and Pleurisy.	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Pneumonia and pleurisy	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Pneumonia and other complications.	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—
Bronchitis and other complications.	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	1	—	—	—	—
Pleurisy	—	—	1	—	2	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Other diseases of respiratory system.	1	—	4	1	3	1	2	1	1	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—
Other complications	—	2	4	3	9	1	—	1	2	—	—	1	1	—	—	—	1	—	—	1	—	—	—	1	—
Total	2	5	20	12	25	11	3	9	5	1	1	2	1	1	1	1	2	1	2	1	1	1	2	2	1

Table G.

Incubation Period.

Table compiled from replies to question: "If attacked within 12 months, give Date of exposure; give Date of Attack."

College.	Number of Days between Date of Exposure to Infection, and Date of Attack.													
	Same Day.	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	12 and over.
1. Christ's	1	2	3	1	2	–	–	–	–	–	1	–	–	–
2. Sidney	–	3	1	–	–	–	–	–	–	1	–	–	–	1
3. Jesus	–	–	1	–	–	–	–	–	–	–	–	–	–	1
4. Magdalene	1	2	1	2	–	–	–	–	–	–	–	–	–	–
5. St. John's	–	2	2	2	3	1	–	1	–	1	–	1	–	–
6. Trinity	–	1	4	1	2	–	1	1	1	–	–	–	–	1
7. Caius	–	1	5	–	2	2	1	–	–	1	–	–	–	–
8. Trinity Hall	–	1	–	–	–	–	–	–	–	–	–	–	–	1
9. Clare	–	2	3	–	1	–	1	–	–	–	–	–	1	–
10. King's	2	–	3	1	–	1	–	1	–	–	1	–	–	–
11. St. Catharine's	–	1	–	–	–	–	–	–	–	–	–	–	–	–
12. Corpus Christi	–	–	1	1	1	–	–	–	–	–	–	–	–	–
13. Selwyn	1	1	–	–	2	–	–	–	–	–	–	–	–	–
14. Queen's	–	–	–	–	2	–	2	–	–	–	–	–	–	2
15. Pembroke	–	–	–	1	2	–	1	1	–	–	–	–	–	–
16. Peterhouse	–	1	–	1	–	–	1	–	–	–	–	–	1	–
17. Fitzwilliam Hall (non-collegiate).	–	–	–	–	–	1	–	–	–	–	–	–	–	–
18. Emmanuel	–	1	1	–	–	–	–	–	–	–	–	–	–	3
19. Downing	–	1	–	–	–	–	–	–	1	–	–	–	–	–
20. Naval Contingent	1	6	8	4	2	3	2	–	–	1	–	–	1	2
21. Sidney (Officers)	2	–	–	–	–	–	–	1	–	–	–	–	–	–
Total - 144	8	25	33	14	19	8	9	5	2	4	2	1	3	11

Table H.

Complete List.

Period of Incubation.

Others Infected.

No.	College.	Age.	Date of Attack.	Date of Exposure of Person Infected.	Date of Attack of Person Infected.
1	Corpus	36	20th Nov.	Wife. 25th Nov.	22nd Nov.
2	Clare	23	1st Nov.	Whole family infected Oct.	At 3 days interval Oct.
3	,,	18			
4	,,	34	17th Feb.	Wife, 15th Feb.	24th Feb.
5	Naval	19	12th Oct.	12th Oct.	20th Oct.
6	,,	20	30th Sept.	Epidemic throughout ship, 30th Sept.	—
7	,,	20	10th Oct.	10th Oct.	16th Oct.
8	,,	20	5th July	5th July	7th July.
9	,,	20	7th Aug.	7th Aug.	9th Aug.
10	,,	20	25th May	27th May	30th May.
11	,,	20	20th Oct.	2nd Class steward. Died in hospital, 20th Oct.	—
12	,,	20	6th Nov.	6th Nov.	8th Nov.
13	,,	21	9th Oct.	3 officers, 7th, 8th. 9th Oct.	9th, 10th, 11th. Oct.

No.	College.	Age.	Date of Attack.	Date of Exposure of Person Infected.	Date of Attack of Person Infected.
14	Naval	21	6th Oct.	6th Oct.	9th Oct.
15	,,	21	24th Jan.	24th Jan.	22th Jan.
16	,,	21	Oct.	Oct.	Oct.
17	,,	21	June	June	June.
18	Caius	18	Nov.	Nov.	Nov.
19	,,	22	19th Oct.	23rd Oct.	27th Oct.
20	Trinity	21	10th Jan.	10th, 20th Jan.	20th Jan.
21	,,	24	July and Sept.	Someone in France, July and Sept.	—
22	Christ's	28	4th Nov.	4th Nov.	10th Nov.
23	,,	43	9th Feb.	9th Feb.	12th Feb.
24	St. Peter's	25	18th Oct.	2 officers, 18th Oct.	20th Oct.
25	Selwyn	24	1st Nov.	4th Nov.	6th Nov.
26	,,	38	26th Feb.	1 maidservant, 26th Feb.	5th March.
27	Jesus	18	19th July	3 Repton school boys	—
28	Fitzwilliam Hall	21	1st July	1st July.	5th July.
29	,,	22	4th July	4th July	6th July.
30	Downing	20	20th June	20th June	26th June.
31	,,	25	4th Feb.	4th Feb.	6th Feb.
32	Queen's	20	14th June	14th–16th June	16th June.
33	,,	22	1st Feb.	14th Feb.	17th Feb.
34	King s	18	5th Oct.	5th Oct.	7th Oct.
35	,,	33	16th Feb.	Nurse, 18th Feb.	21st Feb.
36	,,	41	25th Oct.	25th Oct.	27th Oct.
37	St. John's	18	17th Nov.	17th Nov.	21st Nov.
38	,,	20	7th Dec.	7th Dec.	11th Dec.
39	,,	23	6th Feb.	6th Feb.	9th Feb.
40	,,	24	3rd Nov.	3rd Nov.	5th Nov.
41	,,	53	19th Feb.	21st Feb.	26th Feb.
42	Sidney Sussex	18	13th July	13th July	14th July.
43	Naval	20	30th Jan.	30th–31st Jan.	31st Jan.
44	,,	26	25th July	25th July	29th July.
45	Magdalane	18	3rd Dec.	4th Dec.	8th Dec.
46	,,	19	15th Oct.	2 officers, 15th-20th Oct.	21st Oct.
47	,,	21	June	June	June.

Table J.

List of Persons who have had Two Attacks of Influenza during 1918–19.

College.	No.	Age.	Date of First Attack.	Date of Second Attack.	Com-plication.	Date of Attack thus com-plicated.
Trinity - -	1	19	July	Oct.	—	—
,, - -	2	19	1st Nov.	1st Mar.	—	—
,, - -	3	22	20th Aug.	10th Nov.	—	—
,, - -	4	22	7th Dec.	14th Feb.	Bronchial pneumonia.	7th Dec.
,, - -	5	22	14th Nov.	3rd Mar.	—	—
,, - -	6	23	5th June	24th Nov.	—	—
,, - -	7	24	July	Sept.	—	—
,, - -	8	24	,,	Feb.	—	—
,, - -	9	26	4th Nov.	15th Feb.	—	—
St. Catharine's -	10	24	June	Feb.	—	—
,, -	11	32	Oct.	20th Feb.	Bronchitis	20th Feb.
Pembroke - -	12	18	July	Nov.	Jaundice	Nov.
,, - -	13	18	13th Nov.	20th Mar.	—	—
,, - -	14	19	7th Aug.	28th Nov.	Heart trouble.	28th Nov.

College.	No.	Age.	Date of First Attack.	Date of Second Attack.	Complication.	Date of Attack thus complicated.
Pembroke - -	15	25	7th July	10th Oct.	—	—
St. John's - -	16	17	28th Dec.	10th Feb.	Congestion of lungs.	10th Feb.
,, - -	17	19	2nd Oct.	7th Feb.	—	—
,, - -	18	20	Nov.	Feb.	—	—
,, - -	19	25	1st July	1st Dec.	—	—
Downing - -	20	24	July	Oct.	—	—
,, - -	21	24	31st July	12th Jan.	—	—
St. Peter's - -	22	20	25th Sept.	30th Oct.	—	—
,, - -	23	24	Nov.	7th Mar.	—	—
,, - -	24	24	July	12th Dec.	—	—
,, - -	25	24	17th Nov.	25th Feb.	—	—
King's - - -	26	19	10th Aug.	20th Nov.	Infantile paralysis.	10th Aug.
,, - - -	27	20	25th Oct.	1st Mar.	—	—
,, - - -	28	22	June	12th Dec.	—	—
,, - - -	29	26	,,	Dec.	—	—
,, - - -	30	33	Oct.	16th Feb.	Bronchitis, lumbago.	16th Feb.
,, - - -	31	34	July	Feb.	—	
Queen's - -	32	18	19th Oct.	16th Feb.	—	—
,, - -	33	18	27th June	3rd Jan.	Colic	3rd Jan.
,, - -	34	24	,,	17th Feb.	—	—
,, - -	35	33	,,	27th Sept.	—	—
,, - -	36	35	11th Oct.	20th Feb.	—	—
Emmanuel - -	37	18	10th July	25th Oct.	—	—
,, - -	38	19	27th July	3rd Oct.	—	—
,, - -	39	21	4th Aug.	26th Feb.	—	—
Corpus Christi -	40	20	7th Nov.	17th Feb.	—	—
,, -	41	26	Oct.	20th Feb.	—	—
,, -	42	29	Aug.	Feb.	—	—
Caius - - -	43	18	30th July	28th Oct.	—	—
,, - - -	44	18	15th Sept.	15th Feb.	—	—
,, - - -	45	19	20th July	7th Nov.	—	—
,, - - -	46	19	July	24th Dec.	—	—
,, - - -	47	21	,,	20th Feb.	—	—
,, - - -	48	27	10th July	10th Oct.	—	—
Magdalene - -	49	28	6th Oct.	10th Feb.	—	—
Sidney Sussex -	50	21	Sept.	Nov.	—	—
,, -	51	22	June	Oct.	—	—
,, -	52	22	4th July	17th Nov.	—	—
,, -	53	26	1st July	13th Oct.	—	—
Sidney (Officers) -	54	25	Aug.	5th Feb.	—	—
,, -	55	28	July	Dec.	—	—
,, -	56	31	24th July	10th Feb.	—	—
Clare - - -	57	18	Oct.	10th Mar.	—	—
,, - - -	58	20	1st July	Oct.	—	—
,, - - -	59	30	30th Oct.	22nd Feb.	Loss of memory.	22nd Feb.
,, - - -	60	34	7th Dec.	17th Feb.	Inflammation of drum of left ear.	7th Dec.
Christ's - -	61	18	14th Oct.	20th Nov.	—	—
,, - -	62	25	26th Oct.	4th Mar.	—	—
Jesus - -	63	31	1st July	2nd Oct.	Jaundice	2nd Oct.
Naval - - -	64	19	17th July	25th Feb.	—	—
,, - - -	65	19	Sept.	Feb.	—	—
,, - - -	66	19	Aug.	9th Nov.	—	—
,, - - -	67	20	,,	4th Feb.	—	—
,, - - -	68	20	Oct.	Jan.	Pneumonia	Oct.
,, - - -	69	20	,,	2nd Feb.	Glands	2nd Feb.
,, - - -	70	20	26th July	25th Oct.	Bronchitis	25th Oct.
,, - - -	71	20	Nov.	23rd Jan.	—	—
,, - - -	72	20	,,	28th Jan	—	—
,, - - -	73	20	June	20th Oct	—	—

College.	No.	Age.	Date of First Attack.	Date of Second Attack.	Complication.	Date of Attack thus complicated.
Naval - - -	74	20	1st Jan.	20th Feb.	—	—
„ - - -	75	20	21st July	Sept.	—	—
„ - - -	76	20	2nd July	23rd Oct.	—	—
„ - - -	77	21	June	Oct.	—	—
„ - - -	78	21	28th Aug.	2nd Feb.	—	—
„ - - -	79	21	22nd July	6th Oct.	Pneumonia and bronchitis.	6th Oct.
„ - - -	80	21	3rd Oct.	6th Feb.	—	—
„ - - -	81	21	10th Oct.	2nd Feb.	—	—
„ - - -	82	21	19th July	4th Jan.	Neuralgia	4th Jan.
„ - - -	83	22	Sept.	Dec.	—	—
„ - - -	84	22	20th July	3rd Feb.	—	—
„ - - -	85	25	Aug.	5th Nov.	Pneumonia	6th Nov.
Selwyn - - -	86	19	1st Aug.	3rd Mar.	„	1st Aug.
„ - - -	87	21	14th July	9th Feb.	—	—
„ - - -	88	24	2nd Aug.	25th Oct.	—	—
„ - - -	89	26	1st July	29th Nov.	—	—
Trinity Hall - -	90	17	7th Dec.	10th Feb.	—	—
„ „ - -	91	18	28th Oct.	18th Feb.	Deafness	18th Feb.
„ „ - -	92	25	1st Aug.	Feb.	—	—
„ „ - -	93	40	11th Oct.	20th Feb.	—	—
Fitzwilliam Hall -	94	24	29th Nov.	8th Feb.	—	—
„ „ -	95	26	21st Oct.	18th Feb.	—	—
„ „ -	96	28	30th July	10th Mar.	—	—
„ „ -	97	30	Oct.	1st Jan.	—	—
Trinity - - -	98	24	28th July	3rd Dec.	-	—

List of Persons who have had Three Attacks of Influenza during 1918–19.

College.	Age.	Date of First Attack.	Date of Second Attack.	Date of Third Attack.	Complication, and Date of Attack thus complicated.
St. Catharine's	24	12th June	10th Oct.	5th Mar.	Bronchitis, 5th Mar.
Queen's - -	21	Aug.	11th Oct.	1st Feb.	Bronchitis, pneumonia, 1st Feb.
„ - -	27	July	Nov.	Feb.	——
Naval - -	19	„	Oct.	1st Jan.	Septic nass. pharynx, 1st Jan.
„ - -	21	„	Nov.	Feb.	——
„ - -	22	„	Aug.	20th Oct.	Catarrh, Aug.; bronchitis, 20th Oct.
Trinity Hall -	22	Aug.	12th Nov.	19th Jan.	Jaundice, 12th Nov.
Fitzwilliam Hall.	34	1st Aug.	Dec.	28th Feb.	Bronchitis, 1st Aug.

List of Persons who have had Four Attacks of Influenza during 1918–19.

College.	Age.	Date of First Attack.	Date of Second Attack.	Date of Third Attack.	Date of Fourth Attack.	Complication, and Date of Attack thus complicated.
King's -	21	July	Sept.	Nov.	Dec.	—
St. John's	21	1st Aug.	19th Sept.	7th Nov.	Jan.	Pleurisy, Jan.

Double Attacks, 1918–1919.

Table showing date of onset of 1st and 2nd Attacks, and whether they were Severe or Mild.

——	June.	July.	August.	September.	October.	November.	December.	February.	Total 2nd attacked.
September - -	28 27	*31* ? 15 ?	——	—	——	——	——	—	3
October - -	? ? ? 20 ? 30	2 22 20 ? *1* *1* 1 *10* 27 30 ? *29* *23* *6* *25* ? *2* ? *13* 25 3 28 *10* ? 10	2 25	*25* *30*	——	——	——	—	18
November - -	5 *24*	*1* 20 ? 29 7 ?	? ? 20 7 10 9 *6* 10 28 20	30 *20*	14 20	——	——	—	11
December - -	7 ? *12* ?	? 6 28 ? 1 ? 24 3 12 1	——	? ?	——	——	——	—	8
January - -	27 3	*19* 31 4 12	——	—	? ? ? 1	30 1 23 28	——	—	7
February - -	? 27 ? 17	17 20 24 14 3 ? ? 25 3 *10* *9* 20 ? ?	*28* 1 ? 1 ? 4 *2* 4 5 ? 15 *26*	? *15* ? 15	? *3* 10 *21* 28 11 ? 30 6 ? 2 ? *19* *11* 2 6 *2* 18 *18* 20 20 22 28 *20* 7 16 16 20	29 7 9 17 ? 8 17 15 25 ?	7 7 7 28 10 17 14 *10*	1 20	41
March - - -	—	30 10	1 3	—	23 *30* *25* *4* 10 1	1 14 ? 13 1 3 7 20	——	—	9
Total 1st attack -	10	33	13	5	20	11	4	1	97

The top figures indicate date of 1st attack, *i.e.*, 28, reads, 1st attack 28th June = mild attack.
„ lower „ „ „ 2nd „ *i.e.*, 27, „ 2nd „ 27th Sept. = „ attack.
Nature of attack indicated as follows:— Severe = figures in italics. Mild = Figures in roman type.

TABLE L.

Number of Persons who have suffered from "Common Colds."

(Total number of Returns received = 1,729.)

College.	Have been Attacked by Influenza.			Have not been Attacked by Influenza.		
	Multiple.	Occasional.	Never.	Multiple.	Occasional.	Never.
1. Christ's	3	38	21	3	21	8
2. Sidney Sussex	11	37	7	2	18	5
3. Jesus	7	22	7	1	4	10
4. Magdalene	8	21	5	8	2	1
5. St. John's	21	44	13	13	14	9
6. Trinity	8	64	24	3	19	12
7. Caius	30	46+1	25	11	9	14
8. Trinity Hall	5	20	4	2	3	1
9. Clare	8	34	15	1	10	6
10. King's	5	44	14	1	6	3
11. St. Catharine's	2	17	3	—	8	5
12. Corpus	8	11	3	5	—	4
13. Selwyn	7	15	5	1	5	2
14. Queen's	8	32	8	4	13	8
15. Pembroke	10	32	11	3	12	9
16. Peterhouse	6	17	4	2	7	3
17. Fitzwilliam Hall	6	15	9	1	6	8
18. Emmanuel	6	27	3	4	15	6
19. Downing	3	32	13	1	16	7
20. Naval Contingent	75	167	52	17	64	20
21. Sidney (Officers)	7	9	7	1	8	3
Total	243	745	253	84	260	144
Percentage of Total	19·6	60·0	20·4	17·4	53·2	29·4

TABLE M.

Table showing Daily Admissions to 1st Eastern General Hospital of Cases of Influenza among Military Cadets, &c., from the various Colleges, &c., during June 1918.

College.	June																												
	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.	28.	29.	30.
Downing	–	–	–	–	3	2	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Peterhouse	–	–	–	–	–	–	2	–	1	8	13	–	3	2	2	1	2	–	1	–	–	–	–	–	–	–	–	3	–	–
Trinity	–	–	–	–	–	–	–	–	1	–	–	–	1	–	1	9	28	6	9	3	8	6	4	11	4	4	2	–	–	–
Jesus	–	–	–	–	–	–	–	–	–	–	–	1	1	2	2	1	12	6	7	–	–	–	–	–	–	–	–	–	–	–
Corpus	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	1	2	–
Pembroke	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	2	1	1	–	1	11	2	1	1	1	3	2	–	1	–
Trinity Hall	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	1	–	–	2	–	–	–	–	–	–	–	–
Ridley Hall	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	2	–	1	3	1	2	6
Queen's	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3	–	–	–	–	–	–	2	–	–	–	–	2	–
Christ's	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	–	–	–	–	–	–	–	–
St. John's	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	1	–	–	–	–	–	–	–	1	–	2
Emmanuel	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	–	–	1	–	–
Magdalene	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–
Not given	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	–	1	–	–	–	–
Total Cadets from Cambridge	–	–	–	–	3	2	2	–	2	9	13	3	5	4	6	14	50	14	18	7	19	10	5	19	5	9	7	7	7	8
O.C.B.'s from Newmarket	–	–	–	–	–	–	–	–	–	2	1	2	3	–	3	–	4	1	4	1	–	–	–	–	–	–	–	–	2	–
R.A.F.	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	–	–	–	1	4	1	5	11	7	8
Army	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	—	2	1	3	1	–	6	–	–	–	4
Total of daily admissions to 1st Eastern General Hospital	–	–	–	–	3	2	2	–	2	11	14	5	8	4	9	14	54	15	22	10	21	11	8	21	9	16	12	18	16	20

28*th June* 1918.—One death occurred at 1st Eastern General Hospital registered as from Influenza plus double lobar pneumonia. (The only death among cadets admitted as above.)

TABLE N.

Influenza Cases—1st Eastern General Hospital, 1919.
(Cadets no longer in Residence.)

——	Jan.		February.																											March.			Total.
	28.	30.	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.	28.	1.	2.	3.	
Naval :—																																		
Date of onset.	–	1	3	9	6	5	3	2	1	–	–	–	–	–	–	–	–	1	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	32
Admitted to hospital.	–	–	1	–	5	9	2	3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	20
Total -	–	1	4	9	11	14	5	5	1	–	–	–	–	–	–	–	–	1	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	52
Military :—																																		
Date of onset.	1	2	1	1	2	1	6	1	1	8	–	4	1	5	1	4	4	–	5	1	–	4	1	4	1	–	–	5	–	2	–	1	–	67
Admitted to hospital.	–	–	–	–	–	–	–	–	–	1	–	1	–	–	–	–	–	–	1	1	–	–	–	1	13	–	2	–	–	–	–	–	–	20
Total -	1	2	1	1	2	1	6	1	1	9	–	5	1	5	1	4	4	–	6	2	–	4	1	5	14	–	2	5	–	2	–	1	–	87
R.A.F. :—																																		
Date of onset.	–	–	–	1	2	–	–	–	–	–	–	–	1	–	–	–	–	–	–	2	1	1	1	2	2	1	–	1	–	1	–	–	–	16
Admitted to hospital.		–	–	–	1	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1	–	–	4
Total -	–	–	–	1	3	–	–	–	–	–	–	–	1	1	–	–	–	–	–	2	1	1	1	2	2	1	–	1	1	1	1	–	–	20
Prisoners of war :—																																		
Date of onset.	–	–	–	–	–	–	–	–	–	1	6	6	1	1	1	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	17
Admitted to hospital.	–	–	–	–	–	–	–	–	–	–	–	–	1	8	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	9
Grand Total -	2	3	5	11	16	15	11	6	2	10	6	11	4	15	2	4	4	1	6	4	1	6	2	7	17	1	2	6	1	3	1	1	–	185

LIST OF MEMBERS OF THE UNIVERSITY WHO DIED FROM INFLUENZA FROM 1ST JANUARY 1918–31ST MARCH 1919.

Date of Death.	Age.	Address.	Complication.
3rd Nov.	23	Undergraduate, Magdalene College -	Septic pneumonia.
5th Nov.	22	Undergraduate, Caius College - -	Pneumonia.
23rd Nov.	25	Law Student, Emmanuel College -	,,
24th Nov.	39	University Lecturer, 7, Harvey Road	Broncho-pneumonia.
1919.			
15th Feb.	34	Librarian, Trinity College - - -	Pneumonia.
19th Feb.	18	Undergraduate of Pembroke College, 5, Botolph Lane.	Broncho-pneumonia.
28th Feb.	29	Fellow of King's College - - -	—

LIST OF DEATHS OF CADETS IN 1ST EASTERN GENERAL HOSPITAL.

28th June.—T. H. C., 28, Cadet, 2nd O.C.B.
22nd October.—G. M. R., 36, Cadet, 2nd O.C.B.
29th October.—G. E. Y., 28, Cadet, 2nd O.C.B.
30th October.—G. W. M., 29, Cadet, 2nd O.C.B.
1st November.—J. H C., 27, Cadet, 2nd O.C.B.
1st November.—J. G. P., 25, Cadet, 2nd O.C.B.
2nd November.—J. E. C., 23, Cadet, 2nd O.C.B.
8th November.—T. C. L., 24, Cadet, 2nd O.C.B.
9th November.—W. F., 25, Cadet, 2nd O.C.B.
11th November.—H. J. S., 32, Cadet, 2nd O.C.B.
11th November.—W. S., 23, Cadet, 2nd O.C.B.

EPIDEMIC OF INFLUENZA, 1918–19.

Report by Dr. Andrew J. Laird.

The following report upon the recent epidemic of influenza in Cambridge is submitted for the information of the Health Committee and the Local Government Board.

On 3rd November 1918 the Board issued a circular letter to medical officers of health desiring that certain information should be collected. This has been done as far as possible, and is given below.

Influenza not being a notifiable disease like diphtheria, the available sources of information are chiefly the reports of school teachers and the copies of death returns received from registrars.

In June it was known that the disease had appeared among the cadets in Queen's* College, and although one or two secondary cases among civilians are known to have occurred then there was no general spread the infection.

The source of introduction in the autumn could not be so clearly traced. It is possible that the two outbreaks were connected by a chain of cases, but there may have been a fresh invasion from abroad by returning soldiers. The first intimation of its recrudescence among civilians was on 23rd September when two cases were reported from St. Paul's Infants', two from Sturton Street Infants', and four from East Road Infants' schools. No further reports were received until 14th October, when 63 children and two teachers were notified from Sturton Street Infants', two from St.

* First in Downing College, *see* Table M.

Philip's Infants', 79 from St. Barnabas, and 144 from East Road Infants' schools. At first there was some doubt as to the precise nature of the illness, but inquiry by the school nurses showed that while a number of children were suffering from "colds" and a few were kept away by their parents as a precaution, the majority of the cases reported were genuine influenza.

The information obtained from the schools up to the date of closure gives some indication of the violence of the outbreak. The total number notified by the head teacher was 296, as follows:—

——	Sept. 23.	October.					
		1.	14.	15.	17.	18.	21.	22.
Cases - -	2	2	81	17	27	29	98	40

It is impossible to give more than a rough estimate of the total number of cases, but from information received from medical practitioners the number of cases attended by them was approximately 6,000. There were in addition, no doubt, a considerable number not medically attended.

On 22nd October it was decided to close the whole of the schools, including the continuation classes, in the borough, and at the same time steps were taken to have the Sunday schools closed also. The two county schools and the Perse Girls' School were also closed, the Perse Boys' School remaining open till 4th November.

A request made to the managers of the cinemas and theatre to exclude children was immediately acquiesced in, and persons known to be arranging public meetings, such as scout gatherings, concerts, &c., were at once communicated with and the meetings abandoned for the time. It is desirable that some acknowledgment should be made here of the ready co-operation accorded by all who were approached in any way in the matter.

The total number of deaths was 120, distributed as follows:—

District.	Week ending										Total.
	Oct.		Nov.					Dec.			
	19.	26.	2.	9.	16.	23.	30.	7.	14.	21.	
Castle End - -	—	2	1	2	—	—	—	—	—	—	5
Centre of town -	1	1	6	4	1	4	—	—	—	—	17
New Town - -	1	3	5	6	2	1	1	1	—	—	20
New Cherryhinton -	—	—	2	4	—	3	—	—	—	—	9
Romsey Town -	1	1	6	6	—	1	1	—	—	—	16
Sturton Town -	1	1	6	6	3	1	—	—	—	—	18
Newmarket Road and district.	—	4	3	1	5	—	—	—	—	—	13
New Chesterton -	—	1	6	6	1	1	1	1	—	1	18
Old Chesterton -	—	—	—	—	—	1	—	—	—	—	1
Newnham - -	—	1	2	—	—	—	—	—	—	—	3
Total - -	4	14	37	35	12	12	3	2	—	1	120

No district entirely escaped, but from the table of deaths it will be seen that the outlying parts of the town, more especially Old Chesterton and Newnham, came off better than the central parts.

This table also shows that the first week of November was the worst, and that the number of deaths began to fall soon after that, the last death occurring on 15th December.

The age and sex distribution of deaths was :—

——	Males.	Females.	Total.
Under 1 year	—	—	—
1 and under 5	1	3	4
5 „ 10	1	7	8
10 „ 15	3	1	4
15 „ 20	5	10	15
20 „ 25	7	10	17
25 „ 35	12	31	43
35 „ 45	5	5	10
45 „ 55	2	6	8
55 and over	5	6	11
Total	41	79	120

The excessive incidence of fatal cases among females is possibly accounted for by the absence of a very large proportion of the male population on military and other duty connected with the war, and also by the unusual number of women engaged in occupations outside their homes.

The occupations of the fatal cases were :—

Males.		Females.	
Army pensioner	1	Brush maker	1
Boot repairer	1	Clerks	2
Builder's machine hand	1	College bedmakers help	1
Carpenter and joiner	1	Domestic service	10
Clerks	4	Dressmaker	1
College students	3	Factory hands	4
College kitchen hand	1	Glove makers	2
Farmer	1	Grocer's assistants	2
General labourers	5	Munition workers	3
Grocer's assistant	1	School teacher	1
Hairdresser	1	Waitress	1
House painter	1		
Laboratory attendant	1		
Miller's store keeper	1		
Munition worker	1		
Motor driver	1		
Newsagent	1		
Oil and colour warehouseman	1		
Pianoforte tuner	1		
Porters	2		
Scientific instrument maker	1		
Theatre attendant	1		
University lecturer	1		
Works officer, Board of Trade	1		

In the remaining cases they were registered as son, (7); daughter, (20); wife, (26); widow, (5).

The disease seems to have been spread entirely by personal contact. There is no evidence whatever of any other method of spread.

I have only heard of one second attack in 1918. The patient was a medical practitioner. The second attack was mild, but is considered by him to have been influenza.

Death resulted in 92·5 per cent. of the fatal cases from some secondary complications. These are shown in the following table:—

——		0–1.	1–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45–55.	55 and over.	Total.
Influenza -	M.	—	—	—	—	—	1	—	—	—	—	1
	F.	—	—	—	1	2	1	1	1	1	1	8
+Pneumonia -	M.	—	1	1	1	3	3	6	—	2	3	20
	F.	—	2	4	—	5	5	17	2	2	—	37
+Septic pneumonia.	M.	—	—	—	1	—	1	—	—	—	—	2
	F.	—	—	—	—	—	—	1	—	—	—	1
+Broncho-pneumonia.	M.	—	—	—	—	2	2	3	3	—	1	11
	F.	—	1	3	—	2	4	7	2	2	1	22
+Asthma and pneumonia.	M.	—	—	—	—	—	—	—	1	—	—	1
	F.	—	—	—	—	—	—	—	—	—	—	—
+Congestion of the lungs.	M.	—	—	—	—	1	—	—	—	—	—	1
	F.	—	—	—	—	—	—	—	—	—	—	—
+Bronchitis -	M.	—	—	—	—	—	—	—	1	—	—	1
	F.	—	—	—	—	—	—	3	—	—	1	4
+Meningitis -	M.	—	—	—	—	—	—	—	—	—	—	—
	F.	—	—	—	—	—	—	1	—	—	—	1
+Cardiac failure.	M.	—	—	—	1	—	—	1	—	—	1	3
	F.	—	—	—	—	—	—	—	—	—	2	2
+Toxæmia -	M.	—	—	—	—	—	—	—	—	—	—	—
	F.	—	—	—	—	—	—	1	—	—	—	1
+Nephritis -	M.	—	—	—	—	—	—	—	—	—	—	—
	F.	—	—	—	—	—	—	—	—	1	—	1
+Diabetes -	M	—	—	—	—	—	—	1	—	—	—	1
	F.	—	—	—	—	—	—	—	—	—	—	—
+Carcinoma of breast, &c.	M.	—	—	—	—	—	—	—	—	—	—	—
	F.	—	—	—	—	—	—	—	—	—	1	1
+Pulmonary tuberculosis.	M.	—	—	—	—	—	—	1	—	—	—	1
	F.	—	—	—	—	—	—	—	—	—	—	—
Total -	M.	—	1	1	3	6	7	12	5	2	5	42
	F.	—	3	7	1	9	10	31	5	6	6	78

The staffs of public institutions suffered severely, with the exception of the Infectious Diseases Hospital and the Chesterton Workhouse, where not a single case occurred. This immunity from attack in the case of the Infectious Diseases Hospital might be attributed to the restricted intercourse between the hospital and the town. It was not due to any immunity acquired by inoculation or previous attacks.

The incubation period was short, usually from two to three days, the exact period in one case, where it is accurately known, being 64 hours.

The only exception I have discovered to this was the occurrence of two cases in a ward at Addenbrooke's Hospital 24 hours after the admission of a pneumonic influenza case to the ward.

The symptoms of onset were not those with which we have become familiar during recent years, viz., headache and pains in the limbs chiefly. During the present outbreak patients complained chiefly of headache, backache, and pains in the abdomen, which at times simulated appendicitis. Epistaxis was fairly common; cough, with pain behind the sternum, and, in severe cases, expectoration of fresh blood were early symptoms. The

temperature rose rapidly to 104° or 105° F., and in a few cases a macular rash, which at first suggested measles, was seen.

With regard to the duration of infectivity, the only evidence procurable on the point is: (1) at Addenbrooke's Hospital, when the pressure on their accommodation was greatest, influenza cases were transferred to the general wards after two days of normal temperature, and no subsequent cases followed; and (2) in two or three instances domestic servants, who had influenza at home, returning to Cambridge, apparently were the source of infection, the only symptom persisting being cough.

The administrative and other action taken included the following measures:—

(1) The local newspapers published precautionary advice, which was considered to be more effective than the distribution of leaflets. This was repeated, and consisted chiefly of extracts from the official leaflet of the Board. The continued publication of the progress of the outbreak throughout the country generally led to widespread knowledge, and resulted in precautions being taken chiefly in the avoidance of public gatherings. This was particularly noticeable in the diminished attendances at cinemas, the theatre, &c.

(2) Places of public entertainment were placed out of bounds by the military authorities, and the proprietors excluded all children during the epidemic.

(3) All public elementary, evening, and county and other schools were closed, as well as all Sunday schools.

(4) Cases were admitted to Addenbrooke's Hospital and the Cambridge Union Infirmary. It is unfortunate that the difficulty experienced during the war of maintaining a satisfactory nursing and domestic staff made it impossible to receive cases in the Borough Hospital for Infectious Diseases.

(5) Nursing at home was undertaken in 137 cases by the District Nursing Association.

(6) Help was given in the home in several instances by the Home Helps who are registered with the Local Authority.

So far as I am aware, no bacteriological examination of material from patients was made, nor was there any attempt at prophylaxis by means of vaccines.

The cases referred to occurred in the civilian population, but the Registrar's returns contained particulars of the deaths in the 1st Eastern General Hospital of 97 soldiers in the period from June 28th to December 14th. The patients admitted to this hospital come not only from Cambridge but are drawn from a very wide area in the Eastern Counties.

Pathological Examinations.—Dr. Aldren Wright, Visiting Physician to Addenbrooke's Hospital, has kindly supplied the information upon which the following note is based.

The lower portions of the lungs examined were consolidated, but in a patchy fashion and not to the extreme degree found usually in ordinary lobar pneumonia. Naked-eye inspection of cut surfaces showed numbers of minute white points, which were the bronchioles filled with secretion. A few cases showed infarction of the lungs, and two had empyema.

Sections made by Dr. Wright for microscopical examination were sent to the University Laboratory, Pembroke Street. These have been examined by Professor Sims Woodhead, to whom I am indebted for the following notes:—

> "The main features of all these lungs—I have not seen anything but the microscopic sections—are those indicating intense inflammation, apparently set up by the toxins of micro-organisms. I say

micro-organisms, because more than one type of micro-organism is present.

"It is sometimes stated that this is not a pneumonia, but I maintain that, as it is an extremely rapid and acute inflammatory process, it must be looked upon as an acute pneumonia of a specially acute and virulent form.

"Evidence of this is as follows :—

"(1) In all cases the vessels are greatly congested.

"(2) There is marked œdema of the lung, many of the alveoli being filled with albuminous fluid in which is a small amount of fibrin.

"(3) There is evidently migration of leucocytes.

"(4) Some, but not very marked, proliferation of epithelial cells in certain of the areas.

"(5) There is some exudation of fibrin, but this is in patches only, and is obscured by the œdematous condition.

"(6) The condition is toxic in a large degree. (Note the condition of the cells where they can be carefully studied.)

"(7) The material of the alveoli is crowded with bacilli, mostly of the Pfeiffer type, with a few pneumococci here and there.

"(8) Œdema and congestion of the pleura and interstitial tissue—Trabeculæ.

"(9) Evidence of toxic action on endothelial cells of the vessels.

"The consolidation which in all cases is the result of the inflammatory process occurs in patches, but is more extensive in this form of pneumonia than in catarrhal pneumonia, where the cells cannot be carried from point to point, as can the œdematous fluid. Moreover, the patient dies long before the catarrhal proliferation of the epithelium of the alveoli can go very far, and before large amounts of the materials derived from breaking down cells can combine with the exuded fluid portion of the blood. Consequently consolidation can never be marked, either in large areas or small lobular patches, as in other forms of pneumonia.

"It may be looked upon as true pneumonia, the patient succumbing during the very early stages of a most acute process.

"The bacilli not only multiply very rapidly but they produce toxin, deriving their nutriment from damaged cells and perhaps from altered fluids, and inducing 'inflammatory' œdema. The action on the cells is intense. In some cases the vessel walls are so damaged that the red corpuscles escape in large numbers, whole areas of the alveoli appearing to be filled with these 'cells.'"

II.

Report on an Inquiry into the recent Epidemic of Influenza in the County Borough of Leicester,

By

M. B. Arnold, M.D.

The facts recorded in this report were obtained during the visits made to Leicester County Borough and Leicester County at various dates from the beginning of November 1918 to March 1919. A note of the sources of information is appended (A).

Leicester County Borough.

From the table of deaths appended (B) it will be seen that Leicester County Borough was affected in the summer wave of influenza in 1918, in the autumn wave in 1918 and in the wave in the early part of 1919, the weeks in each wave with the greatest number of deaths being those ending July 20th, November 2nd, and March 1st.

Definite facts as to the incidence and case mortality were lacking; with the assistance of the Medical Office of Health and his staff a house to house inquiry was undertaken. Five areas of the town were selected which were considered to include all types of housing in Leicester with the exception of the largest houses. The inquiry was made between the 4th and 17th March. The February–March wave was of course still active.

Several streets in each area were visited and information obtained so far as possible at every fifth house. The information asked for was—

(*a*) Number of rooms in house.

(*b*) Number of occupants with age, sex, and occupation of each.

(*c*) Dates of attacks of influenza from May 1918.

(*d*) With regard to each attack it was ascertained how long the patient had been in bed, how long ill, whether the patient had been seen by a doctor, whether there had been any complications, and how the illness had ended.

An attempt was also made to ascertain the previous health of the occupants, but this was found to be impracticable without a great increase in the time expended.

With regard to the figures it may be noticed that there are a few slight discrepancies in totals. This arises from the fact that certain data have occasionally not been noted. In no case are the results affected appreciably. A note on certain factors likely to have affected the constitution of the sample population concerned is appended (C).

The number of houses from which particulars were obtained was 1,061. The number of persons concerning whom particulars were obtained was 4,619.

A table is appended (D) showing the grouping into houses with 1, 2, 3, &c., persons per house.

The number of inhabited tenements in Leicester in July 1917 was 54,147. The population at the Census 1911 was 227,242, and the estimated population 1917 was 217,537. The sample is therefore probably about 2 per cent. of the population. The number of influenzal attacks amongst these persons from May to the date of the inquiry was 1,387; 164 of these were in persons attacked twice, and six in persons attacked three times. The number of persons attacked was therefore 1,302.

The attacks were distributed in time as follows:—

		Incidence. Per cent.
Wave period May, June, July, August - -	295	= 6·3
Intervening period of September - - -	30	
Wave period of October, November, December -	678	= 14·6
Intervening period of January - - - -	14	
Wave period of February and part of March -	370	= 8·0
Total - - - -	1,387	= 30·0

The figures for each month are given in Table (E). The dates for 1918 are, in many cases, probably not accurate, as in the absence of some event fixing the date of illness, I found many persons uncertain as to the month in which they had been ill.

It may be noted that if the incidence on the population of the sample population is taken as approximately correct for Leicester, it gives a striking example of the danger of accepting "impressions." During the height of the October–November wave I was given several estimates of the incidence on the population. None were as low as that found in the sample population, and some were several times higher.

The distribution of the population into age and sex groups with attacks and deaths is given in Table (F) appended.

It will be noted that the males of military age are comparatively few in number. This group tends to be a source of error in house to house inquiries at present. A considerable number of men of army ages have been at home for a fraction of the previous year, and many have returned during the past two months More than 200 such were noted and were not included in the sample population, but I am not confident that some such were not included. Amongst the 200 there were some cases of influenza, and some deaths.

The incidence on certain age groups was as follows:—

Age Group.	Persons.	Cases.	Incidence.
			Per cent.
Under 5 - - -	449	113	25·1
„ 15 - - -	1,089	338	31·0
„ 25 - - -	810	290	35·8
„ 35 - - -	625	243	38·8
„ 45 - - -	546	171	31·3
„ 55 - - -	526	135	25·6
55 and over - - -	574	97	16·8
Total - - -	4,619	1,387	30·0

The incidence in each wave was—

Age Group.	1st Wave.	2nd Wave.	3rd Wave.
	Per cent.	Per cent.	Per cent.
Under 5 - - -	3·1	14·6	7·3
„ 15 - - -	6·0	16·6	6·7
„ 25 - - -	10·1	17·2	6·9
„ 35 - - -	9·1	18·8	10·5
„ 45 - - -	6 5	13·3	10·6
„ 55 - - -	5·7	11·0	7·7
55 and over - - -	1·7	7·3	7·3

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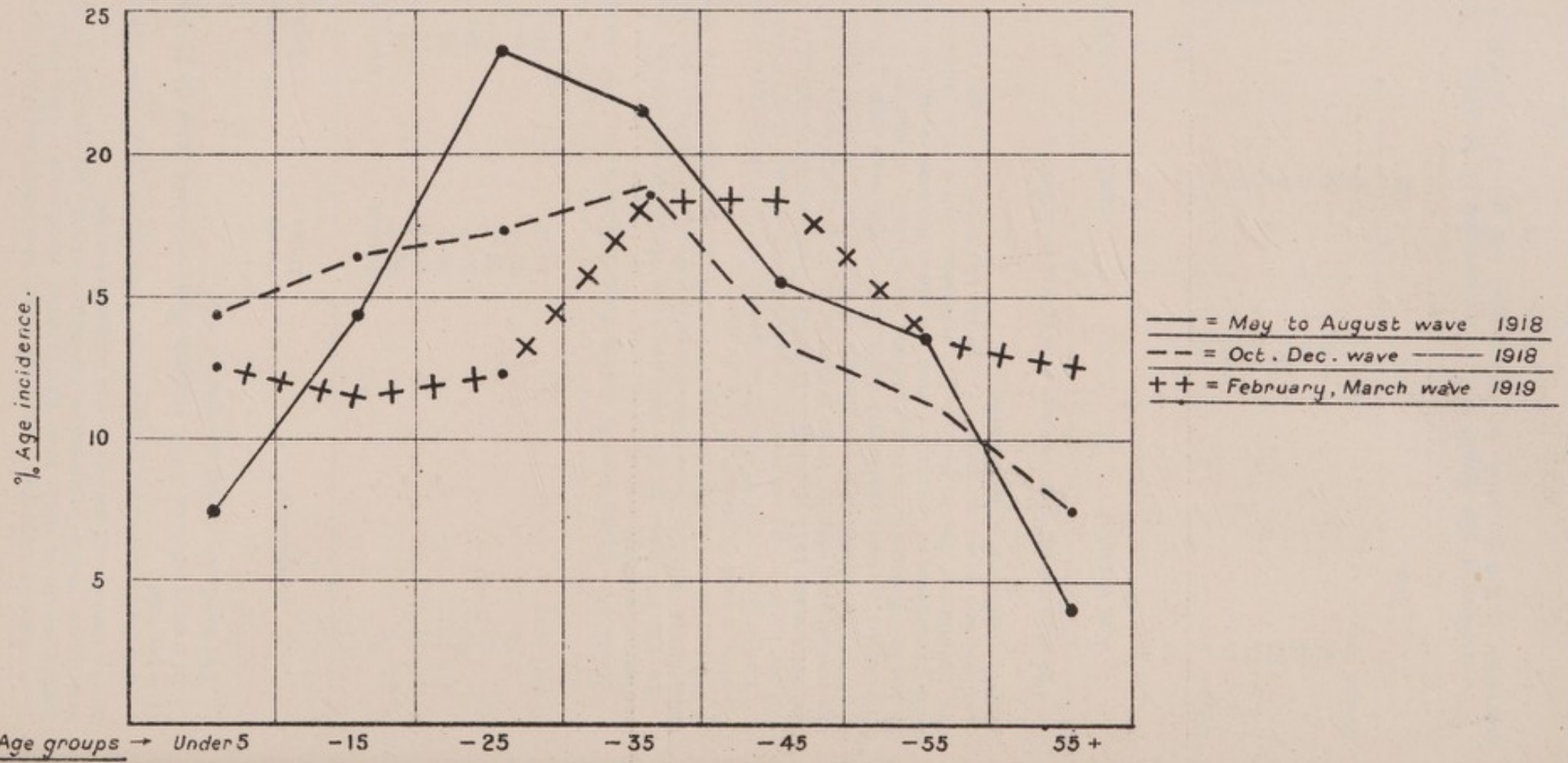
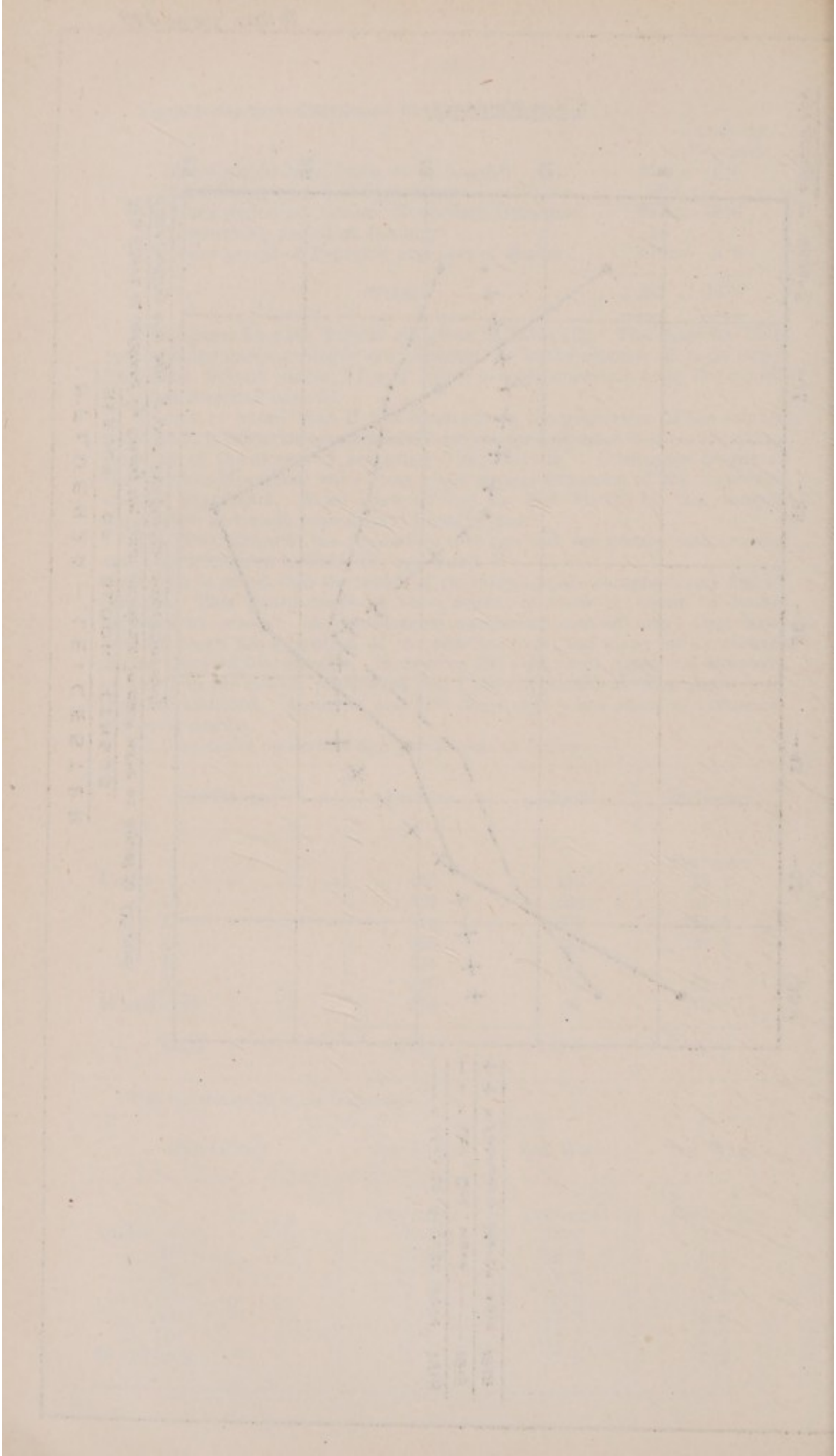
INFLUENZA — LEICESTER

RESULT OF BLOCK CENSUS.

