LOCAL GOVERNMENT BOARD.

FURTHER REPORT

AND

PAPERS

ON

EPIDEMIC INFLUENZA, 1889-92.

WITH AN INTRODUCTION BY THE MEDICAL OFFICER OF THE LOCAL GOVERNMENT BOARD.

Presented to both Houses of Parliament by Command of Her Majesty.

LONDON:
PRINTED FOR HER MAJESTY'S STATIONERY OFFICE,
BY EYRE AND SPOTTISWOODE,
PRINTERS TO THE QUEEN'S MOST EXCELLENT MAJESTY.

And to be purchased, either directly or through any Bookseller, from
EYRE AND SPOTTISWOODE, East Harding Street, Fleet Street, E.C.; and
32, Abingdon Street, Westminster, S.W.; or
JOHN MENZIES & Co., 12, Hanover Street, Edinburgh, and
90, West Nile Street, Glasgow; or
HODGES, FIGGIS, & Co., Limited, 104, Grafton Street, Dublin.

1893.

[C.—7051.] *Price* 2*s.* 8*d.*

CONTENTS.

PART VII.—Prophylaxis.

REPORT ON INFLUENZA IN ITS CLINICAL AND PATHOLOGICAL ASPECTS, By Dr. KLEIN.

INTRODUCTION BY THE MEDICAL OFFICER.

TO THE RIGHT HONOURABLE THE PRESIDENT OF THE LOCAL GOVERNMENT BOARD.

SIR,

ON the 30th May 1891 my predecessor, Sir George Buchanan, presented to the Board an exhaustive report by Dr. Parsons upon the Influenza epidemic of 1889–90.* Owing to the recurrence of the disease in epidemic form in the spring of 1891 and again in the winter of 1891–92, opportunity was afforded for further observation of its behaviour, and early in 1892 it was decided that more detailed study of the natural history of the disease in its clinical, pathological, and bacteriological aspects should be undertaken. As the result, I now have the honour to submit to you two further reports on Influenza; one by Dr. Parsons, the other by Dr. Klein.

Since the issue of Dr. Parsons's first report, the mortality returns for England and Wales during 1890 and 1891, and for London during 1892, have become available; and Dr. Parsons has, in consequence, been enabled to submit a considerable body of statistical information bearing on such matters as the incidence of the disease, in its fatal form, on age, sex, and locality, and as to its relation to season, to the general sanitary history of the places affected, and to mortality from other diseases. But whilst much interesting matter is thus forthcoming, it must be remembered that caution is necessary in drawing definite conclusions from the information available. As yet we are not furnished with the mortality returns of 1892 for England and Wales as a whole, and the Census report for 1891 may, when it comes to be issued, modify provisional inferences as to age and sex, in their relation to the incidence of Influenza. Moreover, comparison of Influenza in one and another locality in the several epidemics has to be very guardedly instituted, for the reason that the term Influenza as a cause of death has varied not only in different localities during the

* Report on the Influenza Epidemic of 1889–90 by Dr. Parsons, with an Introduction by the Medical Officer of the Local Government Board (C.—6387), 1891.

same epidemic, but also in the same locality in different epidemics. But after making allowance for such sources of fallacy it is possible to draw certain general inferences from the information which Dr. Parsons has set forth.

Three epidemic periods come under consideration. First, that of the winter 1889–90, secondly, that of April—June 1891, and thirdly, that of the winter 1891–92. Study of Dr. Parsons's report shows distinctly that these epidemics followed different courses, and that their incidence on locality varied considerably. So far as the metropolis is concerned, the "Influenza" death-rates in the three calendar years 1890, 1891, and 1892 were 146, 544, and 531 per million living respectively. But the onset of the last two epidemics was much less sudden than was the case in the winter of 1889-90; and whilst it is probable that there were fewer attacks in the more recent prevalences than in the first one, yet the later epidemics were both more protracted and more fatal. Whilst, however, this was the case in London, almost the opposite took place elsewhere, notably in Sheffield, where the first outbreak was mild and protracted, whilst the epidemic of 1891 was sudden, severe, and fatal. No conditions of site, soil, climate, sanitary circumstances, occupation, or other, have yet been elicited which afford any satisfactory explanation of these differing incidences. At the same time there is some evidence pointing to the influence of a severe epidemic as serving to grant a certain immunity against another in the same locality.

With regard, further, to the mortality occasioned by the disease in different communities, it is noteworthy that the rate of death due to Influenza was substantially greater in rural and sparsely populated areas than in large towns. Thus, whereas in the metropolis and in 90 towns having populations varying from 20,000 to 80,000 each, the Influenza death-rate varied from 0·52 to 0·58 per 1,000 living, this rate on nearly 3,000,000 people living in 192 rural sanitary districts reached 0·73 per 1,000. This, at first sight, appears opposed to what might have been expected as regards a disease which has been held to be communicable from person to person; but it receives an explanation when it is remembered that the death-rate from Influenza increases with advancing age, and that whilst so many of our rural areas have been more or less denuded of young people and adolescents, the old people have remained at home in their villages.

The further experience which has been obtained goes strongly to confirm the view that Influenza is essentially propagated from person to person. Indeed the evidence which is now forthcoming tending to show that the disease has followed the lines of human

intercourse, so far outweighs any that has been put forward in favour of its being due to "atmospheric causes," as to suggest that where these have been accepted as accounting for outbreaks, all the circumstances of the local prevalences in question have not been forthcoming.

During both 1890 and 1891 the death-rate from Influenza was greater amongst males than amongst females, and this especially at the middle period of life; and having regard to the serious influence of the disease on the nervous system, it is highly probable that this result may be mainly due to the fatigue, mental as well as physical, and to the exposure to which men are more especially subjected.

The heavier mortality from Influenza experienced in the later epidemics as compared with that of 1889–90, has appeared to be in part due to the fact that whereas in the former epidemic disturbances of the circulatory and cerebro-spinal systems were prominent manifestations; the stress of the malady in the more recent prevalences fell especially upon the lungs. This has led Dr. Parsons to raise in his present report the question as to whether inflammatory affections of the lung, and especially pneumonia, are an integral part of the disease or merely superadded complications.

Clinical study of disease has not, as yet, succeeded in clearing up this point, but the bacteriological investigations which have been carried on have a distinct bearing on the question.

In January 1892, Dr. Klein, referring to the observations of Pfeiffer and Kitasato on the constant and copious occurrence, in the bronchial secretion of cases of Influenza, of a species of minute bacillus, as also to the observations of Canon on the occurrence of bacilli in the blood of Influenza cases, gave an account in the "British Medical Journal" of certain experiments which he had carried out as soon as the epidemic reached this country. Examination of the blood was made in six cases; in one instance only were a few minute bacilli, resembling those described by Pfeiffer, discovered, and in all six cases tubes inoculated with the blood remained sterile. But it was otherwise with the bronchial sputum of three cases which had passed a few days previously through the acute febrile stage of the disease. The sputa in each instance contained bacilli as described by Pfeiffer and Kitasato; in a recent case they were so numerous as to amount almost to a pure cultivation; and both cultures and subcultures of the characteristic organism were successfully made.

Soon after the publication of these data the Board decided that, in view of the recurrence of epidemic Influenza in this country, it

was desirable that special inquiry should be made into the clinical, pathological, and bacteriological aspects of the disease, and a grant of money having been placed at the disposal of the Board for this purpose, Dr. Klein was instructed to undertake the work, employing such assistance as he deemed necessary. The result is the subjoined special report on Influenza in its clinical and pathological aspects which has been prepared by Dr. Klein with the co-operation of Dr. Andrewes, Dr. Cautley, and others.

Having regard to the clinical features of Influenza, it seemed probable that the blood and also the tissues involved in the respiratory tract would give most promise in connexion with bacteriological research. But on examination of blood taken from 43 typical cases of the disease no bacterial forms could be discovered in 37 of them. In the other six, bacilli, always of the same form, were found, but their amount was variable, and in one case only did they admit of cultivation, and Dr. Klein concludes that although bacteria which he accepts as the bacilli of Influenza do at times appear in the blood, yet he is of opinion that they do not occur in that fluid with any constancy, and that they rapidly lose their vitality after gaining access to the circulation. In this respect he looks upon his results as confirmatory of those of previous observers, amongst whom he specially quotes Canon, to the effect that the blood cannot in any sense be regarded as the primary nidus of the microbe of Influenza.

Examination of the bronchial sputum in Influenza led to very different results. Minute non-mobile bacilli having a characteristic appearance occur in greater or less amount in the sputa of all cases of Influenza; during the acute stage of the disease they are generally present in abundance, occasionally almost in pure culture; and they tend to disappear as the disease passes off. These bacilli, which are the same as those described by Pfeiffer and Kitasato, do not occur in the bronchial secretion of any other disease; they must be regarded as pathognomonic of Influenza; and their life-history conforms, as Dr. Klein puts it, with what we believe to be the facts about the contagium of the disease.

After describing in considerable detail the clinical features of Influenza and giving an account of certain experiments connected with the cultivation of the Influenza bacillus, Dr. Klein proceeds to discuss the question of experiments made on animals with these bacilli in sterile salt solutions of sputum, and in cultivations. With rabbits the result of these experiments was altogether negatory. Inoculation into monkeys was hardly more successful. Out of 12 on which experiment was made, definite organic disease

only occurred in one. The disease was pneumonia, and in the inflamed lung quantities of the Influenza bacilli were discovered. But bacilli of another kind, which are described in detail in the report, were also found along with the Influenza bacilli, and it is impossible to say to which of the two forms of bacillus the pneumonia was related. No useful inferences can, under these circumstances, be drawn from the inoculations made in the two groups of lower animals referred to.

The question of expense in itself prevented any extension of these experiments to horses, amongst which animals disease held by some to be Influenza has at times prevailed. But careful watch was kept during the progress of the inquiry for indications of any disease that might be Influenza amongst the lower, and especially amongst domestic, animals. None could be met with, and Dr. Klein evidently inclines to the opinion that the disease amongst horses to which, for want of a better name, the name of Influenza is commonly given, cannot be identified with Influenza in the human subject. In this respect his views are in accordance with the evidence obtained by Dr. Parsons as regards the absence of any special incidence of Influenza amongst persons who live in close relation with horses and stables.

One prominent lesson seems indicated as the result of Dr. Klein's study of Influenza from the bacteriological point of view. It is this: the sputa of the sick are, especially in the acute stages of the disease, invariably charged with the micro-organism which is pathognomonic of Influenza, and it may be hoped therefore that when these sputa come to be recognised as infectious and are dealt with, as is held necessary in the case of discharges from the throat, mouth, and nostrils of scarlatina and diphtheria patients, the spread of Influenza from person to person may be to a corresponding extent controlled.

I have the honour to be,

Sir,

Your obedient Servant,

R. THORNE THORNE.

May 1893.

FURTHER REPORT AND PAPERS

ON

EPIDEMIC INFLUENZA, 1889-92.

A further Report on the Influenza Epidemics of 1889–90, 1891, and 1891–92 ; by H. Franklin Parsons, M.D.

On the Influenz Epidemics of 1889–90, 1891, and 1891–92 ; by Dr. Parsons.

In the winter of 1889–90 the British Isles, in common with nearly all other known countries of the world, were revisited, after a lapse of many years, by an epidemic of the historical, but then to most practitioners unfamiliar, disease to which the name of "Influenza" was originally applied. A report on this epidemic was made by me to the Local Government Board in 1891.*

Since the termination of that epidemic the British Isles, together with other countries, have suffered from two further epidemics of the same disease, viz., one in the spring and summer of 1891, (partly described in an addendum to my former report), and the other in the winter of 1891–2. Indeed there is reason to suspect that the disease has never been entirely absent from this country since its appearance late in 1889.

The present report continues the recent history of Influenza, and includes (I.) some statistical studies of the epidemic of 1890 on the basis of the Registrar-General's Annual Report for that year; (II.) an account of the more recent epidemics in England and Wales; (III.) a history of Influenza abroad in 1891 and 1892 ; (IV.) some considerations respecting the etiology of the disease, chiefly indicating directions in which further observations are needed; (V.) some notes on the clinical features of the later epidemics; (VI.) reports on outbreaks of Influenza in 1891 in certain institutions and establishments; (VII.) some remarks on the prophylaxis of the disease, to which is appended the Provisional Memorandum issued by the Board on the subject in January 1892.

Part I.—Statistical Studies of the Epidemic of 1889–90 in England and Wales.

Since my previous report was written the issue of the Registrar-General's annual report for 1890 has afforded some statistical information, previously unattainable, respecting the mortality from Influenza in that year throughout England and Wales, such information having previously been available only for London. The Registrar-General's figures do not enable the Influenza mortality to be studied as regards its distribution in time throughout different periods of the year, nor as regards its local distribution in areas smaller than registration counties.

Mortality from Influenza in England and Wales, 1890;

The mortality occasioned by the epidemic of 1889–90 may be taken as comprised within the latter year. In the year 1889 only 55 deaths were registered as from "Influenza" in England and Wales, of which 8 were in Lincolnshire, 6 in Lancashire, and the remainder scattered through various other counties.

* Report on the Influenza Epidemic of 1889–90, by Dr. Parsons; with an Introduction by the Medical Officer of the Local Government Board. Published by the Queen's Printers. Parliamentary Paper, 1891, C.—6387.

The following table shows the apportionment of the deaths in England and Wales attributed to Influenza in 1890 among persons of different ages and sexes.

Sex.	All Ages.	Age at Death. 0-3 Months.	3-6 Months.	6-12 Months.	Total —1 Year.	1—	2—	3—	4—	Total —5 Years.	5—	10—	15—	20—	25—	35—	45—	55—	65—	75—	85—
Males -	2,415	59	48	63	170	42	32	33	14	291	45	38	86	107	250	345	357	382	305	174	35
Females	2,108	50	22	53	125	54	27	24	10	240	50	51	62	79	203	227	246	282	361	254	53
Both sexes.	4,523	109	70	116	295	96	59	57	24	531	95	89	148	186	453	572	603	664	666	428	88

at different ages and in the two sexes,

The deaths from Influenza among males were therefore more numerous than those among females at all ages except in later childhood and in old age. The larger number of deaths from Influenza among females than among males over 65 is, no doubt, due to the fact that more females than males survive to the later ages of life. In all previous recorded years, however, whether epidemic or otherwise, the deaths from "Influenza" have been more numerous among females than among males, as will been seen from Table A. on page 3 of my former report.

In proportion, however, to the numbers of the population living at different ages the mortality from Influenza among males exceeds that among females at nearly all ages, and notably so at the middle periods of life, viz., from 25 to 65 years. This is shown by the following table calculated from the annual report of the Registrar-General for 1890 thus:—

Total deaths at each age. : Influenza deaths at each age. :: Death-rate per 1,000 living at each age. (Preliminary Table 14.) : Influenza deaths per 1,000 living at each age.

Tables pp. 107-121.

England and Wales. Deaths from Influenza in 1890, per 1,000 Persons living at different Ages.