This Chart is obtained by taking the total incidence in each wave as equal to 100 and distributing the age group incidences proportionately.





A chart is apppended in which these incidences have been scaled to be proportionate in each wave to the total incidences. The chart suggests that there was a progressive tendency towards an equality of incidence on the various age groups in the successive waves.

Sex Incidence.—In a population of 1,894 males, there were 523 cases, or 27·6 per cent.

In a population of 2,725 females, there were 864 cases, or 31·7 per cent.

The incidence on the age groups was—

Age Group.	Incidence on Males.	Incidence on Females.
	Per cent.	Per cent.
Under 5	26·3	24·1
„ 15	30·9	31·1
„ 25	30·1	38·1
„ 35	35·9	39·9
„ 45	28·6	53·0
„ 55	25·6	25·7
55 and over	14·3	18·8

Mortality.—There were 32 deaths from influenza in 1,387 cases, or 2·3 per cent mortality.

In the 295 cases in the summer wave there was no death; in the 678 cases in the October–November–December wave, 26 deaths, a case mortality of 3·8 per cent. In the 370 cases of the February–March wave there were 6 deaths, a case mortality of 1·6 per cent.

The occurrence of 32 deaths in a population of 4,619 gives a mortality of 6·9 per 1,000. The estimated population of Leicester in 1917 was 217,537, and the census 1911 population 227,242. Taking the population at 220,000, the anticipated deaths from influenza and its complications would have been, on the ascertained proportion in the house to house inquiry, 1,518. The deaths actually registered from June to the week ending March 15th, as due to influenza or its complications, were 1,140. If the deaths registered from pneumonia in the same period are added the total would be 1,426. As the pneumonia deaths follow the influenza waves, this addition seems justified, and gives an index of how far the house to house inquiry made may be considered a fair sampling.

The case mortality in the age groups was as follows:—

Age Group.	Cases.	Deaths.	Case Mortality.
			Per cent.
Under 5	113	7	6·1
„ 15	338	5	1·4
„ 25	290	7	2·4
„ 35	243	9	3·7
„ 45	171	2	1·1
„ 55	135	2	1·4
55 and over	97	0	0
Total	1,387	32	2·3

Twenty-two of the deaths were in females and 10 in males. The case mortality is 2·5 per cent. in females and 1·9 per cent. in males. The

difference in age constitution of the male and female populations would nullify any argument from the last figures, and the numbers are too small to justify a further analysis.

All deaths occurred in first attacks.

Complications.—The only complications inquired for were pneumonia and bronchitis and pleurisy. 47 or 3·3 per cent. of cases surviving had pneumonia and 86 or 6·2 per cent. had bronchitis. The incidences of pneumonia and bronchitis in cases of influenza surviving were—

——	Pneumonia.	Bronchitis.
	Per cent.	Per cent.
May to August wave - - -	2·0	5·0
October to December wave -	4·5	4·7
February to March wave - -	2·7	10·2

The age groups were affected as follows:—

Age Group.	Bronchitis Cases.	Percentage of Influenza Cases affected.
	Per cent.	Per cent.
Under 5 - - - - -	7	6·1
„ 15 - - - - -	9	2·6
„ 25 - - - - -	10	3·4
„ 35 - - - - -	16	6·5
„ 45 - - - - -	13	7·6
„ 55 - - - -	15	11·1
55 and over - - - -	16	16·4

If all influenzal pneumonia cases are included, *i.e.*, those that ended fatally as well as those that recovered, all the deaths but three have to be added.

The pnemonia rates then are:—

May to August wave - - - - 2 per cent. of cases.
October to August wave - - - 7·9 „ „
February to March wave - - - 4·3 „ „

The age grouping of all cases of pneumonia is:—

Age Group.	No. of Cases.	Percentage of Influenza Cases with Pneumonia.	
		All Cases.	Oct.–Dec. Wave.
		Per Cent.	Per Cent.
Under 5 - - -	12	10·6	12·1
„ 15 - - -	21	6·2	7·7
„ 25 - - -	17	5·8	10·0
„ 35 - - -	13	5·3	10·1
„ 45 - - -	7	4·0	4·1
„ 55 - - -	5	3·7	3·4
55 and over - - -	1	1·0	2·3
Total - - -	76	5·4	—

Immunity.—Of a population of 4,619, 295 or 6·3 per cent. were attacked in the May to August wave. There were no deaths in this wave in the houses visited.

In considering immunity, groups of cases in September and January have been omitted in order to obtain definite wave periods.

Of the population of 4,619 of May 1918, there were at the beginning of October, 295 who had been attacked in the summer wave and 30 who had been attacked in September, leaving a population of 4,294 persons. Amongst these there were 662 attacks during October, November and December, or 15·4 per cent. Amongst the 295 persons attacked in the summer wave there were 16 cases, or 5·4 per cent.

The population not previously attacked at the beginning of February was 4,619 less those attacked in the two previous waves, *i.e.*, 295 and 662, and those attacked in September and January (30 and 14*) 3,624. Of these 309 were attacked, or 8·5 per cent. There were also 295—16 persons previously attacked only in the May to August wave, and of these 22 were attacked, or 7·8 per cent.

There were also 662 persons attacked in the October–December wave. Of these, 26 had died, leaving 636, and six of these had been attacked again in the interwave period of January, leaving 630, of whom 37 were attacked, or 5·8 per cent.

There were 16 persons who had been attacked previously, both in the summer and autumn waves, and of these, two were again attacked.

It is, however, clear that the age grouping of those previously attacked is very different from that of the population not previously attacked, so that the attacked and unattacked cannot be fairly compared in subsequent waves.

An analysis was made of the age groups of females, 20–40 years, with the following result:—

Of 929 women of these age groups, 86, or 9·2 per cent., were affected in the May–August wave.

At the beginning of the October wave the number of women at these ages not previously attacked was reduced to 838.

Among these, 162 attacks occurred in the October–December wave, of which three were in persons previously attacked in the May–August wave. There were, therefore, 159 attacks in 838 persons not previously affected, or 18·9 per cent., and three in 86 persons previously attacked, or 3·4 per cent.

At the beginning of February 1919, there were 838 persons, less 159 and less two (January cases), or 677 persons not previously attacked. Ninety-one cases occurred in the February–March wave, of which 18 were in persons previously attacked, leaving 73 attacks in 677 persons not previously attacked, or 10·7 per cent. Of the 83 persons previously attacked only in the May–August wave, seven were affected, or 8·4 per cent. Of the 159 persons previously attacked only in the October–December wave, eight had died, leaving 151 persons, and amongst these were 11 attacks, or 7·2 per cent. Amongst the three persons attacked both in the May–August and October–December waves there was no attack in the February–March wave.

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* Of these 14, six were second attacks in persons already attacked in October, and therefore only eight are subtracted here.

No death from influenza was recorded in a second or third attack in this census. Deaths in this population from other causes than influenza were recorded so rarely as not to affect the percentages.

SUMMARY OF FIGURES RELATING TO IMMUNITY.

———	All Ages.	Age Group 20–40, Females.
	Per Cent.	Per Cent.
Incidences in May-August wave - - -	6·3	9·2
Incidences in October–December wave on those—		
1. Not previously attacked - - -	15·4	18·9
2. Previously attacked in May–August wave.	5·4	3·4
Incidences in February–March wave on those—		
1. Not previously attacked - - -	8·5	10·7
2. Previously attacked only in May–August wave.	7·8	8·4
3. Previously attacked only in October–December wave.	5·8	7·2
4. Previously attacked in both May–August and October–December waves.	2 in 16	0 in 3

Ward Distribution.—There are 16 wards in Leicester. If these are grouped from the influenzal death rates into order of severity of incidence in the October–December wave, and again for the February–March wave, it does not appear that the wards most heavily hit in October–December suffered any less than the others in February–March.

Occupation.—The staple trades at Leicester are hosiery and boot and shoe making, Visits to two factories, which were said to be typical, showed the following possible means of transference of particles from one worker to another:—

(1) Roller towels in both factories.
(2) Enamel drinking cups in one factory.
(3) Washing of teacups together in one factory.

I was informed that there are canteens in some factories.

(4) Passing of goods in various stages of manufacture from one worker to another. There may be an interval of a day or more between the completion of one process and the beginning of the next, but where there is an urgent demand for one type of finished article, goods may go immediately from one worker to the next.

The possibility of droplet infection through the air varies considerably in the various processes. Of those I saw, machining in the shoe factory seemed to offer the greatest opportunity. Workers sit on each side of a long bench and face one another. The distance across the bench is about 5 feet and the lateral distance between the workers about 3 feet.

No useful figures were available at the factories I visited.

The effect of occupation is difficult to determine as comparable age groups of house-workers and workers away from homes are not large enough.

Of women of the age group 15 to 55, 56 per cent. went out to work. Amongst those going out to work there were about 34 per cent. of attacks and amongst those not going out to work about 30·5 per cent. The difference might easily be accounted for by the fact that the ages in the out-worker group tend to be lower than in the house-worker group.

One employer told me that he had two factories on similar work. One in Leicester employing about 150 had not had more than 20 cases and never more six away at any one time. The other factory, a little way from Leicester, had had to close for a time owing to the numbers away with influenza. However infection may be spread it does not seem that it is by a method distinctively active in the Leicester factories.

Housing.—597 houses or 55·8 per cent. were invaded. 125 were invaded twice and 15 three times. The percentages of houses invaded in each wave were approximately 15 per cent. in the May–August wave; 30 per cent. in the October–December wave, and 22 per cent. in the February–March wave.

The population was considered in two groups according to density of distribution of persons in individual houses. 2,122 persons lived in houses with one or more persons per room, and amongst these there were 655 cases, or 30·8 per cent. 2,480 persons lived in houses with less than one person per room, and amongst these there were 725 cases, or 29·2 per cent.

The presence of returned soldiers affects the precision of this calculation, but a second count, in which some returned soldiers were included, gave practically the same result.

Of 71 houses with eight or more inhabitants, 47 were invaded, and in 12 of these there was only one case. This is in striking contradiction to a widespread belief that the introduction of the disease into a household is almost always followed by a general infection of the household.

In a population of 627 persons living eight or more in a house there were 27·5 per cent. attacks, amongst 407 persons living eight or more per house in houses which were invaded there were 42·9 per cent. attacks.

In a population of 318 persons living two or less in a house there were 26·7 per cent. attacks.

It should be remembered that the attacks are those occurring from May 1918 to March 1919, and some are second attacks in persons already once attacked. Also it may be noted that the age groups in houses with eight or more per house and in houses with two or less per house differ from the age group constitution of the total population, there being a tendency for the ages to be lower in houses with many persons and higher in houses with few.

After making all allowances, however, it is difficult not to believe that the importance of aerial droplet connection from person to person has not been exaggerated.

Medical Attendance.—Of 931 cases, 609, or about 65 per cent., were said to have been seen by a doctor. The distinction is not so definite in practice as might be expected.

Some persons saw a doctor only when they had recovered sufficiently to go to his consulting-rooms. Others say they obtained medicine for secondary cases in the house, though the doctor saw only the first case. Many who did not call in a doctor refrained because of notices in the press explaining that medical men were working at great pressure and should be consulted only when the need was urgent. The proportion, however, of one-third of the cases not seen by a medical man is probably not excessive in epidemic

times of influenza; in fact, I assume that the proportion not so seen is probably rather greater, as many of the informants seemed to suspect an accusation in the question on this point, especially when the patients concerned were children.

I was told by a physician of a remarkable outbreak in the summer in a small military hospital in Leicestershire which he attended. Its total population was about 60. He visited it one afternoon and all were well. He was informed that night that there were 20 cases of influenza, and next morning everyone, excepting one member of the staff, had influenza. The source of infection was not traced.

There is with regard to influenza an undiscovered factor which occasions among certain groups of persons an infectivity of a remarkable quality. This factor is by no means always, or even usually, perceptibly in action. In many homes only one case occurs.

It seems either that—

(*a*) persons suffering from influenza are markedly different from each other in their power of giving infection, or that

(*b*) they vary markedly and rapidly in their infective power at stages of their illness, or else that

(*c*) there is some link or perhaps a special degree of receptivity required for the consummation of infection which is present somewhat erratically, but which may be remarkably effective when present.

Conclusions.

1. The age groups 15 to 35 appear to have had both a greater incidence and, excepting those under 5, greater case mortality. The incidence was greater on females than on males.

2. Groupings into those occupied at home and those occupied away from home and groupings according to density of home populations did not show significant differences of influenza incidence.

3. Considerable immunity to the October–December wave was shown by those who had already suffered in the May–August wave.
   In the February–March wave only slight immunity of those who had previously suffered is suggested by the figures.

4. The total number of persons in Leicester who had attacks of influenza during the period covered by the inquiry is probably considerably less than was generally believed.

8th May 1919.

Addendum A.

I am much indebted to the following persons, amongst others, for information and assistance in this inquiry—

Dr. C. Killick Millard, Medical Officer of Health for the County Borough of Leicester.

Dr. T. Robinson, County Medical Officer of Health, Leicestershire.

Dr. J E. O'Connor, Medical Officer of Health for the Leicestershire combined sanitary Districts.
The Medical Officers of Health for Coalville Urban District and Loughboro' Urban District.
Col. Astley Clarke, M.D., Senior Physician to the Royal Infirmary, Leicester.
Dr. E. C. Hadley, Medical Superintendent of the Poor Law Infirmary, Leicester.
The Secretary to the Leicester Medical Society.
The Superintendent of the Leicester County Borough Asylum.
The Superintendent of the Leicester District Nursing Association.
Dr. W. Mackarell, Pathologist to the Royal Infirmary, Leicester.
And to members of the staffs of the Public Health Departments of the County Borough of Leicester and of the County of Leicester.

Addendum B.

Influenza and Pneumonia Deaths from June 1918 to March 22nd, 1919, Leicester.

(No Influenza Deaths in weeks omitted.)

Week ending		Influenza.	Pneumonia.
June	15	1	4
July	6	5	7
,,	13	22	11
,,	20	27	6
,,	27	7	5
August	3	1	3
,,	10	2	5
,,	17	3	2
,,	24	1	2
September	7	1	1
,,	28	1	1
October	12	5	11
,,	19	54	19
,,	26	194	24
November	2	262	31
,,	9	147	24
,,	16	65	13
,,	23	20	10
,,	30	18	4
December	7	18	7
,,	14	9	5
,,	21	10	2
,,	28	3	3
January	4	2	0
,,	11	3	1
,,	18	1	2
,,	25	0	4
February	1	1	7
,,	8	2	8
,,	15	12	8
,,	22	60	20
March	1	91	14
,,	8	50	24
,,	15	41	7
,,	22	19	6

Addendum C.

On possible Causes of Deviation from the Normal of the Sample Population.

1. The military age groups of males resident in Leicester are much depleted.

The men in these age groups who have been on military service have returned to their homes at various times throughout the past 12 months and in considerable numbers since the armistice. It was frequently difficult to decide whether a returned soldier should be included, especially in relation to the density of the population in individual houses.

2. Houses which were found closed at the time of visit were ignored in this census. Houses in which there are young children are rarely found closed and this probably tended to affect the age grouping of the population recorded.

3. Families which had been severely affected in the epidemic, *i.e.*, in which there had been more than one death amongst wage earners were more likely to have left an industrial centre such as Leicester. In my own inquiries I was twice told that this had happened.

Addendum D.

Number of houses with—

One occupant	27
Two occupants	155
Three „	220
Four „	217
Five „	170
Six „	130
Seven „	71
Eight „	39
Nine „	12
Ten „	14
Eleven „	5
Twelve „	1
	1,061

Note.—This grouping relates not to the actual number of occupants at the time of visit, but to the inhabitants who had been resident so far as could be ascertained since the beginning of the influenza epidemic.

It is only approximate.

Addendum E.

Monthly incidence of influenza—

May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March (part of).
17	61	188	29	30	202	447	29	14	303	67

Addendum F.

Age Groups.	Males.	Cases of Influenza.	Females.	Cases of Influenza.	Total Males and Females.	Total Cases.	Deaths.	Population re-grouped.	Cases.	Incidence.
										Per cent.
– 2 - -	67	8	72	12	139	20	2	449	113	25·1
– 5 - -	142	47	168	46	310	93	5			
–10 - -	300	97	253	81	553	178	4	1,089	338	31·0
–15 - -	272	80	264	80	536	160	1			
–20 - -	184	65	288	123	472	187	4	810	290	35·8
–25 - -	55	7	283	96	338	103	3			
–30 - -	66	17	239	87	305	104	3	625	243	38·8
–35 - -	98	42	222	97	320	139	6			
–40 - -	93	25	185	66	278	91	1	546	171	31·3
–45 - -	120	36	148	44	268	80	1			
–50 - -	130	39	160	46	290	85	2	526	135	25·6
–55 - -	116	24	120	26	236	50	0			
–60 - -	74	12	118	22	192	34	0	574	97	16·8
60+ -	177	24	205	39	382	63	0			
Totals	1,894	523	2,725	865	4,619	1,387	32	—	—	30·0

III.

Analysis of the results of a Block Census undertaken in Manchester in December 1918,

By

T. Carnwath, D.S.O., M.B.

The method of enquiry is described by Dr. Niven in Appendix IV.

The results are briefly as follows:—The inquiry covered 1,018 houses with a population of 4,666. 500 households or almost exactly one half were invaded in either one or other epidemic: the total number of cases was 1,100 or 25 per cent. of the population. 14·8 per cent. of the population were attacked in summer and 10·3 per cent. in winter. There is record of only two deaths among the summer cases, but 14 deaths occurred in the autumn; the case mortality in the autumn epidemic was therefore 2·9 per cent. Of these 14 only one had the disease in the summer. One of the most noteworthy facts elicited by inquiry into deaths from the disease in Manchester is the small proportion of fatal cases which suffered from previous attack.

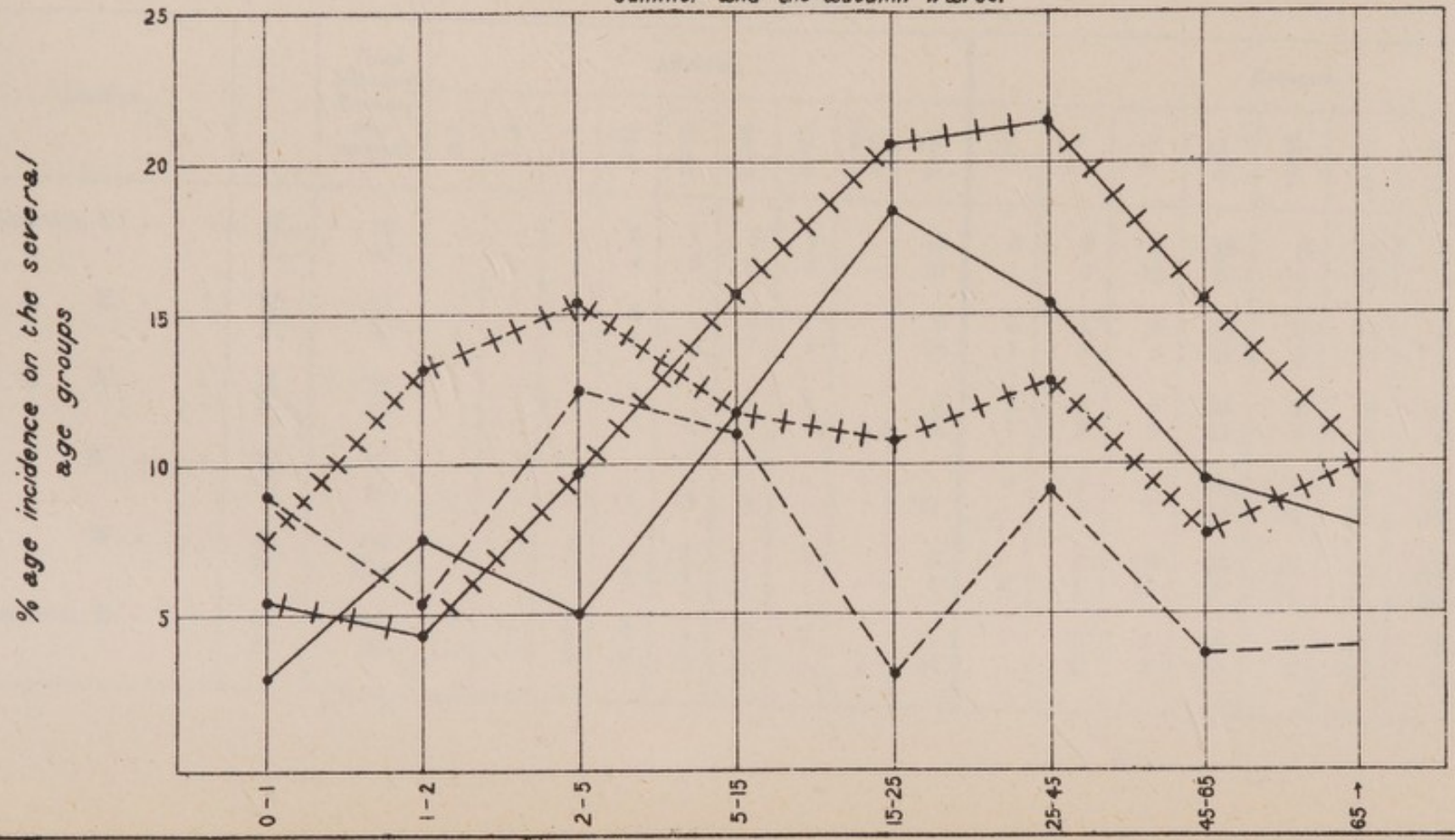
The autumn epidemic affected 268 households with a population of 1,358. 480 persons were attacked including 73 who had had the disease in the summer. 102 households suffered in both epidemics. The history of these 102 households throws some light on the question of immunity. They comprised a population of 553 persons—of whom 199 had suffered in summer and 354 had escaped. In the autumn epidemic 73 (or 37 per cent.) of the presumably "protected" persons succumbed again whereas only 115 (or 32·5 per cent.) of the "unprotected" suffered. Of the former, however, only one died while three of the latter terminated fatally.

The age period most heavily affected in the summer epidemic was from 15 to 45 years, whereas in the autumn there is evident a considerable shifting towards the extremes of life and particularly towards the younger years. At first sight this would seem to argue a certain degree of immunity following a previous attack in summer, but this conclusion is not borne out by closer analysis, as the autumn diminution in the middle-age period affected equally those who suffered in summer and those who escaped.

INFLUENZA—MANCHESTER

RESULT OF BLOCK CENSUS

Curves showing percentage incidence of the disease on the several periods during the summer and the autumn waves.



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H. & C. GRAHAM LTD LITHOGR LONDON, S.E.

Result of Block Census in Manchester (Summer and Autumn Epidemics).

Age and Sex Incidence.

Summer.

District.		Total Number Persons concerned.	Affected.									Escaped.								
			0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.
Ancoats, C. - -	M.	82	–	–	–	5	1	8	1	–	15	5	3	6	26	3	15	9	1	67
	F.	89	–	–	1	4	6	6	4	–	21	1	3	12	15	10	20	4	3	68
„ E. - -	M.	51	–	–	–	2	–	2	1	–	5	3	1	4	10	3	20	4	1	46
	F.	83	1	–	2	4	–	7	4	–	18	1	3	8	19	9	20	4	1	65
„ N. - -	M.	38	–	–	1	2	–	2	1	–	6	2	2	3	14	3	6	2	–	32
	F.	58	–	–	–	–	3	9	3	–	15	1	3	2	14	5	9	9	–	43
„ S. - -	M.	78	–	–	–	–	–	–	–	–	–	8	2	6	28	8	16	8	2	78
	F.	95	–	–	–	4	3	4	–	–	11	–	2	7	19	15	27	12	2	84
„ W. - -	M.	90	–	–	1	–	1	–	1	–	3	–	3	10	24	14	13	17	6	87
	F.	134	–	–	–	2	1	1	–	–	4	6	5	10	28	26	37	13	5	130
Ardwick, E. - -	M.	65	–	–	1	4	1	3	3	–	12	1	2	6	18	3	18	3	2	53
	F.	70	–	–	2	6	4	6	–	1	19	–	2	9	10	3	19	6	2	51

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Result of Block Census in Manchester (Summer and Autumn Epidemics)—continued.

Age and Sex Incidence.

Summer—continued.

District.		Total Number Persons concerned.	Affected.									Escaped.								
			0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.
Ardwick, N. - -	M.	59	–	–	–	2	1	2	–	–	5	1	1	7	24	4	12	5	–	54
	F.	74	–	1	1	2	3	6	1	1	15	3	1	12	11	9	19	4	–	59
,, S. - -	M.	90	–	–	1	1	–	2	–	–	4	3	3	9	25	10	20	14	2	86
	F.	91	–	–	–	1	1	2	–	–	4	–	2	3	20	16	24	18	4	87
Beswick - -	M.	74	–	–	–	–	1	4	1	1	7	2	1	6	30	9	13	6	–	67
	F.	97	–	–	1	6	2	9	1	–	19	2	3	8	19	10	27	9	–	78
Blackley - -	M.	51	–	1	1	1	1	1	2	–	7	–	–	3	12	1	15	9	4	44
	F.	71	–	–	–	1	7	4	4	1	17	–	1	3	13	4	19	9	5	54
Bradford - -	M.	71	1	–	–	1	1	2	1	–	6	5	1	4	26	6	15	8	–	65
	F.	101	–	–	–	4	1	5	–	–	10	5	3	14	25	10	27	7	–	91
,, No. 1 -	M.	71	–	–	–	1	1	2	1	1	6	–	3	3	22	7	15	12	3	65
	F,	75	–	–	1	1	–	2	–	1	5	1	2	3	10	17	19	15	3	70
Chorlton-on-Medlock, E.	M.	87	–	–	–	3	2	1	1	–	7	1	2	5	27	8	15	16	6	80
	F.	118	–	–	–	1	2	2	4	–	9	–	2	6	20	22	34	17	8	109
Chorlton-on-Medlock, S.	M.	66	–	–	–	3	2	1	–	–	6	2	1	7	17	2	18	12	1	60
	F.	76	–	–	2	1	1	7	1	–	12	2	2	3	8	9	26	13	1	64

Gorton - - -	M.	74	–	1	–	2	4	3	3	–	13	1	–	4	18	7	21	8	2	61
	F.	108	–	–	–	4	5	3	5	–	17	2	–	8	28	14	28	6	5	91
„ S. - -	M.	88	–	–	–	1	–	–	1	–	2	5	5	9	28	8	27	4	–	86
	F.	85	–	–	1	1	–	3	–	–	5	2	5	12	22	10	25	3	1	80
„ W. - -	M.	75	–	1	–	4	2	4	3	–	14	–	1	5	15	5	20	11	4	61
	F.	95	–	–	–	1	5	5	3	–	14	–	4	5	11	17	28	15	1	81
Harpurhey - -	M.	56	–	–	–	3	1	6	–	–	10	2	1	6	9	4	18	6	–	46
	F.	74	–	–	–	6	–	8	1	–	15	–	3	6	10	8	28	2	2	59
Hulme, C. - -	M.	81	–	–	–	1	1	4	3	–	9	2	3	10	21	9	11	15	1	72
	F.	110	–	–	–	1	3	6	–	–	10	3	2	10	22	23	22	15	3	100
„ E. - -	M.	34	–	–	–	2	2	2	–	–	6	–	2	3	12	4	2	5	–	28
	F.	59	–	–	–	4	3	3	2	1	13	1	1	4	12	9	13	4	2	46
„ N. - -	M.	78	–	–	–	1	–	–	–	–	1	1	–	4	22	18	16	13	3	77
	F.	69	–	–	1	2	1	1	1	2	8	–	–	1	12	13	23	9	3	61
„ S. - -	M.	68	–	–	–	3	1	6	1	–	11	3	–	9	19	4	12	9	1	57
	F.	82	–	–	2	2	5	6	1	–	16	3	1	4	13	9	22	13	1	66
„ W. - -	M.	79	–	–	–	2	4	1	1	–	8	–	–	10	28	5	14	13	1	71
	F.	90	–	–	–	2	6	6	2	–	16	1	1	5	26	10	18	9	4	74
London Road -	M.	85	1	1	2	4	–	–	1	–	9	3	6	11	23	8	17	7	1	76
	F.	90	1	1	2	4	6	3	1	–	18	3	3	7	18	10	23	6	2	72
Newton - -	M.	63	–	–	1	5	3	7	–	–	16	1	–	6	7	6	14	11	2	47
	F.	92	1	–	2	7	4	15	1	–	30	3	1	7	8	7	21	12	3	62

Result of Block Census in Manchester (Summer and Autumn Epidemics)—continued.

Age and Sex Incidence.

Summer—continued.

District.		Total Number Persons concerned.	Affected.									Escaped.								
			0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.
Newton, E. - -	M.	31	–	–	–	3	4	–	1	–	8	2	–	2	4	3	7	5	–	23
	F.	33	–	–	–	6	2	3	2	–	13	–	–	4	5	2	5	4	–	20
St. George's, C. -	M.	91	–	–	1	11	3	3	–	–	18	2	3	9	27	6	15	8	3	73
	F.	118	–	–	1	11	6	13	1	1	33	5	4	8	21	14	23	8	2	85
„ E. -	M.	66	–	–	–	–	–	5	–	–	5	2	1	8	21	7	13	9	–	61
	F.	83	–	–	1	–	1	4	2	–	8	–	2	4	27	11	19	8	4	75
„ N. -	M.	56	–	–	1	5	4	4	1	–	15	3	3	6	14	3	7	5	–	41
	F.	79	–	1	2	5	4	20	2	–	34	3	2	7	16	5	7	5	–	45
St. George's, Monsall	M.	68	–	–	–	5	–	5	–	2	12	3	–	4	16	7	16	9	1	56
	F.	101	–	–	–	2	3	10	3	1	19	1	3	9	22	12	22	9	4	82
Totals - -	M.	2,066	2	4	10	77	41	80	28	4	246	63	50	185	587	185	441	263	46	1,820
	F.	2,600	3	3	22	95	88	179	49	9	448	49	66	201	504	339	654	268	71	2,152
Grand Totals -		4,666	5	7	3 2	172	129	259	77	13	694	112	116	386	1,091	524	1,095	531	117	3,972

Ancoats, C. - -	M.	82	–	1	2	6	1	3	–	–	13	5	2	4	25	3	20	10	–	69
	F.	89	–	1	1	3	2	6	1	1	15	1	2	12	16	14	20	7	2	74
„ E. - -	M.	51	–	–	–	1	–	3	–	–	4	3	1	4	11	3	19	5	1	47
	F.	83	–	–	2	4	1	2	–	–	9	2	3	8	19	8	25	8	1	74
„ N. - -	M.	38	–	–	–	–	–	1	–	–	1	2	2	4	16	3	7	3	–	37
	F.	58	–	–	–	–	–	1	1	–	2	1	3	2	14	8	17	11	–	56
„ S. - -	M.	78	–	1	1	4	–	2	–	–	8	8	1	5	24	8	14	8	2	70
	F.	95	–	–	1	–	2	1	1	–	5	–	2	6	23	16	30	11	2	90
„ W.- -	M.	90	–	–	–	1	–	–	3	–	4	–	3	11	23	15	13	15	6	86
	F.	134	–	–	–	–	2	–	–	–	2	6	5	10	30	25	38	13	5	132
Ardwick, E. - -	M.	65	–	–	–	–	–	1	–	–	1	1	2	7	22	4	20	6	2	64
	F.	70	–	–	–	1	–	1	–	–	2	–	2	11	15	7	24	6	3	68
„ N. - -	M.	59	–	–	2	4	–	2	–	–	8	1	1	5	22	5	12	5	–	51
	F.	74	–	–	1	1	–	5	–	–	7	3	2	12	12	12	20	5	1	67
„ S. - -	M.	90	–	–	–	2	–	–	–	–	2	3	3	10	24	10	22	14	2	88
	F.	91	–	–	1	2	1	–	–	1	5	–	2	2	19	16	26	18	3	86
Beswick - -	M.	74	–	–	1	1	–	–	–	1	3	2	1	5	29	10	17	7	–	71
	F.	97	–	2	1	5	1	5	1	–	15	2	1	8	20	11	31	9	–	82
Blackley - -	M.	51	–	–	1	3	–	2	–	–	6	–	1	3	10	2	14	11	4	45
	F.	71	–	–	–	2	2	4	2	1	11	–	1	3	12	9	19	11	5	60

Result of Block Census in Manchester (Summer and Autumn Epidemics)—continued.

Age and Sex Incidence.

Autumn—continued.

District.		Total Number Persons concerned.	Affected.									Escaped.								
			0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65	65 and over.	Total.
Bradford - -	M.	71	1	–	–	3	–	2	–	–	6	5	1	4	24	7	15	9	–	65
	F.	101	–	1	5	5	3	5	–	–	19	5	2	9	24	8	27	7	–	82
„ No. 1 -	M.	71	–	–	–	–	–	1	–	1	2	–	3	3	23	8	16	13	3	69
	F.	75	–	–	1	–	2	1	3	1	8	1	2	3	11	15	20	12	3	67
Chorlton-on-Medlock, E.	M.	87	–	–	2	4	1	2	–	–	9	1	2	3	26	9	14	17	6	78
	F.	118	–	1	2	4	5	4	4	–	20	–	1	4	17	19	32	17	8	98
Chorlton-on-Medlock, S.	M.	66	1	–	1	2	–	2	–	–	6	1	1	6	18	4	17	12	1	60
	F.	76	–	–	–	3	1	5	1	–	10	2	2	5	6	9	28	13	1	66
Gorton - - -	M.	74	–	–	–	4	1	3	–	–	8	1	1	4	16	–	21	11	2	66
	F.	108	1	–	1	5	3	7	2	1	20	1	–	7	27	16	24	9	4	88
„ S. - -	M.	88	1	–	4	5	–	4	1	–	15	4	5	5	24	8	23	4	–	73
	F.	85	1	1	5	4	–	4	1	–	16	1	4	8	19	10	24	2	1	69
„ W. - -	M.	75	–	–	3	2	–	2	–	–	7	–	2	2	17	7	22	14	4	68
	F.	95	–	1	–	1	3	5	2	1	13	–	3	5	11	19	27	16	–	81
Harpurhey - -	M.	56	–	–	–	2	–	1	–	–	3	2	1	6	10	5	23	6	–	53
	F.	74	–	–	1	2	1	9	–	–	13	–	3	5	14	7	27	3	2	61

Hulme, C. - -	M.	81	–	–	1	2	–	1	–	–	4	2	3	9	20	10	14	18	1	77
	F.	110	–	1	–	4	1	2	–	1	9	3	1	10	19	25	26	15	2	101
„ E. - -	M.	34	–	–	–	–	–	–	1	–	1	–	2	3	14	6	4	4	–	33
	F.	59	–	–	1	–	1	1	–	–	3	1	1	3	16	11	15	6	3	56
„ N. - -	M.	78	–	–	–	5	–	1	1	–	7	1	–	4	18	18	15	12	3	71
	F.	69	–	–	–	1	1	5	–	–	7	–	–	2	13	13	19	10	5	62
„ S. - -	M.	68	–	–	–	2	1	3	1	–	7	3	–	9	20	4	15	9	1	61
	F.	82	–	–	–	1	1	1	–	–	3	3	1	6	14	13	27	14	1	79
„ W. - -	M.	79	–	–	–	3	2	1	1	–	7	–	–	10	27	7	14	13	1	72
	F.	90	–	–	–	7	3	6	3	–	19	1	1	5	21	13	18	8	4	71
London Road -	M.	85	2	–	3	4	–	1	–	–	10	2	7	10	23	8	16	8	1	75
	F.	90	1	–	–	2	3	3	–	–	9	3	4	9	20	13	23	7	2	81
Newton - -	M.	63	1	–	1	1	–	4	1	–	8	–	–	6	11	9	17	10	2	55
	F.	92	–	–	3	3	1	6	1	–	14	4	1	6	12	10	30	12	3	78
„ E. - -	M.	31	–	–	–	1	–	–	–	–	1	2	–	2	5	7	7	6	–	29
	F.	33	–	–	–	–	–	–	–	–	–	–	–	4	11	4	8	6	–	33
St George's, C. -	M.	91	–	–	–	5	–	4	–	–	9	2	3	10	33	9	14	8	3	82
	F.	118	1	–	2	1	1	6	–	1	12	4	4	7	31	19	30	9	2	106
„ E. -	M.	66	–	–	2	1	–	–	1	–	4	2	1	6	20	7	18	8	–	62
	F.	83	–	–	–	3	3	2	–	–	8	0	2	5	24	9	21	10	4	75
„ N. -	M.	56	–	1	–	–	–	–	1	–	2	3	2	7	19	7	11	5	–	54
	F.	79	–	–	2	1	1	3	1	–	8	3	3	7	20	8	24	6	–	71

Result of Block Census in Manchester (Summer and Autumn Epidemics)—continued.

Age and Sex Incidence.

Autumn—continued.

District.		Total Number Persons concerned.	Affected.									Escaped.								
			0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.	0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.
St. George's, Monsall	M.	68	–	–	–	5	2	2	–	–	9	3	–	4	16	5	19	9	3	59
	F.	101	–	1	4	5	2	8	1	–	21	1	2	5	19	13	24	11	5	80
Totals - -	M.	2,066	6	3	24	73	8	48	11	2	175	59	51	171	590	218	473	280	48	1,890
	F.	2,600	4	9	34	70	47	108	25	8	305	48	60	189	529	380	724	292	72	2,294
Grand Total -	—	4,666	10	12	58	143	55	156	36	10	480	107	111	360	1,119	598	1,197	572	120	4,184

Summer and Autumn.

Ancoats, C. - -	M.	82	–	–	–	–	–	–	–	–	–	5	2	4	20	2	12	9	–	54
	F.	89	–	–	–	–	1	1	1	–	3	1	2	11	12	9	15	4	2	56
,, E. - -	M.	51	–	–	–	–	–	–	–	–	–	3	1	4	9	3	17	4	1	42
	F.	83	–	–	1	2	–	–	–	–	3	1	3	7	17	8	18	4	1	59
,, N. - -	M.	38	–	–	–	–	–	–	–	–	–	2	2	3	14	3	5	2	–	31
	F.	58	–	–	–	–	–	–	–	–	–	1	3	2	14	5	8	8	–	41
,, S. - -	M.	78	–	–	–	–	–	–	–	–	–	8	1	5	24	8	14	8	2	70
	F.	95	–	–	–	–	–	–	–	–	–	–	2	6	19	12	27	11	2	79

„ W. - -	M.	90	–	–	–	–	–	–	1	–	1	–	3	10	23	14	13	15	6	84
	F.	134	–	–	–	–	–	–	–	–	–	6	5	10	28	24	37	13	5	128
Ardwick, E. - -	M.	65	–	–	–	–	–	–	–	–	–	1	2	6	18	3	17	3	2	52
	F.	70	–	–	–	1	–	–	–	–	1	–	2	9	10	3	18	6	2	50
„ N. - -	M.	59	–	–	–	–	–	–	–	–	–	1	1	5	20	4	10	5	–	46
	F.	74	–	–	–	–	1	1	–	–	2	3	1	11	10	10	15	4	–	54
„ S. - -	M.	90	–	–	–	1	–	–	–	–	1	3	3	9	24	10	20	14	2	85
	F.	91	–	–	–	–	1	–	–	–	1	–	2	2	18	16	24	18	3	83
Beswick - -	M.	74	–	–	–	–	–	–	–	1	1	2	1	5	29	9	13	6	–	65
	F.	97	–	–	1	–	–	1	–	–	2	2	1	8	14	9	23	8	–	65
Blackley - -	M.	51	–	–	–	–	–	–	–	–	–	–	–	2	9	1	13	9	4	38
	F.	71	–	–	–	–	–	–	–	–	–	–	1	3	11	2	15	7	4	43
Bradford - -	M.	71	–	–	–	–	–	1	–	–	1	4	1	4	23	6	14	8	–	60
	F.	101	–	–	–	2	–	2	–	–	4	5	2	9	22	7	24	7	–	76
„ No. 1 -	M.	71	–	–	–	–	–	–	–	–	–	–	3	3	22	7	14	12	2	63
	F.	75	–	–	–	–	–	–	–	–	–	1	2	2	10	15	18	12	2	62
Chorlton-on-Medlock, E.	M.	87	–	–	–	1	–	–	–	–	1	1	2	3	24	7	13	16	6	72
	F.	118	–	–	–	–	1	–	1	–	2	–	1	4	16	18	30	14	8	91
Chorlton-on-Medlock, S.	M.	66	–	–	–	–	–	–	–	–	–	1	1	6	15	2	16	12	1	54
	F.	76	–	–	–	–	–	2	–	–	2	2	2	3	5	8	23	12	1	56
Gorton - - -	M.	74	–	–	–	–	–	1	–	–	1	1	–	4	14	6	19	8	2	54
	F.	108	–	–	–	–	–	–	1	–	1	1	–	7	23	11	21	5	4	72

Result of Block Census in Manchester (Summer and Autumn Epidemics)—continued.

Autumn—continued.

District.		Total Number Persons concerned.	Affected.									Escaped.								
			0–1.	1–2.	2–5.	5–15.	15 25.	25–45.	45–65.	65 and over.	Total.	0–1.	1–2.	2–5.	5–15.	15–25.	25–45.	45–65.	65 and over.	Total.
Gorton, S. - -	M.	88	–	–	–	–	–	–	–	–	–	4	5	5	23	8	23	3	–	71
	F.	85	–	–	–	–	–	–	–	–	–	1	4	7	18	10	21	2	1	64
„ W. - -	M.	75	–	–	–	–	–	–	–	–	–	–	1	2	13	5	18	11	4	54
	F.	95	–	–	–	–	–	–	1	–	1	–	3	5	10	14	23	14	–	69
Harpurhey - -	M.	56	–	–	–	1	–	–	–	–	1	2	1	6	8	4	17	6	–	44
	F.	74	–	–	1	1	–	2	–	–	4	–	3	6	9	7	21	2	2	50
Hulme, C. - -	M.	81	–	–	–	–	–	–	–	–	–	2	3	9	19	9	11	14	1	68
	F.	110	–	–	–	–	1	–	–	–	1	3	1	10	18	22	21	15	2	92
„ E. - -	M.	34	–	–	–	–	–	–	–	–	–	–	2	3	12	4	1	5	–	27
	F.	59	–	–	–	–	1	–	–	–	1	1	1	3	12	9	12	4	2	44
„ N. - -	M.	78	–	–	–	1	–	–	–	–	1	1	–	4	18	18	15	12	3	71
	F.	69	–	–	–	–	–	1	–	–	1	–	–	1	11	12	19	9	3	55
„ S. - -	M.	68	–	–	–	–	–	2	–	–	2	3	–	9	17	3	11	8	1	52
	F.	82	–	–	–	–	–	–	–	–	–	3	1	4	12	8	21	13	1	63
„ W. - -	M.	79	–	–	–	–	–	–	–	–	–	–	–	10	25	3	13	12	1	64
	F.	90	–	–	–	2	3	2	–	–	7	1	1	5	21	10	14	6	4	62

London Road -	M.	85	1	–	2	2	–	1	–	–	6	2	6	10	21	8	17	7	1	72
	F.	90	1	–	–	–	2	–	–	–	5	5	3	7	16	9	20	6	2	66
Newton - -	M.	63	–	–	–	1	–	1	–	–	2	–	–	5	7	6	11	10	2	41
	F.	92	–	–	–	1	–	1	–	–	2	3	1	4	6	6	16	11	3	50
,, E. - -	M.	31	–	–	–	–	–	–	–	–	–	2	–	2	3	3	7	5	–	22
	F.	33	–	–	–	–	–	–	–	–	–	–	–	4	5	2	5	4	–	20
St. George's, C. -	M.	91	–	–	–	1	–	–	–	–	1	2	3	9	23	6	11	8	3	65
	F.	118	–	–	–	1	1	2	–	–	4	4	4	6	21	14	19	8	1	77
,, E. -	M.	66	–	–	–	–	–	–	–	–	–	2	1	6	20	7	13	8	–	57
	F.	83	–	–	–	–	1	2	–	–	3	–	2	4	24	9	19	8	4	70
,, N. -	M.	56	–	–	–	–	–	–	–	–	–	3	2	6	14	3	7	4	–	39
	F.	79	–	–	–	–	–	1	–	–	1	3	2	5	15	4	5	4	–	38
St. George's, Monsall	M.	68	–	–	–	2	–	–	–	–	2	3	–	4	13	5	14	9	1	49
	F.	101	–	–	–	–	–	3	–	–	3	1	2	5	17	10	17	8	4	64
Totals - -	M.	2,066	1	–	2	10	–	6	1	1	21	58	47	163	523	177	400	253	45	1,666
	F.	2,600	1	–	3	10	13	21	4	–	52	46	57	170	444	304	569	247	63	1,899
Grand Total -	—	4,666	2	–	5	20	13	27	5	1	73	104	104	333	967	481	969	500	108	3,565

Influenza.

Results of Block Census in Manchester. (*Summer and Autumn Epidemics.*)

Household Incidence.

District.	Total number Households concerned	Summer.		Autumn.		Summer and Autumn.	
		Affected.	Escaped.	Affected.	Escaped.	Affected.	Escaped.
Ancoats, C. - -	36	17	19	12	24	3	10
,, E. - -	33	12	21	10	23	5	16
,, N. - -	21	11	10	3	18	0	7
,, S. - - -	37	5	32	8	29	2	26
,, W. - -	47	2	45	6	41	1	40
Ardwick, E. - -	31	12	19	2	29	1	18
,, N. - -	30	10	20	9	21	3	14
,, S. - -	40	8	32	6	34	3	29
Beswick - - -	37	12	25	10	27	5	20
Blackley - - -	35	10	25	9	26	0	16
Bradford, No. 1 - -	32	7	25	8	24	1	18
,, - - -	32	9	23	10	22	4	17
C/M, E. - - -	42	11	31	14	28	5	22
,, S. - - -	35	11	24	9	26	5	20
Gorton - - -	36	14	22	14	22	4	12
,, S. - - -	33	4	29	12	21	1	18
,, W. - - -	38	14	24	10	28	4	18
Harpurhey - - -	36	15	21	9	27	4	16
Hulme, C. - - -	37	10	27	9	28	5	23
,, E. - - -	22	10	12	4	18	2	10
,, N. - - -	38	5	33	8	30	1	26
,, S. - - -	35	15	20	7	28	4	17
,, W. - - -	36	13	23	16	20	9	16
London Road - -	36	9	27	8	28	4	23
Newton - - -	36	18	18	11	25	4	11
,, E. - - -	12	6	6	1	11	0	5
St. Georges, C. - -	36	20	16	12	24	6	10
,, E. - -	34	10	24	8	26	6	22
,, N. - -	28	22	6	7	21	3	2
,, Monsall -	37	16	21	16	21	7	12
Totals - -	1,018	338	680	268	750	102	514

Results of Block Census in Manchester. Summer and Autumn Epidemics. Incidence in Households affected by both Epidemics.

District.	Households affected in both Epidemics.				Summer.						Affected in Autumn.					
	No. of House-holds.	Members of Households.			Affected.			Escaped.			Affected in Summer.			Escaped in Summer.		
		M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.
Ancoats, C.	3	8	11	19	1	7	8	7	4	11	—	3	3	3	2	5
,, E.	5	10	17	27	3	9	12	7	8	15	—	3	3	2	3	5
,, N.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
,, S.	2	5	3	8	—	2	2	5	1	6	—	—	—	2	—	2
,, W.	1	2	4	6	1	—	1	1	4	5	1	—	1	—	—	—
Ardwick, E.	1	3	3	6	—	1	1	3	2	5	—	1	1	—	—	—
,, N.	3	8	3	11	1	2	3	7	1	8	—	2	2	—	1	1
,, S.	3	8	6	14	1	2	3	7	4	11	1	1	2	1	2	3
Beswick	5	11	13	24	2	5	7	9	8	17	1	2	3	1	5	6
Blackley	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Bradford	4	9	13	22	3	5	8	6	8	14	1	4	5	2	5	7
,, No. 1	1	4	3	7	1	—	1	3	3	6	—	—	—	—	1	1
Chorlton-on-Medlock, E.	5	14	15	29	4	2	6	10	13	23	1	2	3	4	7	11
,, ,, ,, S.	5	12	14	26	3	4	7	9	10	19	—	2	2	3	5	8
Gorton	4	10	16	26	2	6	8	8	10	18	1	1	2	1	5	6
,, S.	1	4	1	5	—	1	1	4	—	4	—	—	—	2	—	2
,, W.	4	12	11	23	1	3	4	11	8	19	—	1	1	3	4	7
Harpurhey	4	7	10	17	4	5	9	3	5	8	1	4	5	1	2	3
Hulme C.	5	10	14	24	7	4	11	3	10	13	—	1	1	1	5	6
,, E.	2	1	4	5	—	2	2	1	2	3	—	1	1	1	—	1
,, N.	1	3	2	5	1	2	3	2	—	2	1	1	2	—	—	—
,, S.	4	8	11	19	3	2	5	5	9	14	2	—	2	4	1	5
,, W.	9	20	29	49	6	13	19	14	16	30	—	7	7	5	6	11
London Road	4	11	6	17	5	4	9	6	2	8	5	4	9	—	—	—
Newton	4	11	16	27	6	9	15	5	7	12	2	2	4	3	4	7
,, E.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
St. George's, C.	6	23	24	47	12	13	25	11	11	22	1	4	5	2	3	5
E.	6	13	18	31	4	4	8	9	14	23	—	3	3	1	4	5
,, N.	3	9	10	19	4	4	8	5	6	11	—	1	1	—	2	2
,, Monsall	7	16	24	40	4	9	13	12	15	27	2	3	5	2	4	6
Totals	102	252	301	553	79	120	199	173	181	354	20	53	73	44	71	115

Influenza—Manchester.

Results of Block Census.

Figures showing susceptibility during the autumn wave of persons who were attacked in summer.

A —Compared with the sample population as a whole (1,018 households = 4,666 total population) :—

		Attacked in Autumn.
694 attacked in summer.	Males, 246	21 (8·5 per cent.).
	Females, 448	52 (11·6 „).
3,972 escaped in summer.	Males, 1,820	154 (7·8 „).
	Females, 2,152	253 (11·7 „).

B —Compared with other members of the same households (102 households invaded in both summer and autumn epidemics = 553 total population of these households) :—

		Attacked in Autumn.
199 attacked in summer.	Males, 79	21 (25·4 per cent.).
	Females, 120	52 (44 „).
354 escaped in summer.	Males, 173	44 (25·2 „).
	Females, 181	71 (39 „).

Influenza—Manchester.

Results of Block Census. Age Incidence in Summer and Autumn Waves.

	Summer.		Winter.	
	Males.	Females.	Males.	Females.
0—1	·3	5·7	9·0	7·7
1—2	7·4	4·3	5·5	13·0
2—5	5·0	9·8	12·3	15·2
5—15	11·6	15·8	11·0	11·6
15—25	18·1	20·6	3·1	11·0
25—45	15·3	21·4	9·2	12·9
45—65	9·6	15·4	3·9	7·9
65 and over	8·0	11·2	4·0	10·0
Totals	11·9	17·2	8·4	11·7

IV.

Report on the Epidemic of Influenza in Manchester, 1918-19.

By

James Niven, M.A., M.B., LL.D., Medical Officer of Health.

Towards the end of June 1918 an epidemic of influenza burst on the City of Manchester, as on other large towns, flared up with extraordinary rapidity, and sank away at the beginning of August. Although, perhaps, its incidence was most evident in South Manchester, at all events as regards the schools, yet it was universally diffused over the whole city. If we consult the Registrar's weekly returns, simultaneity of incidence is observed for the different towns. The explanation may be found, perhaps, in the prevalence of the disease amongst the troops in France in May and June. No doubt it would be brought over to a great variety of localities by men on furlough about the same time; but this hardly accounts satisfactorily for the absolute agreement in time of the outbreaks in so many different centres of population. It must be borne in mind, however, that there was much movement of soldiers about that time, and that railways and trams were much crowded then and later.

Unhappily there are no data available here for the first outbreak to show the latent period of the disease, and, therefore, its speed of travel. It is certain, however, that it swept over schools and works with very great rapidity. Still a certain amount of time was required and great towns were affected, so to say, in the lump.

Assistance is given in understanding how the epidemic in this country may have arisen from that in the Army, by figures given in the report of the Medical Research Committee on Influenza in Hospitals of the British Armies in France, page 10.

We there see that the wave of influenza began to ascend definitely in the week ending June 8th and rose most energetically in the weeks ending June 15th and June 22nd. The largest number was admitted to hospital in the week ending June 29th, viz., 46,273.

If we may assume that the disease was disseminated in this country by men on furlough, it would follow that a large number of men must have come over from June 8th to June 29th, and also that the subsequent outbreak in Great Britain took some time to start into full operation. It is probable from the curves, that the infectious material from cases in the weeks ending June 15th and June 22nd possessed an enhanced power of diffusion.

This, on the whole, appears to be the most likely explanation of the outbreak in this country which started in the week ending June 29th, with faint indicia in the previous week.

We must otherwise assume some meteorological factor which stirred into activity the existing agencies causing influenza, acting at different times in France and Great Britain. This is a most unlikely hypothesis.* There can be no doubt that either some new infective agent was at work, or else that a specially infective strain (or strains) of a common agent of infectious disease was responsible for the outbreak in France, and very little doubt that the infection thus started was passed on, accompanied by a number of other more common agents of disease, all stirred into activity, possibly by the pabulum presented to them by the operations of the primary agents, possibly by the changes in the constitution of the blood caused by it.

The only common agent of which one is aware, which is normally highly infective, and is capable at times of causing rapid diffusion of disease with high mortality, is that responsible for the so-called "common cold."

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* It does not follow, however, that meteorological conditions have no effect in promoting rapid spread of this and other diseases.

The bacteriology and behaviour of common colds, especially when they appear as they often do, in a highly infectious form, appears, therefore, to deserve special study, and it will probably be desirable to bring all respiratory diseases into the sphere of public health at an early period. Unless this is done, and definite machinery provided for the study of different forms of respiratory disease and of influenza, epidemiologically and bacteriologically, little progress will be made, since the existing machinery is inadequate to the steadily growing tasks to which it is put. Rough work, in a matter of this kind, is of little value.

When the first outbreak occurred the medical officer of health, in the first week, issued a leaflet of precautions, which to the number of 30,000 was distributed chiefly to a number of works in the city, to the press, and in houses, through the health visitors. Five hundred large posters were also put up in conspicuous positions throughout the city. The newspapers were very obliging in giving publicity to this and other matters connected with the disease.

The medical officer of health, having no assistant in the public health department, was unable to give much attention to individual cases, but visited two works affected. In one of these he interviewed a number of girls who had just been taken ill, and contracted the disease, the latent period being two days. The attack was a mild one.

As a rule, the persons attacked at this establishment were not very severely affected. The attack was sudden, marked by chill, headache, pain in the back and limbs, temperature of 101° F. to 103° F., and, in some instances, by a feeling of prostration.

The disease appeared to spread rapidly through the works, but how it was conveyed was not clear. In my own case droplet infection could scarcely have accounted for it, but I took their temperatures, and in some cases handled the patients. It is probably in this way that nurses suffer so severely, that is to say, from contamination of the hands. As regards the operatives themselves, the use of common towels and common washbasins would give opportunity for spread of the disease. Also those working contiguously to affected persons, and handling the same articles, would be liable to infection. The effect of droplet infection may be exaggerated, as there is not much coughing in the early stage of the disease, but it may have more to do with the subsequent spread in households. By the courtesy of Dr. Ritchie, I had the opportunity of seeing influenza occurring in schools. At one school I observed the children falling ill. They simply dropped on their desk like a plant whose roots have been poisoned, the attack being quite sudden, and drowsiness a prominent symptom. This aspect of the onset was said to be common. Another much larger school adjoining was practically free from the disease. This was as Dr. Ritchie had mentioned, and it has been a striking feature in these outbreaks how widely the incidence has varied in different institutions, even when close together.

For example, one of the works in which I witnessed the occurrence of cases, a small one, was quite near the Fine Cotton Spinners Association employing a large number of people. I was assured that the latter was quite free from the disease.

It is not a mere matter of hygiene, good light and good ventilation, as one large business, admirable in all these particulars, as in their care for their workpeople, suffered rather severely.

The difference cannot be due to difference in droplet infection.

It is possible that difference would arise in schools from the use of closets, and of common towels; but as these would be common to schools severely and slightly infected, the suggestion scarce seems adequate. It is probable that differences arise in schools from the differing extent to which children are grouped closely in classes. But it is also suggested by the different rates of diffusion in one and another outbreak that differences in infectivity, and by the history of households that differences in receptivity may have to do with the varying amount of spread in different institutions.

It may be that facility of growth of the same infecting agent is correlated with mildness of attack. To some extent this would appear to apply to scarlet fever. When different diseases are compared we see that the more destructive, viz :—typhoid fever, typhus, plague, cholera, tuberculosis, are all difficult of spread. The fact that they attain massive dimensions in no way interferes with this observation, This applies still more to cerebro-spinal fever, pneumonia and streptococcal diseases.

It may be that there are different strains of the infecting agent, and that the more slowly acting strains were those introduced into works in which, at that time, the disease did not make headway.

Possibly individuals affected infect others with a like strain of the disease.

At all events it is evident that an enormous number of persons were affected in the first outbreak, although the fatality was considerably less than in the second and third outbreaks.

Particulars were obtained from a number of works, though not in such a form as to admit of brief presentation; but some idea of the extent of prevalence of the outbreak commencing in the end of June may be obtained from the following figures :—

A.—Letter, dated August 13th, 1918, from Armstrong and Whitworth's.
Total who have been absent through influenza—1,591, out of a total of 7,656.
Ditto. Shell factory, 313 out of 1,380,

B.—Messrs. Lewis's.
Total No. 720; absent on July 8th, 108.
Total number attacked not given.

C.—Crossley Brothers.
Absent at the height of the epidemic—333 (about) out of a total of 1,976.

D.—Manchester Tramways.
No. of employees, 4,613; absent for the week ending July 4th, 765, of whom 363 supplied medical certificates that they were suffering from influenza.

Writing on July 19th, 1918, Dr. Ritchie stated that the number of schools which had been closed on account of the disease was 76, all of which we may assume to have been severely affected. On 18th July, acting on his recommendation, the Education Committee closed all the Elementary Schools till August 26th. This course of action we had previously discussed and were agreed upon. The number of children affected could not then be given, but in a letter, dated November 1st, 1918, Dr. Ritchie informed me that the number of school children known to have been affected in the first epidemic was 39,255.

The deaths amongst school children were few, absolutely as well as relatively, being 28 at ages 5–14, giving a case mortality of less than ·1 per cent. The total number of deaths ascribed to influenza in this outbreak was 332, 177 in males and 155 in females.

The Block Census made for Dr. Carnwath by the Health Visitors Department in Manchester shows the number of attacks in summer to have been 694 in a population of 4,666, so that there must have been well over 100,000 attacks in summer alone, assuming that the sample is a fair one. This agrees with Dr. Ritchie's figures. In the autumn attack there were 407 attacks amongst those who escaped in the summer. The preponderance of attacks in summer agrees with the experience of the B.E.F. in France, though in the latter the difference is greater.

The total number of men on service admitted into hospital in the weeks ending 25th May 1918 to 10th August 1918 is stated to have been 226,615, as compared with 86,636 or 93,670 in the weeks ending October 12th, 1918, to January 18th, 1919.

The latter outbreak was, however, far more deadly than the former, the number of deaths being 5,555 ascribed to influenza and broncho-pneumonia.

The number of deaths ascribed to influenza primarily was 2,613. Taking account of the difference in age constitution between men on service and the general population of Manchester, and of the intensity of mortality at ages 25–29 and 30–34, it would appear that the intensity of mortality was much the same in Manchester as in the Army. In Manchester the corresponding number of deaths was 1,715, though the number attacked was probably not so high as in the Army. Similarly the number of deaths in the Army was about 1,400 for 11 weeks, including the wave of June and July, out of a total number of stated cases 226,615. In Manchester the corresponding number was 332 deaths out of over 100,000 attacks. The Manchester mortality in the first outbreak was lower than in the Army.

The milder character of the outbreak in summer would be conditioned by various causes, one of which would, no doubt, be that men who came over suffering from influenza would bring a comparatively mild disease with them The rise of the wave in France preceded that in England and Wales by three weeks. As regards the second wave, this appears to have begun simultaneously in France and in this country, and may, therefore, have developed from the preceding wave in the same manner in both countries. Evidently the two waves, though differing from each other, were the same in England and France in summer and autumn respectively. Further on it is suggested that the second outbreak may have been introduced from America.

During the summer outbreak numerous applications were made for advice, especially as to cleansing premises, and all the assistance possible was given. As a rule, the corporation disinfecting staff were not employed; where disinfection was advisable the recommendation was to cleanse the premises and use a formalin spray of 2 per cent.

The disease appeared to have sunk into quiescence by August 3rd, but in the week ending October 19th the number of deaths ascribed to influenza began to increase, and the numbers ascended week by week more slowly than in the first outbreak, but culminated in a terrible fatality in the week ending November 30th. In this week the number of deaths ascribed to influenza was 383, as against 120 in the week ending July 19th. Moreover, the distress was prolonged, so that it was realized that a real calamity had befallen the city. Mothers and fathers were often stricken together. The children, themselves ill, could not receive attention, and for a time it seemed as if it would not be possible to get coffins for the dead, or grave-diggers to dig the graves. This outbreak of disease lasted till the week ending January 25th, 1919, or a period of 15 weeks.

The comparative length of this wave by itself strongly suggests that a new strain or strains of disease had been added, or at all events that some change had occurred from the milder type of the summer outbreak. There died in all in this outbreak 1,715 persons, of whom 702 were males and 1,013 females.

The question as to what determines the length of the epidemic wave may prove to be an important one when the bacteriology of the disease is better understood. It may be that the virulence of the organisms causing a wave, and their relative amounts, may give the necessary warning as to the steps which require to be taken, and conversely the character of the wave may give the clue to the organism concerned. Some clue to the position may be furnished by an accurate determination of the latent period between exposure and attack, and comparison in this respect of different infecting agents. It does not follow, of course, that a short latent period means a short wave. If, however, we assume that the end of an outbreak means the exhaustion of a strain, that strain is likely to become most speedily exhausted which has the shorter latent period. If two strains are present together, and both in considerable amounts, the wave length may correspond to that which causes the longer latent period. The shorter latent period will probably correspond also to the milder form of the disease. With a mixture of strains, the duration in virulent form and the degree of virulence will follow the dominant strain.

Many members of the staff contracted the disease, but no fatalities occurred in the summer outbreak. On the other hand two clerks in the Public Health Office died in the course of the autumn outbreak, and also one of the tuberculosis nurses, while, notwithstanding exceptional exposure, there was no death amongst the health visitors.

During the summer outbreak a number of the patients at Baguley Sanatorium contracted the disease, and the admission of cases was suspended. One of the medical assistants was ill for some time. Later on, in the autumn outbreak, a number of nurses had influenza; but, fortunately, there was no death.

Monsall also was severely affected, and the course of events can be followed by means of a return kindly supplied by Dr. Dickinson, at that time acting as medical superintendent.

Incidence of Influenza on the Nurses and Maids at Monsall Hospital, 1918 and 1919.

1918.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
	2	1	1	6	14	2	1	7	23	6
1919.	Jan.	Feb.	March							
	0	10	8							

The cases occurring in June 1918 were all in the week ending June 29th. On March 18th, 1919, the matron died of influenza, and on March 27th, one of the nurses. The attacks varied greatly in severity, but, on the average, appeared to become more severe in the winter outbreak.

Great reluctance was felt to take cases of influenza into Monsall Hospital, but as the epidemic increased, it was felt that this reluctance must give way before the urgency of the need, and a ward was opened for cases of pneumonia following influenza on March 8th, 1919, and closed on April 19th, 1919. During this time 25 cases were admitted, among whom there were five deaths.

Similarly great reluctance was felt to divert the health visitors from their regular and most useful work to the visiting of influenza cases, but a small portion of whom they could hope to overtake; but this reluctance also had to give way, and the visiting of houses in which death had occurred, or in which they knew otherwise of the presence of influenza was begun on December 4th, 1918.

The Public Health Committee also sanctioned from this period the giving of assistance in nursing and otherwise. Fortunately there was a large stock of coal which had been given by a generous donor to the Child Welfare Department, and food, chiefly milk, was granted by the committee.

Every grant of food and coal was signed for by the chairman of the hospitals sub-committee and the medical officer of health, after investigation; but in some cases the family was in extreme distress, perhaps both parents and all the children were ill, and no nurses were available from any quarter.

Under these circumstances the following nurses generously volunteered to nurse, either by day or night, patients who could not otherwise receive the necessary nursing, Miss Sizer, Miss Appleton, Miss Oliver, and Miss Hall.

Others would have done so, but for difficulties connected with their lodgings.

Altogether an excellent spirit was shown.

It may be asked how it was that nurses could not be obtained from the Manchester and Salford District Nursing Association. There were, at the greatest time of need, many of their nurses ill of influenza, and at one home to which we applied the matron died from the disease. It is to this cause that we must ascribe the impossibility of securing nurses from the Association for continuous nursing. The nurses were too few and were over-worked. It

was no part of their duty to do continuous nursing, but that would not have hindered them from assisting us. Otherwise the nurses of the Association did most valuable service.

Later on in 1919 there seemed a prospect of getting assistance in nursing cases from the V.A.D. nurses of Red Cross Society, and one lady, Mrs. Needham, gave valuable help; but this class of work, viz., continuous nursing, does not accord well with their home obligations, and their services were available rather for work which does not exact prolonged strain and absence from home. I have a list of names and addresses, through the kindness of the Local Secretary, Mr. Oliver, so that, in any emergency I can call upon them for assistance.

Further, the Manchester and Salford District Nursing Association will (in emergencies) supply nurses to work under the Medical Officer of Health, and to report direct to him. We shall, therefore, in future possibly be better able to meet a similar heavy stress, though it must be pointed out, as regards the Manchester and Salford District Nursing Association, that in such a period of stress it is possible that they may have difficulty in supplying nurses, for the same reasons as prevailed during the autumn outbreak of 1918.

The difficulties experienced were materially relieved by the action of the Royal Infirmary, which received cases of pneumonia, whether suffering from influenza or not, to the limit of their accommodation. The cases were conveyed by the Public Health Department.

But it must not be supposed that the emergency was fully met. The greatest need experienced was a sufficient body of trained nurses to visit the homes, and nurse the cases when required.

Home helps to go in and clean up, attend to the children, &c., were very difficult to procure. In some cases neighbours met this need as far as they could; in one or two cases we were able to find someone to give the service required; but people were afraid to go to houses in which the cases were numerous and severe, as they had their own families to consider. No doubt the difficulty could be overcome, and, indeed, from December 1918 onwards, was fairly met.

The forms in connection with the health visitors are given herewith:—

FORM 1.

Practitioner's Form.

CITY OF MANCHESTER.

Influenza Distress.

Name.....Address.....District.....

*Assistance needed:—Attendance.

Coal.

Food.

Otherwise.

Doctor's Signature.....

Address.....

Date.....

* Please strike out particulars in which assistance is not required.

For Form 2, *see* following page.

FORM 3.

METHOD OF GRANT.

Copy of Form filled up to illustrate.

Public Health Office,
Civic Buildings, 1, Mount Street.
Manchester,
9th December 1918.

Influenza Allowances.

Address.—13, Back Factory St., Acts C.

Name.—Barlow.

Members ill.—2, Grandmother and child, (child has pneumonia).

Members dead.....

Allowances.—Milk or glaxo, 1 lb. per 2 days - } for 1 week.
Coal, 2 cwt. - - - - }

Standard income.—1*l.* 3*s.* 4½*d.*

Actual income.—18*s.* 6*d.*

Recommended to Nursing Association.—Already attending.

Signatures { Medical Officer of Health - JAMES NIVEN.
Chairman - - - - M. J. O'LOUGHLIN.

It is to be understood that practically the whole staff of the Health Visitors was engaged in this work up to the end of the third outbreak in March, during which time much valuable material in the shape of reports, has been accumulated. These relate, however, chiefly to homes severely visited.

No fatality occurred amongst Health Visitors, but the number of attacks was considerable, viz. :—

1918	Mar	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
	—	—	—	2	10	—	1	2	6	3
1919	Jan.	Feb.	Mar.				July.			
	2	2	6	—	—	—	1	—	—	—

It has been mentioned that great difficulty occurred in getting the dead buried at the height of the epidemic, partly because of the lack of grave diggers, partly because of the difficulty in getting coffins. Bodies were left as long as a fortnight unburied, partly at home, partly at the public mortuaries, and partly at the premises of undertakers.

A great amount of work was thus entailed in ascertaining where coffins could be most readily procured, and in bringing pressure to bear on individual undertakers. The difficulty was in part due to want of knowledge as to where coffins could be most readily obtained; partly it was due to depletion of the undertakers' staffs.

The services of a detachment of the Labour Corps from the Western Command were secured by Mr. Pettigrew, Superintendent of Parks and Cemeteries, and in this way the difficulty, as regards grave diggers, was overcome.

No doubt other towns had a similar experience, but the facts are here recorded to show the conditions under which the civil population laboured in November and December, 1918.

Allusion has already been made to the manner in which the nurses met the calls upon them during this outbreak. Needless to say, the medical practitioners, many of them ill themselves, though much overworked, did all that was possible to meet the emergency.

FORM 2.

HEALTH VISITORS ENQUIRY FORM.

Copy filled up to illustrate.

CITY OF MANCHESTER.

Investigation into Influenza.

Address, 13, Bk. Factory St. District, Ancoats C. No. in Family, 3.

Surname.*	Christian Name.	Age.	Sex.	Taken ill. (Date).	Early Symptoms.	Later Symptoms.	Date of Death.	Had they Influenza in Summer.
Barlow -	Lily.	48	F.	28.11.18	Vomiting, bronchial symptoms. Pains in head and limbs.	Dr. diagnosed pneumonia, 2.12.18. Had been getting up to attend to grandchild.		
,, -	Frederick.	34	M.	—	Soldier on Active Service.	District Nurse attending.		
McDougal -	Andrew.	3 11/12	M.	16.11.18	Pains in limbs and body. Vomiting, bronchial symptoms.	Pneumonia 26.11.18.		

* All Members of family, whether ill or not, to be entered above.

Amount and kind of assistance needed:—Attendance. District Nurse already attending.
Coal - Needed very badly.

Standard Income, 23*s.* 4½*d.*

No. of Rooms $\frac{2}{2}$ Total Income of family, 12*s.* 6*d.* and 6*s.* from S.S.F.A. promised.

No payment for Andrew.

House, Clean. Isolation, F. Good.

Signature of Investigator M. DUDLEY.
Date of Visit, 6.12.18.

On November 14th a phial of prophylactic vaccine, received from Sir Arthur Newsholme, was forwarded to Monsall hospital for trial. The Acting Medical Superintendent, Dr. Dickinson, offered inoculation, and five of the nurses accepted. Of these, one, 15 days afterwards, developed an attack of influenza.

It is possible that, if a suitable prophylactic were used at a critical time, when exposure is acute, an effect would be produced in the way of prevention or mitigation of an attack.

The mortality in school children was high in the second outbreak, and though the disease spread in the schools more slowly than in the first outbreak, Dr. Ritchie informed me on November 4th, 1918, that it had then become general throughout the schools all over the city. At that comparatively early period in this outbreak, he gave a list of 25 schools severely and 43 slightly affected.

At a later period, viz., on December 19th, 1918, the schools were again closed on his advice, till after the Christmas holidays.

On November 20th I sent a circular letter to superintendents of Sunday schools, requesting the managers to close the schools. All addresses could not be ascertained, but, in addition, there were managers who declined to close the schools.

An announcement was also given in the Press that Sunday schools should be closed. It is difficult to understand the perversity of those who neglected, or withstood the request to close.

By the Public Health (Influenza) Regulations, No. 122, dated November 18th and November 22nd, 1918, admission to public entertainments was regulated, with a view to securing good ventilation. These were discussed at a meeting of the Watch Committee held on November 25th, 1918. After discussion, the Committee received a deputation of the licensees and managers of many of the theatres, cinemas, &c., in the city, and slight modifications in the regulations were agreed to, consistent with their main object.

The managers and licensees agreed to exclude children under 14 from their entertainments, though this restriction was afterwards withdrawn by the Medical Officer of Health on December 17th, as the epidemic was then declining. About the same period the closure of Sunday schools was withdrawn.

On 22nd January 1919, the Medical Officer of Health sent a statement to the Chief Constable, advising that the requirements of these regulations might be relaxed. The date was an unfortunate one, as a third outbreak began early in February.

By the Regulations No. 2 the managers, &c., of places of public entertainment were bound to exclude school children under 14, if the public schools were closed.

The President of the Local Government Board, Sir Auckland Geddes, took a share in the education of the public by offering to Public Authorities the loan of a film, "Dr. Wise" on Influenza. This was shown at a number of cinemas in Manchester and was greatly appreciated. A report on the subject was prepared by Inspector Higginbotham, who made the arrangements.

The following handbill was prepared and issued as soon as possible after the commencement of the second outbreak. It is, in effect, the same, though longer and more detailed than the first. 150,000 copies in all were printed and distributed.

(*Copy Handbill.*)

CITY OF MANCHESTER.

Precautions against Influenza.

Influenza is again prevalent in Manchester. It is a highly infectious and very fatal disease, frequently leading to inflammation of the lungs. If

neglected, it is often followed by prolonged weakness and depression. The disease is generally marked by its sudden onset, by severe headache, with pains in the back and limbs, and by fever. Wherever possible the following precautions should be taken :—

1. The spread of disease is favoured by the crowding together of the healthy and the sick. Those desirous of escaping infection should therefore, avoid crowded rooms and assemblages of any kind. The use of towels common to a number of persons should be avoided.

2. THOSE TAKEN ILL SHOULD BE AT ONCE ISOLATED. This is especially important in the case of first attacks in a household, factory, workshop, or home.

In this way the spread of disease will be delayed. Those infected will, on the average, not have the disease so severely, and workshops may avoid the dislocation of work which will ensue if this action is not taken.

For persons taken ill at a workshop or factory who are too ill to walk home, an effort will be made to provide an ambulance on receipt of a telephone message at Tel. City 8680, Medical.

3. ISOLATION FROM THE REST OF THE COMMUNITY IS TO BE FOLLOWED BY ISOLATION AT HOME.

When possible the sufferers must have a separate room. Whether this is or is not possible the room should be ventilated by means of an open window, so as to diminish the risk to those waiting on the sick. But as the sick are very liable to develop pneumonia, they must be kept warm by a sufficiency of clean bedclothes.

If there is fog the window is to be closed, and a fire maintained in the sick room.

If a separate room cannot be provided, it may be possible to rig up a screen, so as to separate the sick from the healthy.

When any member of a household is taken ill, the others should as far as possible, avoid close personal contact.

4. PERSONS WHO ARE ATTACKED SHOULD AT ONCE SEEK REST, WARMTH, AND MEDICAL TREATMENT.— Any delay may result in dangerous complications. There is also great risk of relapse and, generally, there is serious weakness in convalescence. Sick persons should therefore not return to work except under medical advice.

5. DISCHARGES FROM THE NOSE AND MOUTH SHOULD BE AT ONCE DESTROYED.— At home they should be collected in a clean rag or paper, which should then be burnt. Matters coughed up may be collected in a cup or mug when they cannot be immediately destroyed, in which case a little Condy's fluid should be poured into the cup, which should afterwards be emptied into a drain. The cup is then to be cleaned with boiling water and soda.

6. DURING THE OUTBREAK A GENERAL EFFORT SHOULD BE MADE TO OBTAIN THOROUGH CLEANLINESS IN THE WORKSHOP AND HOME.

Any dust or dirt in either may cause the illness to be more severe than it would otherwise be.

If it is desired to go further in the way of prevention, the easiest way will be to spray all surfaces with a solution of Formalin in a strength of 2 per cent.

7. THOSE ATTACKED EVEN SLIGHTLY SHOULD ON NO ACCOUNT JOIN ASSEMBLAGES OF PEOPLE FOR AT LEAST A PERIOD OF TEN DAYS FROM THE COMMENCEMENT OF AN ATTACK, AS THEY MAY CONVEY THE DISEASE TO OTHERS.

The most infectious period, however, appears to be at the outset of the attack.

If the epidemic advances it will be advisable for the healthy to avoid crowded gatherings, since their neighbours may quite well have a slight attack of influenza without being aware of the fact.

8. IN SEVERE CASES, THE PERSON ATTACKED SHOULD REMAIN ISOLATED FOR A PERIOD OF AT LEAST THREE WEEKS FROM THE BEGINNING OF THE ATTACK.

9. During the outbreak, workpeople are advised to wear warm clothing and to avoid unnecessary exposure.

10. A SUFFICIENCY OF PROTEID FOOD IS NEEDED, BOTH FOR HEALTHY WORKERS AND SCHOOL CHILDREN TO ENABLE THEM TO RESIST THE DISEASE WHEN IT COMES.

The cheapest sources of proteid are oatmeal, flour, peas, beans, and lentils, herrings, mackerel, and milk. Fresh milk may be replaced by good dried milk made from unskimmed cows' milk.

Flour, peas, beans, and lentils require the addition of fat. Some vegetable, such as turnips, carrots, and onions, should also be used.

By Order,
JAMES NIVEN,
Medical Officer of Health.

December 5th, 1918.

Besides the issuing of this handbill information was given to the Press for their use from time to time. A memorandum prepared for the Local Government Board on Influenza, by Sir Arthur Newsholme, was also distributed to the Press and to members of the Public Health Committee. This most valuable memorandum arrived too late to be used in the preparation of the handbill then issued.

Considering the character of the infection and experiments made on the conveyance of catarrhs, as well as other facts in the conveyance of disease, one would now feel inclined to lay more stress on certain points.

In the handbill the use of common towels is deprecated, it is true; but it should probably be made a very prominent feature of a precautionary leaflet. Of great importance, also, is it to avoid the use of common wash-basins during an outbreak, unless these have been sterilised after each use. It is important, also, to avoid the use of eating utensils which have not been disinfected with boiling water before each time of use. This should be strongly impressed on the keepers of restaurants.

During this outbreak my attention was drawn to the general custom of handing practically uncleansed glasses, containing beer, to customer after customer. The time available did not appear to the bar-keeper to admit of more complete cleansing. I communicated with the Clerk to the Licensing Justices, who did all that was possible to remedy the practice complained of; but matters concerning food and drink are probably not so important as those which bring infected matters in contact with the nose, as occurs from infected towels and hands. Hence, the care of the hands is all important. This is well recognised in connection with the conveyance of tuberculosis, scarlet fever, and other infectious diseases. It has its application at the workshop, where infected persons handle articles which are then handled by others, as yet free from disease. In this way the hands become contaminated.

Also, if there is a dining room, articles of food, especially bread, are infected by the hands of persons already attacked. Persons attacked by no means give up work in all cases. Indeed, I was assured that it was very difficult, owing to home conditions, to force some workers to leave work, however ill.

The question of droplet infection, that is to say, conveyance through the air, has also to be considered. Having regard to the large number of tram employees affected, and to the manner in which common colds are caught by one person from another who does not use the same towels, wash-basins, food, &c., it would seem evident that, if this is, in any given case, not the

readiest means of transference, in the aggregate it is the most formidable. We have, therefore, to take precautions against giving or receiving infection from all these sources. At the same time, in a period of acute catarrhs, there is much dispersion of infection from handkerchiefs used by persons suffering.

Personally, I am disposed to think that direct droplet infection is not always the most important agency of conveyance. Some of the sources of infection could be guarded against. The danger from common towels could be guarded against by requiring each worker to provide his or her own towel; that from wash-basins by requiring them to be cleansed after each use, preferably with very hot water; the danger from transference at work by washing the hands after work, and taking care to avoid touching the face during work.

The danger from droplet infection can only be escaped by avoiding all crowded places, which makes the use of trams and trains impossible. Hence, it cannot be altogether avoided, unless the public will consent to the wearing of face masks. Persons travelling by tram or train should carefully avoid shaking their handkerchiefs.

The value of prophylactic injections has yet to be determined, and in any case they are scarcely available for a whole population.

It will be seen, however, that the above modes of infection tend to limit outbreaks to particular schools and works, being all contained within the individual establishment.

The same is true of the home, and it is certainly remarkable how the disease spreads through a particular house and spares others in its vicinity. All the above risks of infection are contained within the house itself. The inmates are, it is true, probably in more intimate relation with each other than they are with neighbours; but this special intimacy of relation has concern chiefly with the common slopsink or wash-basin, the common towel, and the common eating utensils and articles of food.

Having regard to the manner in which the disease spreads in households Dr. Ritchie was inclined to think that the spread was from household to household, and that schools were not themselves places of dissemination.

This, however, is contradicted by the fact that the same difference in dissemination occurs between school and school, as between house and house.

It is not necessary to understand intimately the bacteriology or epidemiology of influenza to see that more might have been done to limit the spread of the disease, and that the public health authorities may be expected, in future outbreaks, to press for further elementary precautions to be taken, in presence of a severe outbreak, in works, vehicles, and private houses.

In the week ending February 1st a third outbreak of influenza commenced, and the facts are briefly set forth in a report to the public health committee, dated 4th March 1912, which is here reproduced.

Needless to say, the Public Health Committee has been advised, by reports, of the whole course of these epidemics and the steps taken have been sanctioned by the Committee directly or retrospectively, or, in emergencies, by the Chairman, or Deputy Chairman. The following is an example :—

Copy.

Public Health Office,
Civic Buildings,
1, Mount Street, Manchester,
4th March 1919.

REPORT TO COMMITTEE.

Influenza.

The present epidemic of influenza is the third within the year. In the first outbreak in July 1918 there were 332 deaths, in the second, 1,715. The

first outbreak lasted 6 weeks, the second 15. In the first outbreak the incidence of mortality on children was comparatively light, in the second comparatively heavy. Thus, among boys under 10 in the July outbreak 8·6 per cent of the total mortality in males occurred, 4·5 per cent. among females. In the Autumn outbreak, of the total male mortality, 29·7 per cent. occurred among boys under 10, and of female mortality 23·3 per cent. among girls under 10. As regards the present outbreak individual cases do not appear to differ from those of the previous outbreak. It is noticeable, however, that there have been some cases lately of encephalitis lethargica, and of cerebro spinal fever. Pneumonia is much in evidence as in the Autumn, but the incidence, so far, has been comparatively light on school children.

The course of the deaths is shown on the attached slip, which gives the number of deaths week by week from influenza, bronchitis and pneumonia. The measures brought into action in the Autumn outbreak have been at once resorted to in the present one, viz. :—

(1) The whole of the health visitor staff has been turned on to visiting where a death has occurred and elsewhere, when information of a severe outbreak in a house has been obtained. Several of the nurses have volunteered actually to nurse cases, and their services have already been used.

(2) The Sanitary Inspectors are engaged in visiting cases of nfluenzal and other pneumonias reported under the new order.

(3) The British Red Cross Society has sent in the names of Voluntary Aid Nurses, who are willing to assist in nursing cases. So far one has been placed on duty, and two more are coming to-morrow.

(4) 50,000 handbills of advice have been distributed through schools and works and health visitors.

(5) The ambulance service has been made available for moving cases of influenza, especially those taken suddenly ill.

(6) A ward block will be opened at Monsall Hospital on Monday next for the reception of cases of pneumonia.

(7) Cases needing home nursing are referred tc the Manchester and Salford District Nursing Association.

(8) Cases visited by the health visitors and reported to need coal and food are considered and provision is made so that they may purchase what is necessary, or, where they are unable to do so provision is made by the Public Health Committee.

At the same time an effort is made to secure home-helps, whose services, in case of need, will be paid for by the Corporation, and also to secure nursing; but the former is usually the greater need.

(9) The Chief Constable was communicated with on February 19th to the effect that influenza was increasing, and that the Local Government Board's orders relating to places of amusement should be again brought into effect.

(10) The press has again been good enough to give prominence to advice in connection with the disease.

Further measures will be taken as may seem necessary.

JAMES NIVEN.

The following figures show the course of the mortality from influenza in this outbreak, up to date.

Week ending	Influenza.	Bronchitis.	Pneumonia.
Feb. 1, 1919 - -	11	36	33
„ 8 - - -	28	42	37
„ 15 - - -	44	56	41
„ 22 - - -	127	66	52
„ 29 - - -	191	78	63

It is not possible to give an exact forecast of the present outbreak, but I am disposed to think that it will be neither so prolonged, nor so fatal as the Autumn outbreak.

The forecast given was based on the rapid ascent of the numbers of deaths week by week, in this respect resembling the first outbreak. As a matter of fact this outbreak was neither so prolonged, nor so severe as the second. Its features, so far as mortality is concerned, will be considered further on.

As regards the assistance given in the previous outbreak, the following brief statement made by Miss Seed, Superintendent of Health Visitors, shows the manner and amount of material aid given from December 4th, 1918, to January 11th, 1919. It will be noted that there was no expense put down for coal, for the reason stated.

Statement by Miss Seed on the Procedure used with regard to assistance given in the recent Epidemic of Influenza, and the amount expended.

The very earliest cases of distress were found by the health visitors in the course of their work in the district.

Later, every house wherein a death from influenza had taken place, was visited, and an investigation sheet made out (*see* Forms attached).

A further number of cases (not many) were notified by the doctors as needing assistance, chiefly in the form of coal and milk.

Every application was subjected to the standard income test before help was given, and every application where help was given was signed by the Medical Officer of Health and the Chairman of the Hospitals Sub-Committee, or of the Sanitary Committee. When coal was needed the supply was obtained from the remainder of the gift of coal of December 1916, which is, consequently, nearly exhausted.

The health visitors investigated the cases in their own areas, and the outer and unworked districts were allocated to the sanitary inspectors.

All the assistance needed was discovered by the health visitors.

The investigation of family circumstances was made by the health visitors, but the medical officer of health took into account all the circumstances of each case, as far as was practicable.

9th February, 1919.

EXPENDITURE IN CONNECTION WITH VISITS TO INFLUENZA CASES AND ASSISTANCE GIVEN, DECEMBER 4th, 1918, TO JANUARY 11th, 1919.

	£	*s.*	*d.*
Total number of pints of milk given, 322 at 4½*d.* -	6	0	9
Total number of cwts. of coal given, 246½ at 2*s.* - -	24	13	0
Total number of lbs. of Glaxo given, 348 at 1*s.* 10*d.* -	31	18	0
Money for food expenses - - - - - -	4	0	9½
Total expenditure - - - - -	£66	12	6½

The total expenditure for the autumn and winter outbreaks was 124*l.* 8*s.* 10½*d.*

The following may be given as an example of various memoranda prepared for the use of the press, though these were but variants on one theme.

19.2.19.

OUTBREAK OF INFLUENZA.

Additional observations.

The public are reminded of certain points in connection with influenza, which has extended rapidly within the last few days, and threatens again to exact a high mortality.

The disease is communicated from the sick to the healthy, usually by direct infection, which is projected in coughing, sneezing, and speaking, for a distance of some feet.

Hence, persons who desire to escape infection, especially where there are others already ill, should wear a mask of butter muslin, as already described in the newspapers.

Persons suffering from colds should avoid coughing or sneezing amongst others, without using their pocket handkerchief, which should not be shaken when used. At present it is to the public interest and his own, that anyone suffering from cold, with raised temperature, should stay at home, at all events until there is no fever. Influenza itself requires a more prolonged rest, but it is necessary to remember that what is regarded as a common cold may be influenza.

From these remarks, it will be seen how dangerous it is at the present time, to come into close contact with a number of other persons brought together from various quarters.

Ordinarily, such considerations would raise a smile; but, unfortunately, last year some 2,000 deaths occurred in Manchester alone, and there is a prospect of high mortality from the present outbreak.

All dances and private entertainments should, just now, be avoided.

The present crowding into licensed premises at certain hours is a ready means of spreading disease, especially as the glasses are by no means always cleaned before passing from one customer to another. The disease is thus taken in by two channels, nose and mouth.

The strictest cleanliness as regards eating and drinking utensils should, for the same reason, be carried out at public restaurants.

Unfortunately, it is impossible for many people to avoid exposure to infection, as they are obliged to travel to their work in trains and trams, and when they go to their places of work they are again exposed to infection in a like manner, and from fresh causes. It is customary for people employed at works to use towels in common, as is also the case at offices, restaurants, and schools. This should be avoided. As already mentioned, persons travelling should wear masks, and persons coughing should use their handkerchiefs to cough or sneeze into.

All assemblages involving crowded meetings should, as far as possible, be avoided until this wave of influenza has passed over.

There are, however, many activities which cannot be interfered with, except in the last resort, and with the agreement of all concerned, such as churches, hospitals, out-patient departments, meetings on 'Change, public schools.

Sunday schools are important, no doubt, but stand in a different category, and should at once be suspended when any cases are known to have occurred.

The Local Government Board has dealt in a special manner with places of amusement, but it lies with the individual, both for his own sake and for that of others, to take no unnecessary risks.

At home, the danger of infection is at once greater and more easily escaped, with ordinary care. A new risk comes into view. Members of the family attacked with the disease, as well as those waiting upon them, are very liable to place their hands on bread without cleaning them. The

bread thus gets infected and carries on the disease to other members of the family.

It is, therefore, important that any one handling food such as bread, cheese, &c., should wash their hands carefully before doing so. This remark applies to all catering establishments.

It is important at such a time as the present, that the body should be kept warm, and that a sufficient amount of good food should be available. The former object may be attained by warm underclothing and good boots, coupled with exercise, or by a sufficiency of fuel; but it seems necessary to point this out, as the disease not only infects more easily, but is more severe, where deficiency of food, clothing and fuel is present.

As regards the home the Public Health Department, Civic Buildings, 1, Mount Street, would be glad of an intimation when influenza has appeared in a family, which is not able to procure sufficient fuel, food or attendance.

The Bacteriology of Influenza.

No attempt has been made to obtain bacteriological investigations in the case of persons dying from influenza. Three swabs were submitted to Professor Delepine from cases occurring at Baguley Sanatorium, and one was submitted by me. In none of these was the bacillus of influenza found. On the other hand three swabs, submitted by Dr. D'Ewart, at my request, from school children, all showed the presence of Pfeiffer's bacillus.

Professor Dean, also, on November 1st, 1918, was good enough to give me a short account of his experince obtained amongst the American troops at the Pavilion, Old Trafford. In the majority of cases the influenza bacillus was found in the sputum. He made blood cultures from five of the worst cases, and in three obtained a pure culture of the pneumo-coccus. In three of the cases he made a post-mortem examination and found in each very extensive confluent broncho-pneumonia and purulent bronchitis.

In the British Medical Journal, January 5th, 1919, Colonel Muir described the results of examination of 30 bodies of persons who died of influenza at Glasgow, of whom 26 were American soldiers. In the great majority of these the bacillus of influenza was present, viz., in 22 out of 26, and in all the broncho-pneumonic cases.

The outstanding feature from the point of view of morbid anatomy was the broncho-pneumonic lesion. The number of septicæmic cases was high.

The organisms most frequently associated with the influenza bacillus were pneumo-cocci and streptococci.

Similar findings are recorded in the publication of the Medical Research Committee. In one of the researches there given the close association of the influenza bacillus with streptococcus viridans is a prominent feature. The influenza bacillus appears to become more abundant some time after the commencement of the illness.

The influenza bacillus is frequently not present, in the experience of other observers, and is present in conditions having no relation to influenza.

It is not communicable to animals, so that proof of its causative relation to influenza is wanting.

In a publication by the Medical Research Committee, Major Gibson, R.A.M.C., Major Bowman, C.A.M.C., and Captain Connor, A.A.M.C., record a research into a filtrable organism which they believe to be the cause of the disease.

They show that the filtrate from sputum obtained early in the course of the disease, is capable of producing, when inoculated in animals (monkeys, rabbits, guinea-pigs and mice) lesions in the lungs similar to those occurring in man, more particularly as regards the hæmorrhages which were so prominent a feature in the lungs of patients dying in the second outbreak, to which period their research relates.

These appearances were found even in animals which presented no sign of ill-health after inoculation.

By adopting Noguchi's technique they obtained cultures of a minute coccoid body, which they were able to subculture, and which, when inoculated into animals, were able to transmit disease.

By employing rabbits they were able to pass on disease from one animal to another.

This research followed the lines of one by Dr. G. Foster recorded in the (American) Journal of Infectious Diseases, 1917, 21, on Common Colds.

The above workers did not venture to use human material to test the cultures obtained, but Dr. Foster could more safely do so, and by instilling the filtrate from sputum in common colds, and the cultures which he obtained, into the nostrils of volunteers, he found that acute catarrh was produced in periods varying from 6 to 48 hours.

Gibson, Bowman, and Connor found that, when the inoculated animals showed signs of illness, it was not until the 6-7th day after inoculation.

This may be accounted for by difference in the channel of entrance, but is more likely to have been due to a difference in the infecting agent.

The cultures which these observers obtained were, in appearance, exactly like those obtained by Foster; but no proof exists that those cultures had anything to do with the results obtained, or that they constituted the filtered virus. This may quite well have been in each case some organism which not only was in the first instance, but remained ultra-microscopic,

On the other hand the behaviours of "common cold" and of "influenza" are remarkably alike. As with influenza a "common cold" is at one time infectious, at another non-infectious, at one time a member of a family gets a cold and it stays there; at another it passes through the household with extraordinary rapidity. It behaves differently in different households.

The results of a cold are much more severe at one time than another, as regards the respiratory troubles which it stirs up.

It may well be that a close study of the common cold is necessary to the elucidation of influenza.

Another feature of these outbreaks is the manner in which micro-organisms ordinarily more or less quiescent took on infective and malignant properties. Thus pneumococcal, streptococcal, staphylococcal, and even meningococcal infections all became more active.

This is perhaps more likely to happen if they are associated with a very minute filtrable body than as the result of association with the influenza bacillus.

PART II.

Epidemiology.

In studying the epidemiology of the disease the only material which I possess is the Register of Deaths. Fortunately, by the kindness of Dr. Carnwath and Dr. Arnold, of the Ministry of Health, I have had the opportunity of seeing the results of investigations made by different observers, Dr. Carnwath in Manchester, Dr. Arnold in Leicester, Dr. Macewen on schools, Dr. Hutchinson in a rural district, and other observers on the effects of special occupations on the incidence of influenza.

Further, Dr. Major Greenwood has kindly furnished me with the results of his calculations on the facts for these and other districts. I much regret now that I did not apply for a special staff to carry out observations on the course of the epidemics on the lines pursued by Dr. Carnwath, Dr. Arnold, Dr. Hutchinson, and in Cambridge; but it did not occur to me to use the staff for this purpose until Dr. Carnwath requested that a block census should be taken. The best was done in Manchester which then appeared possible, and Dr. Carnwath had the services of the health visitors for a week. The material thus collected will be useful in various ways.

Areas were selected so as to obtain a total of about 1,000 houses, distributed in such a way as to be representative of different districts in the city.

At the time of Dr. Carnwath's investigation only the facts for summer and autumn could be given. The names and ages of each member of the household were recorded, the date and character of the attack in autumn, and the fact of a previous attack in summer. The summer outbreak covers the period from the week ending July 20th to the week ending August 3rd, the autumn outbreak extending from the week ending October 19th, 1918, to the week ending January 25th, 1919. The facts for the winter outbreak have been subsequently obtained.

As my figures are arranged in age groups of five, it is proposed to re-arrange Dr. Carnwath's data in the same manner, The mortality curves exhibit striking peculiarities when thus arranged, which do not appear otherwise, and they lend themselves better to the construction of frequency curves.

On the other hand their value would not be nearly so great, unless the facts of incidence collected for Dr. Carnwath were there for comparison.

When the Manchester and Leicester experiences are compared in this manner, I expect the frequency curves of incidence for age groups will be found to differ. There is also the gross fact for Manchester of the much larger incidence in summer than in autumn, while the incidences in Leicester show the opposite relation. The two experiences are, probably, in important respects different.

This is important as regards the comparative immunity shown in the two cities of persons exposed in the autumn who had been infected in summer.

The investigations made for the Ministry of Health on the block census method show for Leicester immunity against autumn attacks in those infected in summer, and immunity against winter attacks in those infected in autumn.