Sex.	All Ages.	Ages. 0-5.	5-10.	10-15.	15-20.	20-25.	25-35.	35-45.	45-55.	55-65.	65-75.	75-85.	85+
Males -	·17	·16	·02	·02	·06	·08	·12	·22	·31	·47	·72	1·20	2·1
Females -	·14	·12	·03	·03	·04	·06	·09	·15	·19	·31	·70	1·38	2·0

The higher death-rate from Influenza among males, especially at the middle periods of life, may not improbably be connected with the influence of the fatigue and exposure incidental to men's vocations.

in the several counties.

The deaths in 1890 attributed to Influenza, and the death-rate therefrom, are given by counties in the following table, together with certain other rates which may be considered in connexion with them:—

England and Wales. 1890.

Registration County.	Deaths from Influenza.	Death-rates per 1,000 from					Birth-rate per 1000 Population.	Infant Mortality per 100 Births.
		Influenza.	Diseases of Respiratory Organs.	Diseases of Circulatory Organs.	All Causes.			
					1890.	Average 10 years, 1880-9.		
London	624*	·15	4·91	1·69	21·0	20·5	30·7	16·3
Surrey } Extra-	91	·16	2·62	1·72	15·4	15·6	25·8	11·4
Kent } Metropolitan	151	·19	2·80	1·74	16·1	16·8	27·7	12·7
Sussex	94	·17	2·59	1·71	15·5	15·9	24·5	11·5
Hampshire	94	·14	2·72	1·84	16·1	17·1	27·3	11·6
Berks	65	·24	2·52	1·99	15·8	16·4	26·9	10·4
Middlesex } Extra-	91	·16	3·13	1·45	16·6	16·5	29·5	13·6
Herts } Metropolitan	43	·20	2·70	2·17	16·7	17·0	26·7	10·6
Bucks	41	·25	3·19	1·68	16·2	17·0	27·7	11·6
Oxford	40	·21	3·11	1·98	17·1	16·9	27·6	12·1
Northampton	66	·21	3·27	1·65	17·2	17·3	29·2	14·2
Huntingdon	3	·06	2·95	1·48	16·6	16·6	25·9	12·6
Bedford	27	·16	2·61	1·81	16·2	17·7	27·8	12·7
Cambridge	40	·20	2·94	2·02	18·4	17·3	27·1	13·1
Essex	108	·14	3·40	1·57	17·3	17·2	31·3	13·7
Suffolk	49	·13	2·82	1·70	17·4	17·4	28·7	11·7
Norfolk	69	·15	2·76	1·68	18·2	18·5	28·0	14·4
Wilts	72	·28	3·03	2·23	16·2	17·1	27·5	9·8
Dorset	48	·25	3·01	2·19	15·7	16·3	25·4	9·5
Devon	118	·19	3·48	2·09	18·8	18·4	26·6	13·3
Cornwall	47	·15	3·27	1·97	19·6	19·1	26·7	15·5
Somerset	131	·26	3·38	2·20	17·5	17·4	27·7	11·7
Gloucester	11	·21	3·44	2·08	17·8	17·9	27·6	13·0
Hereford	32	·28	3·35	2·21	17·1	17·3	26·3	12·0
Salop	72	·28	3·12	2·12	17·4	17·4	26·8	11·5
Stafford	164	·15	4·85	1·46	20·3	19·7	34·6	16·6
Worcester	74	·18	3·67	1·80	17·4	17·1	28·9	15·1
Warwick	72	·09	4·55	1·89	20·1	19·0	30·8	16·4
Leicester	44	·12	3·27	1·72	18·0	18·8	31·3	16·2
Rutland	3	·13	2·61	2·21	16·2	16·5	23·4	8·7
Lincoln	78	·17	3·29	1·79	18·1	17·5	27·7	14·2
Nottingham	82	·16	3·84	1·66	18·8	19·9	31·5	15·2
Derby	52	·12	3·61	1·71	18·3	17·9	30·8	14·4
Cheshire	93	·13	4·03	1·84	19·5	19·0	30·2	15·6
Lancashire	401	·10	6·12	1·74	23·4	22·5	32·1	17·4
York { West Riding	326	·13	5·31	1·71	21·1	20·1	30·5	16·2
York { East Riding	96	·24	4·10	1·78	19·4	19·7	30·5	14·6
York { North Riding	45	·13	3·65	1·67	18·5	18·3	31·0	14·4
Durham	140	·14	4·35	1·56	20·5	20·2	36·5	15·9
Northumberland	76	·15	3·68	1·99	20·5	19·9	33·0	15·3
Cumberland	93	·35	3·85	2·00	18·6	18·6	31·8	12·9
Westmoreland	13	·20	2·56	2·22	15·4	15·8	26·0	10·0
Monmouth	49	·18	4·41	1·53	2·00	19·8	34·0	15·9
South Wales	163	·16	3·91	1·55	19·7	19·4	32·9	15·6
North Wales	120	·28	3·47	1·87	18·9	18·7	25·7	12·5
England and Wales	4,523	·157	4·23	1·76	19·5	19·2	30·2	15·1

* According to the figures given in the weekly returns for 1890, the number of deaths from Influenza in London during that year was 648, while in the Registrar-General's Annual Summary it is given as 652.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

On examination of the above table it will be found that the highest rates of mortality from Influenza were by no means in the parts of England in which, according to our previous information, its prevalence had seemed to be greatest, viz., in the east of England and the neighbourhood of London; but on the contrary its greatest proportional fatality was in the southern and south midland agricultural counties of England, and in the hilly regions near the west coast. It was not greatest in the most unhealthy parts of the country, for on the contrary the eight counties with the highest general-death rate (above 20·0 per 1,000 inhabitants per annum), viz., London, Stafford, Warwick, Lancashire, the West Riding of Yorkshire, Durham, Northumberland, and Monmouth, had all, with the exception only of that last-named, death-rates from Influenza below the average of the whole kingdom.*

Similarly with regard to another test of sanitary and social conditions unfavourable to health, viz., the mortality of infants in their first year, the six counties which had an infant mortality of over 16 per 100 born, viz., London, Stafford, Warwick, Leicester, Lancashire, and the West Riding had all an Influenza death-rate more or less under the average for the whole kingdom.

It might have been anticipated that the death-rate from Influenza would exhibit in the different counties some degree of parallelism with the death-rate from diseases of the respiratory organs, more especially in view of the fact that a large increase in the mortality from diseases of this class is always observed during an epidemic of Influenza, but such is not the case. On the contrary, the nine counties in which the mortality in 1890 from diseases of the respiratory organs exceeded 4 per 1,000 inhabitants had all Influenza death-rates below the average, with the exceptions of the East Riding and Monmouth; while of the 13 counties in which the mortality from diseases of the respiratory organs was under 3 per 1,000, eight had Influenza death-rates above the average; two of the remainder being the very small counties of Huntingdon and Rutland, in each of which Influenza mortality was represented by 3 deaths only.

But with the mortality from diseases of the heart and other circulatory organs that from Influenza is in the several counties distinctly parallel. In the 12 counties in which the mortality from diseases of the organs of circulation exceeded 2 per 1,000 the Influenza death-rate was above the average in all, with the exception again of the little county of Rutland, while of the seven counties in which the mortality from diseases of the circulatory system fell below 1·6 per 1,000 the Influenza death-rate was below the average in four; it exceeded it only slightly in Middlesex and South Wales, and rather more in Monmouth.

A similar local parallelism between the mortality from Influenza and that from cancer is indicated in the Registrar-General's tables, though the figures are not here reproduced.

Local differences partly explicable by proportions of inhabitants at different ages.