In Wigan, where the investigation was carried out by Dr. Hutchinson, there was, in addition, immunity against winter attacks of those attacked in summer.

In Cambridge University there was immunity for summer attacks in autumn, and against winter attacks of those attacked in summer.

On the other hand the Finchley Elementary Schools show protection in autumn of those attacked in summer, and in winter for autumn attacks, but not in winter for summer attacks, in this agreeing with Leicester. The experience of Finchley Elementary Schools was also that of Clifton College.

In the Manchester figures no protection is apparent in autumn for summer attacks. There would thus seem to be some variable factors.

So far as my own experience goes it is intended to submit the deaths to a careful examination, and then to compare the results obtained with corresponding figures for incidence obtained by Dr. Carnwath, and a further set of additional figures obtained afterwards.

First, then, as regards deaths. The deaths are recorded in males and females respectively, week by week, and for 16 five-yearly groups of ages, from the beginning of January 1917 to the end of June 1919, registered as due to influenza, pneumonia, broncho-pneumonia, bronchitis, and tuberculosis respectively. Those ascribed to tuberculosis have not been used in drawing the curves submitted to study, since there was but little effect produced on tuberculosis.

The following curves have been constructed for deaths :—

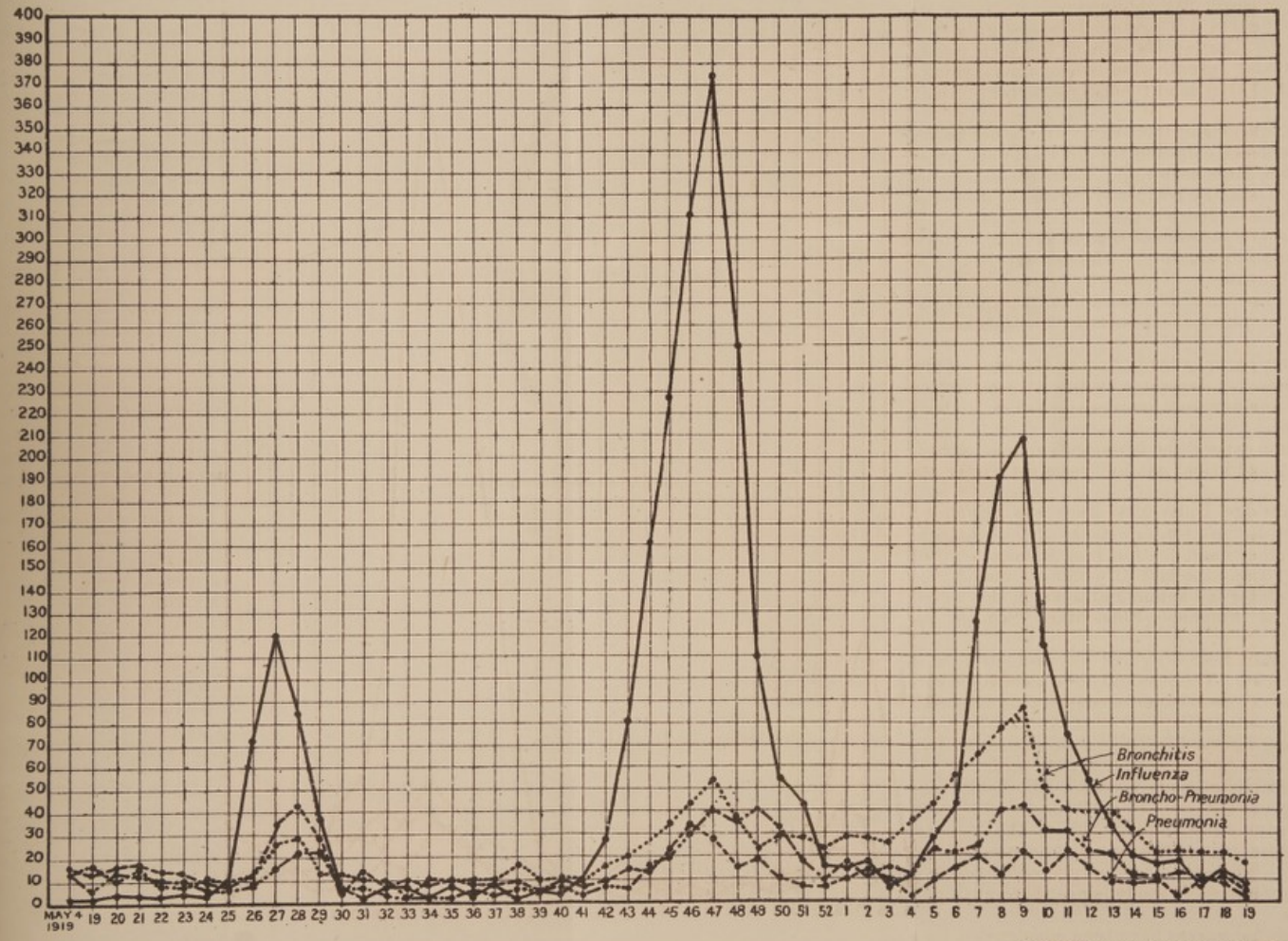
Curves 1.—Continuous frequency curves on the same scale from "influenza" from the 18th week in May 1918 to the 19th week in 1919, showing deaths from influenza, pneumonia, broncho-pneumonia and bronchitis.

We thus see that three waves of influenza mortality occurred, the first lasting from the 25th week, 1918, to the 30th. The duration of this wave was six weeks. It will be seen that the mortality sustained was far slighter than in the second and third waves.

To face p. 488.

FREQUENCY OF DEATHS FROM INFLUENZA, BRONCHITIS, BRONCHO-PNEUMONIA AND PNEUMONIA IN WEEKS FROM THE 19TH WEEK 1918 TO THE 19TH WEEK IN 1919.

CURVES I (See p. 506)



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The second wave lasted from the 42nd week in 1918 to the 4th week in January 1919, a period of 15 weeks. This was by far the most fatal visitation.

The third outbreak lasted from the 5th to the 16th week in 1919, a period of 12 weeks, and was intermediate in severity between the first and third.

Roughly the second wave was twice as severe as the third, and five times as severe as the first.

In appearance they are very much alike, so far as influenza alone is concerned. The third wave is somewhat flattened at the top, and is narrower than the second, except at the base.

It will be remembered that notwithstanding the great fatality in the second wave, there is reason to believe that far more persons were attacked in the first wave, many of them, no doubt, so slightly as to make them forget that they had been attacked.

An important difference was observed in the second wave in the greater number of persons who suffered from bleeding at the nose, and, when post-mortem examinations were made, in the comparative frequency of hæmorrhages into the lung.

Important differences also are observed in the curves for pneumonia, bronchitis, and broncho-pneumonia.

The curve of pneumonia mortality reached its highest point in the first wave, and in this wave it resembled, more than the waves of the other two diseases, that for influenza. In the second wave the pneumonia mortality shows a well marked wave, but it is now lower than that from bronchitis or broncho-pneumonia. In the third wave it again rises above the level, but the rise is not a sustained one, and does not attain nearly the height of either broncho-pneumonia, or bronchitis.

It may be observed that, in the first wave, the crest of the pneumonia wave follows that of influenza, by a week, in the second it precedes it, in the third the pneumonia wave has no distinct crest though its upper part corresponds roughly with that of influenza mortality.

It would thus seem as if the impulse towards the creation of an epidemic pneumonia diminished from the first wave onwards.

In the first outbreak the broncho-pneumonia mortality shows also a distinct wave, considerably less than that of pneumonia, which attained its crest a fortnight after influenza, and a week after pneumonia. The wave of broncho-pneumonia is throughout greater than it appears, however, as it rises from a lower base than pneumonia or bronchitis.

In the second outbreak broncho-pneumonia rises well above pneumonia, its wave having two crests, one coinciding with that of pneumonia, the other a fortnight later; but it only rises in a conspicuous manner in the latter part of the wave, in a part of which it rises above bronchitis.

As a separate cause of death broncho-pneumonia thus becomes much more important in the second outbreak than in the first. Its importance relative to pneumonia is much more emphasised in the third wave, where it separates itself from "pneumonia" entirely, and as a cause of death is as important as in the second outbreak. This is the more remarkable since this outbreak coincides with the season at which lobar pneumonia is most in evidence.

There is, however, not much difference between the mortalities from broncho-pneumonia in the second and third waves.

Bronchitis presents a different picture. Like the other two respiratory conditions the mortality ascribed to it has a well-marked wave; but this wave has very definite features throughout.

In each wave the crest coincides with that of influenza, and the course of the mortality pursues a similar course though flattened down. In the first outbreak this wave precedes that of broncho-pneumonia, as its does in the second. It is not materially greater than that from broncho-pneumonia in the first wave, though smaller than the wave of pneumonia. In the second it is somewhat greater than the wave of broncho-pneumonia. In the third

it rises far above both pneumonia and broncho-pneumonia as a cause of death.

In order, however, to be able to form an opinion as to the relation which the frequency curves from those respiratory conditions bear to iufluenza, it is necessary to consider the number of instances in which each of them occurs as a complication of influenza. It is manifest, of course, that the waves affecting these conditions, when given as independent causes of death, are in reality waves of influenza, though the most prominent feature and character may be those of the cause of death assigned; but it is at least possible that these waves, when compared one with the other, may be raised or depressed according as the number of complications in the corresponding period from that particular cause is low or high.

Moreover, a study of the complications in fatal cases may throw light on the character of the respective outbreaks.

These conditions, when occurring as complications of influenza, have, therefore, been taken out in 16 age groups for four periods, viz., January to June 22nd, 1918, June 23rd to August 3rd, 1918. October 23rd, 1918, to January 25th, 1919, and January 26th to April 26th, 1919.

The second, third and fourth of these periods cover the three epidemic waves.

For convenience of reference the corresponding facts for influenza are given at the head of the table.

Table showing four Periods for Deaths from Influenza, Deaths Registered simply as from Influenza, and Deaths Registered from a Variety of Complications, divided into 18 Age Groups, each of five Years.

Influenza age frequencies in fatal Cases.

——	Period.	0–5.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.	50–55.	55–60.	60–65.	65–70.	70–75.	75–80.	80–85.	85 and above.	Total.
	Jan.–June 22, 1918.	2	1	3	1	3	2	2	3	2	8	5	8	8	10	4	1	2	1	66
	June 23–Aug. 3	14	8	17	28	27	42	31	27	23	23	21	19	17	14	15	3	2	1	332
	Oct. 13–Jan. 25, 1919.	304	130	65	96	127	181	155	105	80	93	80	78	71	73	42	19	10	5	1,714
	Jan. 26–April 20	109	31	22	48	44	112	94	57	53	73	64	44	49	50	43	29	13	1	936

Complications of Deaths from Influenza—1918–1919.

Complication (Secondary Cause).	Period.	0–5.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.	50–55.	55–60.	60–65.	65–70.	70–75.	75–80.	80–85.	85 and above.	Total.
Broncho Lobular, Catarrh, Pneumonia	Jan.–June 22 1	1	—	—	1	—	—	—	--	—	—	—	—	—	3	1	—	1	—	7
	June 23–Aug. 3 2	4	3	3	8	2	9	4	4	5	4	4	4	5	3	1	—	1	—	64
	Oct. 13–Jan. 25 3	161	73	16	19	22	44	43	24	21	24	28	10	18	16	7	2	1	—	529
	Jan. 26–Apr. 26 4	54	13	5	8	12	33	18	15	10	16	17	13	11	9	14	4	1	—	253
Other Pneumonia	Jan.–June 22 1	—	1	1	—	—	1	2	1	—	2	2	3	1	2	1	—	—	1	18
	June 23–Aug. 3 2	2	2	11	15	18	19	19	14	12	11	8	10	3	3	4	1	1	—	153
	Oct. 13–Jan. 25 3	77	35	35	61	83	107	86	62	28	46	30	26	21	14	9	1	1	—	722
	Jan. 26–Apr. 26 4	30	10	12	24	25	56	56	28	22	39	24	9	11	13	11	9	—	—	379
Bronchitis	Jan.–June 22 1	—	—	—	—	1	—	—	—	1	2	1	2	2	—	—	—	—	—	9
	June 23–Aug. 3 2	—	—	—	1	—	3	2	2	1	3	4	2	2	4	6	—	—	—	30
	Oct. 13–Jan. 25 3	11	2	2	3	3	11	5	4	10	13	10	22	19	25	13	12	3	1	169
	Jan. 26–Apr. 26 4	10	1	—	2	2	8	2	3	13	6	17	9	17	19	10	9	5	—	133

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Complications of Deaths from Influenza—1918–1919.—continued.

Complication (Secondary Cause).	Period.	0–5.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.	50–55.	55–60.	60–65.	65–70.	70–75.	75–80.	80–85.	85 and above.	Total.
Other Respiratory Dis.	Jan.–June 22 1	—	—	1	—	—	—	—	1	—	1	1	1	1	1	1	—	—	—	8
	June 23–Aug. 3 2	1	—	—	—	1	1	—	1	—	2	—	—	—	—	—	—	—	—	6
	Oct. 13–Jan. 25 3	2	1	—	—	2	2	2	1	3	1	2	1	1	1	2	—	1	—	22
	Jan. 26–Apr. 26 4	—	—	—	—	—	—	1	1	1	3	—	1	1	—	1	—	—	—	9
Tuberculosis	Jan.–June 22 1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	June 23–Aug. 3 2	1	—	1	—	—	2	1	2	2	—	—	2	1	—	—	—	—	—	12
	Oct. 13–Jan. 25 3	—	2	5	4	10	8	3	3	4	2	1	1	2	—	—	—	—	—	45
	Jan. 26–Apr. 26 4	—	—	1	3	1	4	1	1	—	3	1	2	—	1	—	—	—	—	18
Meningitis	Jan.–June 22 1	—	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	2
	June 23–Aug. 3 2	3	1	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	6
	Oct. 13–Jan. 25 3	14	5	1	1	2	1	1	—	—	2	—	1	—	—	—	—	—	—	28
	Jan. 26–Apr. 26 4	—	1	1	2	—	1	1	1	—	—	—	—	—	1	—	—	—	—	8
Nervous Dis.	Jan.–June 22 1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	June 23–Aug. 3 2	2	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	3
	Oct. 13–Jan. 25 3	9	2	—	1	—	—	—	—	—	—	1	1	—	—	—	—	—	—	14
	Jan. 26–Apr. 26 4	1	—	—	—	—	—	—	—	—	1	—	—	—	—	1	—	—	—	3
Digest Syst.	Jan.–June 22 1	1	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	2
	June 23–Aug. 3 2	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
	Oct. 13–Jan. 25 3	8	—	1	1	1	1	—	1	—	—	1	—	1	1	—	—	—	—	16
	Jan. 26–Apr. 26 4	—	1	—	1	—	—	—	1	—	—	—	—	—	—	1	—	—	—	4
Heart and Blood Vessel Dis.	Jan.–June 22 1	—	—	—	—	—	—	—	1	—	2	—	1	3	4	2	1	—	—	14
	June 23–Aug. 3 2	—	1	1	1	3	6	5	3	2	2	3	1	6	3	3	1	—	—	41
	Oct. 13–Jan. 25 3	5	4	—	4	2	1	9	7	10	4	2	9	6	7	7	1	2	—	80
	Jan. 26–Apr. 26 4	2	1	1	5	2	6	7	5	4	4	3	9	4	3	2	5	6	1	70

Measles and Whoop. Cough	Jan.–June 22	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	June 23–Aug. 3	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Oct. 13–Jan. 25	3	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2
	Jan. 26–Apr. 26	4	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
Diarrhœa	Jan.–June 22	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	June 23–Aug. 3	2	—	—	—	1	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	3
	Oct. 13–Jan. 25	3	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
	Jan. 26–Apr. 26	4	3	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	4
Septicæmia	Jan.–June 22	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
	June 23–Aug. 3	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Oct. 13–Jan. 25	3	—	—	1	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	2
	Jan. 26–Apr. 26	4	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
Urin. Syst.	Jan.–June 22	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	1
	June 23–Aug. 3	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Oct. 13–Jan. 25	3	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	1
	Jan. 26–Apr. 26	4	—	—	—	—	1	—	1	—	—	—	1	—	—	—	—	—	—	—	—	3
Rheumatism	- - -		—	—	—	—	—	—	—	1 (3)	—	—	—	—	—	1 (3)	1 (4)	—	—	1 (3)	4
Childbirth	- - -		—	—	—	—	—	2 (4)	2 (4)	1 (3)	—	—	—	—	—	—	—	—	—	—	5
Anæmia	- - -		—	—	—	—	—	—	—	1 (4)	—	1 (3)	—	—	—	—	—	—	—	—	2
Arthritis	- - -		—	—	—	—	—	—	—	—	—	—	—	1 (3)	—	—	—	—	—	—	1
Ulcer	- - - -		—	—	—	—	—	—	—	—	—	—	—	—	—	1 (2)	—	—	—	—	1
Syphilis	- - -		1 (4)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
Encephalitis	- - -		—	—	1 (4)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1

From the above table we find that the following complications form the percentages of deaths from influenza in the respective periods given underneath :—

Percentages of Influenzal Deaths.	First Period. Jan. 1918 to Week ending June 22.	Second Period. First Wave, 26th–31st week inclusive.	Third Period. Second Wave.	Fourth Period. Third Wave.
Pneumonia - - -	27·27	46·08	42·12	40·49
Broncho-pneumonia -	10·61	19·30	30·86	27·03
Bronchitis - - -	13·64	9·04	9·86	14·21

To compare this with the corresponding causes of death registered as primary during the second, third and fourth periods, it is necessary to remove that part of the waves which is permanent and occurs in other years. This can be done approximately by subtracting the figures for the corresponding weeks in the year before; we thus get :—

Disease.	Second Period.	Third Period.	Fourth Period.
Pneumonia - - - -	136	209	165
Less	49	155	144
	87	54	21
Broncho-pneumonia - - -	78	310	303
Less	32	181	245
	46	129	58
Bronchitis - - - - -	111	419	629
Less	67	360	295
	44	59	334

The percentages which these residues form of the deaths from influenza in the corresponding period as shown in the above table are :—

Disease.	Second Period.	Third Period.	Fourth Period.
Pneumonia - - - -	25·30	3·15	2·24
Broncho-pneumonia - - -	13·86	7·53	6·20
Bronchitis - - - - -	13·25	3·77	35.68

On reference to the curves of frequency it will be seen that this mode of treating the figures meets the case very well.

It shows the powerful influence of the first wave on pneumonia, broncho-pneumonia and bronchitis. This is much less conspicuous in the second wave, even as regards broncho-pneumonia, which is the condition most affected.

The striking characteristic of the third wave is the dominance of bronchitis, which may, presumably, be taken to mean absence of consolidation.

It would thus appear as if some more fatal influence had become dominant in the second wave, and taken the place of the more common complications of the disease, perhaps a hæmolytic streptococcus.

Adding, now, the residues of the waves of pneumonia, bronchitis and broncho-pneumonia obtained above to each of the influenza waves, we obtain the figures given below for the approximately correct number of deaths from influenza and its complications.

When we treat the figures in the way suggested we get:—

Number of deaths.	First Wave.	Second Wave.	Third Wave.
Influenza - - - - -	509	1,956	1,349
Having pneumonia - - -	240	776	400
Having broncho-pneumonia -	110	658	311
Having bronchitis - - -	74	228	467

This gives for the percentage of complications in:—

——	First Wave.	Second Wave.	Third Wave.
Pneumonia - - - -	47·15	39·67	29·65
Broncho-pneumonia - - -	21·61	33·64	23·05
Bronchitis - - - - -	14·54	11·66	34·62

On this mode of treatment it will be seen that pneumonia, which includes lobar pneumonia and other pneumonia not specifically named, but usually lobar, is more in evidence in the first wave than in the second, and still less in the third. Broncho-pneumonia, on the other hand, is less evident in the first and third waves than in the second.

Bronchitis is least evident in the second wave, most marked in the third.

In the third wave 87 per cent. of the deaths have respiratory disease as a complication, in the second wave, 85 per cent., in the first wave 83 per cent.

Pneumonia, including broncho pneumonia, is present in 73 per cent. of the fatal cases in the second wave, in 69 per cent. in the first, and in 53 per cent. in the third.

It would thus seem that marked differences appeared in the three waves as regards the complicating conditions in the lung, which probably corresponded to changes in the character of the organisms responsible for the fatal issue, and perhaps also for the incidence in the three waves.

It is important to observe that, while the true curve of influenza mortality may probably be constructed by adding to the waves of deaths from influenza, the simultaneous waves of deaths from pneumonia, broncho-pneumonia, and bronchitis, the transference is not all on one side. This will be seen on comparing the control waves in curves 6 for January 1917 to June 1918, with those for June to December 1918 and January to June 1919. It will be seen, on examining the latter curves for pneumonia and broncho-pneumonia at advanced ages, and comparing them with the control curves, that there is evidence of transference from pneumonia and broncho-pneumonia to influenza.

With influenza so prevalent as it was in the summer and autumn of 1918, and in the first-half of 1919, the tendency is, in fact, to regard as influenza every acute fever, especially those of short duration. In this way a large amount of error must have been introduced into double attacks,

sufficient to obscure the facts of immunity, unless these were ascertained with great pains and discrimination. This is especially likely to occur in districts such as Manchester, where the incidence of pneumonia and other acute respiratory affections is always high.

With less severe affections it is not possible to say whether these normally occur in Manchester with exceptional frequency, but it is likely, if we may judge from the high death rate from pneumonia, and also from the very smoky state of the atmosphere, which I have elsewhere shown to be, at times, associated with a high respiratory mortality.

The difficulty in determining in any given case, whether two reputed attacks have been from influenza may be illustrated by the histories which I obtained from four of our health visitors who are said to have had double attacks.

Health Visitors.

(1) First attack. Mrs. S. aet. 30.
Second attack, 1st in June 1918.

First attack, began on July 1st, in Warrington, where she was a health visitor. Severe headache, temperature 102° for several days, pains in the limbs, &c., no epistaxis, no catarrh of the eyes and nose, slight sore throat, anorexia; no diarrhœa. Away a fortnight. Had cough, but no pneumonia; picked up very quickly; felt quite fit on returning.

Second attack began October 26th. Very well in morning; afternoon gradually felt ill; severe headache, very fatigued; anorexia, chill; temperature not taken; no epistaxis; no actual sickness; no diarrhœa; no chest affection, but cough. Went to bed, but got up and tried to work it off. Was away a fortnight. Second week had palpitation very badly.

(2) Miss B.C., Higher Broughton.

I. At Radcliffe, nursing soldiers at the Radcliffe Military Hospital.

Took ill in July; came on suddenly. Shivering, general pains, headache, prostration, temperature, 101°. Vomited, no appetite, no epistaxis, running eyes, sore throat or diarrhœa. Did not go to bed but continued duty. Ill about a month, but no cough. Feeling of lassitude.

II. Took ill about November 1st. Came on rather suddenly; chill, general pains, great depression, severe headache, temperature, 100° twice; cough, pain in the chest, went to bed and stayed in bed three or four days, then came down to the office. Has a cough now and does not seem to me fit, but will not stay at home (seen on her return).

(3) Mrs. B.

I. Took ill July 1918. Having dinner, directly after took suddenly ill, like fainting fit. Went home; temperature, 103·8°; was then a health visitor at Oldham. Severe headache, sore throat, ending in tonsilitis. Pains all over, vomited; no diarrhœa. Went to bed and stayed there a week. Very weak; off work three weeks.

II. Took ill November 6th. Felt heavy, severe cold in the head. November 7th, severe diarrhœa; chief symptom was headache. Came down November 8th, was sent home. Temperature, 101·4°. Had another sore throat; not sick; had cough; two days in bed. Got up the day the Armistice was signed, November 11th.

(4) Miss C.

I. Took ill on July 18th. Ill, out of sorts, headache and pains. No rise of temperature. Very quick pulse. Went to bed, stayed in bed a week. Very ill three days; temperature, third day 102°. Vomiting, pains in hands and toes; epistaxis third day. Running eyes and nose, cough afterwards; no pains in the chest; no sore throat. Very weak on getting up. Doctor prescribed a week's rest; felt all right when she got up.

II. Home at Llanberis. Began with pains in her chest about October 27th, followed by a cold. No severe headache or general pains. Temperature not taken. Returned to work on November 5th.

It is very doubtful how many of these attacks were really influenzal. Thus it is doubtful whether the second attack was influenza in cases 1, 3, and 4, and not merely common colds, and if that can be said to be so in the case of health visitors, it is much more likely that untrained persons would call every feverish attack influenza.

On the other hand, it has been necessary to make further inquiries at houses to bring the cases up to date, and I am informed that the tendency now is for people who formerly stated that they well ill with influenza in the summer of 1918 to deny that there was then anything the matter with them, being afraid that the continued enquiry implies blame imputed in regard to their previous statements, so easily may these statements swing to one side or another. No doubt their first statements were what they believed, allowing for some natural exaggeration.

But such transference is most likely, at all events as regards cases of such diseases as tuberculosis, enteric fever, scarlet fever, and ordinary catarrh.

The rest of the table of complications may be stated briefly. There is little evidence that influenza caused any rise in the number of deaths from other causes than those named above. We may, while giving the facts, also state what number of deaths were to be expected had the disease exerted the same influence on all of the causes of death as it did in the first outbreak. For this purpose we may multiply the deaths in the first outbreak by 5 to give the expected number in the second, and by 3 to give the expected number in the third.

——		Broncho-Pneumonia.	Pneumonia.	Bronchitis.	Other Respiratory Diseases (mostly Pleurisy).	Tuberculosis.
Actual number	1	64	153	30	6	12
	2	529	722	169	22	45
	3	253	379	133	9	18
Expected number	2	320	765	150	30	60
	3	192	459	90	18	36

——		Meningitis.	Nerve Diseases.	Heart Disease and Diseases of Blood-Vessels.	Digestive System.
Actual number	1	6	3	41	1
	2	28	14	80	16
	3	8	3	70	4
Expected number	2	30	15	205	15
	3	18	9	123	3

We thus see that under other respiratory diseases (mostly pleurisy) the numbers in the second and third outbreak are fewer than we should expect.

Under tuberculosis also the numbers indicate the greater influence of the first outbreak on mortality. The same is true of meningitis and other diseases of the nervous system.

It is, however, under heart diseases that the influence of the first outbreak is most conspicuous, which again may indicate pneumo-coccal septicaemia.

Probably these unexpected facts may be related to the larger numbers attacked in the first outbreak.

It has been said that it is by no means clear that, in the aggregate, influenza had any influence on deaths from tuberculosis. The matter would seem to be easily tested. There were 2,096 from influenza in 1918, and 1,390 deaths from tuberculosis in the year out of a total of 12,181. The chance that a death from tuberculosis would coincide with a death from influenza on the hypothesis of independence of the two is therefore—

$$\frac{2096}{12181} \times \frac{1390}{12181},$$

and the number of deaths which might be expected to coincide is—

$$\frac{2096}{12181} \times \frac{1390}{12181} \times 12181 = 239{\cdot}2.$$

The number to be expected in the six weeks of the first outbreak is—

$$\frac{6}{52} \times 239{\cdot}2 = 27{\cdot}6,$$

in the second—

$$\frac{15}{52} \times 239{\cdot}2 = 69,$$

in the third—

$$\frac{13}{52} \times 239{\cdot}2 = 59{\cdot}8.$$

The actual numbers were 12, 45, and 18. This seems to give some support to the view that there is an actual antagonism between the two diseases. But this is open to fallacy, since deaths from tuberculosis might quite easily be ascribed to concurrent influenza.

These figures need some revision. Strictly speaking, the figures should not be applied to the third outbreak at all. Moreover, the calculation should be restricted to the second half of the year when influenza was prevalent, and when also the mortality from tuberculosis is least. It is, however, clear enough to enable us to say that, in the aggregate influenza had little effect on tuberculosis mortality.

The corrections mentioned, when applied, give the following figures. The chance of influenza occurring in a case of tuberculosis in the second half of 1918 is—

$$\frac{2004}{6342} \times \frac{619}{6342},$$

and the number which may be expected to occur in six weeks out of 6,342 deaths is—

$$\frac{2004 \times 619}{6342} \times \frac{6}{26} = 44,$$

the expected number in 15 weeks is 110. The actual numbers are 12 and 45 respectively.

If we take the experience of the first half year of 1919 the corresponding expected number is 43, the actual number being 18. The results do not differ materially from those already given.

Tables have been prepared showing the numbers of deaths occurring week by week from the 19th week in 1918 to the 19th week of 1919, from influenza, pneumonia, broncho-pneumonia, bronchitis, and tuberculosis. If we refer to the deaths from tuberculosis during the influenza wave periods we find that no definite increase occurs in July 1918, or during the second wave. In this instance the figures for 1919 have not been given.

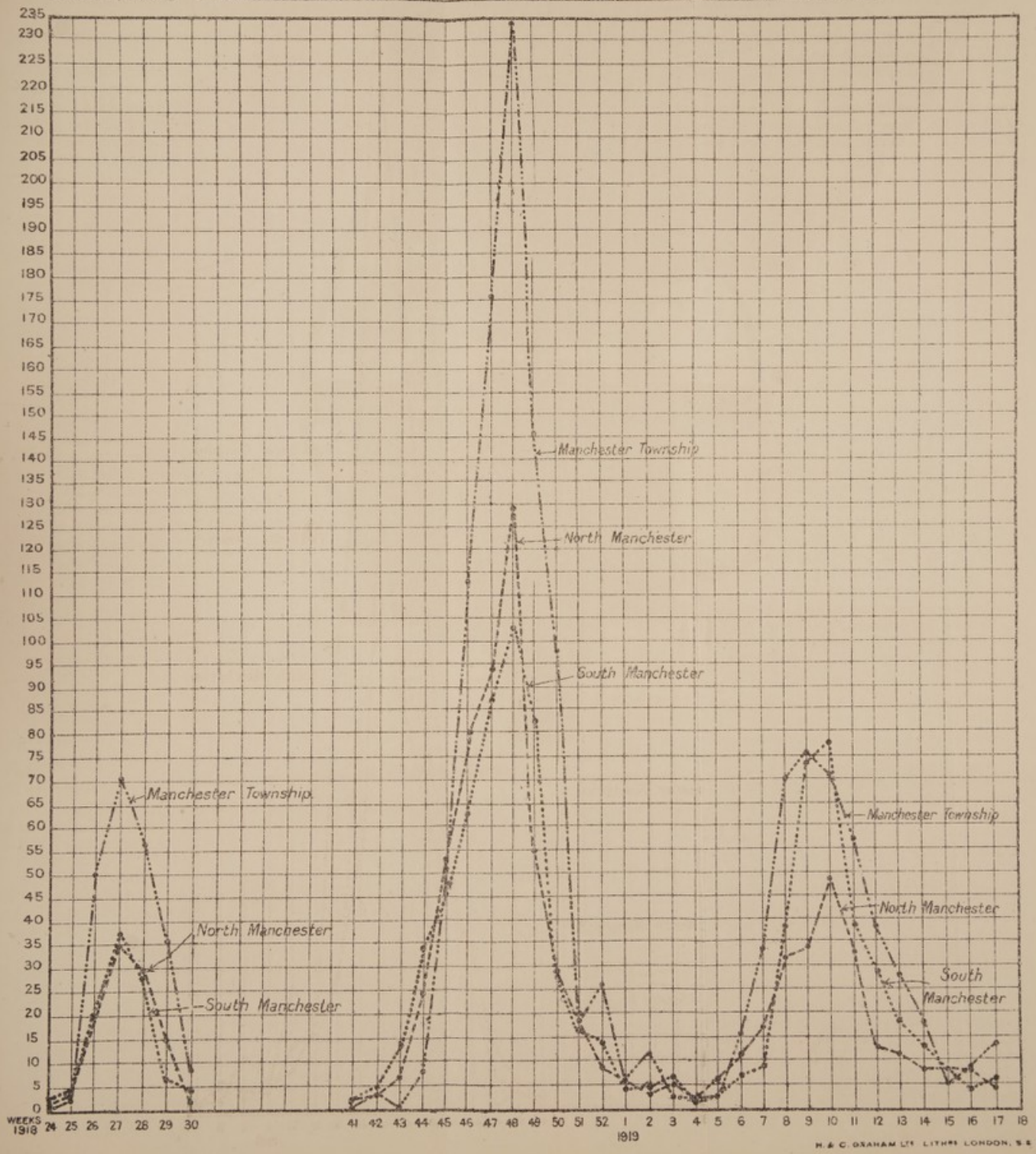
Curves 2.

Curves 2 have been prepared showing the death-rates from influenza in each of the principal divisions of the city from the week ending June 22nd

To face p. 498.

FREQUENCY IN WEEKS OF INFLUENZA DEATHRATES IN THE THREE PRINCIPAL DIVISIONS OF THE CITY FOR EACH WAVE.

CURVES 2 (See p. 507)



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1918, to the week ending April 19th, 1919. Unfortunately the populations have become very uncertain, so that the accuracy of the death-rates is somewhat uncertain absolutely, though not so much relatively. In order to appreciate the constant differences in mortality rates between the three divisions of the city, it is necessary to have figures showing the death-rates from all causes, and from the main causes of death in which large differences are shown every year. The following figures will suffice for this purpose :—

Death Rate.	All Causes.		Pneumonia.		Bronchitis.		Heart Disease.		Tuberculosis.	
	1901.	1917.	1901.	1917.	1901.	1917.	1901.	1917.	1901.	1917
Manchester Township.	27·52	20·63	2·94	2·22	2·55	2·70	2·93	2·72	4·63	3·77
North Manchester.	17·57	11·62	1·64	0·98	1·64	1·18	2·21	2·05	1·63	1·57
South Manchester.	21·08	12·55	2·21	1·10	1·86	1·27	2·50	2·33	2·75	1·87

South Manchester has been extended since 1901, and its death rates ought now to be lower relatively than in that year.

Passing to the frequency curves from influenza it will be seen that the death rates from influenza in the first outbreak rose simultaneously in all three divisions to a peak in the same week, and declined simultaneously. The space between the curves and the base line may be taken as showing the number of deaths per 100,000 in the outbreak.

In the first and second outbreak the relative death-rates from influenza correspond generally to the relative death-rates from respiratory disease, except that we should expect the area embraced by North Manchester deaths to be rather smaller than that embraced by South Manchester deaths. In the first two outbreaks the relation is the reverse of this. This might be taken to indicate some effect of industrial occupation, North Manchester being the industrial part of the city. There is, however, but little difference in mortality between these two divisions. In the third outbreak, however, the South Manchester death-rates attain the same height as those in the Township, although the aggregate mortality per 1,000 is not so great in the former. North Manchester has now a much lower death-rate than either of the other two divisions.

We may conclude that there was exceptional incidence in South Manchester in the third outbreak.

In the second outbreak the mortality rate in the Township does not begin to ascend till a week after the other two divisions, and it commences more abruptly in South than in North Manchester.

This abrupt commencement in South Manchester may have to do with the presence of a number of American soldiers suffering with influenza who were treated on the south side of Manchester in October. It is conceivable that the difference in these curves supplies the explanation of the second outbreak. Large numbers of American soldiers visited this country and France in September and October, and it is known that some ships witnessed very painful scenes, the men suffering from influenza in a very severe form. It appears likely that this outbreak was introduced from America and that an increased virulence was imported at the same time, possibly by a new race of streptococci, or pneumococci, or both, possibly by some as yet unknown micro-organism.

On examination of the Registrar General's weekly return it would appear that the influenza fatality started to ascend at Southampton and Portsmouth, as well as at Exeter, Bath and Gloucester in the week ending October 5th, that is a fortnight before it commenced in Manchester, and that the ascent began in the week ending October 12th in Liverpool, Birkenhead, Bootle, St. Helens, Blackpool and Southport, slightly also in Salford. In Manchester

there was no definite ascent till the week ending October 19th. There was certainly an impression here that the disease travelled from Liverpool, *viâ* Bolton to Manchester, although, as we have seen, there was material in Manchester from which it could have spread. At all events it appeared to start in Southampton, Portsmouth and Liverpool before it reached Manchester. If the suggestion made with regard to its importation from America be the true explanation of the change of character of the outbreak from summer to autumn, the circumstances would be easily explained, since there were a great many American soldiers moving about this country in September and October. This would explain why the disease appeared simultaneously in France and in this country generally, and why it began to be fatal a little before this time at English ports.

When we pass to the third outbreak we find that all three waves embrace a much less area than in the second, and that the curves have somewhat changed their outline, two of them having now flat tops as if the attending respiratory conditions tended to take precedence of the specific infecting agent as a cause of death, thus spreading out the curve.

On this occasion the curve begins to ascend abruptly in the Manchester Township as in the first curve, and the onset of fatality and presumably of spread is delayed in South Manchester, though this is made up for in subsequent weeks. In North Manchester the curve rises and descends more slowly, besides attaining less height than in the other two divisions.

There is nothing in the manner of ascent taken by itself to account for the differences of these curves from those in the first two outbreaks. So far as respiratory diseases is concerned, it is associated with the rise of bronchitis as a cause of death, though it is necessary to remember that, at this season, bronchitis is normally, at its highest point as a cause of death. For this allowance has already been made.

Curves 3.

The third set of curves show the deaths in each week of each outbreak from influenza, pneumonia, broncho-pneumonia and bronchitis as percentages of the total deaths from the particular cause occurring in the course of that outbreak. Naturally the longer curves are flattened. The particular points brought out are:—

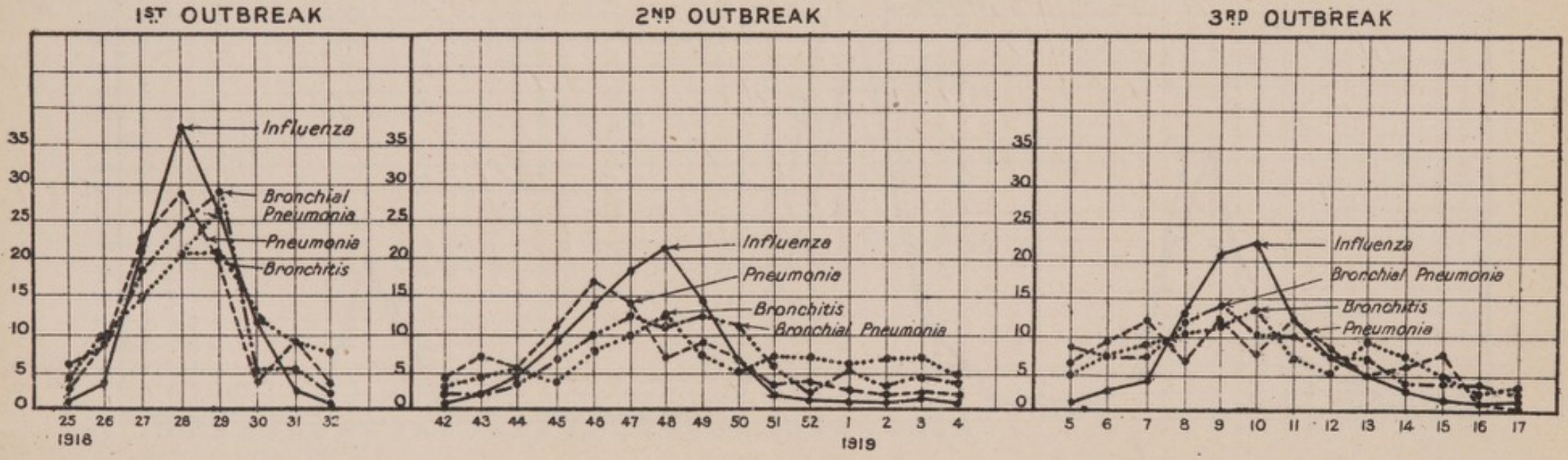
1. Each cause of death above enumerated swells into a wave.
2. The crest of the wave is pointed for influenza.
3. The pneumonia wave is peaked in the first and second outbreak, but appears only as a general swell in the third.
4. The broncho-pneumonia wave is crested in the first outbreak appears as a rounded swell in the second, and as a broken continued ridge in the third.
5. The bronchitis wave in each outbreak rises continuously to a crest simultaneously with the influenza wave, falls more quickly than it rose, and has a dip two-thirds of the way down, after which, in the second and third waves, it rises again. This wave corresponds throughout more closely with the influenza wave than do the other two, and comes into increasing prominence with each successive wave, while pneumonia undergoes a steady decrease, if we exclude broncho-pneumonia.

Curves 4.

Frequency curves for influenza deaths week by week, similar to the foregoing have been constructed for males and females respectively. In the first outbreak male outnumber female deaths, though not by much. In the second the male mortality falls below the female throughout especially in the middle part of the outbreak. In the third the female mortality exceeds the male, though not by much; but throughout the first half of the wave it is in advance of the male. It is evident that the advent of bronchitis as a cause of death does not explain the relative position of the sexes in the third curve, which is like the first, with male exchanged for female deaths. In Manchester female deaths from bronchitis normally exceed male to some extent, and assuming an unknown factor for the greater mortality of females

MODE OF DISTRIBUTION OF THE DEATHS FROM INFLUENZA, BRONCHITIS, BRONCHO-PNEUMONIA, AND PNEUMONIA IN EACH WAVE, THE TOTAL NUMBER OF DEATHS FROM EACH DISEASE IN THE RESPECTIVE EPIDEMICS BEING TAKEN AS 100.

CURVES 3. (See p. 508)



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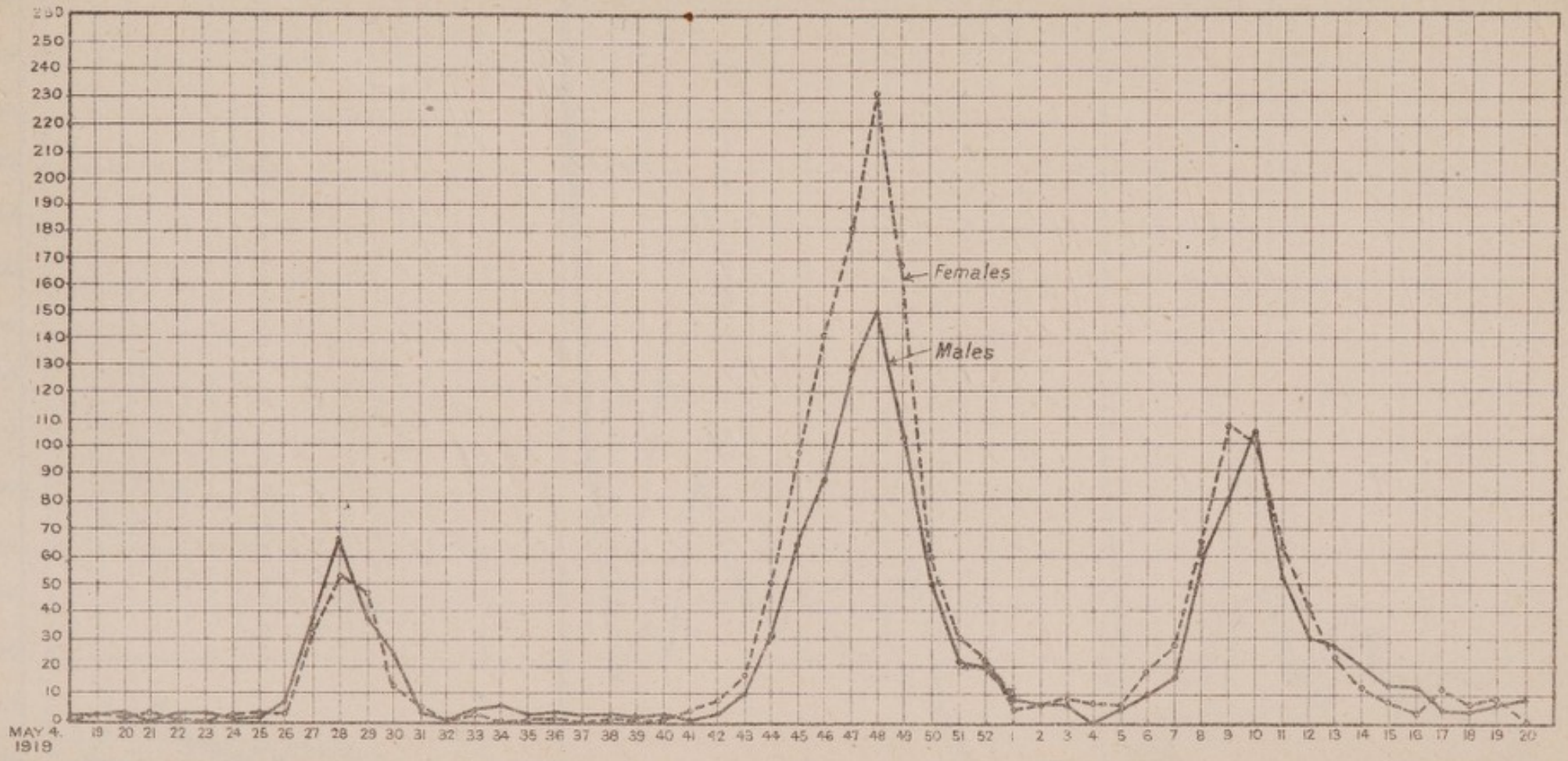
To face p. 500.

INFLUENZA DEATHS. MALES & FEMALES.



To face p. 500.

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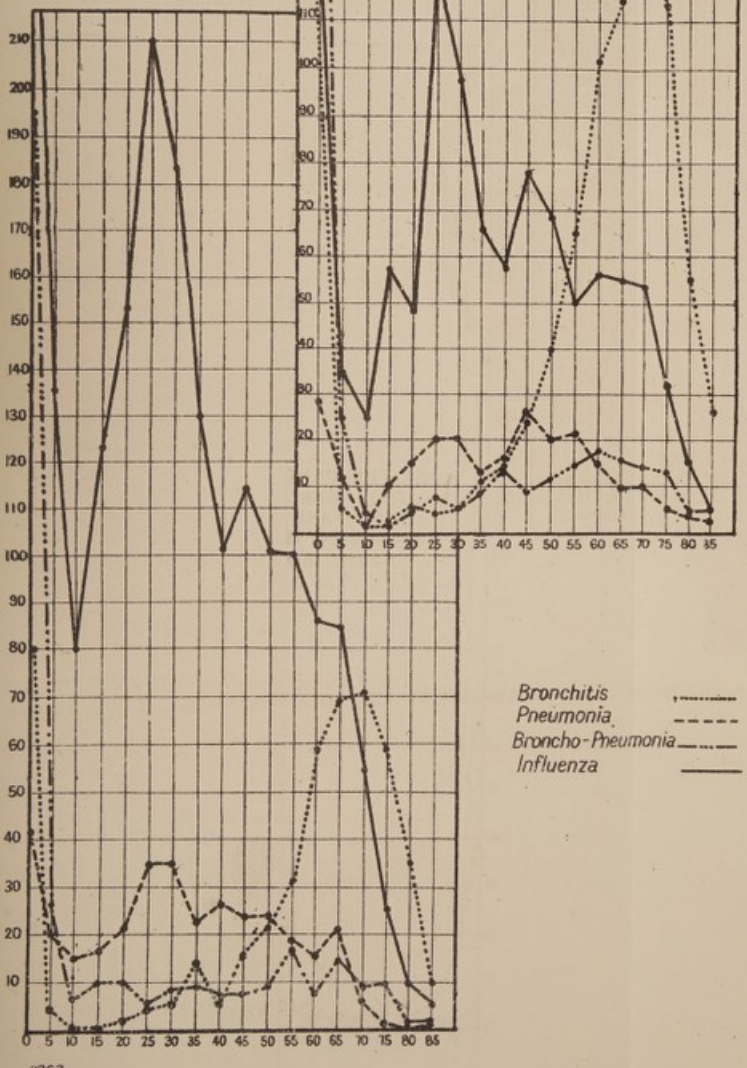
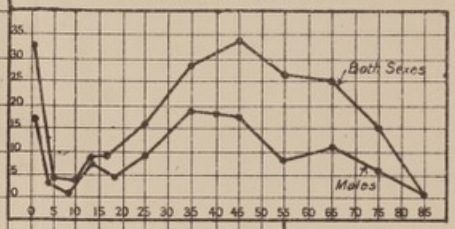
CURVES 6. (See p. 512)

CURVES OF AGE FREQUENCIES OF DEATHS FROM INFLUENZA, PNEUMONIA, BRONCHITIS, BRONCHO-PNEUMONIA. JAN–JUNE. 1919.