The explanation seems partly to be that the question is one of age. Influenza is a disease comparatively harmless to young people after the first year and especially dangerous at the later periods of life, and therefore, like heart disease and cancer, most fatal in populations containing a larger proportion of elderly persons, and least so in those which contain the largest proportion of children and young persons.

The report on the census of 1891, showing the age-distribution of the inhabitants of the several counties of England and Wales, is not yet published, but an indication of the relative proportions of older and

* It will be seen in a later part of this report (pp. 24 & 25) that the local fatality of Influenza in 1891 has by no means coincided with that in 1890.

younger persons in a community may be gained from a comparison of the birth-rates. A high birth-rate in a community indicates (*a*) a relatively large proportion of young adults constituting the parents, and (*b*) a relatively large proportion of children. On the other hand, a population in which the birth-rate is small will ordinarily contain a relatively large proportion of elderly persons. This seems to be, in part at least, the explanation of the high mortality from Influenza in the agricultural counties, from which many of the younger people emigrate to the towns or manufacturing and mining districts, leaving the old people behind.

In the preceding table it will be seen that the general rule is that the counties which have a low birth-rate have a high Influenza death-rate, and vice versâ. In five counties only in which the birth-rate is above the average for the whole kingdom is the Influenza death-rate above the average, and in two of these, viz., Nottingham and South Wales, only slightly. In six only does a low birth-rate coincide with a low Influenza death-rate; two of these are the small counties of Rutland and Huntingdon, and another is Hampshire, in which local circumstances, such as the existence of military stations containing a large proportion of unmarried persons at the prime of life, may render the birth-rate low in proportion to the age-distribution of the inhabitants.

The Influenza death-rate exceeded or equalled 2·00 per ten thousand inhabitants in 15 counties, viz.:—

Cumberland	3·5	Berkshire	2·4
Wilts	2·8	East Riding	2·4
Hereford	2·8	Northampton	2·1
North Wales	2·8	Oxford	2·1
Salop	2·8	Gloucester	2·1
Somerset	2·6	Cambridge	2·0
Dorset	2·5	Hertford	2·0
Buckingham	2·5		

In these countries, with an aggregate population, estimated to the middle of 1890, of 4,318,527, the birth-rate in 1890 averaged 27·7.

The Influenza death-rate was under 1·40 per 10,000 in the following countries, viz.:—

Huntingdon	·6	Cheshire	1·3
Warwick	·9	West Riding	1·3
Lancashire	1·0	Rutland	1·3
Leicester	1·2	Suffolk	1·4
Derby	1·2	Durham	1·4
North Riding	1·3		

In these counties, with an aggregate population reckoned to the middle of 1890, of 10,467,593, the birth-rate in 1890 averaged 31·6. Lest it should be thought that the local severity of the Influenza epidemic might have been the cause of the low birth-rate in the former counties in 1890, it may be mentioned that in the groups of counties above mentioned the birth-rates maintained much about the same proportion as in other years.

On further examination, however, it does not appear that the difference between the death-rate from Influenza in the agricultural and in the manufacturing and mining counties can be wholly explained by considerations of age and sex-distribution. On the basis of the table already given, on page 3, of the death-rates from Influenza in England and Wales in 1890 among persons of different ages, and taking the numbers of persons of different ages and sexes given in the third volume of the Report on the Census of 1881, I have calculated for certain counties the number of deaths from Influenza which should have occurred on the hypothesis that the distribution of the disease was

but not wholly so explicable.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

uniform over the kingdom, and that the differences in its local fatality were solely due to differences of age and sex constitution of the population. The results are as follows :—

Registration County.	Deaths from Influenza.			Rate per 1,000 Population.	
	Calculated on Hypothesis of uniform Rate over England and Wales.		Number registered in 1890.	Calculated.	Observed.
	On Population in 1881.	On Population in 1891.			
Durham - -	135	156	140	·15	·14
Cumberland - -	40	43	93	·16	·35
Wilts - - -	44	46	72	·18	·28
Shropshire - -	46	43	72	·17	·28
North Wales - -	81	79	129	·17	·28
Monmouth - -	36	42	49	·15	·18
Hampshire - -	95	110	94	·16	·14
London - -	567	627	624	·15	·15

It will be seen that, so far as these figures go, the differences in the death-rates from Influenza in different counties cannot be fully explained by differences in the proportion of persons living at different ages, but for a fuller consideration of the question we must wait until the figures of the census of 1891 and of Influenza mortality in 1892 are before us.

Part II.—Course of the recent Epidemics of Influenza in England and Wales.

Reappearance of epidemic Influenza in 1889-90 after lapse of 41 years.

In my report on the epidemic of 1889–90 it was shown that cases subsequently believed to have been identical with the true Influenza occurred in London and in the eastern counties, (in some instances forming local epidemics), in October and November 1889, and that the disease was somewhat prevalent in London in December 1889, attaining the dimensions of an epidemic in London and the neighbouring counties about January 1st, 1890. Other portions of the kingdom, for the most part, began to be epidemically affected rather later; the west of England about the latter part of January, and the large Lancashire towns about the middle of February; while some remote villages in mountainous districts were not reached until the beginning of March. The disease in its epidemic form had practically died out at the end of the first quarter of 1890, and as more than 40 years had elapsed since the last previous similar epidemic occurred, viz., that of 1847–8, it was hoped that some such period might again pass before the country underwent another visitation.

This hope, however, was not destined to be fulfilled, for in the spring of 1891 another epidemic of Influenza occurred, severer and more fatal than that of the previous year. This second epidemic was first heard of, so far as the British Isles are concerned, in the Yorkshire towns, Hull being the first to be affected, viz., in March, and the other large Yorkshire towns in April. The return of Influenza in Hull was suspected by some to be due to introduction by Russian emigrants, but it seems quite as likely to have been due to a local revival of activity of infection already present, local outbreaks having been observed in December and February in North Yorkshire, and in January at Durham.

Second epidemic in spring of 1891 commenced in Yorkshire,

Influenza seems also to have revived independently about the same time in another part of the country, viz., in South Wales and on the Welsh border, where, since my former report was issued, I have heard of its return in an epidemic form in several districts at a date apparently too early to be consistent with its introduction from Hull.

on Welsh border.

Thus the medical officer of health of Tredegar speaks of its reappearance there at the end of 1890. At Bridgend it began early in the first quarter of 1891, and at Abertillery there were many cases in February 1891, and sporadic ones up to the end of the year. It was prevalent at St. David's in February and March 1891, and at Pontypool in the end of March. In the Pontypool rural district it is stated that an epidemic of Influenza ushered in 1891, then ceased for a few weeks, recurred about the end of March, lasting to the middle of May; ceased again for a very short time to reappear in June, continuing to end of September, again to disappear and break out about middle of November, this time with increased violence and mortality, and continuing to end of year. In the Madley division of the Dore Union, Herefordshire, the first cases are stated to have been observed at the end of February and the beginning of March 1891, they increased in number up to the end of June, and then a few sporadic ones occurred up to the end of November. At Coleford (Forest of Dean) Influenza was epidemic in February, March, and April, and indeed the district is said to have been hardly ever free from it in 1891, and in the adjoining Monmouth rural district it was never quite absent during the earlier half of 1891. At Wrexham and in the neighbourhood Influenza was epidemic in the first quarter of 1891.

and in southern counties of England.

Influenza is also said to have been present in the first quarter of 1891 in some inland rural districts in the southern counties of England. In the Hambledon rural district it was prevalent in January and February 1891, and again in December. At Whitchurch (Hants) Influenza is stated to have been prevalent from January to August 1891, the largest number of cases being in May, June, and July. There had been cases in August, September, and November 1890. At Wilton there were a few cases in February, March, and April. It was extremely prevalent from May to July, and again in December. In the Alderbury rural district it commenced in March 1891, and was very prevalent in May and June. It prevailed throughout the Romsey district in the earlier half of the first quarter of 1891. In the Wincanton rural district there were a few cases in the beginning of February 1891, and it continued prevalent all through that month. At Rye Influenza, which was at no time entirely absent during 1890, began to increase in virulence in the early part of January 1891, the cases increasing in number until the third week of February, when it began to subside again, but was not altogether lost sight of even through the summer months.

Epidemic in London, April to June 1891.

In London Influenza began to be epidemic about the end of April 1891, and as before, when London had been invaded, the disease soon became general all over the country. A postscript to my report already mentioned brought the history of the second epidemic, so far as it could be then learnt, up to June 4th, 1891. This second epidemic seems

to have been more severe and fatal than the first one in London and in the large northern towns and in North Wales, but to have been comparatively mild in the south-west of England.

As an epidemic Influenza seems to have subsided about July, though in many districts a succession of scattered cases is reported to have still occurred. The respite, however, was even shorter than before, for in the autumn of 1891 a third epidemic of Influenza began both in the British Isles and abroad, and occasioned a very great mortality in the winter of 1891–92 in London as well as in many of the large cities of continental Europe which had escaped the earlier epidemic of 1891.

Third epidemic in winter of 1891-2 commenced in autumn of 1891 in Scotland and North of England.

The third epidemic in Great Britain seems to have started in October 1891 in two distinct quarters, viz., in Scotland and in the south-west of England, whence it extended southward from the first and eastward from the second.

This third epidemic seems to have begun in the northern parts of the British Isles, being early established in Scotland at Dundee. It is stated in the "British Medical Journal" of October 31st, 1891, that Influenza had been prevalent in Dundee since the second week in July, causing two or three deaths in each month, but that since the beginning of October it had spread rapidly. In Edinburgh and Aberdeen the first epidemic of 1891 subsided about July, so that it may be looked upon as probable that the second epidemic of 1891 originated in infection remaining from the earlier one of that year. The later epidemic was especially severe in and around Dundee, which up to the end of November seemed to be its chief focus in Scotland, though by that time there was a widespread prevalence over the whole of that country, except the extreme west and north ("British Medical Journal," November 28, 1891). In Aberdeen cases were observed in October, and were more numerous in November, but, on the whole, this city escaped lightly, only 13 deaths occurring up to December 16th. In Edinburgh and Glasgow cases of Influenza were observed in the first week of November, and the disease was severe in the former city, 202 deaths occurring up to the end of January, at which time the epidemic was declining. Glasgow, however, as in the previous epidemics, appears to have escaped comparatively lightly, 110 deaths from Influenza occurring in the eight weeks ending January 23rd, 1892, in a population $2\frac{1}{2}$ times as large as that of Edinburgh. By the beginning of December the epidemic had affected the Orkney Islands, but the Shetlands remained free at the end of January 1892. On January 30th it was still very prevalent in the north of Scotland and the Isles. It was said that in many districts in the Highlands in which scattered cases were noticed in November the disease did not take on an epidemic form until the occurrence of a snowstorm, when whole districts were prostrated with it ("British Medical Journal," January 30, 1892).

In Ireland the first case of Influenza at Londonderry is said to have been noticed on October 4th. At Belfast it began to be epidemic early in November, and at Dublin in the latter part of November.

Of the large English towns, Newcastle-on-Tyne was the first to be severely attacked, the epidemic being severe there in November. The death-rate in this town rose gradually from 15·6 in the week ending October 17th to 45·1 in that ending December 26th, 1891. In Leeds cases of Influenza were observed in the first week of November, but no noteworthy increase in the death-rate took place until the week ending December 26th.

The second epidemic of 1891 appears to have been early established in the county of Durham. It is reported by the medical officer of health of the South Shields rural district to have been continuously present in that district from the end of March 1891 to the close of the year; it was widely spread in August, and about the middle of autumn amounted to a

perfect epidemic. In West Hartlepool it is stated to have been prevalent and steadily increasing from September 1891 to the end of the year. In the Wolsingham division of the Weardale rural district the medical officer of health states that two outbreaks of Influenza took place in 1891 : a mild one in July, not more than 50 or 60 cases in a population of 3,169 ; and a very severe one of about 700 cases, which began on October 18th and continued to the end of December. This latter began simultaneously in several isolated houses, without apparent communication one with another ; but it was found that the people attacked had been three days before to some services at a certain place of worship. In the Sedgefield rural district Influenza is reported as prevailing from the end of October to the close of the year, and other Durham districts are reported as invaded in November.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92 ; by Dr. Parsons.

In Maryport, Cumberland, Influenza, which had been prevalent in May and June, but had subsided in July, reappeared in November, having been introduced from Scotland, and quickly became epidemic, continuing so up to February 1892.

At Standish, near Wigan, Influenza, which had been epidemic earlier in the year, but had subsided in July, was again present in October, but was chiefly confined to children, of whom over 100 were attacked within a few days. In November the disease declined among children, but it attacked adults severely at the end of the year.

Another part of the kingdom in which Influenza revived in the autumn of 1891 was that bordering on the Bristol Channel, viz., the counties of the West of England and South Wales.

and in S.W. England and South Wales.

The medical officer of health for Truro states that Influenza was prevalent in the spring of 1891, but much more so in October, November, and December, when a very large per-centage of the inhabitants suffered, but few children were attacked. In Bath Influenza is reported as prevalent from October 1891 to February 1892.

At Panteg the medical officer of health reports that the Influenza epidemic broke out in October 1891, when almost every house was affected. The medical officer of health for the Nantyglo and Blaina district says that at no time during 1891 was Influenza entirely absent; but the greater number of cases appeared in October and November. Similarly, the medical officer of health for Tredegar, Mr. G. Brown, known as a careful observer, says : "At no time since the second re-
" appearance of Influenza in the end of 1890 has the district been
" entirely free from it, numbers of sporadic cases occurring, sometimes
" one at a time sometimes several. In October 1891 the disease
" began to be epidemic, and raged in every part of the place, reaching
" its highest intensity about the middle of November. It then began
" to decline, and by Christmas had ceased to be epidemic, though cases
" have continued to crop up from time to time."

At Bridgend Mr. Randall, medical officer of health, says : "As in the
" year before, epidemic Influenza attacked the neighbourhood in 1891,
" beginning early in the first quarter and continuing until the summer.
" We then had a lull until the end of September or beginning of
" October, when a recrudescence occurred, which lasted, attacking
" numerous victims, until the end of the year, but which I hope and
" believe is now dying out at the time of writing, early in the first
" quarter of 1892."

In many places in the West of England Influenza is stated to have become epidemic in November 1891, and to have attained very formidable proportions during that month and in December. Examples are Bristol, Taunton, Wellington (Somerset), Newport (Monmouth), and the St. Germans, St. Columb, Dursley, and Pontypool rural districts.

Other local recrudescences of Influenza occurred elsewhere in the autumn of 1891. Thus the medical officer of health for the Bourton

On the Influenza Epidemics of 1889–90, 1891, and 1891–92; by Dr. Parsons.

division of the Stow-on-the-Wold rural district reports that Influenza was epidemic in different parts of the district at different times during 1891, viz., at Bourton-on-the-Water from March to the end of June, and in April, May, and June at Upper and Lower Slaughter. In July it had nearly disappeared, but it returned in September in Great Rissington.