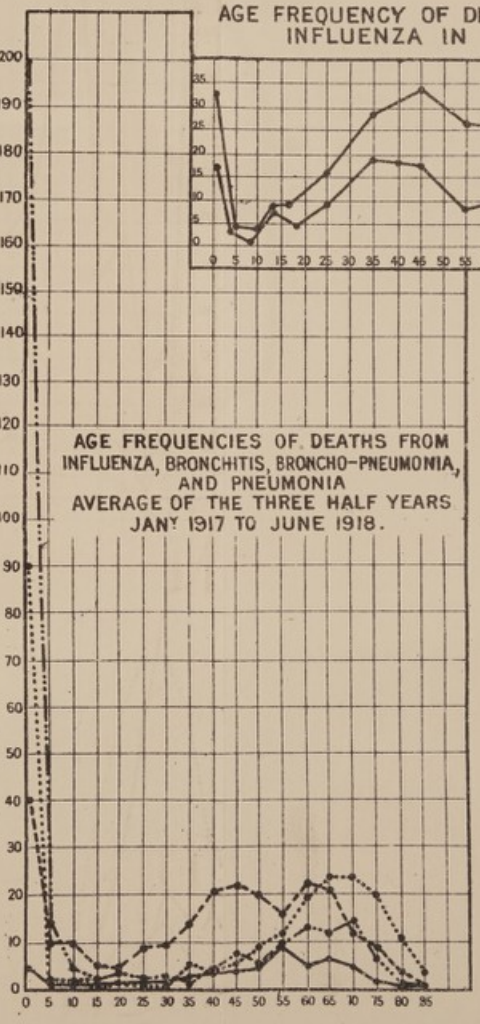
CURVES OF AGE FREQUENCIES OF DEATHS FROM INFLUENZA PNEUMONIA, BRONCHITIS, BRONCHO-PNEUMONIA JULY–DEC^{R} 1918



AGE FREQUENCY OF DEATHS FROM INFLUENZA IN 1891.



AGE FREQUENCIES OF DEATHS FROM INFLUENZA, BRONCHITIS, BRONCHO-PNEUMONIA, AND PNEUMONIA AVERAGE OF THE THREE HALF YEARS JAN^{Y} 1917 TO JUNE 1918.



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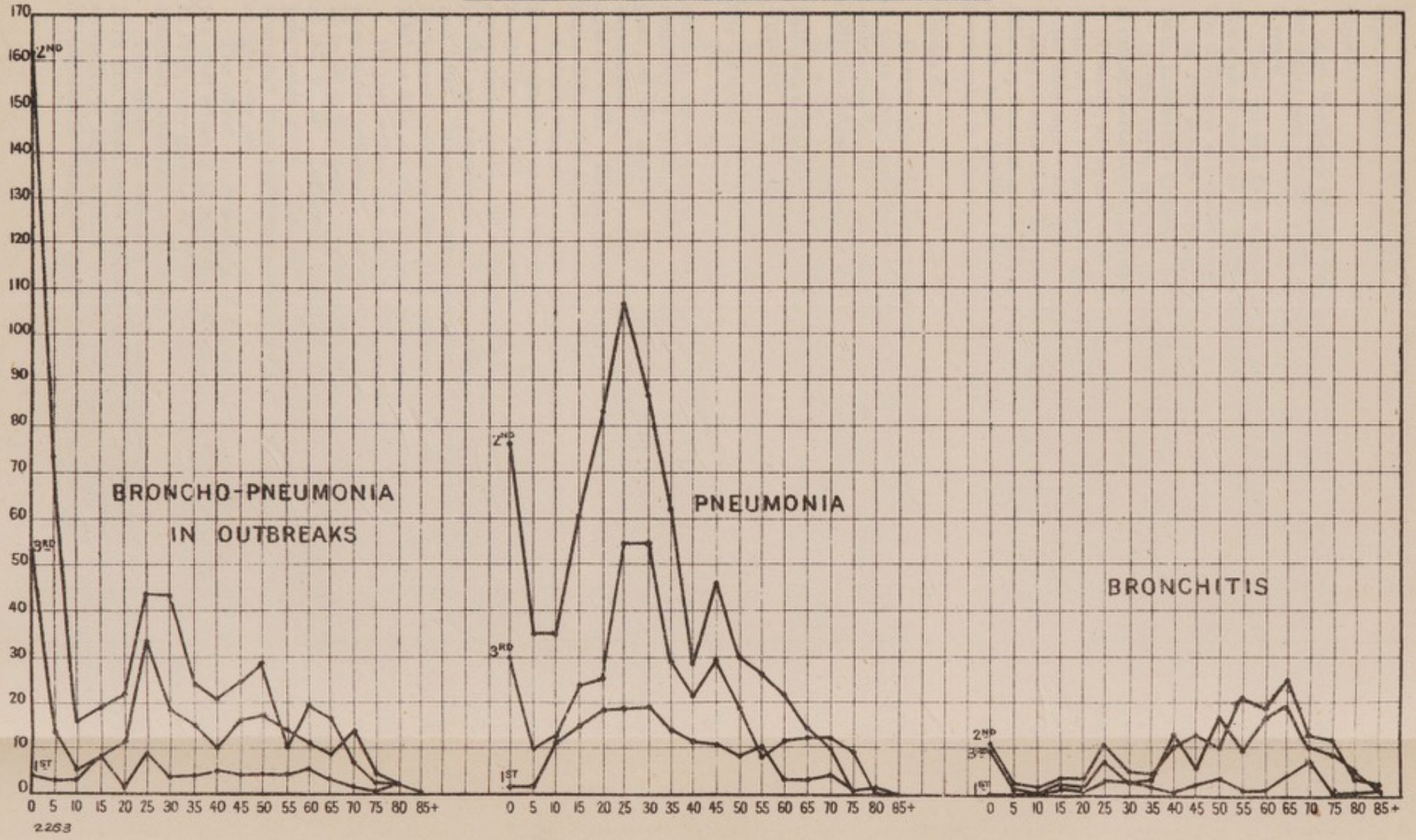
AGE FREQUENCES OF THE CHIEF COMPLICATIONS IN DEATHS REGISTERED AS FROM INFLUENZA, IN EACH OF THE THREE OUTBREAKS, SUMMER AND AUTUMN 1918 AND WINTER 1919.



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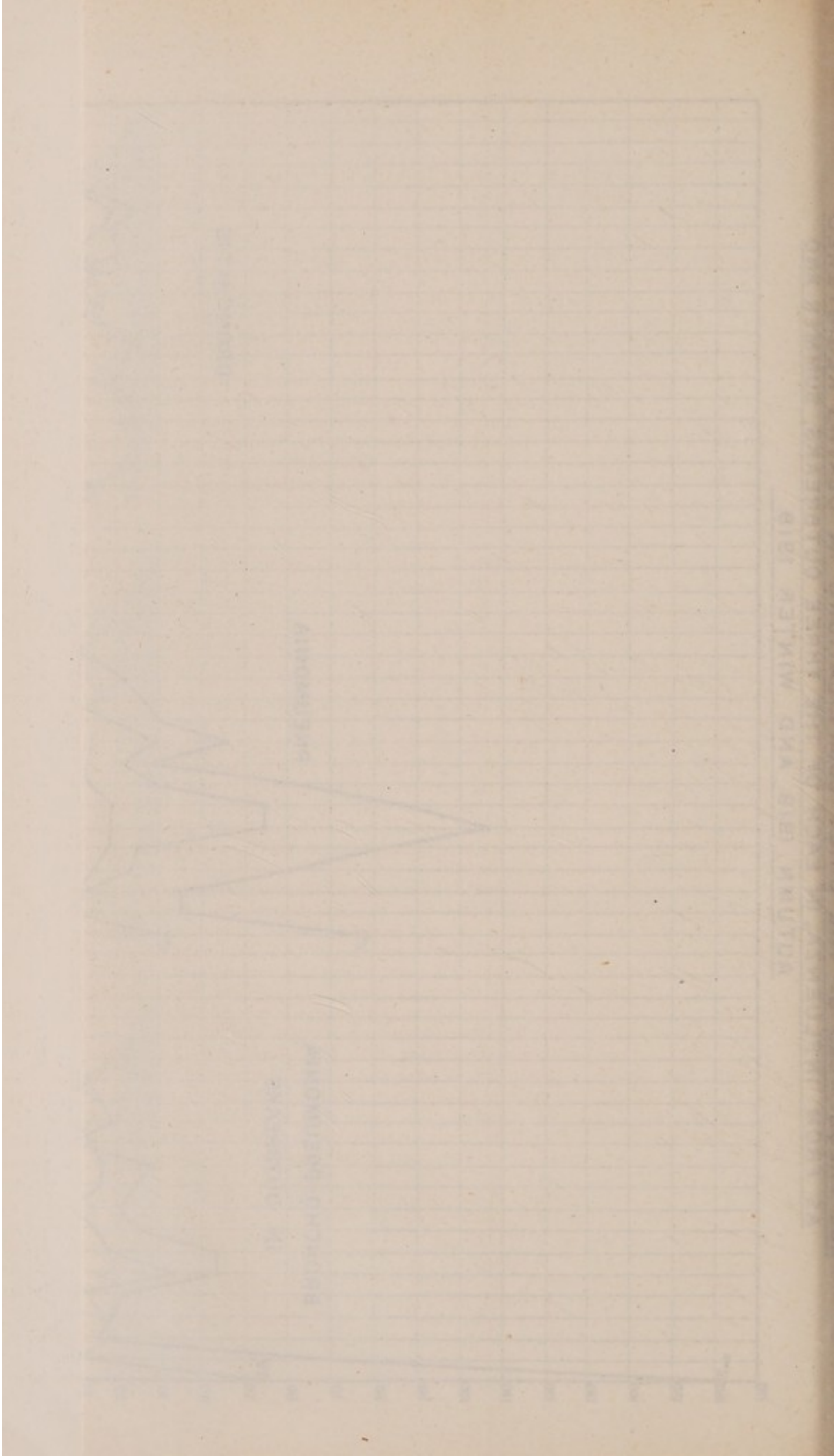
To face p. 500



in autumn, we might regard the third curve as a partial return to the first type, modified by bronchitis.

Curves 5.

Age frequency curves have been constructed for each of the outbreaks for males and female respectively, at 18 groups of ages, each of a period of 5 years.

That for the first outbreak shows that the female mortality rises above the male at ages 10 to 30, and falls below it at ages 45 to 85. In both cases the difference is considerable.

In the second outbreak the female mortality rises greatly above the male, immensely at ages 0 to 45, but also considerably up to the age 75.

In this curve, broncho-pneumonia shows signs of being a potent factor, if infant mortality be any test.

Usually, at ages 0 to 4, broncho-pneumonia is more fatal to males than females; but the excessive fatality of females in the second outbreak affects even this age. All these curves, males and females, show a maximum fatality at ages 25 to 29; and any explanation of this epidemic must explain this very unusual and unlikely occurrence. The only exceptions to this rule are the high fatalities in infancy in the second outbreak, and the fatality from males in infancy in the third outbreak.

Curves 6.

Curves showing the number of deaths occurring at each age group of 5 years have also been constructed for July to December, 1918, thus embracing the first and second outbreaks, and for the first half-year of 1919, for influenza, pneumonia, broncho-pneumonia, and bronchitis.

In order to afford a basis of comparison with the normal, the three half-years, January 1917 to June 1918, have been averaged, and corresponding frequency curves prepared.

Also a curve has been prepared showing the age frequency of deaths in the influenza year 1891.

The two first set of curves show the high death rate from influenza in children under 5 years, especially in 1918. The peak at ages 25 to 30 is manifest in both curves. Both curves show a secondary, but much lower peak, at the ages 45 to 50. This low peak corresponds to the influenza experience of 1891, which shows nothing to correspond with the peak at ages 25 to 30. These outbreaks are, therefore, in some essential particular, quite different from the wave in 1891.

Indeed, when we compare the 1891 experience with the curves constructed for comparison and control, we see that the age distribution of influenzal mortality in 1891 corresponds very much with that of pneumonia in the comparison curves. It is permissible to suppose that the dominant influence in 1891 was pneumonia, but that other and disturbing factors enter in 1918 and 1919. Moreover, the age-frequency curves of influenza for the three half-years preceding the first influenza outbreak, correspond with that of 1891, as does also that for the interval between summer and autumn.

In the curves for July to December 1918 and January to June 1919 pneumonia reaches a maximum at ages 25 to 35 in the former period, at 25 to 30 in the latter, though in the latter it attains a still higher maximum at 45 to 50. Influenzal infection has been able to distort the normal pneumonia curve. This will be made clear by the comparison curve of age frequency from pneumonia; but the effect of influenza is but little manifest at ages over 40.

The effect on bronchitis of the influenzal wave is very different. Here the chief effect is to raise the natural rise of this part of the curve, greatly in 1918, very greatly in 1919, though the base of the enlargement is also extended, especially in the latter.

The effect of influenza on broncho-pneumonia is again different. In 1918 the whole curve is lifted up, except at ages 0 to 4, at which the mortality is high, with or without influenza. At all other ages the impress of influenza is manifest, particularly in the earlier ages. The character of

the age distribution is quite altered. In the first half of 1919 the mortality from broncho-pneumonia at ages 0 to 4, reaches the high figure of 237, but this cannot be ascribed to influenza, especially as the effect of influenza does not begin to be evident until the age 20 to 25. After that, it is general, and affects all ages.

The frequency curves in weeks of the three main complications of influenza have been already dealt with.

From a consideration of all these curves, we see—

1. That the true influenzal curve has to be constructed by subtracting the deaths at each group of ages in the control curve from the corresponding group of influenzal curves from pneumonia, broncho-pneumonia, and bronchitis, and adding the number thus obtained to the number of deaths ascribed to influenza at the corresponding ages.

2. The identity of bronchitis with influenza is very easily overlooked, as may be seen on examining the complications of influenza, and the curves for bronchitis in July to December 1918, and January to June 1919. That is to say, a severe cold cannot easily be distinguished from influenza. Tne essential identity of the curves of influenza and bronchitis has already been pointed out. It appears likely, as already stated, that a study of common colds will throw light on the occurrence of influenza.

3. The proposed change of the curve of age frequencies of deaths from influenza will not, however, alter the peaks at ages 25 to 30 and 30 to 35.

4. The difference in sex distribution of the deaths in the three epidemics seems to indicate some fundamental difference in character, but might, if all the factors were known, receive some explanation from the amount of immunity in the two sexes. This does not, however, appear likely to give an adequate explanation. Other differences in the character of the epidemics have already been mentioned.

The most probable explanation is the introduction of fresh strains or elements.

If we may suppose, for example, that the infective element in influenza attached itself in succession to the pneumococcus, to a streptococcus, and to the influenza bacillus, the occurrence and behaviour of the outbreaks might receive some explanation; but these must be matters for further enquiry.

5. The whole group of respiratory affections needs to be brought under enquiry.

Various accessory inquiries arise in connection with these facts. As streptococci were such a prominent feature in the bacteriology of the disease, it is natural to inquire whether any increase occurs amongst the notified cases of erysipelas, puerperal fever, or scarlet fever. No such occurrence shows itself, as will be seen from the following figures:—

Cases notified in Quarters from—

Erysipelas.				Puerperal Fever.			
—	1917.	1918.	1919.	—	1917.	1918.	1919.
1	52	81	44	1	19	15	22
2	45	65	70	2	23	18	26
3	58	63	98	3	12	25	58
4	77	71	—	4	15	19	—

Thus the increases which occur are in periods outside influenza prevalence, and they are particularly marked in the second half of 1919, as is the the rise of scarlet fever, or appears to be so. This qualification is necessary, as the prevalence of influenza might cloak a rise in all three. But the suggestion already made, that the infective element in influenza may give wings to the infections of other diseases, may have application to scarlet fever, erysipelas, encephalitis-lethargica and other epidemic diseases of short latent period. With this idea we may connect the tendency to epidemicity of other infectious diseases round about influenzal periods.

It is evident that during an outbreak, pneumococci, streptococci and influenza bacilli all gain greatly in infectivity and numbers. It is possible that they become infective by associating themselves with a more minute organism. In that case there would be some restriction on spread by the increased size of the infecting agent.

We may, therefore, bring together the facts available, without attempting meanwhile to interpret them further.

It appears to be worth while also to bring together the periods between the occurrence of primary and secondary cases, with a view to see whether there is any evidence of agencies with different latent periods. The facts are collected without attempting to draw conclusions which would be difficult, unless one also knew the degrees of exposure to infection.

Influence of Severity of Disease of Infectivity.

When a number of deaths are investigated it would at first sight appear as if there were an unduly large number of households with single cases in them. These investigations do not quite all relate to deaths, but they do all relate to severe cases.

The figures obtained from them are :—

Households having Cases.	1.	2.	3.	4.	5.	6.	7.	Total.
Number of households.	234	101	57	30	12	4	2	440
Number of persons.	1,083	513	326	186	78	32	19	2,237
Average number of persons per house.	4·63	5·01	5·72	6·2	6·5	8	9·5	—

Thus in 234 households there is only one case, while in 206 there is more than one case.

Advantage has been taken of the block census made for Dr. Carnwath to control this by ascertaining what occurs in houses as they come along. The figures relate mostly to the autumn outbreak with a few from early winter.

Households with Cases.	1.	2.	3.	4.	5.	6.	7.	Total.
Number of above	162	77	37	10	5	2	2	295
Deaths included above.	7	1	2	—	—	—	—	10

It would thus appear that while more than half the total number of households have as before only one case, amongst those having only one case there are seven deaths as against three deaths amongst the rest of the cases. Notwithstanding the similarity of experiences in the two sets of

households there appears, therefore, to be association between the severity of the case and the failure of the disease to spread. It does not, of course, follow, supposing severity to restrict propagation of the disease, that there should be any difference in the two experiences, nor would there be if the first case were invariably slight. But this is not the case. The question cannot be determined in this way.

A portion of the attacks were therefore analysed, restricting those selected to the autumn outbreak, and putting aside the instances in which a death occurred. The remainder were grouped as before, according to the number of cases in the household, but the classification is according to the character of the attack in the first case.

Character of Attack in the first Case.	Number of Cases in Household.							
	1.	2.	3.	4.	5.	6.	7.	Total.
Broncho-pneumonia.	2	—	1	—	—	—	—	3
Pneumonia -	9	—	2	—	1	—	—	12
Otherwise severe	13	4	3	—	—	—	—	20
First case not severe.	86	59	26	6	4	3	1	185
Total -	110	63	32	6	5	3	1	220

Thus the total number of households in this group was exactly halved by the number in which there was only one case in the house. But among those in which only one case occurred, viz., 110, the first case was severe in 24, with two cases there were four severe first cases out of 63, with three cases six severe cases out of 32, of the remainder one out of 14.

Summing up, there were 24 severe first cases where only one was attacked out of 110, 11 severe cases out of 110 where more than one was attacked.

When this is added to the experience regarding fatality, a case seems to be made out for association of severity of first case with infectivity.

The point is one of some importance.

Supposing such an association to be made out, the infectivity of the patient is restricted by his being confined to his bed. It is difficult to believe that it is otherwise much restricted on the average. The restriction is plainly on his power of infecting others by handling towels, washbasins, utensils, food, and might be greater than is here shown, since the aggregate judgment of a case which is not severe gives no sufficient picture of the commencement of attack. Experience of the way in which the sick are cared for shows that the danger from droplet infection is at least as great from those confined to bed as from those who can get up, while the danger from the handling of utensils, though much diminished, is by no means abolished.

So far as they go, the figures seem to suggest the importance of severe illness in restricting infection, whatever be the explanation.

But that this is not an adequate or complete explanation of the failure in many cases of influenza to spread in the household is shown by the large number of single cases whose attacks were not severe.

Interval between Exposure and Development of the Disease.

This has been taken out for 278 instances, which relate almost entirely to households in which a death occurred, but in all of which severe cases have occurred.

The previous analysis shows that the results will not differ greatly from those obtained as the result of a census.

These results are as follows :—

Interval in days between First and Second Attack.

——	0	1	2	3	4	5	6	7	8	9	10	11
Number of cases occurring in each group.	48	39	46	26	18	17	18	18	6	3	6	5

——	12	13	14	15	16	17	18	19	20	20 and above.
Number of cases occurring in each group.	3	6	4	2	2	0	1	0	2	8

It thus appears that the intervals are gathered in a group in the first four days, then follows a group from the fourth to the seventh days inclusive, in which there is a marked fall from the first four days, but in which the intervals stand at a level, indicating apparently a different level of infectivity. After this point there is a drop, the intervals again remaining fairly level for another week, after which a marked drop again occurs.

Although this may be taken as fairly representative of what happens, it needs to be checked by an indifferent group, irrespective of severity of individual cases occurring in the household.

The Significance of the Incidence on Ages 25–29 *and* 30–34.

Perhaps undue stress has been laid on the incidence of mortality at the above ages. The most evident suggestion is that a large degree of immunity remains over from the epidemic period 1889–1891. This explanation has to contend with the apparent slightness of protection conferred on persons attacked in the summer and autumn outbreaks. Even if we assume that older persons may have been protected by repeated attacks in the previous epidemic years, all the features of age incidence would not be explained.

CURVES 1.—*Number of Deaths Week by Week from the Causes given below in the Weeks named.*

1918.

——	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
Influenza	2	1	2	3	2	2	2	9	71	120	85	38	9	2	5	4	2
Pneumonia	14	16	16	13	12	5	5	12	33	42	29	7	13	4	6	6	5
Broncho-pneumonia	8	11	14	11	10	4	5	7	16	21	24	5	5	2	1	2	1
Bronchitis	16	10	15	11	8	8	6	12	19	27	28	14	11	9	6	2	8

1918—*cont.*

——	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
Influenza	4	1	3	—	3	2	9	29	83	163	229	313	383	250	109	55	42
Pneumonia	4	3	8	2	4	5	9	15	12	22	34	28	14	19	10	7	7
Broncho-pneumonia	3	2	4	4	4	2	6	6	14	21	31	41	35	41	32	19	10
Bronchitis	6	7	8	16	10	10	9	15	21	19	34	44	55	35	22	29	29

1919.

——	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Influenza	14	14	16	6	11	28	44	126	191	206	114	74	52	35	20	17	18	6	12
Pneumonia	9	12	9	2	10	15	19	13	22	15	21	15	9	9	11	3	3	7	10
Broncho-pneumonia	16	11	15	12	24	21	23	40	41	31	31	22	21	12	14	14	9	11	9
Bronchitis	24	29	28	26	36	43	56	67	77	87	50	41	55	39	32	23	23	24	24

CURVES 2.—*Death Rates from Influenza in the Manchester Township, North Manchester and South Manchester, in the Weeks shown above, to the nearest decimal.*

1918.

——	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
Township - - - - -						0	0·5	10	14	11	7	2					
North Manchester - - - -						0	0·5	4	7	6	3	0					
South Manchester - - - -						0·2	0·7	4	7	4	1	1					

1918—*cont.*

——	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
Township - - - - -						0·0	0·5	0	1·5	10	23	35	47	29	19	4	5
North Manchester - - - -						0·2	0·5	1·2	5	11	16	19	27	11	6	4	2
South Manchester - - - -						0·1	0·6	2·5	7	10	13	17	21	17	5	3	3

1919.

——	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Township - - -	1	2	·5	·5	·5	3	7	14	15	14	12	8	6	4	1	2	3		
North Manchester -	1	1	1	·5	1	2	3	7	8	10	6	3	2	2	1·4	1	1		
South Manchester -	1	1	1	·4	·6	1	2	8	15	16	8	6	4	3	1·4	1	1		

CURVES 3.—*First Wave Percentages.*

Weeks.	Influenza.		Pneumonia.		Broncho-Pneumonia.		Bronchitis.	
	Deaths.	Percentages.	Deaths.	Percentages.	Deaths	Percentages.	Deaths.	Percentages.
25	3	0·89	5	3·45	5	5·88	6	4·76
26	9	2·67	12	8·27	7	8·24	12	9·52
27	71	21·07	33	22·76	16	18·82	19	15·08
28	120	35·61	42	28·97	21	24·71	27	21·43
29	85	25·22	29	20·00	24	28·24	28	22·22
30	38	11·28	7	4·83	5	5·88	14	11·11
31	9	2·67	13	8·97	5	5·88	11	8·73
32	2	0·59	4	2·76	2	2·35	9	7·14
TOTAL	337	—	145	—	85	—	126	—

Second Wave Percentages.

Weeks.	Influenza.		Pneumonia.		Bronchitis.		Broncho-Pneumonia.	
	Deaths.	Percentages.	Deaths.	Percentages.	Deaths.	Percentages.	Deaths.	Percentages.
42	9	0·53	9	4·31	9	2·15	6	1·94
43	28	1·65	15	7·18	15	3·58	6	1·94
44	81	4·76	12	5·74	21	5·06	14	4·52
45	163	9·58	22	10·53	19	4·53	21	6·77
46	229	13·46	34	16·27	34	8·11	31	10·00
47	313	18·39	28	13·40	44	10·50	41	13·23
48	373	21·92	14	6·70	55	13·13	35	11·29
49	250	14·69	19	9·09	35	8·35	41	13·23
50	109	6·40	10	4·78	22	5·25	32	10·32
51	55	3·23	7	3·35	29	6.92	19	6·13
52	42	2·47	7	3·35	29	6·92	10	3·23
1919								
1	14	0·82	9	4·31	24	6·73	16	5·16
2	14	0·82	12	5·74	29	6·92	11	3·55
3	16	0·94	9	4·31	28	6·68	15	4·84
4	6	0·35	2	0·96	26	6·21	12	3·87
TOTAL	1702	—	209	—	419	—	310	—

Third Wave Percentages.

Weeks.	Influenza.		Pneumonia.		Bronchitis.		Broncho-Pneumonia.	
	Deaths.	Per-centages.	Deaths.	Per-centages.	Deaths.	Per-centages.	Deaths.	Per-centages.
5	11	1·16	10	6·06	36	5·72	24	7·92
6	28	2·99	15	9·09	43	6·84	21	6·93
7	44	4·70	19	11·52	56	8·90	23	7·59
8	126	13·46	13	7·88	67	10·65	40	13·20
9	191	20·41	22	13·33	77	12·24	41	13·51
10	206	22·01	15	9·09	87	13·83	31	10·23
11	114	12·18	21	12·73	50	7·95	31	10·23
12	74	7·91	15	9·09	41	6·52	22	7·26
13	52	5·56	9	5·45	55	8·74	21	6·93
14	35	3·74	9	5·45	39	6·20	12	3·96
15	20	2·14	11	6·67	32	5·09	14	4·62
16	17	1·82	3	1·82	23	3·66	14	4·62
17	18	1·92	3	1·82	23	3·66	9	2·97
	936	—	165	—	629	—	303	—

CURVES 4.—*Influenza Deaths, Males and Females, from 18th Week* 1918 *to 19th Week* 1919.

1918.

———	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.
Males - - - -	1	1	1	0	2	2	1	1	7	36
Females - - -	0	1	0	2	1	0	1	1	2	35

1918—*cont.*

———	28.	29.	30.	31.	32.	33.	34.	35.	36.	37.
Males - - - -	66	39	25	4	1	3	4	2	3	1
Females - - -	54	46	13	5	1	2	0	0	1	0

1918—*cont.*

———	38.	39.	40.	41.	42.	43.	44.	45.
Males - - - -	2	0	2	0	3	11	33	65
Females - - -	1	0	1	2	6	18	50	98

1918—*cont.*

	46.	47.	48.	49.	50.	51.	52.
Males - - - -	88	130	150	103	51	23	20
Females - - -	141	183	233	147	58	32	22

1919.

	1.	2.	3.	4.	5.	6.	7.	8.	9.
Males - - - -	8	7	8	2	5	9	17	61	82
Females - - -	6	7	8	4	6	19	27	65	109

1919—*cont.*

	10.	11.	12.	13.	14.	15.	16.	17.	18.
Males - - - -	106	52	31	28	22	12	13	6	2
Females - - -	100	62	43	24	13	8	4	12	4

CURVES 5.—*Frequency Curves of Deaths in Age Groups, Male and Female, each of 3 Outbreaks.*

SUMMER.

	0–5.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.
Males - - - -	10	5	9	12	8	20	16	13	13	9
Females - - -	4	3	8	16	19	22	15	14	10	14

	50–55.	55–60.	60–65.	65–70.	75–80.	75–80.	80–85.	85 and above.	Total.
Males - - - -	15	13	12	8	9	3	1	1	177
Females - - -	6	6	5	6	6	—	1	—	155

AUTUMN.

	0–5.	5–10.	10–15.	15 20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.
Males - - - -	142	62	27	37	32	66	48	40	27	47
Females - - -	162	68	38	59	95	115	107	65	43	46

	50–55.	55–60.	60–65.	65–70.	70–75.	75–80.	80–85.	85 and above.	Total.
Males - - - -	36	29	35	25	20	14	4	1	702
Females - - -	44	49	36	48	22	6	6	4	1,013

WINTER.

	0-5.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.
Males - - - -	70	17	9	21	17	41	43	25	32	42
Females - - -	39	14	13	27	27	71	51	32	21	31

	50–55.	55–60.	60–55.	65–70.	70–75.	75–80.	80–85.	85 and above.	Total.
Males - - - -	28	23	21	22	18	10	5	—	444
Females - - -	36	21	28	28	25	19	8	1	492

For Curves 6 *see* following page.

Comparison of the above Facts with those obtained from Block Censuses.

In January 1919 a block census was made in Manchester by the Health Visitors at the request and under the direction of Dr. T. Carnwath, a medical officer of the Ministry of Health, with a view to ascertaining the facts as regards incidence on households and persons of attacks of influenza in the summer and autumn outbreaks, also as regards immunity, if any, conferred by the summer outbreak of persons then attacked and living in the autumn outbreak. To these have now been added the facts for the winter outbreak. In addition a control census has been taken so as to ascertain how representative a block census for 1,000 houses is.

The Summer outbreak lasted from June 22, 1918, to August 3, 1918.
„ Autumn „ „ „ October 12, 1918, to January 26, 1919
„ Winter „ „ „ January 27, 1919, to March 10, 1919.

The facts are arranged in five-yearly age groups, males and females, as in the tables below. (Tables E. and F.)

The figures have been very carefully corrected, and the slight difference between the total and that of Dr. Carnwath is no doubt due to exclusion by him of some of the cases.

The figures are arranged in these groups so as to furnish a comparison with the curves for age frequencies of deaths for the whole city.

The figures at the side of Table C. mean—

1 escaped in summer; 2 attacked in summer; 11 escaped summer and autumn; 12 escaped summer, attacked autumn; 21 attacked summer, escaped autumn; 22 attacked both in summer and autumn, and so forth. Thus 121 means escaped summer, attacked autumn, escaped winter. * means death in summer; † in autumn; ‡ in winter.

CURVES 6.—*Deaths from Influenza, Pneumonia, Broncho-Pneumonia, and Bronchitis—July to December*, 1918.

	0–5.	5-10.	10-15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.	50–55.	55–60.	60–65.	65–70.	70–75.	75–80.	80–85.	85 and above.	Total.
Influenza - - -	301	136	80	123	154	210	183	130	102	117	101	100	87	85	54	24	11	6	2,004
Pneumonia - -	42	20	15	17	22	35	35	22	27	24	24	18	16	22	7	2	0	0	348
Broncho-pneumonia	197	26	7	10	10	5	8	9	6	7	9	17	7	15	9	10	0	0	352
Bronchitis - -	81	5	2	0	2	4	6	14	5	16	23	32	58	69	72	59	35	10	493
January–June 1919.																			
Influenza - - -	126	35	23	58	48	120	98	65	57	79	68	49	57	55	48	31	15	1	1,033
Pneumonia - -	29	12	1	12	15	22	20	13	16	27	19	21	15	10	10	4	3	1	250
Broncho-pneumonia	237	24	3	1	4	8	6	9	14	9	12	15	17	16	14	13	3	3	408
Bronchitis - -	121	5	1	1	4	4	6	11	19	29	42	66	103	116	131	114	55	26	854
January 1917–*June* 1918—*Control.*																			
Influenza - - -	14	5	4	2	2	6	5	8	6	13	14	26	16	21	15	8	4	4	173
Pneumonia - -	120	26	30	17	14	28	28	43	63	65	64	51	70	65	37	28	9	1	759
Broncho-pneumonia	598	40	11	4	8	2	6	7	12	21	16	23	38	36	42	20	3	3	890
Bronchitis - -	266	12	4	0	3	17	14	32	45	53	80	121	172	208	230	194	96	43	1,590
Influenza, 1891 -	29	4	3	9	10	32		58		66		54		51		30		1	—

CURVES 7.—*See* Table of Compilations.

The figures are arranged first without removal of deaths to facilitate checking the figures and the deaths are shown in their place in summer and autumn. In the winter outbreak 111 to 222, all the deaths are shown in their places.

The figures are then re-arranged (Table "C") by omission of deaths in the preceding outbreak, and the deaths are shown in their appropriate places in summer, autumn and winter. There is no material advantage in this correction of the populations as much greater changes occur owing to the interval between one outbreak and another. However, using the re-arranged figures, and glancing over the table, we note that there were two deaths in autumn in persons previously attacked in summer, and one in winter amongst those attacked both in summer and winter. Table C. relates to the second block census. The corresponding table (Table A) for the first block census is not reproduced.

The case mortality rate in summer is 0·71, in autumn 2·75, in winter 2·66. It will be seen that, so far as this census goes, the attacks in autumn and winter were about equally severe This table affords no evidence of protection acquired by cases attacked in summer against attack in autumn. Thus, the chance of a person being attacked in autumn who escaped in summer is $\frac{403}{3,977}$, of a person being attacked in autumn who was attacked in summer $\frac{70}{709}$. The difference is negligible.

We may next enquire what is the evidence of protection in winter for those attacked in summer. The chance of a person being attacked in winter who escaped in summer and autumn is $\frac{(112)}{(11)} = \frac{73}{3574}$. The chance of a person attacked in summer, but not in autumn being attacked in winter is $\frac{(212)}{(21)} = \frac{26}{634}$ which is considerably greater than the above fraction. There would, therefore, appear from these figures to be no protection, but the reverse. The result is not altered if we take instead of the last fraction—

$$\frac{(212)}{(21)+(22)} = \frac{26}{709}.$$

As regards protection in winter of those attacked in autumn, the chance of a person being attacked in winter who was attacked in autumn but not in summer is $\frac{(122)}{(12)} = \frac{11}{403}$, which again is greater than $\frac{73}{3574}$. There is, therefore, the reverse of protection.

We may now examine the distribution of persons in the population. We note the great preponderance in the population of women over men, at ages 15 to 34, and the remarkable reduction amongst both men and women at ages 20–24 (compared with all other ages).

As regards incidence of the disease we note the excessive incidence on females, even taking into account the differences in population, from the age of 10 upwards. This is not reflected in the numbers of death for the whole population in the first outbreak, and to that extent this census is not representative. Doubtless the reason why it is not reflected is because of the special connection between the first outbreak and lobar-pneumonia, a connection which may have been particularised in districts. As is well known lobar-pneumonia is more fatal to males than to females.

This excessive incidence on females is shown in the second outbreak. It is, however, absent in the principal section of the third outbreak, viz.:— 112), though again present in the sections (122) (212) and (222), as might be expected.

TABLE "C."

MANCHESTER.

INFLUENZA, 1918–19. (Second Block Census.)

KEY:—

1 { 11 { 111 Escaped infection.
112 Escaped Summer and Autumn, attacked Winter.
12 { 121 Escaped Summer and Winter, attacked Autumn.
122 Escaped Summer, attacked Autumn and Winter.

2 { 21 { 211 Attacked Summer.
212 Attacked Summer and Winter, escaped Autumn.
22 { 221 Attacked Summer and Autumn, escaped Winter.
222 Attacked Summer, Autumn, and Winter.

——	Sex.	0–1.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40	40–45.	45–50.	50–55.	55–60.	60–65.	65–70.	70–75.	75–80.	80–85.	85 and above.	Totals.
1	M.	105	72	255	351	284	186	76	137	157	180	155	114	89	47	33	21	15	5	3	1	2,286
	F.	90	82	274	340	332	227	212	235	206	210	148	108	79	72	44	27	20	11	8	1	2,726
2	M.	1	2	18	52*	56	37	12	28	34*	33	30*	16*	10	4	3	1	1				338
	F.	2	1	21	61	61	51	41	69	65	56*	31	22	9	13*	4	7	4	4			522
11	M.	99	67	231	320	264	171	71	127	146	163	143	104	85	45	27	21	15	5	3	1	2,108
	F.	83	74	238	300	286	205	188	199	167	184	131	93	72	65	40	24	20	10	8	1	2,388
12	M.	6†	5	24††	31†	20†	15	5	10	11	17	12†	10	4†	2	6						178
	F.	7††	8	36†	40	46	22†	24†	36†	39††	26	17†	15	7	7†	4†	3†		1†			338
21	M.	1	2	18	42	51	35	12	27	29	31	24	14	10	4	3	1	1				305
	F.	2		19	59	58	47	36	67	57	51	26	21	9	10	4	7	4	4			481
22	M.				9†	5	2		1	4	2	5	1									29
	F.		1†	2	2	3	4	5	2	8	4	5	1		2							39

111	M.	97	65	230	317	264	167	71	127	142	162	140	104	84	44	27	20	15	5	3	1	2,085
	F.	82	74	236	298	286	205	188	190	163	180	129	91	70	64	39	22	20	10	8	1	2,356
112	M.	2	2	1	3		4			4	1	3		1	1		1					23
	F.	1		2	2				9	4	4	2	2‡	2	1	1	2					32
121	M.	5	5	22	30	18	15	5	10	11	16	11	10	3	2	6						169
	F.	5	8	35	40	45	20	23	35	37	26	16	15	7	6	3	2					323
122	M.					1					1											2
	F.					1	1‡															2
211	M.	1	2	18	41	50	35	12	27	29	30	24	14	10	4	3	1	1				302
	F.	2		18	56	57	47	36	64	54	49	26	21	9	10	4	6	4	4			467
212	M.				1	1					1											3
	F.			1	3	1			3	3	2						1					14
221	M.				8	5	2		1	4	2	5	1									28
	F.			2	2	3	4	5	2	8	4	5	1		2							38
222	M.																					
	F.																					
Deaths	M.	†		††	*††	†				*		*†	*	†								
	F.	††	†	†			†‡	†	†	††	*	†	‡		*†	†	†		†			

Note { From Classes 11 to 22 Summer Deaths have been extracted.
From Classes 111 to 222 Summer and Autumn Deaths have been extracted.

Deaths shown :—* Summer. † Autumn. ‡ Winter.

TABLE "E."

A Summary of Facts relating to the Age Frequencies of Population, of Attacks, and the Percentages of Attacks to Population in each of Eighteen Groups of Ages, from the Block Census taken in Manchester under the direction of Dr. Carnwath.

Age Groups.	0–4.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.	50–55.	55–60.	60–65.	65–70.	70–75	75–80.	80–85.	85 and above.	Total.
First outbreak—Population (1) -	666	655	600	407	251	366	376	342	280	208	188	123	94	67	39	17	5	2	4,686
Attacks - -	44	92	88	72	59	83	73	68	37	32	20	13	14	8	3	8	1	0	715
Percentage of attacks -	6·61	14·05	14·67	17·69	23·51	22·68	19·41	19·88	13·21	15·38	10·64	10·57	14·89	11·94	7·69	47·06	20	0	
Second outbreak—Population (2) -	666	654	599	407	250	365	376	342	280	207	188	123	94	67	39	17	5	2	4,681
Attacks (2) - -	84	84	52	30	24	47	53	30	21	15	11	5	7	6	3	0	1	0	473
Percentage of attacks to population -	12·61	12·85	8·68	7·37	9·60	12·88	14·63	8·77	7·5	7·25	5·85	4·06	7·45	8·96	7·69	0	20	0	
Third outbreak—Population (3) -	661	653	599	407	249	365	374	342	280	207	188	122	92	66	39	17	5	2	4,668
Cases - - -	12	14	11	9	6	18	5	11	10	5	4	3	3	1	1	0	0	0	113
Percentage -	1·82	2·14	1·84	2·21	2·41	4·90	1·34	3·22	3·56	2·42	2·13	2·46	3·23	1·52	2·56	0	0	0	
Total percentage of Incidences -	21·0	29·0	25·2	27·3	35·5	40·4	35·4	31·8	24·3	25·0	18·5	17·0	25·5	22·4	17·9	47·06	40	0	27·6

Table "F."

SUMMARY TABLE.

Block Census 2.

Outbreaks.	0–4.	5–10.	10–15.	15–20.	20–25.	25–30.	30–35.	35–40.	40–45.	45–50.	50–55.	55–60.	60–65.	65–70.	70–75.	75–80.	80–85.	85 and above.	Total.
1. Population - -	923	804	733	501	341	469	462	479	364	260	187	136	84	56	40	20	11	2	5,872
Attacks - - -	45	113	117	88	53	97	99	89	61	38	17	19	7	8	5	4	0	0	860
Per cent. attacked -	4·9	14·1	16·0	17·6	15·5	20·7	21·43	18·5	16·8	14·6	10·2	12·5	8·3	14·3	12·5	20·0	0	0	—
2. Population - -	923	803	733	591	341	469	461	478	363	259	187	135	84	56	40	20	11	2	5,866
Attacks - - -	89	82	74	43	34	49	62	49	39	27	11	11	10	3	—	1	0	0	584
Per cent. attacked -	9·6	10·2	10·1	8·6	10·0	10·4	13·4	10·2	10·7	10·4	5·9	8·1	11·9	5·4	0	5·0	0	0	—
3. Population - -	916	801	732	500	340	468	459	478	361	259	186	134	83	55	40	19	11	2	5,844
Attacks - - -	9	9	4	5	0	12	11	9	5	2	3	2	1	4	0	0	0	0	76
Per cent. attacked -	0·98	1·12	0·6	1·0	0	2·6	2·4	1·9	1·4	0·8	1·6	1·5	1·2	7·3	—	—	—	—	—

The principal object, however, in rearranging the figures in these age groups was to ascertain what relation the age frequencies bear to the age frequencies of deaths for the whole city, as exhibited in curves. To ascertain this, differences between males and females have been neglected, and the age frequencies for the two added, so as to show for each age group the total population at risk, and the total number of attacks at that age group. When this is done we get the following table in which the percentages of attacks in the corresponding populations are also given. Tables E. and F.

The percentages in the three outbreaks are then added to get a total percentage throughout the epidemic periods. The age frequencies of the total percentages are remarkably like the age frequencies for deaths at ages 20–35.

In all the attack frequency curves there is a swell in childhood, due chiefly to the large numbers at risk. The percentages attacked at these early ages, however, tell a different tale.

The true test, however, is the percentage of attacks to population at corresponding ages, which again brings out quite clearly the peak occurring about the ages 20–35. The particular age period 25–30 will be seen to dominate the picture of attacks in the table, small and large alike, as it does also in the Summary Table, Curves 8.

There is, therefore, both as regards incidence and fatality in these outbreaks as they affected Manchester, a quite special tendency to attack and fatality at the ages 25–29, and 30–34, a new feature, and one requiring explanation.

The striking agreement of the age frequencies of attacks with the age frequencies of deaths for the whole population may be taken as evidence that the facts have been collected with reasonable accuracy.

It may be observed that the figures above given may be taken as showing the number of cases which would have occurred in the City if the sample were representative. Taking the population of the City in 1918 as 730,000, the number of attacks in the first outbreak may be taken as

$$730{,}000 \times \frac{709}{4684} = 110{,}450.$$

The number of cases in the second outbreak would be—

$$730{,}000 \times \frac{473}{4681} = 73{,}704,$$

and in the third—

$$730{,}000 \times \frac{113}{4668} = 17{,}671.$$

The total number of cases would thus be about 200,000; but if females and males are taken separately for a calculation, the number would be greater.

It will be noted that the numbers of attacks in successive outbreaks were 709,473,113. If no protection was afforded by the summer and autumn attacks, this great decline in the number of attacks is not readily explicable. The method of analysis adopted, therefore, would seem to overlook some cardinal facts, and it is possible that slight overlooked attacks play a large part in the production of immunity, as they do, for example, in enteric fever.

There is, however, another way in which we might estimate the number of attacks, viz., from the case fatalities, which leads to very different results. The objection to it is that these case fatalities are founded on small numbers, and on an experience in the total death-rate quite at variance with that of the whole population. However, accepting the case fatality in the first outbreak 0·7, in the second as 3·25, in the third as 2·7, we get the number attacked in the first outbreak, $332 \times \frac{1000}{7} = 47{,}420$, in the second 50,860, in the third 34,630.*

* These case fatalities are founded on the two block censuses taken together.

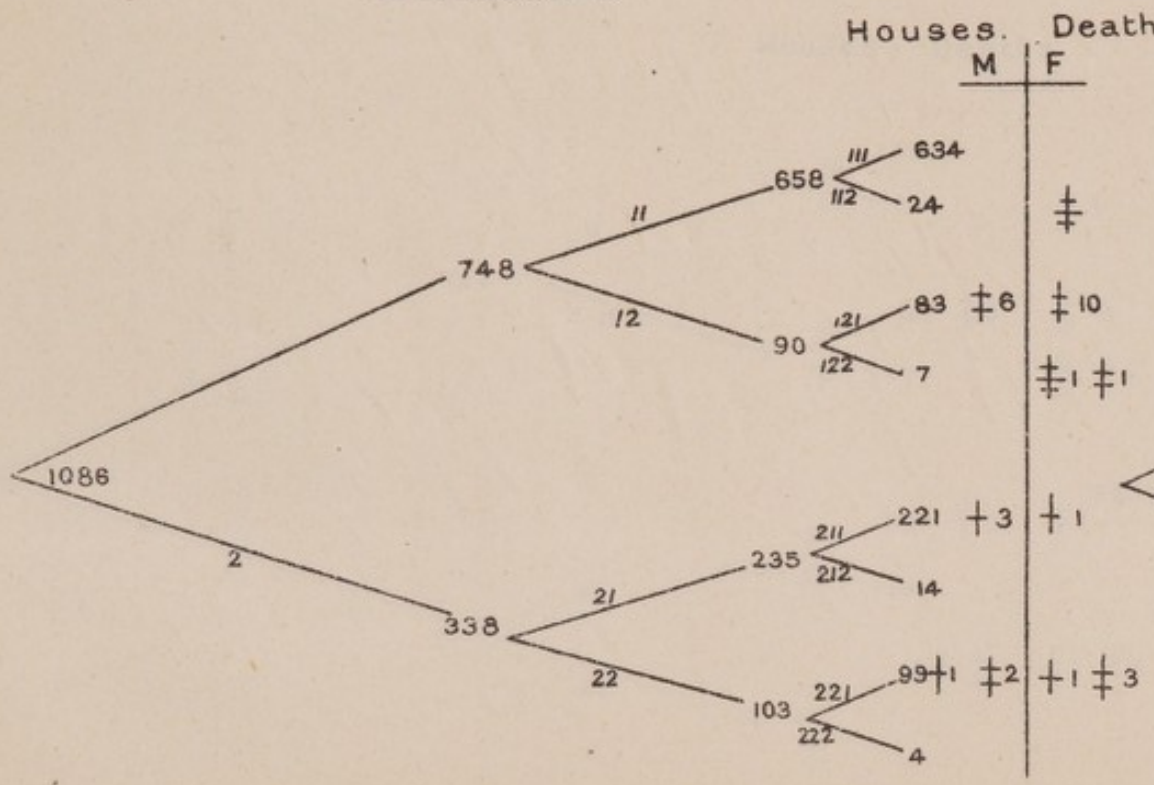
Table B.

MANCHESTER.

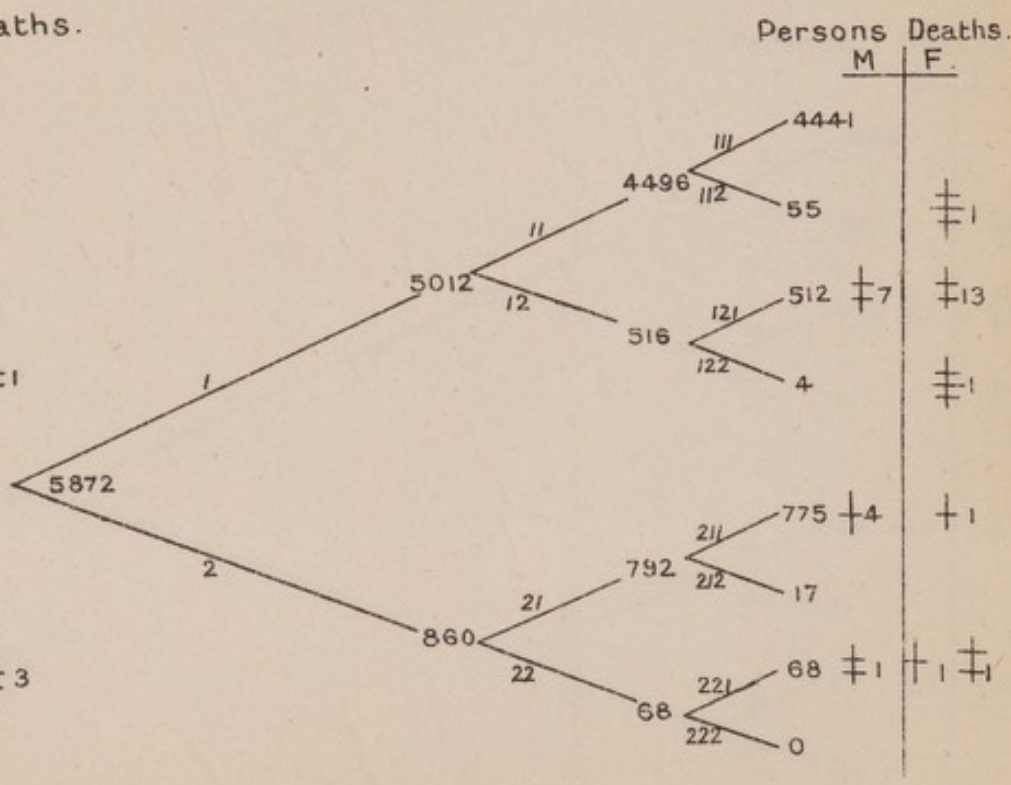
INFLUENZA 1918-19. — SECOND BLOCK CENSUS.

(For Key see Table c.p. 514.)

HOUSEHOLDS.



PERSONS.



To face p. 518.

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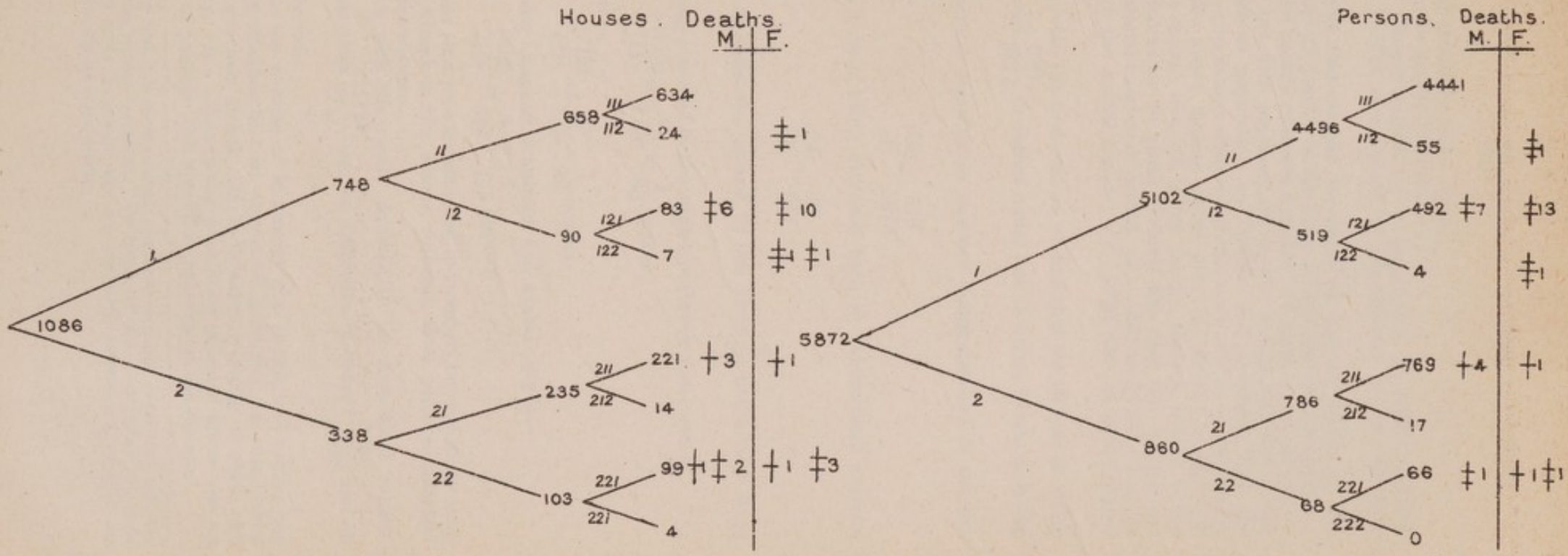
Table D.

MANCHESTER.

INFLUENZA 1918-19.—SECOND BLOCK CENSUS.

(For Key see Table c.p. 514)



Note:- From classes 11 to 22 Summer Deaths have been extracted.
" " 111 to 222 Summer & Autumn. " " " "

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*The other method should be more correct.

The facts may also be looked at in this way.

A second Block Census related to a population of 5,872 in 1,086 households situated in the same parts of Manchester, as in the first Census.

In the first outbreak 860 attacks are recorded and six deaths, in the second, 584 attacks and 22 deaths, in the third, 76 attacks and two deaths.

Taking the first and second censuses together, we have a population of 10,558, 11 deaths in the first outbreak, 35 in the second, and five in the third. Taking the whole population at 730,000, and assuming that the deaths over the whole population occurred at the same rate as in the Health Visitors' Districts, the above figures would give 761 deaths in the first outbreak, 2,420 in the second, and 346 in the third. Having regard to the comparatively high mortality in these districts the first two figures are sufficiently near the actual number of deaths, viz.:—332 and 1,702; but the third is so much below the actual number viz.:—936, that it needs special explanation.

In comparing this with the first census, we find that the chance of being attacked in summer was $\frac{860}{5872}$, or rather under one in seven, herein agreeing with the first census.

The chance of a person escaping in summer being attacked in autumn was—

$$\frac{(12)}{(1)} = \frac{516}{5012}.$$

The chance of a person being attacked both in summer and autumn was—

$$\frac{(22)}{(2)} = \frac{68}{854}.$$

The first chance exceeds the second by less than $\frac{1}{30}$, not a very large amount.

The chance of being attacked in the winter outbreak alone was—

$$\frac{(112)}{(11)} = \frac{55}{4496} = \frac{1}{82} \text{ approx.}$$

in summer and winter—

$$\frac{(212)}{(21)} = \frac{17}{786} = \frac{1}{46} \text{ approx.}$$

The chance of being attacked in autumn and winter only is—

$$\frac{(122)}{(12)} = \frac{4}{516} = \frac{1}{129} \text{ approx.}$$

It would thus seem that persons attacked in summer, but not in autumn, were more liable to attack in the winter outbreak than persons attacked neither in summer nor autumn.

This again agrees with the result obtained in the first block census. $\frac{(122)}{(12)}$, however, is less than $\frac{(112)}{(11)}$, but the difference is under $-\frac{1}{200}$, and no immunity can be inferred.

In the first census the reverse of immunity was shown, and taking the two together we may conclude that no degree of immunity was established in individuals by a previous attack. It is, however, possible to state the facts in another way, viz.:—That in the population selected there was an exceptional number of persons with special receptivity to the disease.

---

* If we may assume that it is more correct, the facts are not consistent with the mortality figures in the winter outbreak. To reconcile the discrepancy we must assume either that the facts for winter are not correctly ascertained, or that the winter incidence fell mainly on fresh areas.

In the mortality figures, however, neither census reflects the death rate for the whole city, and the second less so than the first, at all events as regards the winter outbreak. Thus, out of 860 attacked in summer six died; giving a case mortality of approximately seven per thousand, which is nearly the same as in the first outbreak.

The differences between Block Censuses No. 1 and No. 2 are only such as might be expected for the limited number of houses taken.

The curve of percentage of attacks to population at corresponding groups of ages is shown in Curves 8.

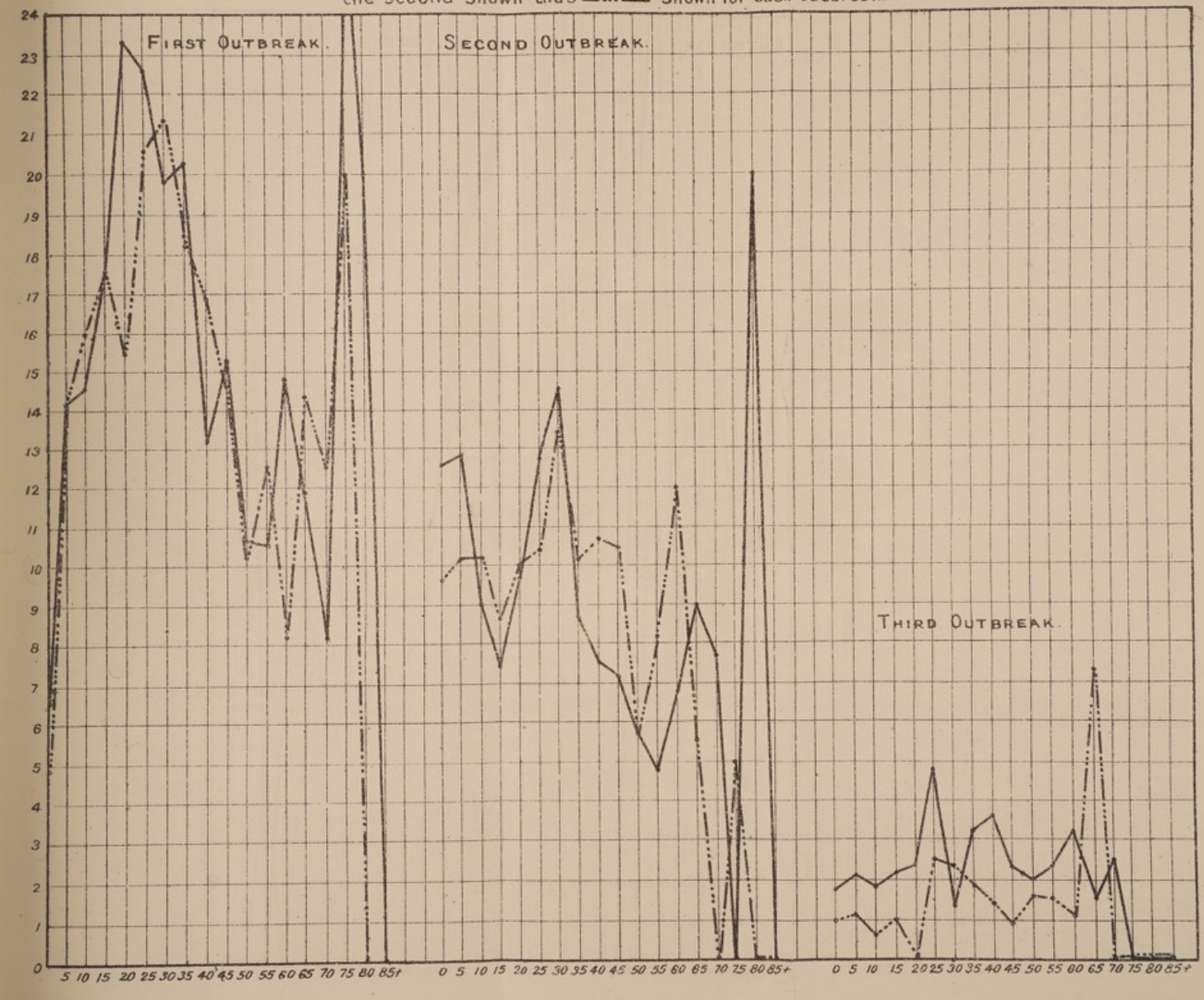
The Summary Tables E. and F., on which the curves are based, are given herewith, pp. 516–17.

CURVES 8. (See p. 518)

To face p. 520.

PERCENTAGE OF ATTACKS TO POPULATION.

in groups of ages. the first census shown thus ——
the second shown thus —·—·— shown for each outbreak.



2263

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V.

Report on Influenza amongst the employees of two firms of chemical manufacturers, Messrs. the United Alkali Company of Widnes, and Messrs the Castner Kellner Company of Runcorn, together with—for control purposes—certain figures relating to the incidence of the disease amongst Ironworkers.

By

J. R. Hutchinson, M.D., and F. S. Carson, M.B.

Method of Investigation.

By the courtesy of the firms mentioned and the Widnes Forge Company the works were visited and the various chemical processes inquired into.

Lists of the names and addresses of employees engaged in the manufacture of chemicals were obtained and the homes visited. At every house the information set out below was sought and recorded :—

(1) Names of occupants with age, sex, and occupation of each.
(2) Number of rooms in house.
(3) Dates and nature of attack or attacks of influenza.
(4) Order in which the occupants were attacked.
(5) The period elapsing between the time the first member became ill, and second cases.
(6) Complications.
(7) Whether patient or patients were medically attended.
(8) Result.

Widnes with a population of 31,541 is a town whose main industries are the manufacture of chemicals, soap, and iron ware. Runcorn's population of 18,000 is engaged largely in similar trades and in tanning. The aggregate number of persons of whom particulars were obtained was 3,417, approximately 7 per cent. of the entire population. The 3,417 persons were made up of 944 employees at one or other of the three works plus their wives and families.

Character of Work in which the Population investigated is engaged.

The chemical workers are divided into process workers and non-process workers. Non-process workers are unskilled labourers who wait upon the process workers. They work in an atmosphere where there are chemical fumes, but they are not constantly employed in the same atmosphere—one day they may be on the chlorine plant, next on the vitriol or some other plant where there are fumes.

The process workers are divisible into—

(*a*) Those engaged in an atmosphere where chlorine is constantly present.
(*b*) Those engaged in the manufacture of vitriol (sulphuric acid).

The chlorine process workers include—

(1) Those employed in the salt cake department.
(2) „ „ „ bleach department.
(3) „ „ „ organic department.

In all these departments the workers are employed in an atmosphere where chlorine is always present.

In the "salt cake" processs (for practical purposes this includes the "bleach" also) common salt is placed in cone-shaped iron pots into which sulphuric acid is then run.

The hydrochloric acid evolved ($NaCl + H_2SO_4 = NaHSO_4 + HCl$) passes direct to the Deacon's decomposers for conversion to chlorine which is passed into condensers—any excess of HCl being absorbed in water. Finally the chlorine is passed over slaked lime to form bleaching powder.

The residue from the furnaces is sulphate of soda or *salt cake.* The gases found are—

HCl and traces of SO_3 in the salt cake department.

HCl and Cl—on the decomposers.
Chlorine—in bleach department.

Organic Process.

Chlorine is generated by the electrolysis of brine and led in pipes to the organic plant where it is passed through benzine to form monochlor benzine. The monochlor benzine is nitrated by nitric acid to form di-nitrochlor benzine which is then converted into di-nitro-phenol by displacement of the chlorine with OH.

The gases that may be found in the neighbourhood of the organic plant are chlorine, monochlor, and benzine vapours, nitric acid, hydrochloric acid. Traces of various nitrous compounds—mono-nitro-chlor-benzine, vapour of acetic acid from monochlor acetic acid.

The workers on vitriol plants may be exposed to any or all of the following gases—SO_2, SO_3, NO_2, HNO_3, N_2O_2, N_2O_3—traces only of the last four are found. The manufacture is carried out in open sheds where natural ventilation only is employed.

In the bleach department some workers use dry masks of several layers of unimpregnated flannel, but this is optional and it is conceded that a man may wear a mask and still be "gassed."

The work done at the Widnes Forge Company's premises is essentially free from anything in the nature of chemical fumes. It consists in the manufacture of castings, &c.

Influenza Incidence on Population investigated.

In the summer outbreak there were 423 attacks = 12·3 per cent.
,, autumn ,, ,, 288 ,, = 8·4 ,,
,, winter ,, ,, 369 ,, = 10·7 ,,

The 1,080 attacks affected 1,036 persons out of a total of 3,417, *i.e.*, approximately ⅓ suffered in one or other wave.

Age and Sex Incidence.—The population of 3,417 comprised 1,737 males and 1,680 females. Of the former 510 or 29·4 per cent., and of the latter 526 or 31·3 per cent. suffered. The incidence on certain age groups was :—

TABLE A.

Age Group.	Males.			Females.		
	Total Males.	No. of Cases.	Per-centage.	Total Females.	No. of Cases.	Per-centage.
0–1 - -	25	3	12·0	23	1	4·3
1–5 - -	142	31	21·8	150	26	17·3
5–15 -	407	105	25·8	396	90	22·7
15–25 -	247	77	31·2	348	98	28·2
25–35 -	225	87	38·7	235	103	43·8
35–45 -	297	108	36·4	222	100	45·0
45–55 -	224	61	27·2	168	73	43·4
55 and over	170	38	22·3	138	35	25·4
Total -	1,737	510	29·4	1,680	526	31·3

The incidence in each wave on the total population investigated was :—

Age Group.	Summer.	Autumn.	Winter.
	Per cent.	Per cent.	Per cent.
0–1	2·0	—	6·2
1–5	4·4	3·7	11·3
5–15	11·5	5·9	6·9
15–25	15·4	10·2	5·3
25–35	17·4	10·4	14·8
35–45	15·2	11·5	16·4
45–55	13·2	9·6	12·7
55 and over	4·2	7·1	13·6

Incidence on certain Age Groups of Process Workers in the Chemical Industry.—Process workers are invariably males.

Those engaged constantly in an atmosphere in which chlorine is always present :—

TABLE B.

Age Group.	No. of Workers.	No. of Cases.	Percentage Incidence.
15–25	21	7	33·3
25–35	84	34	40·5
35–45	131	50	38·2
45–55	87	19	21·8
55 and over	62	12	19·3
Total	385	122	31·7

Those engaged constantly on vitriol plant :—

TABLE C.

15–25	7	3	42·8
25–35	35	14	40·0
35–45	41	15	36·6
45–55	33	12	36·4
55 and over	12	3	25·0
Total	128	47	36·7

Inasmuch as Table B. relates to three times as many persons as does C., the smaller per cent. incidence in favour of the former is more apparent than real, and it will be of advantage to combine the two Tables as

illustrating the age incidence on process workers generally. This is done in Table D.:—

TABLE D.

Age Group.	No. of Workers.	No. of Cases.	Percentage Incidence.
15–25 - - - - -	28	10	35·7
25–35 - - - - -	119	48	40·3
35–45 - - - - -	172	65	37·8
45–55 - - - - -	120	31	25·8
55 and over - - - -	74	15	20·3
Total - -	513	169	32·9

These 169 persons had in all 176 attacks.

Incidence on certain Age Groups of Non-Process Workers.—That is to say, on persons intermittently exposed for varying periods, sometimes to chlorine, sometimes to gases from vitriol plants and at others not exposed at all. Non-process workers may be females.

TABLE E.

15—25 - - - - -	113	41	36·3
25—35 - - - - -	40	17	42 5
35—45 - - - - -	25	3	12 0
45—55 - - - - -	32	12	37·5
55 and over - - - -	20	4	20·0
Total - - -	230	77	33·5

Tables B, C, D, and E therefore set out the incidence of influenza on two classes of chemical workers, one constantly exposed to certain gases and the other exposed intermittently.

Table F, which forms a control, shows the incidence of influenza on the employees of the Widnes Forge Company, who live in the same neighbourhood as the chemical workers of the United Alkali Company and who therefore may be assumed ordinarily to be exposed to the same atmosphere outside working hours.

TABLE F.

15—25 - - - - -	30	15	50·0
25—35 - - - - -	43	19	44·2
35—45 - - - - -	61	25	41·0
45—55 - - - - -	36	11	30·5
55 and over - - - -	31	9	29·0
Total - - -	201	79	39·3

The 79 persons had 85 attacks.

Summarised these figures show that the incidence of influenza on process workers was 32·9 per cent. as compared with 33·5 on non-process workers, 39·3 per cent. on iron workers living under precisely similar conditions, and 30·3 per cent. on *all males* of *same age* in the population investigated.

The incidence in each wave on these several classes of persons was :—

On Process workers.

TABLE G.

Age Group.	Summer.	Autumn.	Winter.
	Per Cent.	Per Cent.	Per Cent.
15—25 - - - - -	21·0	10·7	3·5
25—35 - - - - -	21·8	9·2	10·9
35—45 - - - - -	15·6	10·4	12·7
45—55 - - - - -	10·0	9·1	8·3
55 and over - - - -	4·0	5·7	10·8

On Non-Process workers.

TABLE H.

	Per Cent.	Per Cent.	Per Cent.
15—25 - - - - -	21·7	10·3	3·25
25—35 - - - - -	15·3	8·0	21·8
35—45 - - - - -	4·0	Nil.	8·0
45 and over - - - -	7·8	5·8	17·6

On Ironworkers.

TABLE J.

	Per Cent.	Per Cent.	Per Cent.
15—25 - - - - -	30·0	13·3	10·0
25—35 - - - - -	13·9	13·9	18·6
35—45 - - - - -	18·0	13·1	14·7
45—55 - - - - -	16·6	5·5	11·1
55 and over - - - -	Nil.	16·1	12·9

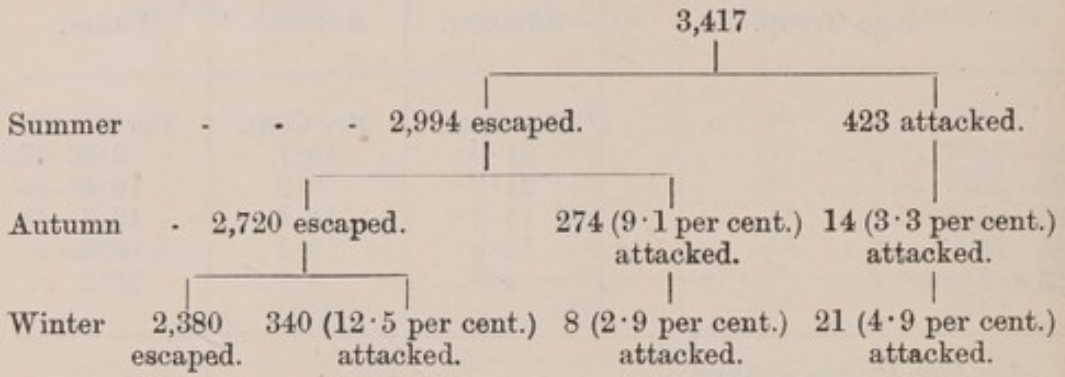
Incidence on certain age groups among females engaged in home duties—including nursing.

TABLE K.

Age Group.	No. of Workers.	No. of Cases.	Percentage Incidence.
15—25 - - - - -	117	28	23·9
25—35 - - - - -	194	87	44·8
35—45 - - - - -	214	95	44·4
45—55 - - - - -	164	71	43·3
55 and over - - - -	133	35	26·3
Total - - -	822	316	38·4

The average incidence therefore was 38·4 per cent. The corresponding figure for all females of like ages in the total population investigated, is 33·1 per cent.

Immunity.—There were 423 attacks in the summer wave, 288 in the autumn and 369 in the winter. 43 persons had two attacks; 14 in the summer and autumn, 21 in the summer and winter, and 8 in the autumn and winter waves. For the whole community of 3,417 persons investigated the table below summarises the result of our enquiries.



Further analysis failed to disclose any difference in this respect between the various classes of workers.

Mortality :—

Of the 1,080 attacks, 16 or 1·5 per cent. proved fatal.
Of the 423 summer cases, 2 died = 0·47 per cent.
Of the 288 autumn cases, 2 died = 0·69 per cent.
Of the 369 winter cases, 12 died = 3·3 per cent.
Of the 169 cases occurring amongst 513 process workers (chlorine and vitriol) none died.
Of the 230 non-process workers, 36 were attacked in summer, 16 in autumn and 24 in winter. There were no deaths in the first two waves, but three occurred in the last; this gives a case mortality of 12·5 per cent.
Of the 201 iron-workers, 32 suffered in the first, 25 in the second and 28 in the third wave. There were no deaths.

The case mortality of the investigated population set out in age groups was :—

Age groups.	Attacks.	Deaths.	Case Mortality.
0–5	61	6	9·9 per cent.
5–15	197	1	0·5 „ „
15–25	185	—	—
25–35	196	1	0·5 per cent.
35–45	224	1	0·4 „ „
45–55	140	1	0·7 „ „
55 and over	77	6	7·8 „ „

Nine males and seven females died. The age periods most heavily affected were 0–5 and 55 and over. There were five deaths of children under three years and four of adults over 60 years.

All the deaths occurred in first attacks, and all but three were attributed to pulmonary complications.

Complications.—It is stated that a large proportion of the chemical works' employees suffered from chronic bronchitis; it is a popular belief that "bad chests" from the "gas" were common. For this reason enquiry was more particularly directed to pneumonia and pleurisy, rather than to bronchitis, as complications. It was found that of the total cases, 58 or 5·3 per cent. had pneumonia of one kind or another and 3 or 0·27 per cent.

had pleurisy. The occurrence of these complications in the several waves was :—

	Pneumonia.	Pleurisy.
In the summer - - - - -	1·18 per cent.	—
In the autumn - - - - -	5·2 „ „	0·69 per cent.
In the winter - - - - -	10·29 „ „	0·27 „ „

The pneumonia incidence on age groups was :—

Age group.	Influenza cases.	Pneumonia as a complication.
0–5 - - -	61	13 or 21·3 per cent.
5–15 - - -	195	7 or 3·6 „ „
15–25 - - -	175	9 or 5·1 „ „
25–35 - - -	190	8 or 4·2 „ „
35–45 - - -	208	7 or 3·4 „ „
45–55 - - -	134	4 or 3·0 „ „
55 and over - -	74	10 or 13·5 „ „

Pneumonia as a complication amongst process workers in chlorine was absent in the summer wave (50 cases), while in the autumn (40 cases) it occurred in 5 per cent. and in the winter (37 cases) in 2·7 per cent.

Among vitriol workers pneumonia as a complication did not occur until the winter outbreak when there was one case, 5·8 per cent., in a man of 61 years. There were 32 influenza cases in the summer and autumn and 17 in the winter.

Non-process workers suffered less severely—

In the summer, of 36 influenza cases, 1 or 2·8 per cent. developed pneumonia.

In the autumn, of 16 influenza cases, 2 or 12·5 per cent. developed pneumonia.

In the winter, of 24 influenza cases, 2 or 8·3 per cent. developed pneumonia.

These figures differ little from those presented by the control cases (ironworkers) among whom there were—

In the summer, 32 influenza cases with no pneumonia.

In the autumn, 25 influenza cases with 3 or 12 per cent. pneumonia.

In the winter, 28 influenza cases with 2 or 7·1 per cent. pneumonia.

Incidence of Influenza on Households.

In all 653 houses in the two towns were visited, and of these 410 or 62·8 per cent. were invaded between June 1918–April 1919. Of 366 houses each containing a process worker in chlorine, 219 or 59·8 per cent were invaded, and in 20 per cent. of these the chlorine worker himself was the first to suffer. Similarly of 167 houses of process workers on vitriol plants, 114 or 68·3 per cent. were affected. Here again in 20 per cent. of instances the first influenza case was the vitriol worker himself. Of 167 control houses (ironworkers) 114 or 68·3 per cent. were invaded, and in 25 per cent. it was the ironworker himself who introduced the disease. Inasmuch as the average number of persons per house was 5·2, the natural chance of any one individual in any house introducing the disease was approximately 20 per cent.

Influence of Overcrowding.—Contrary to expectation we could discover no evidence to show that the incidence on overcrowded houses was greater than that on houses which were not overcrowded.

Incubation Period.—Direct evidence bearing on this point is somewhat scanty owing to the number of factors introduced. Where, however, such was available the period varied from 12–48 hours. Second crops of cases in the same house commonly followed 48 hours after the first.

Influence of Nursing as a factor in the spread.—No appreciable difference was noted between the figures relating to women employed in house duties, including nursing and those engaged in chemical works as non-process workers.

Comments.—In this investigation we were unable to find any artificial means of transference of possibly infected particles from one worker to another in the actual processes carried on.

Owing to the nature of the processes chemical plants cover a comparatively large area of ground The work is carried on in open sheds and in any one department the workers are few and scattered. There is little intercommunication between the different departments.

All the works have canteens, but our information is that they were little used by the male workers, and when they were used it was customary for the men to use as drinking vessels the utensils in which they brought their food.

In connection with the organic plant of one of the works there is a common washing-up place for eating and drinking utensils, but there is no evidence to show that the incidence on the users was either greater or less than on their fellows who had not access to like facilities. The few female workers are provided with dining rooms. Their eating vessels are washed up, &c. all together, but here again evidence of the influence or otherwise of this factor in the spread of influenza is lacking.

We owe our thanks to Messrs. The United Alkali Company, Messrs. The Castner Kellner Company, and to Messrs. The Widnes Forge Company for their courtesy in supplying information concerning their staffs. We are also indebted to Dr. H. E. Annett, Medical Officer of Health, Runcorn Urban District, and Dr. Albert Jones, D.S.O., M.C., Medical Officer of Health, Widnes Borough, for valuable assistance in prosecuting the inquiry.

VI.

Report on an Investigation of Epidemic Influenza in the County Borough of South Shields, 1919,

By

D. Morley Mathieson, M.D., Medical Officer of Health.

For the purpose of this investigation South Shields is divided into two districts. In each district five groups of ten dwellings are selected which represent the various types peculiar to the County Borough. The investigation was carried out by the Assistant Medical Officers, Dr. Lyons and Dr. Martin, each being allotted one district.

The method of inquiry was to visit each dwelling, make notes of name, age, and sex of each occupant, and the disposition of the occupants in the various rooms. Inquiry was next directed to the occurrence of influenza, notes being made of each attack, its date of onset, duration, result, and complications. The date of the doctor's first visit was asked for, also the name of the person who nursed the patient. The previous medical history of each person attacked was obtained and an expression of opinion as to the source of infection was encouraged.

In all, 102 dwellings with 462 occupants were visited. Among these were 72 cases of influenza; 5 of the latter were attacked twice, a total of 77 attacks.

Table 1 shows the age and sex distribution of the area investigated, and Table 2 the age and sex distribution arranged in the various groups of houses with the incidence of influenza, recurrences, and death.

It will be seen that females were more affected than males, the percentage of the population affected being, females, 20·1 per cent., and males, 11·9 per cent., while the age groups most affected among the total cases are from 55–65 and from 25–35. The former produced 31·4 per cent. and the latter 24·2 per cent. of cases. (The total population of the borough obtained from ration cards in 1917 was 105,344.)

The total deaths from influenza in the borough in 1918 and in the first quarter of 1919 are detailed in Table 1.

TABLE 1.

Deaths from Influenza.

Age.	1918.	1919, First Quarter.
Under 1 year	9	10
1	21	5
2	17	6
3	18	3
4	18	4
5	54	16
10	37	3
15	33	9
20	31	9
25	92	34
35	52	30
45	31	19
55	26	17
65	16	6
75	5	2
85 years and over	1	—
Total	461	173*

* Of these 94 were males and 79 females.

TABLE 2.

Age and Sex Distribution of the Population investigated.

Age Groups.	Under 1 year.		1–		2		5–		10–		5 –		20–		25–		35–		45–		55–		65–		75 and over.		Total.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Age and sex incidence.	6	7	5	1	8	19	15	31	23	34	21	30	17	27	27	39	20	30	23	30	21	14	5	5	2	2	193	269
Cases of influenza	-	1	–	–	–	4	3	6	3	7	2	3	1	1	5	11	–	9	4	4	4	7	1	–	–	1	23	54
Per cent. attacked.	–	14·3	–	–	–	21	20	19·3	13	20·6	9·5	10	6	3·7	18·5	28	–	30	17·4	13·3	10	50	20	–	–	50	11·9	20·1
Total number of persons in each age group.	13		6		27		46		57		51		44		66		50		53		35		10		4		–	–
Number of cases	1		–		4		9		10		5		2		16		9		8		11		1		1		–	–
Per cent. attacked	7·7		–		14·8		19·5		17·5		9·8		4·5		24·2		18		15·1		31·4		10		25		–	–
Deaths - -	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1

TABLE 3.

Age and Sex of Persons attacked.

—	Under 1 year.		1–		2–		5–		10–		15–		20–		25–		35–		45–		55–		65–		75 and over.		Total.
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
Hill Street -	–	–	–	–	1	**2**	**2**	3	5	3	2	3	4	2	2	3 (1)	2	2	2	4	2	2 (1)	–	–	–	–	46 (2)
Double Row -	–	–	–	–	1	4	1	4 (1)	3	4	6	5 (1)	2	7 (1)	4	2	1	1 (1)	4 (1)	6 (2*)	5	2 (2*)	2	–	1	–	65 (9)
Egglesfield Road	–	–	–	–	–	1	1	2	3	3	1	3	1	3	1	6	2	4	2 (1)	1	3 (1)	1	2	3	–	–	43 (2)
West Park View	1	–	–	–	**2**	1	1	2	2 (1)	5 (1)	**2**	4	1	**1**	3 (1)	8 (1)	2	4 (2)	4 (1)	6	**2**	1	-	–	–	–	52 (7)
Simon Street -	3	3	1	–	1	5 (2) **1**	4 (1)	6 (1)	3 (1)	3 (1)	3	5 (1)	1	2	2 (1)	2 (1)	4	4 (1)	2	1	1 (1)	–	–	–	–	–	56 (11) **1**
The Lawe - -	–	1	1	–	1	1	1	1	2 (1)	2	1	2	–	2	3	7	3	4 (2)	1	3 (1)	3 (2)	3 (1)	–	1	1	–	44 (7)
Hepple's Court -	1	1	–	–	1	1	1	3	1	6	1	3	1	3	–	1 (1)	1	5	3	1	1	1 (2*)	–	–	–	1	37 (3)
Thames Street -	1	1 (1)	1	–	–	2	2	4 (1)	–	3	1	2	2	–	5 (2)	5 (4*)	1	1	–	1	1	2 (1)	–	1	–	–	36 (9)
Marsden Street -	–	1	**2**	1	–	2 (2)	2 (2)	4 (3)	2	2 (2)	2 (1)	2 (1)	2 (1)	4	5 (1)	2	2	2 (2)	–	1 (1)	1	–	1 (1)	–	–	1 (1)	41 (18)
Osborne Avenue	–	–	–	–	1	–	–	2	2	3 (3)	2 (1)	1	**3**	3	**2**	3 (3)	2	3 (1)	5 (1)	6	2	**2**	–	–	–	–	42 (9)
Total persons -	6	7	5	1	8	19	15	33	23	34	21	30	17	27	27	39	20	30	23	30	21	14	5	5	2	2	462
Total cases of influenza -	–	1	–	–	–	4	3	6	3	7	2	3	1	1	5	11	–	9	4	4	4	7	1	–	–	1	77

Figures in brackets = cases of influenza. Figures in black type = death. * = recurrence.

Seasonal Incidence.—In the first quarter of 1918 there were 4 deaths from influenza and its complications; in the second, 6; in the third, 43; and in the last quarter, 408.

In Graph 2 it will be seen that the age groups chiefly affected in the summer and autumn waves were 10–15 and 25–35 years; while in the winter wave the age group most affected was 55–65 years.

Occupational Incidence in Persons over School Age.—Total, 49 cases; males 17 and females 32:—

Males.		Females.	
Miners	4	Household duties	28
Platers (Shipyard workers)	2	Clerks	2
Driller (Shipyard workers)	1	Teacher	1
Fitter (Shipyard workers)	1	Shop Assistant	1
Sea Captain	1		
Seaman	1		
Fisherman	1		
Marine Superintendent	1		
Undertaker	1		
Shop Assistant	1		
Clerk	1		
Labourer	1		
Collector of dues	1		

It is of interest to note that all those who were attacked twice were females over 15 years of age, and were engaged in household duties.

Incidence among Persons nursing Cases of Influenza.—In the area investigated there were 44 persons who nursed cases of influenza; of these 12, or 27·3 per cent., were attacked, and 32, or 72·7 per cent., escaped. Only those who developed the disease within seven days from the onset of the patient's illness were considered as being infected by the patient. The similarity of the percentage figures of persons infected whilst nursing cases in the two districts is shown in the following table:—

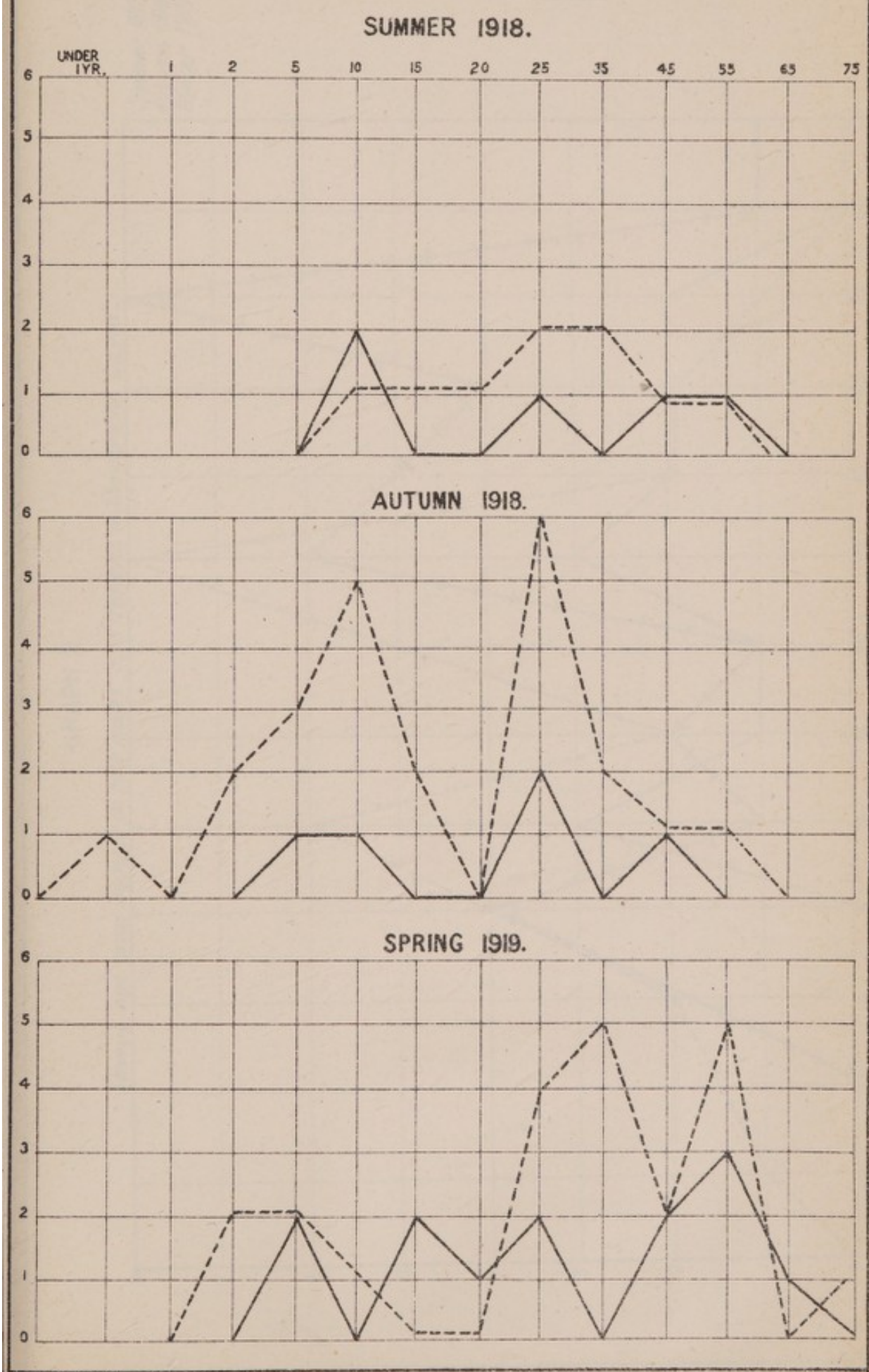
———	Number of persons nursing cases.	Attacked.	Escaped.
		Per Cent.	Per Cent.
Dr. Lyon's district	18	5, or 27·7	13, or 72·3
Dr. Martin's district	26	7, or 26·9	19, or 73·1

Immunity.—The following table shows the number of those attacked once and twice. It lends no support to the view that persons who have been attacked once enjoy any immunity in subsequent epidemics:—

———	Males.	Females.	Total Attacks.
(*a*) Number of persons attacked in summer.	5	9	14
Number of persons escaped in summer.	188	260	—
(*b*) Number of persons attacked in autumn:—			
(1) Of those who suffered in summer.	—	1	28
(2) Of those who escaped in summer.	4	23	

GRAPH I

Showing age and sex incidence in the several waves.
Males ____ Females _ _ _ _ _



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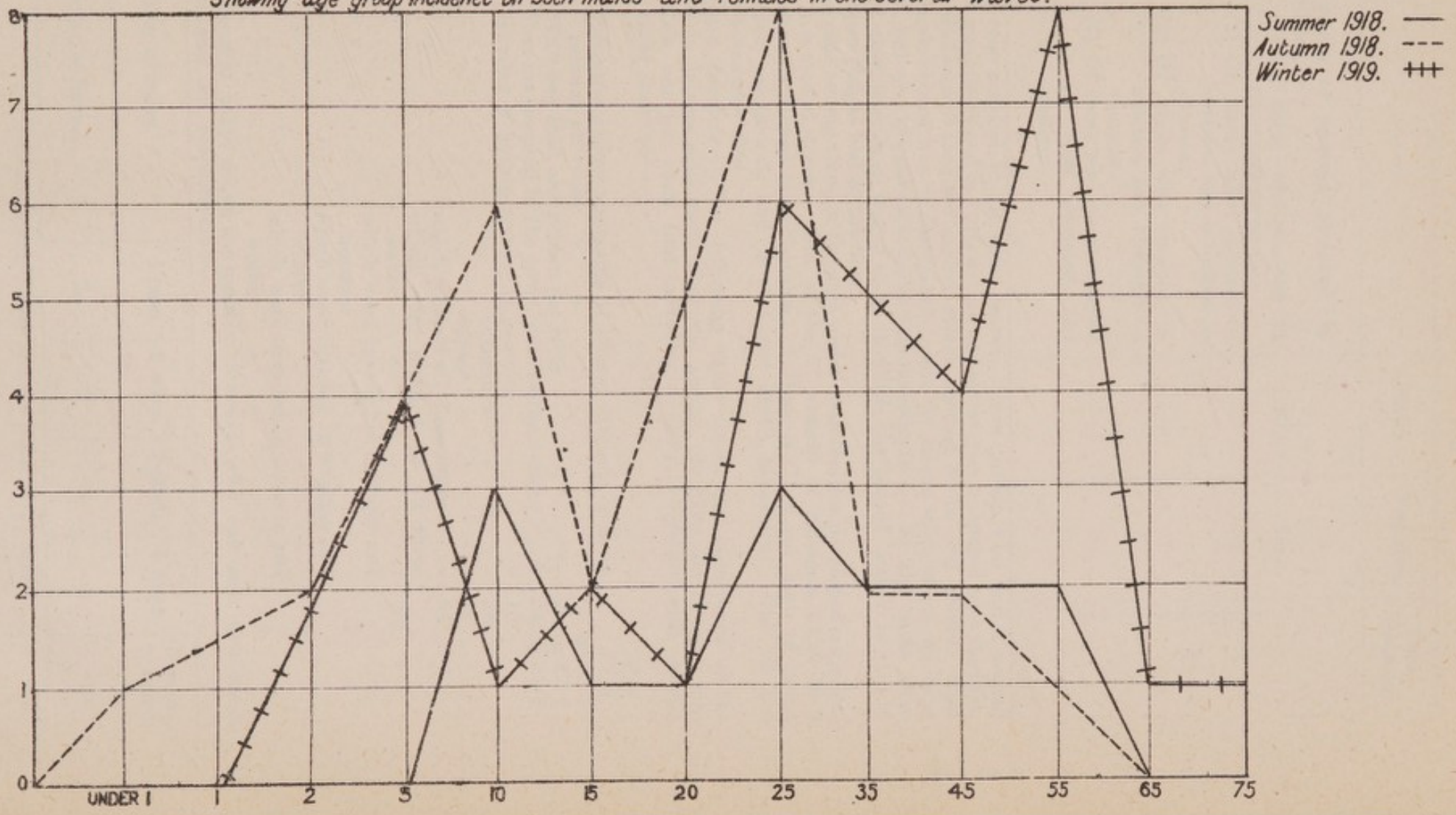
GRAPH 2

Showing age group incidence on both males and females in the several waves.



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	Males.	Females.	Total Attacks.
(c) Number of persons attacked in winter :—			
(1) Of those who escaped in two previous waves.	13	18	35
(2) Of those who escaped in summer but attacked in autumn.	—	3	
(3) Of those who escaped in autumn but suffered in summer.	—	1	
(4) Of those who were attacked in both summer and autumn.	—	—	

No case suffered in all three waves. Each person who had a second attack was questioned as to its nature and severity as compared with the first. They all declared that the second attack seemed to be less severe. This view is also borne out by statements made by others with whom we have talked, but who are not included in this investigation. At the same time several cases have been known to us in which the second attack is said to have been just as bad as the first.

No case among those in the area under consideration developed complications in a second attack. Out of the 77 attacks of influenza, 13, or 16·9 per cent., developed complications; of these 8 (11·2 per cent. of cases) developed pneumonia, and 5 (6·5 per cent.) bronchitis. One of the pneumonia cases died.

House Incidence.—The following table shows the occurrence of the disease in houses with more than one person per room and in those with less than one per room (not including scullery); together with certain other facts observed :—

	Per Cent.
Incidence among occupants of houses with one or more persons per room - - - - - - - - -	56, or 72·7
With less than one person per room - - - -	21, or 27·2
Persons sleeping in the same room as the patient -	24, or 31·2
Number of houses invaded in summer - - - -	10
,, ,, ,, autumn - - - -	14
,, ,, ,, winter - - - -	21
,, ,, which escaped all three waves -	64
,, ,, which were invaded in all three waves - - - - - -	Nil.
,, ,, which were invaded in summer and autumn - - - - -	1
,, ,, which were invaded in summer and winter - - - - -	3
,, ,, which were invaded in autumn and winter - - - - -	3

The next table shows the number of houses with one case, two cases, three cases, &c., respectively :—

Number of cases - -	1 case.	2 cases.	3 cases.	4 cases.	5 cases or more.
Number of houses - -	24	7	4	2	2*

* One house with 6 cases, and one house with 8 cases.

Incubation period.—The longest incubation period encountered in the inquiry appears to be five days; the shortest 12 hours; and the great majority appear to have been about 48 hours. The 12-hour incubation case is interesting. A house of eight rooms was occupied by a husband and wife who employed no maids. It was decided that the spring cleaning and some re-decorating should be undertaken by themselves. The husband went out during the day and worked at painting, &c., at night. The wife spring-cleaned all day and rested in the evenings. She was not out of doors for seven days and saw no person except tradesmen and her husband during that time. On the sixth evening the husband came home feeling ill and with catarrhal symptoms, which developed into an attack of influenza. Early next morning his wife also had symptoms of catarrh followed by a typical attack of the disease. On the morning preceding his attack, the husband expressed himself as feeling particularly fit.

Duration of Infectivity.—It is regretted that it has not been possible to determine how long a case may be infectious, no case being found to yield any useful information on this point. There were indications, however, that the early catarrhal stage of the disease appears to be the most infectious, and this view seems to be substantiated by the fact that the majority of the cases among contacts began usually about 48 hours after the onset of symptoms in the primary case.

Prevention and Treatment.—Little can be said with regard to preventive measures, but it would appear that where home isolation can be carried out the incidence of the disease among contacts is reduced.

Vaccine treatment of the condition, as far as observation went, was not attended by any results more satisfactory than treatment on other lines. Nor did the use of vaccine as a prophylactic appear to be of much value in the prevention of an attack, although its real value could not be estimated on account of the small number of persons so treated.

During the epidemic in the first three months of this year a number of patients were treated in the South Shields Isolation Hospital, and the value of institutional treatment was demonstrated, but only in those cases in which lung complications had not developed. In this connection it is of interest to note that only those members of the staff of the hospital who came into contact with the patients and who did not wear masks contracted the disease.