On October 28th, 1891, I visited an industrial school at Dartford, Kent, where I found 37 boys out of a total of 200 suffering from a very severe type of Influenza, which had broken out during the course of the previous week. (*See* account on p. 73 of this report.) Influenza is reported, on November 26th, to have broken out at East Grinstead Workhouse.

In London, as will be seen from the table on the next page but one, the week ending October 24th was the only one in the latter half-year of 1891 in which no deaths ascribed to Influenza were registered. In August the average weekly number of such deaths was 9·8, in September 5·2, in October 4·2, in November 7·0, and in December 13·2. In the week ending December 12th, 1891, there were 8 such deaths, and they increased in successive weeks to 17, 19, 77, 85, 271, and 506. The latter number, occurring in the week ending January 23rd, 1892, was the highest weekly number attained in this or any recorded epidemic. After that date the number rapidly declined, and the epidemic may be considered to have been over by the end of February, although no subsequent week has been free from deaths ascribed to Influenza.

Recent threatenings of a recrudescence.

It is to be feared that the contagium of Influenza must be regarded as still domiciled among us, and that a renewal of its epidemic activity within the next few years is by no means improbable. Local recrudescences at several places, both in this country and abroad, have been observed during the autumn and winter of 1892. Thus Mr. Hartill, medical officer of health for Short Heath, Staffordshire, states that 26 cases of Influenza occurred during August in that district in a population of 2,514. In the adjoining district of Willenhall the same gentleman records 66 cases of Influenza in the third quarter of 1892. In the neighbourhood of Daventry Influenza is stated ("British Medical Journal," December 17, 1892) to have been continually present since January 1892, but latterly to have considerably increased in prevalence and to have been frequently associated with lung complications. I have also more recently heard of local prevalences in Kent, Surrey, Essex, Derbyshire, Yorkshire, and other parts of the country.

Influenza seems also to have been recently on the increase in London and the neighbourhood; the deaths ascribed to it in the metropolis in the first 8 weeks of 1893 having been respectively 7, 12, 14, 16, 15, 19, 27 and 35, 41.

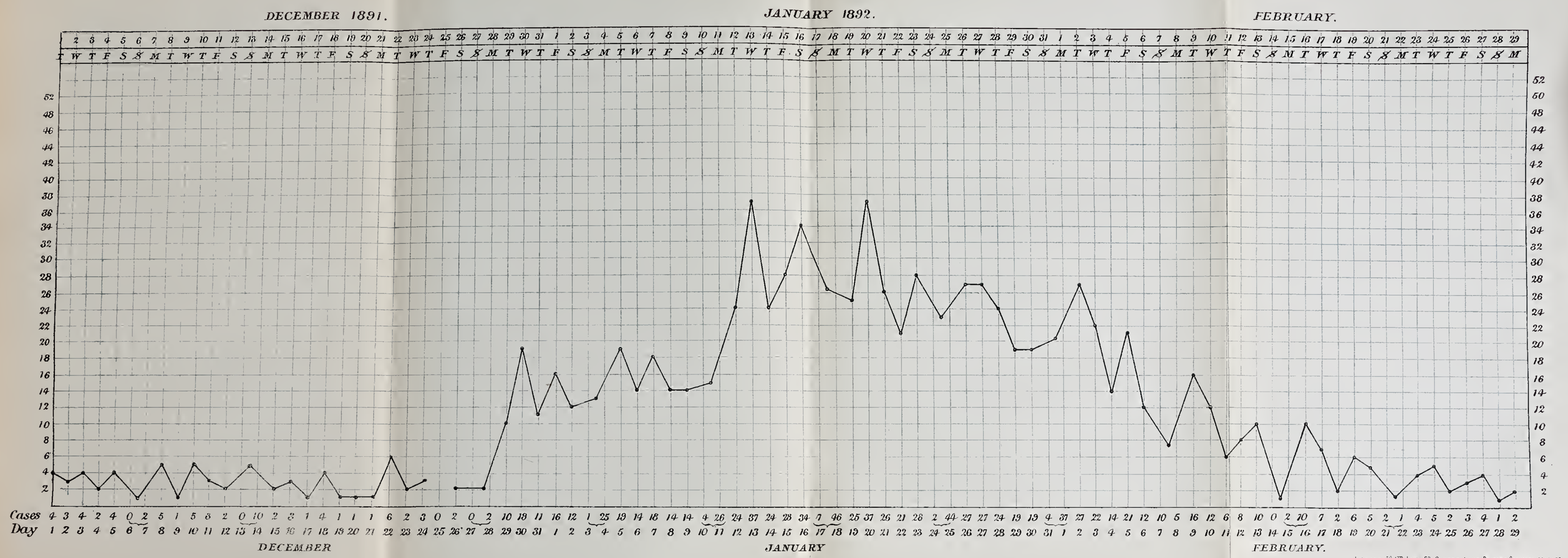
Comparison of three recent epidemics;

The two later epidemics of Influenza seem to have differed from that of 1889–90 in being less rapid in their onset, and, perhaps, also in attacking a smaller proportion of the population; but, on the other hand, they were more protracted in their course, and occasioned a far higher mortality. This mortality was due to the frequency with which in the later epidemics Influenza was complicated with pneumonia and other inflammatory affections of the respiratory organs, and it occurred especially among elderly people.

The course of the rise and fall of the later epidemics in London is illustrated by the annexed diagrams, which show the aggregate daily number of new cases of Influenza coming under treatment at certain London hospitals, and which may be compared with a similar table opposite page 124 of my former report.* It will be seen that the first

* In these diagrams, as in the former, the mean of the figures of Sunday and Monday in each week has been taken in constructing the curve.

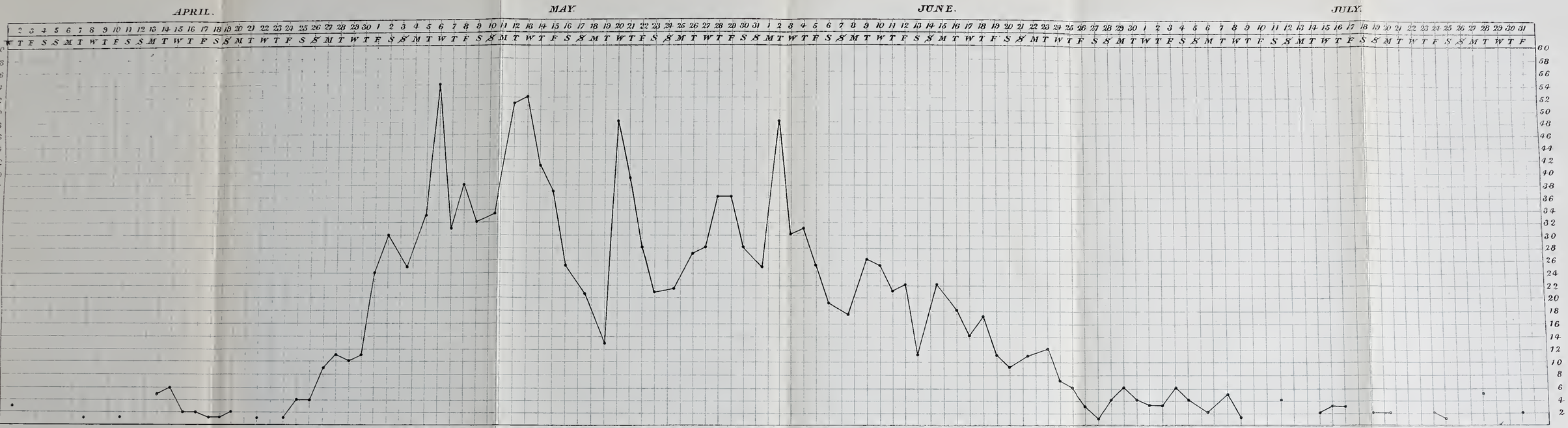
AGGREGATE DAILY NUMBER OF NEW CASES OF INFLUENZA, TREATED AT CERTAIN LONDON HOSPITAL IN 1891-2.