Associated Medical History.—The 72 persons attacked were classified as—

(1) Healthy previous to attack - - - - - - 63
(2) Unhealthy „ „ - - - - - - 9

The latter include five cases of chronic bronchitis, one of chronic hepatitis, one of anæmia, and two of pregnancy, *i.e.*, pregnancy at the time of the attack. Two of the cases of chronic bronchitis were females occupied on household duties and both had two attacks of influenza. As has already been stated, all those who had two attacks were females occupied at home on household duties. It is unfortunate that associated diseases were not met with during the investigation. Numerous cases, however, have recently attended at the Tuberculosis Clinic in South Shields where the patients definitely attribute thier tuberculous condition to an attack of influenza. The fact has been noted even in former years when influenza did not assume epidemic form. Since February 1919, observation has been made of the cases of tuberculosis attending the clinic for treatment for the purpose of discovering what proportion of the patients contracted influenza, one was surprised at the very small number who did so.

With regard to the effect of the disease on pregnancy; there were two women among those investigated who were pregnant at the time of the epidemic. Both had influenza; one aborted and the other had a living child, but it only lived a few weeks. The case of chronic hepatitis is said to have had a recrudescence of liver symptoms (jaundice, &c.) during convalescence from influenza. (NOTE.—The proportion of the cases in the investigation who were medically attended was 84·4 per cent.)

The following Table shows the death rate from the principal pulmonary diseases in 1915, 1916, 1917, 1918, and the first quarter of 1919 :—

———	Bronchitis.	Pneumonia.	Pleurisy.	Phthisis.
1915 - - - -	2·21	2·07	0·05	1·67
1916 - - - -	1·81	1·50	0·01	1·73
1917 - - - -	1·83	1·81	0·05	2·09
1918 - - - -	1·85	2·01	0·02	1·93
1919 (1st quarter) - -	2·84	3·12	—	1·84

INFLUENZA INVESTIGATION—APPENDIX.

(1) *Lawe Cottages Group.*

Fairly good class of self-contained houses, and fair cottages.

Street Number.	Rent. Rated on Net.	Rent. Rated on Gross.	Number of Rooms.	Occupation of Householder.
	£	£		
5½ - -	16	19	6	Dock manager.
6 - -	21	25	6	Pilot.
7 - -	24	28	6	Pilot.
8 - -	24	28	6	Master butcher.
9 - -	20	24	5	—
10 - -	30	36	9	Pilot.
11 - -	20	24	6	Undertaker.
12 - -	20	24	6	—
13 - - (three tenants).	Rent, 7*s.* 6*d.*; 7*s.*; and 5*s.*; plus rates. Rooms divided, 6; 2; 2.		10	Labourer and sailor.
14 - -	33 10*s.*	40	7	Marine Superintendent.

(2) *Thames Street Group.*

Section of street divided into eight one-roomed and two two-roomed tenements. Bad class of property; one common yard; two W.C.'s.

Street Number.	Rent.	No. of Rooms.	Occupation of Householder.
	s. *d.*		
55 - -	2 6	1	
56 - -	2 3	1	
	2 –	1	Cartman.
	2 3	1	Dock labourer.
	2 3	1	Boiler cleaner.
57 - -	2 6	2	Pit labourer.
	2 6	2	Rivetter.
58 - -	2 3	1	Fireman (Marine).
59 - -	2 3	1	Herring curer.
60 - -	2 3	1	Dock labourer.

(3) *Hepple's Court Group.*

(Waterloo Vale.)

Section of cul-de-sac street divided into one and two-roomed tenements. Bad in condition and conception.

Street Number.	Rent.	No. of Rooms.	Occupation of Householder.
	s. *d.*		
2 - -	2 10	2	
2 - -	2 10	2	Gasworker.
4 - -	2 10	2	Gasworker.
2 - -	3 3	1	
2 - -	3 –	2	Pit labourer.
2 - -	2 10	2	
2 - -	2 10	2	Shipyard labourer.
2 - -	2 10	2	Sawyer.
4 - -	3 3	1	T.I.C. labourer.
2 - -	2 10	2	

(4) *Osborne Avenue Group.*

Good class of self-contained houses.

43 - -	Rated on 24*l.* gross	6	
45 - -	,, ,,	6	Butcher.
47 - -	,, ,,	6	Com. agent.
49 - -	,, ,,	6	Collector for Tyne Dues
51 - -	,, ,,	6	Assistant Town Clerk.
53 - -	,, ,,	6	Teacher.
55 - -	,, ,,	6	Teacher.
57 - -	,, ,,	6	Teacher.
59 - -	,, 22*l.* ,,	6	Tailor's Cutter.
61 - -	,, 26*l.* 10*s.* ,,	6	House agent.

(5) *Marsden Street Group.*

Average tenement property in flats (one upper and one lower flat), yard and privy receptacle common to two tenants.

	s. *d.*		
133 - -	4 6	2	Miner.
135 - -	4 3	2	Miner.
137 - -	4 6	2	Shipyard worker.
139 - -	4 6	2	Miner.
141 - -	3 11½	2	Miner.
143 - -	3 11½	2	Miner.
145 - -	3 11½	2	Seaman.
147 - -	3 11½	2	Shipyard worker.
149 - -	4 –	2	Miner.
151 - -	3 8½	2	Miner.

(6) *Hill Street Group.*

Section of street consisting of three houses of 3 floors each (ground 1st and 2nd). Poor class of tenement property; fairly clean tenants; common yard (two levels); two w.c.'s and two privy receptacles.

Street Number.	Rent.	No. of Rooms.	Occupation of Householder.
	s. *d.*		
15 - -	3 7	2	General labourer.
13 - -	4 1½	3	Household duties.
11 - -	3 7	2	Cartman.
9 - -	4 1½	3	Dockyard labourer.
7 - -	4 8	3	Stoker.
14 - -	5 –	3	Household duties.
11 - -	3 4	2	Ship's fireman.
11 - -	2 9	2	Dockyard.
12 - -	3 10½	3	Holder-up.
13 - -	3 10½	3	Various kinds of work.

(7) *Simon Street Group.*

Section of street consisting of two houses. Bad class of tenemented property; common yards and privies.

	s. *d.*		
20	3 0	2	Foyboatman.
Bk. 20	1 9	1	Boiler cleaner.
„ 20	2 0	1	Driller.
18	3 0	2	Boiler cleaner.
Bk. 18	1 9	1	Dock fireman.
18	2 0	1	Boiler cleaner.
16	2 0	1	„ „
Bk. 16	2 0	1	Rivetter.
„ 16	2 0	1	Miner.
„ 16	2 0	1	„

(8) *Eglesfield Road Group.*

Section of street consisting of five houses. Good class of self-contained flats; separate yard and privy for each occupier.

	s. *d.*		
66	6 –	3	Engine man.
68	8 –	6	Ships' carpenter.
70	6 –	3	Iron moulder.
72	Rated on 13*l.* gross	5	Ships' plater.
74	5 6	3	Marine engineer.
76	Rated on 13*l.* gross	5	Household duties.
78	6 0	3	Corporation labourer.
80	Rated on 13*l.* gross	5	Engine fitter.
82	6 0	3	Iron moulder.
84	Rated on 13*l.* gross	5	Engine fitter.

(9) *West Park View Group*

Section of road comprising eight self-contained houses and four self-contained flats. Of modern type and situated in a good residential part of the town.

Street Number.	Rent.	No. of Rooms.	Occupation of Householder.
145	Rated on 30*l.*	11	Draper.
147	,, ,,	6	Boot dealer.
149	,, ,,	7	House furnisher.
151	,, ,,	8	Plater (shipyard).
153	,, ,,	8	Gun manager.
155	,, ,,	8	Builder.
157	,, ,,	8	Master mariner.
159	,, ,,	8	Master plumber.
Flats 161	Rent, 7*s.* 9*d.*	4	Commercial clerk.
Flats 163	Rated on 16*l.*	6	——
Flats 164	Rated on 13*l.*	4	Marine engineer.
Flats 167	Rent, 9*s.* 9*d.*	7	Contractor for scaling ships' boilers.

(10) *Double Row Group.*

(Colliery Cottages.)

Section of road comprising 10 cottages situated in the neighbourhood to fairly good class property. These cottages are old, obsolete in type, but have front garden; two rooms on the ground floor with attic in roof space; large double privy middens and ashpits serve for privy accommodation. *Rent.*—No rent is paid directly, but wages of the miner include allowances of 2*s.* for coal and 3*s.* for rent.

1	Rated on 9*l.* gross	1 up & 2 down.	Coal hewer.
2	,, ,,	,,	Deputy overman.
3	,, ,,	,,	——
4	,, ,,	,,	Deputy overman.
5	,, ,,	,,	Coal hewer.
6	,, ,,	,,	,, ,,
7	,, ,,	,,	,, ,,
8	,, ,,	,,	Deputy overman.
9	,, ,,	,,	,, ,,
10	,, ,,	,,	Coal miner.

VII.

Report on an investigation of the incidence and effects of Influenza among the Population of Warrington (Lancs.).

By

G. W. N. Joseph, M.D., Medical Officer of Health.

General Course of the Epidemic.—The epidemic occurred in three distinct waves.

The first stage commenced in Warrington about the end of June, 1918, and although the whole town was more or less affected simultaneously, probably in the early stages the Orford District was attacked most severely. The only apparent reason that can be assigned for this is the greater congestion of property in this area compared with the rest of the town.

The worst period of this first stage was reached about the middle of July, and the number of cases then diminished until the second week in August.

A second stage involving the whole town commenced about the third week of October, and continued up to the end of December.

The third stage began about the middle of February and lasted to the end of April. Again the whole town was involved.

The death-rate was heavy, and some particulars are given on pages 554 and 555.

Method of Inquiry.—The notification of mortality figures of influenza alone are not of great service. It is, of course, quite as important to have full particulars of those who do not contract the disease during an epidemic as of those who do. In order, therefore, to obtain such information, a house to house inquiry was instituted. It was not possible to visit all the houses in the town, but a fair sample was taken. Altogether 41 streets were chosen in various districts, including residential and industrial quarters, better class and lower class homes, some in open areas, others in congested areas, but quite irrespective of any known incidence of influenza in the neighbourhood. In each street at least half-a-dozen consecutive houses were selected haphazard, and full inquiries made into the medical history of each one of the inmates in these houses.

In the 41 streets, investigations were made in 353 houses regarding 1,626 inmates living therein.

The number of deaths from influenza found to have occurred during the epidemic in these houses was 9 out of the 1,626 persons.

The population of Warrington in October, 1918 (estimated from the Ration Books issued from the Food Control Office), was 68,677.

If, then, the deaths from influenza were spread throughout the population in the ratio found amoung the portion visited, there would have been 379 deaths in all, or a mortality rate of 5·5 per 1,000 population. The actual number of deaths that occurred as shown on the Death Returns was 391, giving a mortality rate of 5·6 per 1,000. So far as the death rate is concerned, therefore, the sample of the population investigated was apparently fairly representative of the whole.

It is hoped that the figures obtained in this inquiry, though small in some instances, will be found useful for comparison with results found in other areas.

I.—Age and sex constitution of the population investigated, together with the cases of Influenza in this area.

1. *Population.*—The total number of persons visited, together with the age constitution, is shown in the following table :—

TABLE I.

Age constitution of Population investigated.

Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over 75.	Total.
37	39	134	219	200	170	97	228	247	138	69	43	5	1,626

The next table shows the sex constitution in addition :—

TABLE II.

—	Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over 75.	Total.
Females -	19	18	78	108	105	94	71	135	135	61	44	22	–	890
Males -	18	21	56	111	95	76	26	93	112	77	25	21	5	736

Total persons visited, 1,626.

The actual population as obtained from the Ration Cards in June 1918 is given in the following table :—

TABLE III.

Population as estimated from Ration Cards, June 1918.

Males :—

Under 1 year	2·4 per cent.	-	745
„ 2 years	2·2 „	-	696
„ 3 „	8·08 „	-	739
„ 4 „		-	791
„ 5 „		-	941
„ 6 „	13·2 „	-	779
„ 7 „		-	882
„ 8 „		-	793
„ 9 „		-	853
„ 10 „		-	832
„ 11 „	13·2 „	-	916
„ 12 „		-	815
„ 13 „		-	846
„ 14 „		-	811
„ 15 „		-	749
„ 16 „		-	709
„ 17 „		-	787
„ 18 „		-	685
18 years and over		-	16,192
			30,561

Females :—

Under 1 year, 2·0 per cent.			765
„ 2 years, 1·7	„		666
„ 3 „			753
„ 4 „	6·3	„	796
„ 5 „			865
„ 6 „			870
„ 7 „			790
„ 8 „	10·8	„	780
„ 9 „			862
„ 10 „			839
„ 11 „			852
„ 12 „			855
„ 13 „	10·7	„	785
„ 14 „			815
„ 15 „			762
„ 16 „			821
„ 17 „			731
„ 18 „			699
18 years and over			23,810
			38,116
Total (Males and Females)			68,677

On contrasting the proportion of persons in the various age periods of the population investigated with the total proportion in the population as shown by the Ration Cards, we find the following :—

TABLE IV.

—	Under 1.	2.	5.	10.	15.	25.	35.	45.	55.	65.	Over 75.
						59·3					
Males :—											
Percentage of population investigated.	2·4	2·8	7·6	15·0	12·9						
Percentage of total population.	2·4	2·2	8·08	13·2	13·2	60·92					
Females :—											
Percentage of population investigated.	2·13	2·02	8·7	12·1	11·7	63·35					
Percentage of total population.	2·0	1·7	6·3	10·8	10·7	68·5					

(11) *Incidence of Influenza among the Population investigated.*

Out of this population of 1,626 the number of persons in which a history of an attack of influenza (occurring between June 1918 and the beginning of April 1919) could be traced was 369, or 22·6 per cent.

TABLE V.

Age Incidence of Persons who contracted Influenza.

Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over 75.	Total.
—	4	20	49	48	34	27	64	74	37	7	4	1	369
Percentage of total persons in each age group - -	10·2	14·9	22·3	24	20	27·8	28·07	29·9	26·9	10·1	9·3	20	22·6

The age and sex incidence of persons who contracted influenza during the period under survey is given in Table V., and of those who did not contract the disease in Table VII.

TABLE VI.

Age and Sex Incidence of Persons who contracted Influenza.

—	Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over 75.	Total.
Females -	–	2	8	17	23	17	22	42	46	19	6	2	–	204
Percentage	–	11·1	10·2	15·7	21·9	18·08	30·9	31·1	34·07	31·1	13·6	9·08	–	22·9
Males - -	–	2	12	32	25	17	5	22	28	18	1	2	1	165
Percentage	–	9·5	21·4	28·8	26·3	22·3	19·2	23·6	25	23·3	4·0	9·5	20	22·4

TABLE VII.

Age and Sex Incidence of those who did not take Disease.

—	Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over 75.	Total.
Females -	19	16	70	91	82	77	49	93	89	42	38	20	–	686
Percentage	100	88·8	89·7	84·2	78·0	81·9	68·9	67·6	65·8	68·8	86·3	90·9	–	-
Males - -	18	19	44	79	70	59	21	71	84	59	24	19	4	571
Percentage	100	90·4	78·5	71·1	73·6	77·2	80·7	76·2	75·0	76·5	96·0	90·8	80·0	–

The total number of those who did not contract influenza was 1,257, or 77·3 per cent., whereas 369, or 22·7 per cent., were attacked. If this, therefore, is a true indication of the incidence of the disease throughout the general population, it is evident that in all probability much less than

25 per cent. of the population suffered in the whole epidemic, as it is unlikely that any genuine cases were missed in the inquiry, although cases would certainly be included that were not true cases of influenza.

Further than this, the proportion of persons affected in each separate wave was apparently much less than the majority of people thought at the time, for it works out at 7·5 per cent. of the population in the summer, 8·2 per cent. in the autumn, and 7·9 per cent. in the winter wave (*see* Table VIII.).

From the foregoing tables it is apparent that among the population investigated—

(1) No cases of illness in children under 1 year were ascribed to influenza.

(2) The greatest proportion of cases occurred in persons between 35 and 45 years of age (29·9 per cent. of total persons at these ages), but the incidence between the ages of 20 and 55 years (varying from 26·9 per cent. to 29·9 per cent. of persons at these age periods) was almost as high.

(3) Between the ages of 1 and 2, 55 and 75, the incidence was low, varying from 9·3 to 10·1 per cent.

(4) The incidence between 10 and 15 years (24 per cent.) was higher than in any age period excepting those between 20 and 25, 25 and 35, 35 and 45, and 45 and 55.

(5) The incidence among the total male population investigated (22·4 per cent.) was very little less than that among the female (22·9 per cent.), but varied considerably at the different age periods.

(6) The largest proportion of cases occurred in females between 35 and 45 years of age (34·07 per cent. compared with 25 per cent. in males). The highest incidence among males was between the ages of 5 and 10 and 10 and 15 (28·8 per cent. and 26·3 per cent. respectively, compared with 15·7 per cent. and 21·9 per cent. in females).

(7) In females in the age groups 1 to 2, 20 to 25, 25 to 35, 35 to 45, 45 to 55, and 55 to 65, the proportion of cases was higher than the average for both sexes combined.

(8) In males in the age groups 2 to 5, 5 to 10, 15 to 20, 65 to 75, the incidence was heavier than the average for both sexes combined.

(9) Females were least susceptible to the infection at the extremes of life from birth to 10 years and after 55. Males between 20 and 25 years of age appear less liable to contract influenza than at any other period between 2 and 65 years.

2. *Age and Sex Incidence in the several Waves.*—The next tables show the age incidence in the three waves of the epidemic.

TABLE VIII.

Age Incidence.

SUMMER.

Age Group.	Under 1.	2i	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over.	Total.
No. of cases	–	1	2	18	14	13	12	22	20	15	3	2	-	122
Percentage of persons in the Age-group investigated	–	2·5	1·4	8·2	7·0	7·6	12·3	9·6	8·09	10·8	4·3	4·6	–	7·5

AUTUMN.

Age Group.	Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over.	Total.
No. of cases	–	1	12	16	17	9	7	24	38	5	1	1	1	134
Percentage of persons in the age-group investigated.	–	2·5	8·9	7·3	8·5	5·2	7·2	10·8	15·3	3·6	1·4	2·3	·20	8·2

WINTER.

No. of cases	–	2	6	15	16	12	8	25	24	17	3	1	–	129
Percentage of persons in the age-group investigated.	–	5·1	4·4	6·8	8	7·05	8·2	10·9	9·7	12·3	4·3	2·3	–	7·9

Apparently the greatest proportion of cases occurred in a different age group in each wave, and each time the incidence was more severe among older people than in the previous wave. In the Summer, persons aged 20—25 years (12·3 per cent.); in the Autumn, 35—45 years (15·3 per cent.); and in the Winter wave, 45—55 years (12·3 per cent.) were most affected.

The distribution according to sex is shown as follows:—

TABLE IX.

Table giving Age and Sex Incidence in the several Waves, with Percentage affected of Persons in the Age Group investigated.

SUMMER.

June, July, August, 1918.

—	Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over.	Total.
Females -	–	–	1	6	7	5	10	15	13	8	2	1	–	68
Percentage	–	–	1·2	5·5	6·6	5·3	14·0	11·1	9·6	13·1	4·5	4·5	–	–
Males -	–	1	1	12	7	8	2	7	7	7	1	1	–	54
Percentage	–	4·7	1·7	10·8	7·3	10·5	7·6	7·5	6·2	9·0	4·0	4·7	–	7·5

AUTUMN.

September, October, November, December, 1918.

Females -	–	1	6	6	7	6	6	14	21	3	1	1	–	72
Percentage	–	5·5	7·6	5·5	6·6	6·39	8·45	10·3	15·5	4·9	2·27	4·5	–	–
Males -	–	–	6	10	12	3	1	10	17	2	–	–	1	62
Percentage	–	–	10·7	9·0	12·6	3·94	3·8	10·7	15·1	2·5	–	–	20·0	8·2

WINTER.

January, Febuary, March, 1919.

Females -	–	1	1	5	9	6	6	15	16	8	3	–	–	70
Percentage	–	5·5	1·2	4·6	8·5	6·3	8·4	11·1	11·8	13·1	6·8	–	–	–
Males -	–	1	5	10	7	6	2	10	8	9	–	1	–	59
Percentage	–	4·7	8·9	9·0	7·3	7·8	7·6	10·7	7·1	11·6	–	4·7	–	7·9

In the summer wave the high incidence in the 20 to 25 years group was mainly due to the excessive number of females affected, whereas in the Autumn wave both sexes in the group (35–45) with the largest incidence appeared to suffer equally. In the groups 45 to 55 in the Winter wave again the proportion of females was larger than that of males.

Children and persons over 55 years of age in all three waves escaped comparatively lightly, although in the Autumn wave the incidence among children of 2–5 years was six times and in the Winter wave was three times as heavy as it was in the Summer wave.

3. *Occupational Incidence.*—The occupations of those affected is shown as follows :—

TABLE X.

Incidence of Occupation.

——	Number of Persons visited.	Number affected.	Percentage affected.
Housewife - - - - - -	395	116	29·3
Scholar - - - - - -	360	82	22·7
Ironworker - - - - -	159	40	25·1
Child under school age - - -	218	19	8·7
Wireworker - - - - -	72	14	19·4
Rubberworker - - - - -	21	10	47·6
Shop assistant - - - - -	22	8	36·6
Railwaymen - - - - -	35	7	20·0
Clerk - - - - - - -	21	7	33·3
Soap maker - - - -	39	7	17·9
Cotton operative - - - -	49	5	10·2
Tannery and leather worker - -	30	5	16·6
No occupation - - - - -	56	5	8·9

——	Number of Persons visited.	Number affected.	Percentage affected.
Joiner and building work - - -	12	4	33·3
Aluminium worker - - - -	9	4	44·4
Hotel and Cafe Waitress - - -	5	3	60·0
Carter - - - - - -	9	2	22·2
Engineer - - - - - -	6	2	33·3
Confectioner - - - - -	4	2	50·0
Chemical worker - - - -	8	2	25·0
Tramway worker - - - -	3	2	66·6
Fustian cutter - - - - -	3	2	66·6
Filecutter - - - - - -	2	2	100·0
Milliner and dressmaker - - -	5	2	40·0
All other occupations in which only one person contracted influenza.	83	17	20·4

Children under school age and persons of no occupation evidently suffered less proportionately than other persons.

It is interesting to note the incidence among women who worked entirely at home compared with those who worked away from home :—

——	Number of Women.	Number of Cases of Influenza.	Per-centage.
Total women in population investigated between ages of 15 and 45 years.	435	132	30·3
Number of these women who worked at home.	281	90	32·0
Number of these women who worked away from home.	154	42	27·2

The percentage of cases was higher among those who worked at home than among the other class, but the difference is not so striking as one might have expected, considering the fact that the housewife in many instances was in contact continuously for many days with severe cases of the disease.

4. *Incidence on Person nursing the Disease.*—Among the cases investigated it was found that in 284 cases the nursing was done by a member of the same household.

In 56 instances (19·7 per cent.) the nurse developed the disease, whilst in 228 cases (80·3 per cent.) influenza was not contracted.

5. *Sleeping Room Infection.*—The total number of persons found to have occupied a bedroom with a patient suffering from influenza was 235. Of these 70 or 29·8 per cent. contracted influenza, and 165, or 70·2 per cent., were not infected.

6. *Multiple Attacks.*—The following figures have a bearing on the question of immunity :

SUMMER WAVE.

	Per cent.	
Number of males attacked - -	54 or 7·3	Of population investigated.
„ females attacked -	68 or 7·6	
Number of males who escaped -	681 or 92·5	
„ females who escaped -	823 or 92·4	

Autumn Wave.

	Per cent.	
Number of males attacked	62 or 8·4	Of population investigated.
„ females attacked	72 or 8·08	
Number attacked of males who had suffered in Summer	2 or 3·6	
Number attacked of females who had suffered in Summer	4 or 5·8	
Number attacked of males who had escaped in Summer	60 or 8·8	
Number attacked of females who had escaped in Summer	68 or 8·2	

Winter Wave.

Number of males attacked	59 or 8·01	Of population investigated.
„ females attacked	70 or 7·8	
Number attacked of those who escaped both previous waves :—		
Males	51 or 6·9 per cent.	
Females	68 or 7·9 „	
Number attacked of those who escaped Summer but attacked Autumn :—		
Males	5	
Females	0	
Number attacked of those who escaped Autumn but attacked Summer :—		
Males	1	
Females	2	
Number attacked of those who were attacked both Summer and Autumn :—		
Males	2	
Females	—	

With regard to the immunising effect of a previous attack it would appear as though females are less liable than males to a second attack.

In the Winter wave 119 persons (51 males, 68 females) contracted influenza who escaped the previous two waves.

In addition, eight males were attacked; one of these had had the disease in the Summer wave, five in the Autumn wave, and two had been attacked before in both waves.

Only two females, however, had previously had the disease among those attacked in the Winter wave, and these had had it during the Summer wave.

7. *House Incidence.*—As might be expected it was found that the incidence of disease was highest in the most overcrowded houses :—

(i) Houses with one or more Persons per Room.	(ii) Houses with less than one Person per Room.
There were 251 houses occupied by 1,358 persons. The number of persons affected was 327 = 24·07 per cent.	There were 102 houses occupied by 268 persons. The number of persons affected was 42 = 15·6 per cent.

The virulence of the epidemic appeared to increase in each wave, and whereas 11·6 per cent. of the houses were invaded in the first wave, 15 per cent. were invaded in the last wave.

More than half the houses escaped without having a case of infection in any of the waves.

Number of Houses invaded Summer only.	Number of Houses invaded Autumn only.	Number of Houses invaded Winter only.	Number of Houses escaped all three Waves.	Number of Houses invaded all three Waves.	Number of Houses invaded Summer and Autumn.	Number of Houses invaded Summer and Winter.	Number of Houses invaded Autumn and Winter.
41	44	54	183	6	8	7	10
Per cent. 11·6	Per cent. 12·4	Per cent. 15·2	Per cent. 51·8	Per cent. 1·7	Per cent. 2·03	Per cent. 1·9	Per cent. 2·8

Multiple Cases.—The number of houses visited was 353.
Of these houses—

		Per cent.
The number without a case was	183	51
„ „ with 1 case was	71	20
„ „ „ 2 cases was	46	13
„ „ „ 3 „ „	18	5·1
„ „ „ 4 „ „	18	5·1
„ „ „ 5 „ „	10	2·8
„ „ „ 6 „ „	4	1·1
„ „ „ 7 „ „	2	0·56
„ „ „ 8 „ „	1	0·02

8. *Incubation Period.*—Out of 385 cases inquired into, the shortest incubation period recorded was 12 hours, and the longest given was six days. The average was two days, and is probably shorter than this.

9. *Medical Attendance.*—The proportion of cases receiving medical attention was high. Out of 385 instances a doctor was in attendance on 323, or 83·8 per cent.

10. *Effect of Attendance at Places of Entertainment.*—In the following table those persons who are given as attending places of entertainment are those only who visited such places regularly at frequent intervals, and in those cases in which the person contracted influenza the period elapsing between the date of last visit and the date of onset of disease did not exceed a week or 10 days :—

(*a*) Total number of persons visited	1,626
(*b*) Number of these who frequented places of entertainment	227
(*c*) No of persons who contracted influenza among those who frequented places of entertainment	49 or 21·5 per cent.
(*d*) Number of persons who did not frequent places of entertainment	1,399
(*e*) Number of persons who contracted influenza among those who did not frequent places of entertainment	320 or 22·8 per cent.

From the above there does not appear to be much difference in the incidence of influenza among the two classes, and if anything, the proportion of persons attacked is slightly heavier among those who did not visit places of amusement.

This incidence, however, varied in the three waves, and is shown as follows:—

——	Summer Wave.	Autumn Wave.	Winter Wave.
Total persons visited -	1,626	1,626	1,626
Number of these who had influenza.	122 or 7·5 per cent.	134 or 8·2 per cent.	129 or 7·9 per cent.
Number who attended places of amusement.	227	227	227
Number of these who had influenza.	8 or 3·4 per cent.	20 or 8·8 per cent.	21 or 9·2 per cent.
Number who did not attend places of amusement.	1,399	1,399	1,399
Number of these who had influenza.	114 or 8·1 per cent.	114 or 8·1 per cent.	108 or 7·7 per cent.

The proportion of those affected who did not attend places of entertainment was fairly constant throughout the three waves (varying only from 8·1 per cent. in the summer to 7·7 per cent. in the winter), whereas amongst those who attended such places the incidence rose from 3·4 per cent. in the summer to 9·2 per cent. in the winter.

There is a possibility that the predisposing effect of a chill due to waiting in queues before the performance may have had something to do with this higher incidence in winter, because, if anything, ventilation of the halls was better then, as special regulations were in force.

11. *Effect of Influenza on Pregnant and Lying-In Women.*—From the cases reported from time to time by doctors and midwives in the district, it appeared that influenza was specially deleterious to pregnant women and women in childbirth. An attempt was made to collect some information on this subject.

In addition to the usual card filled in by the health visitors at the time of their visits under the Notification of Births Act, or in connection with visits under the Midwives Act, a special card was drawn up, divided into two parts, one part of which had to be completed in every instance according to whether the mother *had had* or *had not had* influenza during pregnancy. This card was in use for a period of four months from the 1st December 1918 to 31st March 1919, and during this time inquiries were made in the 475 notifications received.

There was a history of the mother having suffered from influenza during pregnancy in 187, or 39·3 per cent. of the cases.

The incidence of influenza among women of child-bearing age (from 15 to 45 years) found in the general inquiry (*see* page 546) was 30·3 per cent. of the cases.

According to these figures it is evident that the pregnant woman was more liable to the disease than the average woman at the same age. It is probable, too, that many cases of pregnancy that terminated in early abortion would not come to our notice, so that the incidence of influenza among pregnant women was probably higher than the percentage shown. On the other hand, it is quite possible that there might be more exaggeration of the numbers than among the general population owing to

a tendency to ascribe many of the ailments of pregnancy to influenza, especially if there happened to be a genuine case in the house.

Total cases inquired into, 471.

	Women who had Influenza during Pregnancy, 187.		Women who did not have Influenza during Pregnancy, 284.	
	No.	Per Cent.	No.	Per Cent.
Normal confinements	137	73·2	223	78·52
Abnormal confinements, including all forceps confinements.	50	26·8	61	21·48
Deaths of mothers	8	4·2	—	—

Summary of Abnormal Cases of Labour.

	Total Women who had Influenza, 187.		Total Women who did not have Influenza, 284.	
	No.	Per Cent.	No.	Per Cent.
Ordinary forceps cases without complications.	15	8·01	33	11·6
Other abnormality, viz:	35	19	28	9·85
Miscarriage and abortion	7	3·74	9	3·17
Premature births	12	6·42	8	2·83
Still births, premature	9	4·81	3	1·05
„ full time	2	1·07	7	2·46
Postpartum hæmorrhage	3	1·60	1	·35
Phlegmasia alba dolens	1	0·53	Nil	Nil
Retained placenta and collapse	1	0·53	Nil	Nil

Effect on Child.

	Mother suffered from Influenza during Pregnancy (187 = 39·3 per Cent.).		Mother did not suffer from Influenza during Pregnancy (284 = 60·4 per Cent.).	
	No.	Per Cent.	No.	Per Cent.
Healthy full-time babies	137	73·26	232	81·69
Poor or fair babies	21	11·23	25	8·80
Dead or immature	29	15·51	27	9·50

Further contrasting the maternal death-rate and infantile death-rate due to the causes prematurity and still-birth for the past five years with the period under review, we find there is a markedly increased death-rate under these headings during the influenza epidemic.

——	Total known Confinements, &c.	Maternal Deaths.		Infantile Deaths due to			
				Prematurity.		Still Births.	
		No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
1913 - -	221	4	·18	35	1·58	87	3·84
1914 - -	2,271	6	·26	42	1·85	72	3·17
1915 - -	1,980	5	·25	29	1·46	2	1·61
1916 - -	1,850	4	·22	26	1·40	23	1·24
1917 - -	1,576	3	·19	20	1·27	23	1·45
Four months Dec. 1918 to Mar. 1919.	471.	8	1·7	20	4·29	21	4·46

Table showing Months of Pregnancy in which Mothers had attack of Influenza which was followed by Abnormal Confinement.

——	1st.	2nd.	3rd.	4th.	5th.	6th.	7th.	8th.	9th.
Still Births (premature) -	–	1	–	–	2	2	4	–	–
,, (full time) -	–	–	–	–	–	–	–	2	–
Premature Births - -	3	1	–	–	1	2	4	1	–
Abortion or Miscarriage -	1	2	1	–	2	1	–	–	–
Postpartum hæmorrhage -	1	1	–	1	–	–	–	–	–
Phlegmasia alba dolens -	–	–	–	–	–	–	–	1	–
Partially retained placenta and collapse - - -	–	–	–	–	–	–	–	–	1
TOTAL - - -	5	5	1	1	5	5	8	4	1

Effect of an Attack of Influenza occurring at time of Confinement is shown in table.

——	Total No. of Cases.	No. of normal Confinements.	No. in which Child was premature.	No. of Deaths of Mothers.
Cases in which influenza occurred at the time of confinement.	16	4 (25 per cent.).	12 (75 per cent.).	8 (50 per cent.)
Cases in women who did not have influenza at time of confinement.	455	364 (71·1 per cent.).	36 (7·9 per cent.).	—
Cases in women who did not have influenza at all.	284	223 (78·5 per cent.).	20 (7 per cent.).	—
Cases in women who had influenza during pregnancy and not at time of confinement.	187	141 (73·7 per cent.).	28 (14·6 per cent.).	—

There is no doubt that an attack of influenza occurring towards the end of pregnancy is a very serious matter, both for mother and child.

Out of 16 cases of the disease at this period there were no less than eight deaths of mothers.

Effect of Influenza on Pregnant Women treated in an Institution.

From figures kindly supplied to me by Dr. Hodgkinson, Medical Superintendent, Union Infirmary, Warrington, I am able to draw up the following table of cases dealt with in that institution during the six months ending 31st March, 1919 :—

——	No. of Women.	No. of Deaths.
Total number of known pregnant women admitted suffering from influenza.	6	2 = (33·3 per cent.).
Total number of women aged 16 years to 45 years admitted suffering from influenza.	41	14 = (34·1 per cent.).

The risk to life of the pregnant women suffering from influenza when nursed in an institution does not appear, according to this, to be any greater than it is for all women of similar ages.

II.—Deaths from Influenza.

The total deaths from influenza in 1918 was 272, giving a death-rate of 3·9 per 1,000 of the population.

During the period of the epidemic, from June 1918 to April 1919, the number of deaths certified as due to influenza was 391, distributed as follows :—

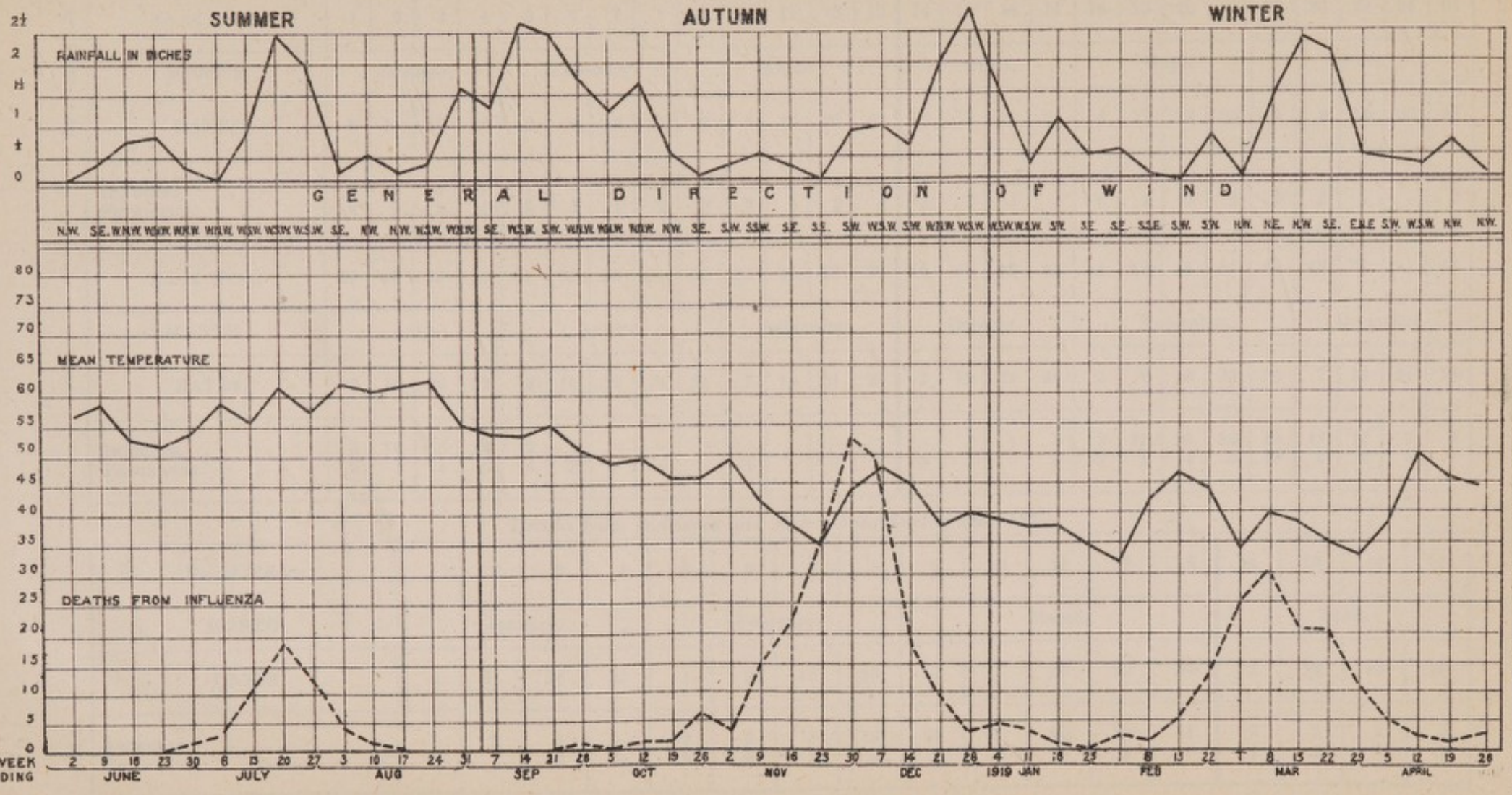
Summer wave - - - - - - - -	47
Autumn - - - - - - - - -	203
Winter - - - - - - - - -	141

The table on page 553 shows the number of deaths each week due to influenza and pneumonia. A chart is given showing the number of deaths at various times of the year and the meteorological conditions so far as rainfall, temperature, and prevailing winds are concerned.

The age and sex incidence of the persons who died is shown in the next table :—

——	Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over 75.	Total.
Females -	4	7	17	15	11	13	18	45	33	17	25	10	8	223
Males -	3	3	20	11	1	5	6	30	33	17	22	13	4	168
Percentage	7	10	37	26	12	18	24	75	66	34	47	23	12	391

WARRINGTON.



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Deaths from Influenza and Pneumonia each week during year 1918.

No. of Week.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
MONTH.	January.				February.				March					April.				May.				June.				
DATES.	5	12	19	26	2	9	16	23	2	9	16	23	30	6	13	20	27	4	11	18	25	1	8	15	22	29
Influenza - -	–	1	1	1	1	–	–	–	–	–	–	1	1	–	–	–	–	–	–	–	–	–	–	–	–	1
Pneumonia - - -	2	3	2	4	3	3	1	1	3	3	3	2	4	5	3	4	3	6	7	7	2	3	2	–	–	2

No. of Week.	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
MONTH.	July.				August.					September.				October.				November.					December.			
DATES.	6	13	20	27	3	10	17	24	31	7	14	21	28	5	12	19	26	2	9	16	23	30	7	14	21	28
Influenza - - -	3	10	18	12	2	1	–	–	–	–		–	1	–	1	1	5	3	15	20	36	49	44	17	9	3
Pneumonia - - -	2	1	4	5	3	2	–	–	–	1	–	1	–	2	–	–	2	4	1	4	9	12	9	2	5	1

Deaths from Influenza and Pneumonia during year 1919.

No. of Weeks.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
MONTH.	January.				February.				March.					April.			
DATES.	4	11	18	25	1	8	15	23	1	8	15	23	30	5	12	19	26
Influenza - - - -	3	3	1	—	2	2	5	12	24	30	20	20	11	5	2	1	2
Pneumonia - - - -	1	1	2	—	5	6	7	4	5	4	8	2	7	4	2	4	2

Influenza Death-Rate per 1,000 of the Population in the Age and Sex Groups obtained from the Statistics of Ration Cards.

——	Under 1.	2.	5.	10.	15.	All other Ages.
Males :—						
Rate per 1,000	4·0	4·3	8·0	2·6	·2	7·2
Females :—						
Rate per 1,000	5·2	10·5	7·0	3·6	2·7	6·4

The death-rate per 1,000 among the female population at any age-group appears to have been higher than among the males except in the case of children 2—5 (males 8 and females 7 per 1,000).

The following tables show the number of deaths that occurred in the various age-periods during the three waves :—

SUMMER.

——	Under 1.	2.	5.	10.	15.	20.	25.	35.	45.	55.	65.	75.	Over 75.	Total.
Females -	1	–	–	3	–	–	2	3	6	4	3	1	–	23
Males -	–	–	3	–	–	–	2	4	8	4	2	1	–	24

AUTUMN.

Fem les -	–	3	8	7	10	9	13	28	18	7	12	5	3	123
Male -	1	–	10	7	1	4	2	11	16	8	11	5	4	80

WINTER.

Females -	3	4	9	5	1	4	3	14	9	6	10	4	5	77
Males -	2	3	7	4	–	1	2	15	9	5	9	7	–	64

Population from Ration Cards :—

Females - - - - - - - -	38,116
Males - - - - - - - -	30,561

Total Female deaths, 223 = 5·8 per 1,000.
„ Male „ 168 = 5·4 „

A striking feature is the fact that there were few deaths among males of the age-periods 10-25 compared with the number of female deaths at the same ages.

From our investigation, however, the actual incidence of illness among males was higher than among females of those ages.

The total death-rate among females was slightly higher than among males (females, 5·8 per 1,000 ; males, 5·4 per 1,000).

In the *Summer wave* the deaths among females were very similar both in number and age-group affected to those occurring among males, but the death-rates worked out on the population estimated from the Ration Cards was : males, ·7 per 1,000 ; females, ·6 per 1,000.

In the *Autumn* there were 123 deaths of females compared with 80 of males, the excess among females being chiefly between the ages of 10–35 years. (The death-rate was 3·1 per 1,000 females and 2·6 per 1,000 males).

In the *Winter wave* the death-rate for males was 2·09, and for females 2·02 per 1,000.

Evidently the Autumn wave was more fatal to females than either the Summer or Winter waves.

Investigations elicited the following facts:—

(*a*) *Occupation of those who died during the epidemic.*—

	Per cent.
Housewife	31
Persons engaged in factories	17
Ironworkers	15
Under school age	14
School children	10
Persons engaged in shops or public places	8
Invalids, &c., of no occupation	4
Soldier	1

(*b*) *Places of Amusement.*—It was found that 11 per cent. regularly attended places of entertainment and had been there within one week of commencement of illness.

(*c*) *Infectivity of Fatal Cases.*—In houses in which a death occurred, 40·04 per cent. of the inmates contracted influenza, whereas in infected houses where no deaths occurred the proportion of cases was 38·4 per cent. of the inmates.

Deaths from other Diseases of the Respiratory Organs.

During the epidemic period of 10 months the following deaths from diseases of the respiratory organs occurred in addition to those from influenza:—

Pneumonia	129
Bronchitis	102
Empyema	2
Acute pleuritis	1
Asthma	1
Total	235

They are contrasted with the total deaths from these diseases during previous years:—

1913	1914	1915	1916	1917	1918
197	204	251	234	252	250

In conclusion, I am much indebted to Dr. J. R. Hutchinson, of the Ministry of Health, for valuable advice, and to Mr. J. Stevens of our Health Department, who has given great assistance both in collecting and summarising the statistics.

October 1919.

VIII.

Analysis of an Influenza Census at Newcastle-upon-Tyne,

By

S. J. Clegg, M.D., Deputy Medical Officer of Health.

Owing to the exceptional prevalence of influenza in Newcastle and the country generally during the past nine months, it was felt that an investigation into the local conditions influencing the Newcastle outbreaks might prove useful, and an "Influenza Census" was therefore begun. This consisted in a house-to-house enquiry in two areas at either end of the city. The areas selected were known to have had a definite influenza prevalence, especially during the January–March (1919) outbreak. Streets in these areas were picked out so as to include flat, tenement, and self-contained households, but apart from this no selection was made.

Nine hundred and seventy households (the majority being flats) containing 4,461 occupants, were investigated and the following results were obtained.

General incidence on Population.—890 cases occurred during the summer, autumn, and winter epidemics (June, July, August; October, November, December; January, February, March), giving an attack rate of 200 per 1,000 population. On these figures there must have been, approximately, 50,000 to 56,000 cases in Newcastle alone during the three epidemic periods. The incidence varied in the three outbreaks, being 62 per 1,000 population in summer, 52 per 1,000 population in autumn, and 86 per 1,000 population in winter; the average attack rate per 1,000 being, therefore, about 67. (The average attack rate for the last three years in Newcastle of enteric fever, scarlet fever, and diphtheria were 0·21, 3·0, and 0·9 per 1,000 population, respectively.)

Mortality Figures.—During the three epidemic periods the case mortality rate was 4·3 per cent., being 2·2 per cent. in the summer, 3·4 per cent. in the autumn, and 6 per cent. in the winter outbreaks. The case mortality rates over the three outbreaks for the different age periods are:—

	Per cent.
Under 1 year	38·4
Between 1 and 5	17·1
„ 5 and 15	2·6
„ 15 and 25	3·5
„ 25 and 45	1·5

At ages over 45 years, the case mortality rate was 2·3 per cent. Of the total deaths, 53 per cent. were in children below the age of five years.

Age incidence in Infected Households only.—Taking the three outbreaks as a whole, the greatest number of cases occurred in the 25–45 age period and the next greatest in the 5–15 age period.

A noteworthy change in the age incidence occurred between the summer and winter epidemics as is shown in Table III. of Dr. Hutchinson's addendum. This was apparently general throughout the country and was not confined to Newcastle alone.

Sex incidence in Infected Households only.—Of 890 cases, 41 per cent. were males and 59 per cent. females. The higher incidence in females occurred exclusively between the ages of 15–45, in which period the proportion of females to males attacked was as 2 : 1; below the age of 15 the proportions were reversed, being as 6 : 7. Whether the excess of female cases over males at the higher-age periods is *entirely* due to the abnormally low proportion of males of those ages at the time is not clear.

Occupation.—A large proportion (70 per cent.) of the female cases between the ages of 15 and 45 were engaged in "home duties," and the majority appeared to have become infected from the nursing of other

patients; many, however, were either first cases in a household and infected others, or were the only cases in the households affected. Apart from this occupation appears to play no part in the incidence.

Incubation Period.—The incubation period, calculated on the period elapsing between the date of onset of the first case in a household and the dates of onset of subsequent immediate contacts, varied from one to six days, in the great majority of cases being within three days.

Period of Infectivity.—No data were obtained bearing on this point owing to the difficulty of excluding other sources of infection.

Period of Immunity.—Second Attacks.—Of the 890 cases, 39 or 3·7 per cent. developed second attacks at the end of six months, 19 developed second attacks at the end of three months, and 2 were attacked in each of the three epidemics. A few cases also were stated to have had two attacks in two successive months. Reliable information could not be obtained as to the influence of the severity of the first attack on the period of immunity.

A certain degree of immunity therefore appears to be conferred by an attack of influenza, varying in different individuals and becoming noticeably less after six months.

Influence of Overcrowding.—(Tables A. and B.).—The evidence as to the influence of overcrowding appears to be inconclusive; the tables would appear to show that there is actually more chance of contracting infection by living in a house with less than one person per room than there is by living in a house with four or more persons per room. The inference would seem to be that overcrowding *quâ* overcrowding is not the prime factor in the spread of infection.

Previous Medical History.—Enquiry was made into the previous medical history of occupants of both affected and non-affected households, with a view to ascertaining whether previously existing disease such as tuberculosis, septic diseases, &c., had any influence either in predisposing to or protecting from attack, but no conclusion could be come to from the information obtained.

Complications.—13 per cent. of all cases developed complications of the respiratory system, of which 64 per cent. were pneumonia.

Of 53 second attacks, 14 were complicated similarly, (or 26 per cent. of cases) 71 per cent. of which were pneumonia.