JUDD AND Co LTD LITH 63 CARTER LANE, DOCTORS COMMONS 3814.1.33

Aggregate Daily Number of New Cases of Influenza, Treated at Certain London Hospitals in 1891.

To face page 10.



Judd and Co Ltd Lith, 63 Carter Lane, Doctors Commons 3814/91

epidemic was comprised within a period of three weeks, that the number of new cases rose rapidly to a maximum, and then nearly as rapidly declined, the rise occupying a week, and the decline two weeks, and both rise and fall being little interrupted. On the other hand, the first epidemic of 1891 lasted about eight weeks, and that of 1891–2 about six weeks, and the course of both was marked by many fluctuations.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

It is to be noted, however, that each of these diagrams is based upon returns from a different number of hospitals; the diagram in my first report including the cases in several hospitals from which I was not able to get lists for the later epidemics. It is likely that, had figures been obtainable from other hospitals, the fluctuations in the diagrams of the later epidemics would have been to some extent smoothed out.

Later ones less sudden and more protracted than first. Cases probably fewer.

For the same reason, the figures of the diagrams cannot be taken as indicating the relative numbers of attacks in the several epidemics as compared one with another.

For one numerously attended hospital, however,—viz., the Middlesex Hospital,—I am able, through the courtesy of Dr. Essex Wynter, Medical Registrar, to give in the following table the figures for each of the three late epidemics; and it will be seen that they fully bear out the statements that the two later epidemics were less sudden in their development than that of 1889–90, and that the cases were less numerous, but that the course of these later epidemics was more protracted and more fluctuating than that of the first.

MIDDLESEX HOSPITAL.—DAILY NUMBERS of CASES of INFLUENZA (In and Out-Patients) treated during the EPIDEMICS of 1889–92.

Day of Month.	Epidemic of 1889-90.			First Epidemic in 1891.			Epidemic of 1891-92.		
	December 1889.	January 1890.	February 1890.	April 1891.	May 1891.	June 1891.	December 1891.	January 1892.	February 1892.
1	—	36	6	—	11	22	3	8	20
2	—	25	3§	—	10	25	3	4	14
3	—	35	1	—	3§	21	4	—§	12
4	—	63	5	—	17	20	2	8	6
5	—	5§	10	—§	11	14	2	1	6
6	—	104	3	—	9	12	—§	3	8
7	—	128	7	—	14	—§	2	2	8§
8	—	85	—	—	17	15	3	5	—
9	—	96	—§	—	13	13	1	5	8
10	—	58	2	—	—§	13	1	2§	4
11	—	60	2	—	21	12	2	11	3
12	—	7§	4	—§	26	12	—	7	3
13	—	93	—	—	25	9	—§	15	5
14	—	54	3	—	28	—§	2	13	—§
15	—	51	2	—	23	21	1	16	1
16	—	40	3§	—	10	11	2	12	—
17	—	32	1	—	—§	10	—	1§	4
18	—	26	1	—	16	7	2	19	—
19	—	4§	2	—§	1	9	—	16	3
20	—	48	1	—	24	8	1§	16	3
21	—	26	1	—	17	—§	1	15	2§
22	—	28	—	—	15	8	3	10	—
23	—	27	—§	—	7	9	1	13	—
24	—	12	3	—	16§	2	—	1§	—
25	—	10	3	1	1	5	—	22	2
26	1	—§	1	—§	9	2	—	13	1
27	—	16	2	1	17	—	—§	10	3
28	—	8	2	9	17	2§	—	11	—§
29	—	9		7	20	—	2	8	1
30	11	4		11	13	3	7	13	
31	7	2			1		4	1	
	1,279			726			437		

§ Sundays.

but mortality greater.

But while the more recent epidemics of Influenza in London as compared with that of 1890 appear to have shown a diminished incidence, as regards number of attacks, they have unquestionably caused a greater mortality.

The mortality in London in 1890 ascribed directly to Influenza according to the Registrar-General's Weekly Returns* was 648, or at a rate of 155 deaths per million of the estimated population. These 648 deaths were distributed through the several weeks of the year as follows:—

Influenza mortality in London in 1890.

LONDON.—DEATHS from "INFLUENZA" in 1890.

First Quarter.		Second Quarter.		Third Quarter.		Fourth Quarter.	
Week ending	Deaths.	Week ending	Deaths.	Week ending	Deaths.	Week ending	Deaths.
Jan. 4	4	Apr. 5	10	July 5	2	Oct. 4	2
„ 11	67	„ 12	7	„ 12	1	„ 11	2
„ 18	127	„ 19	9	„ 19	1	„ 18	1
„ 25	105	„ 26	6	„ 26	1	„ 25	—
Feb. 1	75	May 3	2	Aug. 2	3	Nov. 1	2
„ 8	38	„ 10	3	„ 9	1	„ 8	2
„ 15	30	„ 17	2	„ 16	1	„ 15	6
„ 22	24	„ 24	1	„ 23	1	„ 22	3
Mar. 1	23	„ 31	1	„ 30	1	„ 29	2
„ 8	24	June 7	3	Sept. 6	—	Dec. 6	1
„ 15	11	„ 14	—	„ 13	1	„ 13	1
„ 22	17	„ 21	2	„ 20	1	„ 20	1
„ 29	13	„ 28	1	„ 27	2	„ 27	3
						Jan. 3, 1891	1
Total	558	Total	47	Total	16	Total	27

During 1891 the weekly numbers of deaths in London ascribed directly to Influenza have been as follows:—

in 1891.

LONDON.—DEATHS from "INFLUENZA" in 1891.

First Quarter.		Second Quarter.			Third Quarter.			Fourth Quarter.	
			Deaths.			Deaths.			
Week ending	Deaths.	Week ending	Primary.	Secondary.*	Week ending	Primary.	Secondary.*	Week ending	Deaths.
Jan. 10	3	Apr. 4	7	Not stated.	July 4	56	10	Oct. 3	6
„ 17	3	„ 11	3		„ 11	40	7	„ 10	2
„ 24	1	„ 18	9		„ 18	29	4	„ 17	6
„ 31	2	„ 25	16		„ 25	18	4	„ 24	—
Feb. 7	3	May 2	37		Aug. 1	17	2	„ 31	7
„ 14	2	„ 9	148	33	„ 8	6	1	Nov. 7	2
„ 21	—	„ 16	266	52	„ 15	10	3	„ 14	6
„ 28	2	„ 23	319	60	„ 22	9	Not stated.	„ 21	7
Mar. 7	2	„ 30	310	64	„ 29	7		„ 28	13
„ 14	2	June 6	303	56	Sept. 5	12		Dec. 5	9
„ 21	1	„ 14	249	49	„ 12	4		„ 12	8
„ 28	3	„ 20	182	38	„ 19	1		„ 19	17
		„ 27	117	21	„ 26	4		„ 26	19
								Jan. 2	37
Total	24	Total	1,960	373	Total	213	31	Total	139

* The deaths ascribed, as a secondary cause, to Influenza occurring during the course of other diseases are only stated by the Registrar-General in times of an epidemic.

The total number of deaths registered in London directly ascribed to Influenza in 1891 was therefore 2,336,† equal to a rate of 5·44 per million inhabitants.