The incidence of respiratory complications (pneumonia and bronchitis) was nearly four times as high in the winter outbreak as in the summer one, and twice as high in the autumn as in summer (5·4 per cent., 11 per cent. and 19·6 per cent. of cases respectively).

The higher incidence of complications in second attacks may thus be partly explained inasmuch as the great majority of second attacks occurred in the winter outbreak. Overcrowding appears to have no influence on the incidence of complications.

Incidence and infectivity of Pneumonia.

Seventy-five cases developed pneumonia, 4·3 per cent. of cases in summer, 7·6 per cent. of cases in autumn, and 11·6 per cent. of cases in winter. As previously stated, overcrowding appears to exert no influence on the incidence of pneumonia.

In all except three instances, only one case of pneumonia occurred in any one household, although there were other cases of influenza actually ill at the time. Of the exceptions, in each of two households three cases appeared to develop simultaneously, *all* the influenza cases being affected—in the other household, a second case of pneumonia followed a first at an interval of 3 days. In no case were any special measures taken to protect one case from infection by another, as far as could be ascertained.

The spread of pneumonia from one case to another would, therefore, appear to be uncommon.

TABLE A.

	Less than 1 Person per Room.					1–2 Persons per Room.					2–3 Persons per Room.					3–4 Persons per Room.					4 and more Persons per Room.				
	Affected.			Not affected.		Affected.			Not affected.		Affected.			Not affected.		Affected.			Not affected.		Affected.			Not affected.	
	Households.	Occupants.	Cases.	Households.	Occupants.	Households.	Occupants.	Cases.	Households.	Occupants.	Households.	Occupants.	Cases.	Households.	Occupants.	Households.	Occupants.	Cases.	Households.	Occupants.	Households.	Occupants.	Cases.	Households.	Occupants.
Whole Period	36	104	49 (47·1 per cent.)	65	150	166	665	270 (40·6 per cent.)	222	804	142	777	322 (41·4 per cent.)	150	752	77	482	192 (40 per cent.)	61	369	22	161	60 (37·2 per cent.)	29	197

TABLE B.

Households containing	Total Number of Households investigated.	Number of Households affected in whole Epidemic Period.	Number of Households not affected throughout.	Proportion of affected Households to Total.	Total Number of Cases.	Average Number of Cases per affected Household.	Actual Number of Cases to immediate Contacts.	Percentage number of Cases to immediate Contacts in affected Households.
								Per Cent.
Less than 1 person per room -	101	36	65	1 in 2·8	49	1·36	1 in 2·12	47
1-2 persons per room - -	388	166	222	1 in 2·3	270	1·63	1 in 2·42	40·6
2-3 persons per room - -	292	142	150	1 in 2	322	2·27	1 in 2·41	41·4
3-4 persons per room - -	138	77	61	1 in 1·8	192	2·50	1 in 2·52	40
4 and more persons per room -	51	22	29	1 in 2·3	60	2·72	1 in 2·68	37
Total - - - -	970	443	527		893			

Table C.

Incidence of Complications.

—	Less than 1 per Room.		1—2 per Room.		2—3 per Room.		3—4 per Room.		4 and more per Room.		Totals.		Summer.	Autumn.	Winter.
	Pneumonia.	Bronchitis.	Pneumonia.	Bronchitis.	Pneumonia.	Bronchitis.	Pneumonia.	Bronchitis.	Pneumonia.	Bronchitis.	Pneumonia.	Bronchitis.			
Single attacks -	2	4	13	10	26	15	17	8	7	1	65	38	15 (5·4 per cent.).	26 (11·0 per cent.).	76 (19·6 per cent.).
Second attacks -	2	—	1	1	5	—	1	2	1	1	10	4			
	4	4	14	11	31	15	18	10	8	2	75	42			
Totals - -	8		25		46		28		10		117		—	—	—
Percentage to cases	16·3 per cent.		9·2 per cent.		14·3 per cent.		14·6 per cent.		16·6 per cent.		13·1 per cent.		3·4 per cent.	11 per cent.	19·6 per cent.

Percentage Complications to Second Attacks :—26 per cent. (14 complicated cases in 53).
No. of Second Attacks : 53.

Incidence of Concurrent Diseases, e.g., E.C.S.M., Poliomyelitis.—Enquiry was made as to above, in view of the statements which have appeared in the Medical Journals recently describing organisms obtained from filtrates of material from cases of E.C.S.M., poliomyelitis, typhus, measles, &c., which were apparently morphologically identical.

No evidence of any increase of these diseases concurrently with the influenza outbreaks was noted.

Use of Preventatives.—Numerous drugs and quack remedies, disinfectants, &c., were used but without any apparent effect.

Addendum by Dr. J. R. Hutchinson to Dr. Clegg's Analysis of the Newcastle-upon-Tyne Influenza Figures.

970 households were investigated: of these, 439 houses were invaded. The number of persons attacked was 828; 60 of these had more than one attack; the aggregate number of attacks was 890.

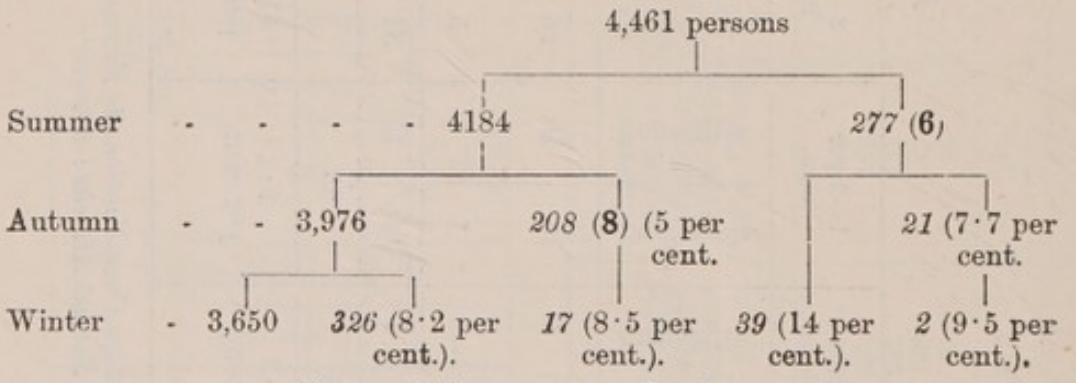
768 had one attack (17·2 per cent. of population investigated).
58 „ two attacks (1·3 „ „ „ „).
2 „ three „ (0·04 „ „ „ „).

Occupational Incidence.—Of the 828 persons attacked:—
34 per cent. were children of or below school age.
39 per cent. were women engaged in household duties.
5 per cent. were labourers.
4 per cent. were ironworkers.
3 per cent. were shopkeepers.
2 per cent. were clerks.

The remainder comprised cobblers, teachers, carpenters, horsemen, railway employees, dressmakers, barmaids, painters, bricklayers, tailors, barbers, coachbuilders, tramway workers, miners, weavers, papermakers, motormen, firemen, &c., &c.

Information as to nursing is not available.

Immunity.—The table below summarises the data on this point.



Figures in heavy type = deaths.
Figures in italics - = attacks.

TABLE I.

Age and Sex Incidence of Cases.

——	Under One Year.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45–55.	55–65.	65–75.	75 and over.	Totals.
Males - - - - -	5	8	42	56	48	32	12	45	43	32	12	6	—	341
Females - - - -	8	12	28	49	40	44	52	98	78	51	19	5	3	487
Total - - -	13	20	70	105	88	76	64	143	121	83	31	11	3	828

41 per cent. were males and 59 per cent. females.

TABLE II.

Mortality Incidence.

—	Under One Year.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45–55.	55–65.	65–75.	75 and over.	Totals.
Summer:—														
Males	1	1	2	—	—	—	—	—	—	—	—	—	—	4
Females	1	—	—	1	—	—	—	—	—	—	—	—	—	2
Autumn:—														
Males	1	—	1	—	—	1	—	—	—	—	—	—	—	3
Females	—	2	2	—	—	—	—	—	1	—	—	—	—	5
Winter:—														
Males	2	—	2	1	—	2	—	1	—	—	—	1	—	9
Females	—	1	3	—	3	1	1	2	—	2	—	—	—	13
Totals	5	4	10	2	3	4	1	3	1	2	—	1	—	36
Case mortality	38·4 per cent.	20 per cent.	14·3 per cent.	2·6 per cent.		3·5 per cent.		1·5 per cent.		1·7 per cent.		7·1 per cent.		4·3 per cent.

TABLE III.

Age and Sex Incidence in the Three Waves.

——	Under One Year.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45–55.	55–65.	65–75.	75 and over.	Totals.	
Summer:—															
Males - - -	—	3	10	12	14	12	2	17	13	8	5	2	—	98	234
Females - - -	2	3	5	13	7	15	15	33	28	8	6	—	1	136	
Autumn:—															
Males - - -	2	1	9	13	15	12	5	13	8	7	2	2	—	89	208
Females - - -	1	3	8	14	9	11	14	26	14	13	4	1	1	119	
Winter:—															
Males - - -	3	4	21	25	17	8	4	11	19	16	5	1	—	134	326
Females - - -	4	5	15	20	21	16	19	29	26	24	8	4	1	192	
Summer and Autumn:—															
Males - - -	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2
Females - - -	—	—	—	—	—	—	—	1	—	1	—	—	—	2	
Autumn and Winter:—															
Males - - -	—	—	—	—	—	—	—	3	2	—	—	—	—	5	17
Females - - -	—	1	—	2	1	—	2	2	2	1	1	—	—	12	
Summer and Winter:—															
Males - - -	—	—	2	5	2	—	1	1	1	1	—	1	—	14	39
Females - - -	1	—	—	—	2	2	2	6	8	4	—	—	—	25	
Summer, Autumn, and Winter:—															
Males - - -	—	—	—	1	—	—	—	—	—	—	—	—	—	1	2
Females - - -	—	—	—	—	—	—	—	1	—	—	—	—	—	1	
							Persons attacked - - - - - - -							828	

28·2 per cent. of the persons suffered in summer; 25·1 per cent. of the persons suffered in autumn; 39·3 per cent. of the persons suffered in winter; while 7·2 were attacked in two or all three outbreaks.

IX.

Summary of Results of an inquiry at Wigan, made under the supervision of J. R. Hutchinson, M.D.

Population, 80,365. Deaths from Influenza, June 18—March 19 (inclusive).

Total number of households investigated = 204.

„ „ „ inhabitants of these houses = 1,027 { 501 males. 526 females.

„ „ „ households invaded = 108.

Streets Included.—Northumberland, Great George, Lime, Brook, Golborne, Duke, Albert, Seed and Platt Lane. These streets are situated in different parts of the borough and were selected as typical.

Age and Sex Analysis of the Population, Cases, &c.

—	Under 1 year.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45–55.	55–65.	65–75.	75 and over.	Totals.
Age and sex constitution of population investigated	14	8	33	70	70	50	33	79	67	67	25	9	1	501 males.
	15	12	36	66	67	49	48	94	64	65	23	9	1	526 females.
Age and sex incidence of persons attacked.	2	1	13	17	8	5	5	23	17	7	6	0	1	105 males.
	1	3	8	16	14	8	13	39	15	15	3	3	0	138 females.
Age and sex incidence of death.	–	–	1	1	–	–	–	2	–	–	–	–	–	4 males.
	1	1	2	2	–	1	1	2	1	–	1	1	–	13 females.
Age and sex of cases in summer wave.	1	–	1	3	1	1	2	8	2	1	–	–	–	20 males.
	–	1	1	2	3	2	4	8	1	2	–	1	–	25 females.
Age and sex of cases in autumn wave.	–	–	6	5	6	–	–	10	5	–	3	–	1	36 males.
	–	2	3	7	4	3	3	14	6	3	–	1	–	46 females.
Age and sex of cases in winter wave.	1	1	6	9	1	5	3	6	10	6	3	–	–	51 males.
	1	–	4	7	8	4	6	17	8	10	3	1	–	69 females.
Total attacks all three waves.	3	4	21	33	23	15	18	63	32	22	9	3	1	247

Two persons had two attacks in both autumn and winter, and one in summer, autumn and winter.

Occupational Incidence.—Of the 105 *males*, 37 were coal miners (hewers, drawers, pit boys); the remainder included ironworkers, firemen, railway workers, bottle-makers, spinners, weavers, candle-makers, horsemen, skepmakers, works managers, in and outdoor labourers and school children. The incidence on coal miners is not excessive having regard to the preponderance of this class of worker.

Of the 138 *females*, 63 or nearly 50 per cent. were engaged in housework; the remainder were spinners, weavers, tailoresses, railway carriage cleaners and school children.

In 105 invaded households the housewife or the person presumably occupied as such escaped infection in 46 or practically 44 per cent. of cases.

In 35 (*ex* 105) instances in which the housewife amongst others was infected.

She was the 1st (or one of the first) cases in 17 instances.
" " 2nd case in 15 instances.
" " 3rd " " 1 "
" " 4th " " 1 "
" " 7th " " 1 "

In 24 instances, the housewife herself was the only case.

Taking only households with multiple cases in one or other wave, the liability of the nurse to infection as compared with that of other contacts was as 39·2 per cent. is to 47 per cent.

The number of persons attacked in Summer was 45.—There were no deaths. The number who escaped was 982. Of the 45, one was again attacked in autumn. Of the 982, 81 were attacked in autumn and six died.

The number of persons attacked in winter :—

(i) Of the 981 who escaped in summer and autumn was 117—11 died.

(ii) Of the 75 survivors of those who escaped in summer, but were attacked in autumn, was two.

(iii) Of those who escaped in autumn, but were attacked in summer, was—nil.

(iv) Of those who were attacked in both summer and autumn waves, was—one.

There is no evidence to show whether there is any correlation between degree of immunity and severity of attack.

The incidence on houses with one or more persons per room as compared with those with less than one person per room was as 1·5 is to one.

No. of houses invaded in summer	15
" " " " autumn	29
" " " " winter	48
" " escaped in all three waves	96
" " invaded " " "	1
" " " in summer and autumn	3
" " " " " winter	3
" " " in autumn and winter	9
No of houses with one case	48
" " " two cases	26
" " " three "	12
" " " four "	11
" " " five "	8
" " " six "	0
" " " seven "	3

In seven instances every member of the family was infected in the one outbreak; the number of persons in the families were 2, 3, 4, 5, 5, 7 and 7, respectively.

The incubation period as gathered from these reports is from one to two days, occasionally three, but seldom more.

There is no information on the question of the duration of infectivity or the utility of preventive measures.

There is one recorded case of premature delivery and death of the mother.

I am indebted to Dr. Wynne for the information on which this summary is based.

X.

Summary of Results of an Inquiry at Blackburn made under the supervision of J. R. Hutchinson, M.D. The Investigation covered the Period of the three Epidemic Waves.

Summer.—June, July, August, 1918.
Autumn —September, October, November, December, 1918.
Winter.—January, February, March, 1919.

1. *Age and Sex Incidence. Whole Period of the Three Epidemics.*

—	Sex.	Under 1 Year.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45–55.	55–65.	65–75.	75 and over.	Total.
Population investigated.	M.	18	7	39	65	64	46	25	67	68	60	48	26	1	533
	F.	8	10	50	73	54	58	52	149	109	76	68	31	13	751
Persons affected -	M.	3	2	10	13	9	7	3	18	19	6	2	9	–	101
	F.	–	2	9	12	16	18	12	44	25	17	16	3	–	174
Per cent. of both sexes affected.	–	11·5	23·5	21·3	18	21·2	24	19·5	28·7	24·9	17	15·5	11·4	–	–
Escaped - - -	M.	15	5	29	52	55	39	22	49	49	54	46	16	1	432
	F.	8	8	41	61	38	40	40	105	84	59	52	28	13	577
Deaths - - -	M.	1	–	1	–	–	1	–	–	–	–	–	–	–	3
	F.	–	1	1	–	–	–	–	–	–	1	2	–	–	5

	Per Cent.
Incidence on population investigated - - - -	21·41
Mortality of population investigated - - - - -	0·62
Mortality among persons affected - - - - -	2·91

2. *Age and Sex Incidence. Summer Epidemic.*

Affected - - -	M.	–	–	–	1	4	6	2	9	8	4	1	4	–	39
	F.	–	–	2	3	7	9	6	22	12	6	4	1	–	72
Per cent. affected -	-	–	–	2·2	2·9	9·3	10·4	10·4	14·3	11·3	7·4	4·3	9	–	–
Escaped - - -	M.	18	7	39	64	60	40	23	58	60	56	47	21	1	494
	F.	8	10	48	70	47	49	46	127	97	70	64	30	13	679
Deaths - -	M	–	–	–	–	–	–	–	–	–	–	–	–	–	–
	F.	–	–	–	–	–	–	–	–	–	1	–	–	–	1

	Per Cent.
Incidence on population investigated - - - -	8·64
Mortality of persons investigated - - - - -	0·07
Mortality of persons affected - - - - - -	0·90

3. *Age and Sex Incidence. Autumn Epidemic.*

—	Sex.	Under 1 Year.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45 55.	55–65.	65–75.	75 and over.	Total.
Persons affected -	M.	–	–	5	10	4	2	1	4	4	2	1	2	–	35
	F.	–	–	5	6	5	3	3	12	7	6	5	2	–	54
Per cent. affected -	–	–	–	6	11·2	11·6	7·6	4·8	5·2	7·4	6·2	5·9	5·2	7·1	–	–
Escaped - - -	M.	18	7	34	55	60	44	24	63	64	58	47	23	1	498
	F.	8	9	45	67	49	55	49	137	102	69	63	29	13	696
Deaths - - -	M.	–	–	1	–	–	1	–	–	–	–	–	–	–	2
	F.	–	1	–	–	–	–	–	–	–	–	–	–	–	1

	Per Cent.
Incidence on population investigated - - - - -	7·01
Mortality of population investigated - - - - -	0·23
Mortality among those affected - - - - - -	3·33

4. *Age and Sex Incidence. Winter Epidemic.*

Persons affected -	M.	3	2	5	4	2	1	–	6	7	4	1	4	–	39
	F.	–	1	2	4	5	10	4	12	7	7	8	1	–	61
Per cent. affected -	–	11	19	8	5·8	6	10·7	5·2	8·3	8	8	7·8	9	–	–
Escaped - - -	M.	15	6	33	61	62	44	25	61	61	56	47	21	1	492
	F.	8	8	48	69	49	48	48	137	102	68	60	30	13	688
Deaths - - -	M.	1	–	–	–	–	–	–	–	–	–	–	–	–	1
	F.	–	–	1	–	–	–	–	–	–	–	2	–	–	3

	Per Cent.
Incidence on population investigated - - - - -	7·81
Mortality of population investigated - - - - -	0·31
Mortality among those affected - - - - - -	4·00

Summary.

Affected summer only.	M.	–	–	–	1	4	4	2	9	8	1	–	3	–	32
	F.	–	–	2	3	7	6	5	21	11	4	4	1	–	64
Autumn only - -	M.	–	–	5	8	2	1	1	3	4	–	–	2	–	26
	F.	–	1	5	5	4	–	3	10	6	6	4	1	–	45
Winter only - -	M.	3	2	5	2	1	1	–	5	7	1	1	3	–	31
	F.	–	1	2	3	4	9	4	11	6	5	7	–	–	52
Multiple attacks -	M.	–	–	–	2	2	1	–	1	–	4	1	1	–	12
	F.	–	–	–	1	1	3	–	2	2	2	1	1	–	13
Total affected - -	M.	3	2	10	13	9	7	3	18	19	6	2	9	–	101
	F.	–	2	9	12	16	18	12	44	25	17	16	3	–	174

5. *Table showing number of Second Attacks among Persons investigated.*

Autumn Epidemic.

—	Sex.	Under 1 year.	1–2.	2–5.	5–10.	10–15.	15–20.	20–25.	25–35.	35–45.	45–55.	55–65.	65–75.	75 and over.	Total.
Total persons affected	M.	–	–	5	10	4	2	1	4	4	2	1	2	–	35
	F.	–	1	5	6	5	3	3	12	7	6	5	2	–	55
Affected summer and autumn.	M.	–	–	–	–	1	1	–	–	–	1	1	–	–	4
	F.	–	–	–	–	–	2	–	1	1	–	–	–	–	4
Affected autumn escaped summer.	M.	–	–	5	8	2	1	1	3	4	–	–	2	–	26
	F.	–	1	5	5	4	–	3	10	6	6	4	1	–	45

6. *Table showing number of Second and Third Attacks among Persons investigated.*

Winter Epidemic.

Total persons affected	M.	3	2	5	4	2	1	–	6	7	4	1	4	–	39
	F.	–	1	2	4	5	10	4	12	7	7	8	1	–	61
Affected autumn and winter escaped summer.	M.	–	–	–	2	1	–	–	1	–	1	–	–	–	5
	F.	–	–	–	1	1	–	–	1	–	–	1	1	–	5
Affected summer and winter escaped autumn.	M.	–	–	–	–	–	–	–	–	–	2	–	1	–	3
	F.	–	–	–	–	–	–	–	–	1	2	–	–	–	3
Affected summer, autumn and winter.	M.	–	–	–	–	–	–	–	–	–	–	–	–	–	–
	F.	–	–	–	–	–	1	–	–	–	–	–	–	–	1
Affected winter only	M.	3	2	5	2	1	1	–	5	7	1	1	3	–	31
	F.	–	1	2	3	4	9	4	11	6	5	7	–	–	52

BLACKBURN.—TABLE SHOWING FREQUENCY OF SECOND ATTACKS.

Total population investigated.

1,284

Summer - - 1,173 — *111* (**1**)

Autumn - 1091 — *81* (**3**) (6·8 per cent.) — *9* (8·2 per cent.)

Winter - 1,008 — *83* (**4**) (7·6 per cent.) — *10* (1·3 per cent.) — *6* (5·4 per cent.) — *1* (11·1 per cent).

Note.—Attacks are in italics.
Deaths in heavy type.

Note.—Four persons were affected twice during one epidemic, viz.:—

One, November and December 1918.

Three, February 1919, and two weeks later. These three comprised the whole of one family, and one (F. 2) died.

Of the eight fatal cases only one (F. 2) had previously suffered from influenza (two weeks earlier).

7. *House Incidence.*

Number of Houses investigated.	Number escaped all Three Waves.	Number affected.	House Incidence	Houses invaded in						
				Summer.	Autumn.	Winter.	Summer and Autumn.	Summer and Winter.	Autumn and Winter.	Summer, Autumn, and Winter.
300	166	134	Per Cent. 44·6	56	50	63	10	10	11	2

8. *Incidence on Occupiers of Invaded Houses.*

Condition of House as regards Number of Inmates.	Number of Houses investigated.	Houses			Population.		Persons affected.				Incidence on Inmates.
		Affected.	Escaped.	House Incidence.	Of Houses invaded.	Of Houses not invaded.	Single Attacks.	Double Attacks.	Triple Attacks.	Total.	
A.—Houses with one or more persons per room - - - -	176	87	89	Per Cent. 49	486	448	163	20	1	184	19·70
B.—Houses with less than one person per room - - - -	124	47	77	38	155	195	83	8	—	91	26·00

9. *Multiple Cases.*

Cases per House.	Type of House.	Summer.			Autumn.			Winter.			Total.		
		Type A.	Type B.	Total.	A.	B.	Total.	A.	B.	Total.	A.	B.	Total.
1	A.—One or more persons per room.	20	14	34	22	12	34	30	10	40	72	36	108
2		11	6	17	4	2	6	11	5	16	26	13	39
3		1	3	4	4	3	7	3	2	5	8	8	16
4	B.—Less than one person per room.	4	1	5	1	2	3	1	1	2	6	4	10
5		1	—	1	1	—	1	1	—	1	3	—	3
6		1	—	1	1	—	1	—	—	—	2	—	2

Of the 134 invaded houses some were invaded in more than one outbreak; the number of invasions of houses was 178:—

In 108, or 60 per cent. of instances, there was 1 case only.
In 39, or 22 „ „ „ were 2 cases.
In 16, or 9 „ „ „ „ 3 „
In 10, or 5·5 „ „ „ „ 4 „
In 5, or 2·75 „ „ „ „ more than 4 cases.

Tables 8 and 9 together show that the incidence on the inmates of invaded houses of Class A. was 13·1 per cent., as compared with 12·7 per cent. for Class B.

Of 193 persons sleeping in same room as patient, 76 were attacked - - - - - - - = 39·3 per cent.
Of 145 persons nursing influenza patients, 43 were attacked - - - - - - - = 29·6 per cent.
Of 73 persons nursing and sleeping with patient, 24 were attacked. - - - - - - - = 32·8 per cent.
In 161 out of 301 attacks (= 53·4 per cent.) a doctor was in attendance.

Occupation of Females over School Age.	Affected.	Escaped.	Incidence.
			Per cent.
Household - - - - - -	59	226	20·7
Factory - - - - - -	63	170	27·0
Other work - - - - -	14	31	31·1

Complications.—47 out of 301 attacks (= 15·6 per cent.) were followed by complications:—

Summer, 7. $\frac{7}{111}$ = 6·3 per cent.

Bronchitis, and abscess, thigh - -	1	
Bronchitis - - - - -	2	Death, F., 53, bronchitis.
Pneumonia - - - - -	1	
Cardiac debility - - - -	1	
Rheumatism - - - - -	1	
Enteritis - - - - - -	1	

Autumn, 16. $\frac{16}{90}$ = 17·7 per cent.

Pneumonia - - - - -	8	Deaths.
Bronchitis - - - - -	2	M., 4, influenza.
Quinsy - - - - - -	2	M., 15, pneumonia.
Rheumatism - - - - -	2	F., 1 5/12, pneumonia.
Cystitis and lumbago - - -	1	F., 55, melancholia (inquest).
Delirium - - - - - -	1	(Lived until March).

Winter, 24. $\frac{24}{100}$ = 24 per cent.

Bronchitis - - - - -	9	Deaths.
Broncho-pneumonia - - - -	1	M., 8/12, bronchitis.
Pneumonia and convulsions - -	1	F., 2, pneumonia.
Pneumonia - - - - -	8	F., 59, influenza.
Epistaxis and hæmoptysis - -	1	
Hæmoptysis - - - - -	1	
Convulsions - - - - -	1	(+ 1, pneumonia and convulsion, noted above).
Slow convalescence - - - -	1	
Pleurisy - - - - - -	1	

Complications among Persons with Multiple Attacks.

Summer and Autumn.—Nil.

Autumn and Winter—

Pneumonia, each attack	2
Bronchitis, each attack	1
Bronchitis and infl., each attack	1
Epistaxis, winter	1
Vulvitis, autumn	1
Broncho-pneumonia, winter	
Pneumonia, winter	1
Bronchitis, winter	1

Thus five persons out of 10 who were attacked both in autumn and winter had some complication in the first attack. These five also had complications in the second attack. Three persons had complications in the second attack only.

Summer and winter—

Bronchitis, summer	1
Pneumonia, winter	
Pneumonia, summer	1 (has chronic bronchitis).
Epistaxis, winter	1
Thacheitis and Laryngitis, winter	1

Thus two persons out of six had in the previous attack suffered from some complication. One suffered from complication in each attack. Two suffered from complication in the second attack only.

Incidence on Persons who were subject to Chronic Catarrh or other Inflammatory Conditions.

Chronic bronchitis	4	
Chronic laryngitis	1	
Chronic cough	1	
Delicate: "weak chest"	4	
"Frequent colds"	2	
Asthma	1	
Tuberculosis (pulmonary)	1	
	14	$= \frac{14}{275} = 5$ per cent.
Diarrhœa	1	
Dysentry	1	
Gastric ulcer	1	
Hemiplegia	1	
Goitre	1	
Pregnancy	4	(Still birth, one).

Thus out of 275 persons affected with influenza, 14 persons were stated to have some form of respiratory catarrh.

Other variations from normal health were noted amongst nine persons.

Of four recorded cases of pregnancy among 174 females affected, one still birth was noted.

Note, re Tables 7 and 8.—At six houses investigated there were resident persons who had suffered from influenza whilst away from home (*e.g.*, in Army). No other member of the family had suffered, the *houses* consequently were *not invaded*, and are therefore not included in the "number of houses invaded" (*134*).

In Table 7, these six houses have been included in the "number of houses escaped," but the six persons affected are included in the number of "single attacks" (163), occurring in house of Type A, with one or more persons per room. The population of these six houses is 32.

XI.

Extract from a Report on the incidence of Influenza on certain classes of Workers in the Southern Section of the Swansea Valley.

By

F. S. Carson, M.B.

The inquiry dealt chiefly with workers in the large copper and spelter works situated on either bank of the River Tawe. The incidence of influenza on the 15–65 year group of the population of the village of Pentrechwyth is given for purposes of comparison.

The whole valley where the works are situated is practically grassless, and a large area of country on the leeward side of the works on the eastern bank of the river is devoid of vegetation. The village of Pentrechwyth is situated on the eastern bank and in the barren area. Some of the spelter workers live in this village.

Accurate classification is difficult, but roughly the workers may be divided into:—

(*a*) Those engaged part or full time in the buildings where the fumes are actually generated.

(*b*) Those not working in fumes, *i.e.*, yard men, &c.

Sulphur dioxide is apparently the chief gaseous impurity found in the atmosphere. The inquiry relates only to the autumn and winter epidemics, as Swansea was practically unaffected by the summer wave of 1918.

Number of Workers of each Class.

Class A.—Workers in fumes full time	634
„ B.— „ „ part time	323
„ C.— „ not working in fumes	883
Total	1,840

Number of Cases according to Class.

Epidemic.	Class A.	Class B.	Classes B.& C.	Class C.
	Per cent.	Per cent.	Per cent.	Per cent.
Autumn (1918)	124 = 20·0	43 = 13·9	167 = 17·9	89 = 10·2
Feb.–March (1919)	75 = 12·0	39 = 12·6	114 = 12·2	65 = 7·4
Total	199 = 32·0	82 = 26·5	281 = 30·1	153 = 17·6

dence of Influenza on other classes of the Population (Village of Pentrechwyth).

r of houses	207
ation (excluding spelter and copper workers):—	
otal	827
Age group 15–65 years	466

Epidemic.	Number of cases.	
	Total of all Ages.	At Ages 15–65
	Per cent.	Per cent
Autumn (1918)	84 = 10·3	64 = 13·4
February–March (1919)	70 = 9·0	53 = 11·3
Total	154 = 19·3	117 = 24·0

Summary of Percentage Case Incidence.

Workers not in fumes	17·6 per cent
„ part time in fumes	26·5 „
„ in fumes	32·0 „
Other classes of population—	
Total	19·3 „
Age group 15–65 years	24·0 „

XII.

Note on the Periodicity of Influenza.

By

John Brownlee, M.D., D.Sc.,

Director of Statistics, Medical Research Committee.

Introduction.

Some time ago I made a short note in the *Lancet** on the periodicity of influenza epidemics, with the intention of returning again to the subject. Owing to the difficulty of obtaining series of statistics with regard to other cities, I have not been able to extend the investigation, but one or two points have emerged. The main part of the investigation stands and is reproduced. Reference, however, is made to more recent papers.

Periodicity and Characteristics of Recorded Epidemics.

In the first place the series of epidemics of influenza between the years 1889 and 1917, beginning with the last great invasion of the disease, in those places for which statistics are accessible, present a singular phenomenon. Epidemics seem to be of extreme rarity between the end of June and the beginning of December, so rare in fact as to be almost non-existent. This is the season of the year in which bronchitis and pneumonia are least frequent. Influenza, then, does not, if epidemic, assume a form which causes death to any extent until either a bronchitic or pneumonic "constitution" has been established. The great epidemic which occurred in October 1919 falls completely out of line with what happened between 1889 and 1917. Some small epidemics, it is true, occurred at this season, but none comparable in size to the last.

The statistics of the epidemics which have been specially investigated are those of London between 1889 and 1896. For this city the weekly number of deaths since 1889 has been published. Also what is of the greatest advantage, the weekly numbers of deaths from bronchitis and pneumonia are available since the year 1870. Considering specially the years 1889 to 1896 as the most typical years, it is seen that the epidemics of influenza have had their maxima from the beginning of January to the end of May. Applying the ordinary method of the periodogram, it is found that the interval between the epidemics is 33 weeks, there being a missed epidemic when an epidemic is due in the autumn. The periodicity is rigid within these years, though later years show some considerable aberration. The method of the periodogram, however, allows us to extend the knowledge acquired with regard to influenza to the associated diseases, bronchitis and pneumonia. There is no question at all that the period between the influenza epidemics between 1889 and 1896 is 33 weeks. Whether this periodicity is accidental or dependent on the special organism of the disease does not affect the next step in the investigation. If it is found that there is no periodicity of 33 weeks with regard to bronchitis or pneumonia in the absence of influenza, and if this periodicity appears with regard to these diseases after the return of influenza it must be taken as associated definitely with that disease. It is found on examination that between 1876 and 1890 there is no such periodicity with regard to bronchitis and pneumonia, but that from 1889 to 1896 it is very marked. The graphs for 33 weeks may then be compared for the three diseases. It is found on inspection that the deaths from pneumonia precede the deaths from influenza by a little over a week, while the deaths ascribed to bronchitis have their epidemic rise a full fortnight before the rise of influenza. The number of these deaths for bronchitis and pneumonia ascertained by this

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* *Lancet*, 8th November 1919.

method of grouping is fully twice the number of deaths ascribed to influenza. It thus seems certain that in these years influenza appeared, on its epidemic onset, first with bronchitic symptoms, slightly later with pneumonic symptoms, and, lastly with those symptoms more definitely associated with influenza proper. When the several sets of deaths are added together in 33-week periods a very typical epidemic makes its appearance. I have not, so far, been able to obtain weekly statistics of any other great city, but I have examined the monthly statistics of Glasgow, Aberdeen, Massachusetts, &c., and have found nothing which differs essentially from the phenomena found in London.

Deductions drawn from the Data.

Before passing from this subject it may be said that what has been ascertained with regard to the sequence of these diseases, bronchitis, pneumonia, and influenza from a consideration of a 33-weeks period, also holds when the annual variations are compared. Between the years 1876 and 1889 the annual curve which gives the variations of bronchitis possesses two maxima, one at the end of January and the second in the middle of March. From this point the decline in the number of deaths from bronchitis is very rapid. The reappearance of the disease dates from the beginning of October. When the eight years 1889 to 1896, however, are examined, it is found that the maximum number of deaths from bronchitis occur in the second week in January and in the last week of February. Both these maxima are a fortnight before the maxima of the epidemics of influenza as before found. This suggests that the advent of influenza has brought a change in the seasonal prevalence of bronchitis, and also supports the view that the earlier portion of the influenza epidemic is associated with bronchitic symptoms. The same phenomenon holds for pneumonia as a like alteration has occurred in the seasonal curve of the death-rates of this disease before and after 1889. The maximum numbers of deaths from pneumonia are thrown earlier into the year and precede the maximum number of deaths from influenza by about a week.

Since the preceding remarks were published, two papers have appeared on the subject, one by Mr. Stallybrass* of the Department of Health in Liverpool, who finds the same phenomena in Liverpool which have already been found to exist in London and elsewhere, and who also gives a number of further details. Both my paper and the preceding have been criticised by Mr. Spear.† He states dogmatically "that the method can only yield correct results when the data to which it is "applied can be described as recurring 'waves' of approximately uniform "'amplitude.'" There is, however, no necessity that the waves of the epidemic be of uniform amplitude as Mr. Spear thinks. He further says: "How then, is the result found by Dr. Brownlee by the periodogram method "to be accounted for? The explanation lies in the fact that the 'ampli-"tudes' of the successive 'waves' of influenza mortality—the number of "deaths during the week of climax—in certain of the epidemics in this "period are so great as to overshadow and practically to eliminate the smaller "outbreaks when the data are subjected to analysis by the periodogram "method."

The fallacies of the method are of course quite familiar to me. Mr. Spear's statement, however, last quoted is quite wrong, there is no evidence that two or more large epidemics have exercised a dominating influence. His error is illustrated exceedingly well if the course of the epidemic of influenza in Boston from 1889 to 1900 are examined.

The facts are illustrated in the accompanying diagram. This diagram has been constructed by the following method: Each division in the diagram refers to the death-rate for two years in months, the first month on the left-hand side being the month of September, and the last month August of the

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* *Lancet*, 14th February 1920. † *Lancet*, 13th March 1920.

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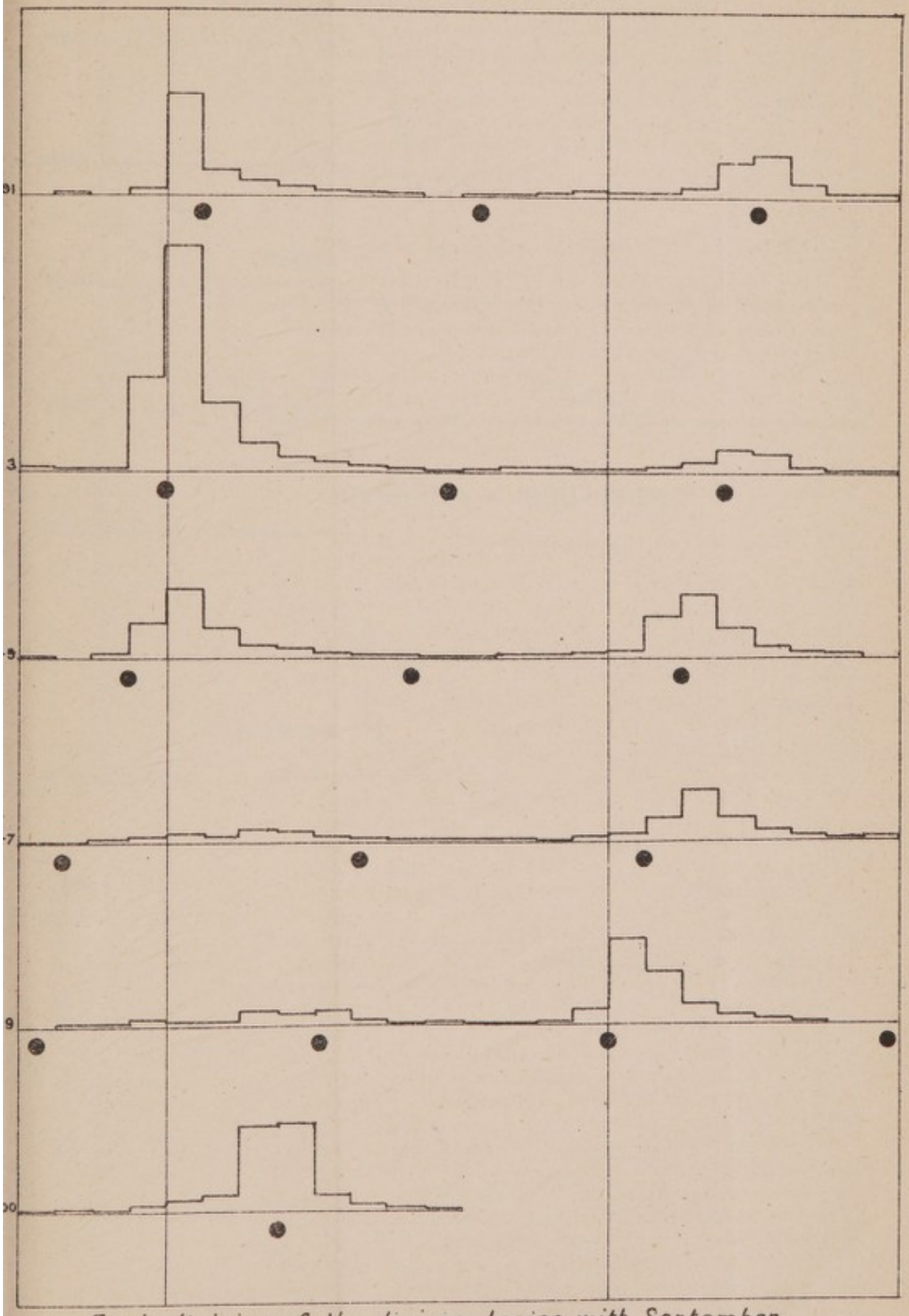
DIAGRAM SHOWING THE COURSE OF THE EPIDEMICS OF INFLUENZA IN BOSTON, U.S.A. 1889–1900.



Each division of the diagram begins with September. The vertical lines give the beginnings of the years, the black circles the times at which epidemics might be theoretically expected to occur.

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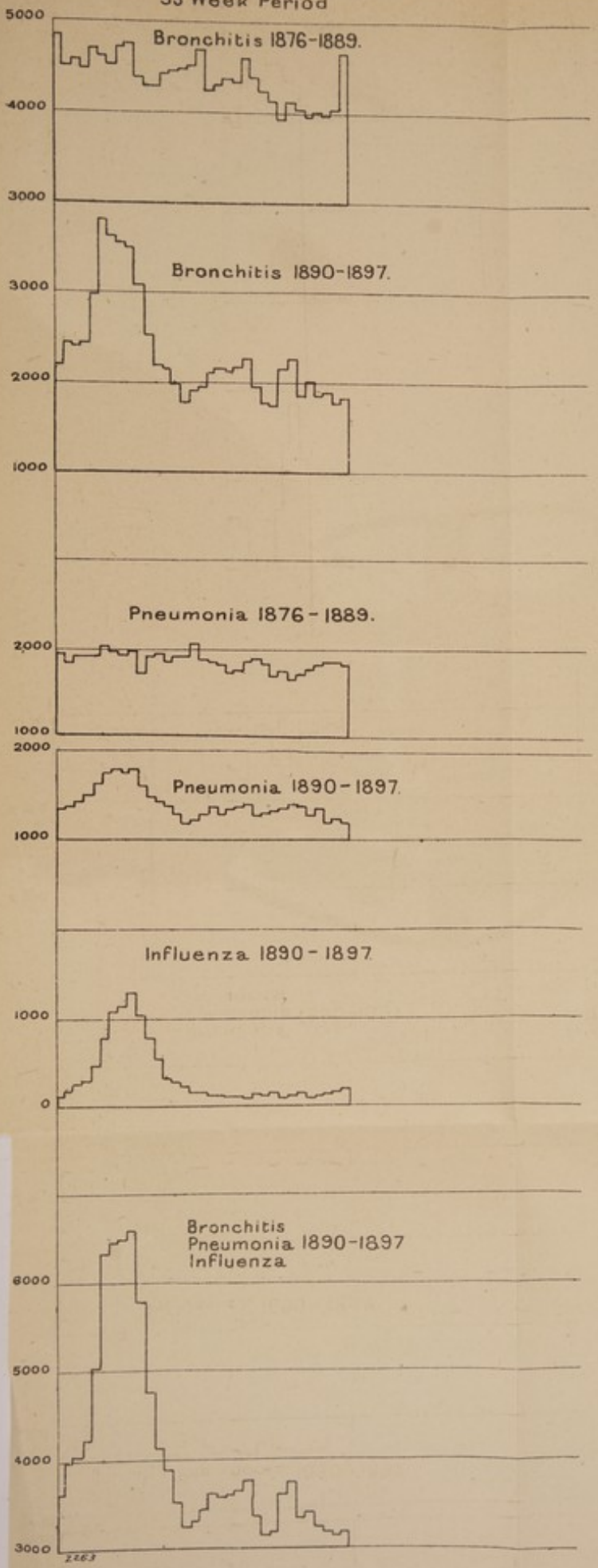
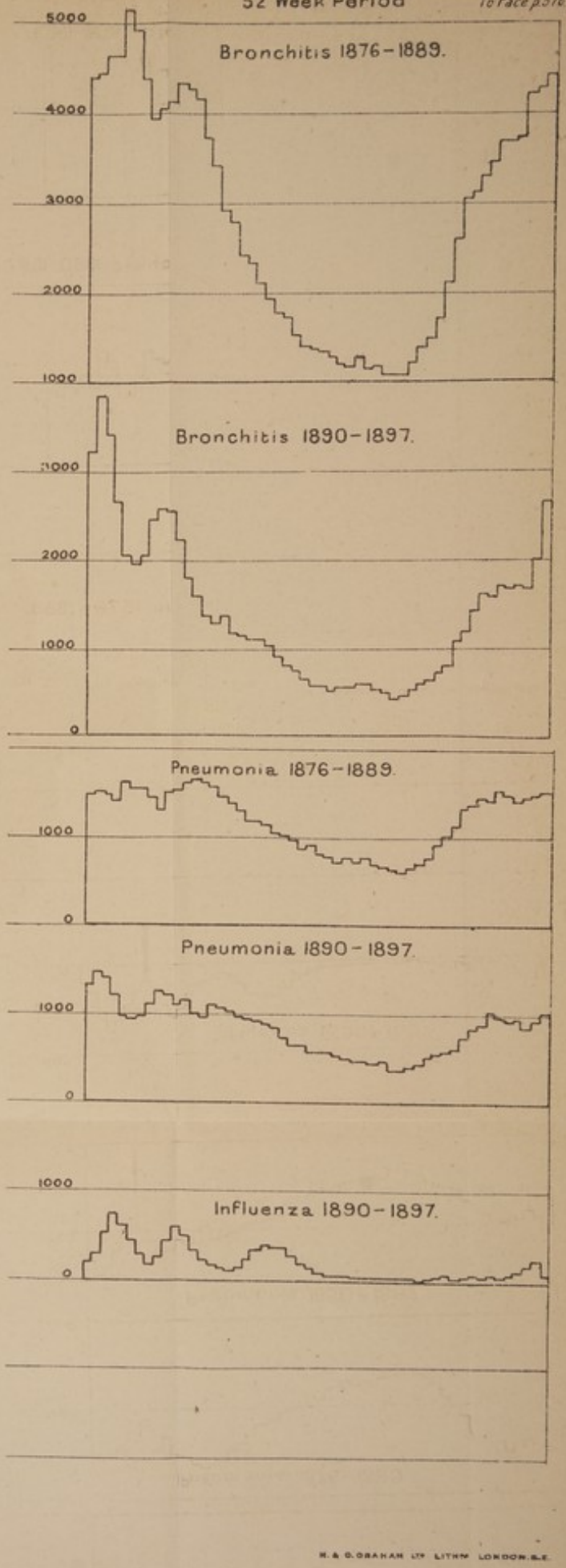
WEEKLY DEATHS IN LONDON FROM BRONCHITIS, PNUEMONIA & INFLUENZA. 1876.-1897.

33 Week Period



52 Week Period

To face p. 576



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next year. The vertical lines mark the beginning of the year and the black dots indicate the points at which the maxima of the epidemics would occur if the period was rigidly 33 weeks. It will be seen that the centre of the main epidemics varies very little from its theoretical placing. In only two cases is it more than a month out of place. The diagram shows further, if the difficulty of an epidemic occurring between the end of June and the middle of December be admitted, that a 33-weeks period explains all the facts. Thus in the years 1895–96 and 1897–98 the theoretical maxima fall in that part of the year and almost no deaths from influenza occurred, but when the following maxima fell in the appropriate season the epidemics re-appeared in their proper place. After 1899 the rhythm broke. The facts for London are identical except that the rhythm broke eighteen months earlier. This diagram is a sufficient refutation of Mr. Spear's statement, that the 33-weeks period arises from conjunction of two or three large epidemics.

Mr. Spear further mentions an amplitude with a 50-weeks period. This amplitude I was fully acquainted with when I wrote my note. If a 33-weeks period exists with the further requirements that every third epidemic is missed, the form of the curve arising if the figure be added up in rows of 50 weeks is identical with that found in this case. This amplitude being thus fully explained, it did not seem necessary to make special reference to it.

When the note was written the following remarks were added.

From 13th July 1918 to 1st March 1919 the maximum points of two of the last epidemics is 33 weeks; from 1st March to 21st October is also 33 weeks. An epidemic is therefore due, but it falls at the unsuitable season of the year and should, therefore, be small. With regard to the aberrant October epidemic, this might be expected to have been followed by an epidemic in June, a season of the year at which an epidemic is still possible but very unlikely. If the October epidemic, therefore, has a 33-weeks sequence, the next epidemic would occur in January or February of the New Year. Of course, it is to be remembered that the intervals between epidemics are never exact; only their average approaches exactitude. A month either way is of no moment.

How far, then, has the prophecy been fulfilled?

Dr. Wladyslaw Szenajch, commenting on the note in the Polish "*Journal of Epidemiology*," says: "This announcement has, on the whole, proved true "in England and in America, but here (in Poland) the epidemic has reached "its highest in January 1920, the interval between the end of one epidemic "and the commencement of the other amounted to 30 weeks, or up to the "greatest prevalence to 33 weeks." So that even in Poland there was no great divergence.

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