* But see note on page 3.

† In the Registrar-General's Annual Report for 1891 the number of deaths from Influenza in London is given as 2,302.

During 1892 the number of deaths in London directly ascribed to Influenza has been 2,264, or 531 per million estimated inhabitants. They have been distributed through the year as follows :—

LONDON. DEATHS from INFLUENZA in 1892.

and in 1892.

First Quarter.			Second Quarter.		Third Quarter.		Fourth Quarter.	
Week ending	Deaths from Influenza. Primary.	Deaths from Influenza. Secondary.*	Week ending	Deaths from Influenza.	Week ending	Deaths from Influenza.	Week ending	Deaths from Influenza.
Jan. 9	95	—	April 9	13	July 9	8	Oct. 8	2
„ 16	271	63	„ 16	7	„ 16	2	„ 15	5
„ 23	503	86	„ 23	8	„ 23	1	„ 22	5
„ 30	436	71	„ 30	8	„ 30	2	„ 29	5
Feb. 6	314	62	May 7	11	Aug. 6	2	Nov. 5	5
„ 13	183	33	„ 14	10	„ 13	2	„ 12	6
„ 20	79	9	„ 21	4	„ 20	2	„ 19	2
„ 27	61	20	„ 28	3	„ 27	2	„ 26	4
Mar. 5	34	5	June 4	7	Sept. 3	2	Dec. 3	6
„ 12	30	5	„ 11	7	„ 10	3	„ 10	5
„ 19	35	9	„ 18	5	„ 17	1	„ 17	3
„ 26	20	5	„ 25	2	„ 24	1	„ 24	9
April 2	14	—	July 2	3	Oct. 1	2	„ 31	11
Total	2,078	368	Total	88	Total	30	Total	68

* The deaths ascribed, as a secondary cause, to Influenza occurring during the course of other diseases are only stated by the Registrar-General in times of an epidemic.

Thus, while the deaths from Influenza in the first three months of 1890 (including the first epidemic period) were 558, those in the three months May, June, and July 1891 (including the second epidemic) were 2,104, and in the first three months of 1892, 2,078. Adding in the last two epidemics the cases, so far as given, in which Influenza was recorded as a secondary cause of death, the numbers become 2,502 and 2,448.

But the deaths ascribed primarily or secondarily to Influenza form but a small proportion of the total mortality due to an epidemic. During the first epidemic the total mortality in London exceeded the average in each of the weeks ending in January 1890 as follows :—

Increase of total mortality during epidemic period. In 1890,

Week ending	Excess of Deaths above Average of the corresponding weeks of previous 10 years. All Causes.	Excess of Deaths above Average of the corresponding weeks of previous 10 years. Diseases of Respiratory Organs.*	Deaths from Influenza.
January 4th	123	301	4
„ 11th	810	522	67
„ 18th	765	464	127
„ 25th	260	167	106
Total	1,958	1,454	304

* Excluding phthisis.

(In the succeeding weeks the deaths from all causes and from lung diseases fell below the average, though a considerable but diminishing number were still ascribed to Influenza week by week.)

It may be noted that the deaths ascribed to Influenza bore to the excess from diseases of the respiratory organs the proportion of 1 to 4·75, and that the deaths ascribed to Influenza together with the excess in deaths from diseases of the respiratory organs accounted for 1,758 out of the total excess of 1,958 deaths, leaving only 200 to other causes.

in 1891,

In the second epidemic the total mortality in London continued above the average for a period of 10 weeks, as follows:—

Week ending	Excess in Deaths from		Deaths from Influenza.	
	All Causes.	Diseases of Respiratory Organs.	Primary.	Secondary.
April 25 - - -	75	83	10	—
May 2 - - -	334	219	37	—
„ 9 - - -	433	240	148	33
„ 16 - - -	661	303	266	52
„ 23 - - -	642	296	319	60
„ 30 - - -	837	316	310	64
June 6 - - -	735	297	303	56
„ 13 - - -	461	216	249	49
„ 20 - -	481	217	182	38
„ 27 - - -	156	82	117	21
Total - -	4,815	2,269	1,941	373

(In the week ending July 4th the deaths from all causes had fallen below the average, but there was still a small excess of 29 deaths in the mortality from affections of the respiratory organs, and 56 deaths were ascribed primarily, and 10 secondarily, to Influenza. In the following weeks the general mortality was considerably, and that from respiratory diseases slightly, below the average.)

In the above period the deaths ascribed primarily or secondarily to Influenza bore to the excess of deaths from diseases of the respiratory organs the proportion of 1 to 2·1, or more than twice that in the previous epidemic. The deaths under these headings accounted for 1,583 out of the total excess of 4,815 deaths during the epidemic period.

and in 1892.

In the third epidemic the total mortality in London exceeded the average during each of the seven weeks ending February 13th, 1892, as below:—

Week ending	Excess in Deaths from		Deaths from Influenza.	
	All Causes.	Diseases of Respiratory Organs.	Primary.	Secondary.
January 2 - - -	1,484	778	37	—
„ 9 - - -	624	455	95	—
„ 16 - - -	1,193	591	271	63
„ 23 - - -	1,762	868	506	86
„ 30 - - -	1,471	654	436	71
February 6 - - -	698	279	314	62
„ 13 - - -	173	45	183	33
Total - -	5,921	2,892	1,805	315

(I have not included in the epidemic period the week ending January 2nd (although the deaths from all causes and from diseases of the respiratory organs registered in that week were excessive), for the following reasons—

1st. The large number of deaths registered in that week is attributed by the Registrar-General partly to the delay in registration, owing to Christmas, of deaths which had occurred in the previous week.

2nd. In the previous week there had been a hard frost, with a dense dark frozen fog over London, meteorological conditions which, apart from the presence of Influenza, give rise to a high mortality, especially from diseases of the lungs. This frost and fog broke up on Christmas Day, and the following week was mild and rainy.

3rd. The number of deaths attributed to Influenza in the week ending January 2nd, though increasing, was still comparatively small.

4th. The mortality from all causes fell in the following week, viz., that ending January 9th, before rising to its maximum.

I have no doubt, however, that Influenza contributed to some extent to the high mortality in London registered in the week ending January 2nd, 1892. In the week ending December 26th, 1891, the total deaths registered were 42 below, but those from disease of the respiratory organs 51 above, the average, and 19 deaths were attributed to Influenza.

In the weeks following February 13th, 1892, the total deaths and those from diseases of the respiratory organs were below the average, though deaths from Influenza still continued to occur.)

In this epidemic period the deaths ascribed primarily or secondarily to Influenza bore to the excess of deaths from diseases of the organs of respiration the proportion of 1 to 2·8. The deaths under these headings made up 5,012 out of the total excess of 5,921 deaths.

Question of certification of deaths from Influenza.

The idea may occur to some whether the apparent greater fatality of the later epidemics of Influenza may not have been merely a matter of certification; that when epidemic Influenza first reappeared in 1889–90 medical practitioners, misled by the common misapplication of the word "Influenza" to severe cases of ordinary catarrh, looked upon it as a trivial ailment, and hesitated to ascribe death to it; whereas in the later epidemics it had become recognised as a formidable disease, in which a fatal termination might occur without discredit to the medical attendant.

On the Influenza Epidemics of 1889–90, 1891, and 1891–92; by Dr. Parsons.

It is not improbable that some such change of nomenclature may have had a share in the increased number of deaths ascribed to Influenza in the later epidemics.*

In the first epidemic the deaths ascribed to Influenza were fewer in proportion to the excess in the mortality from diseases of the respiratory organs than in the later ones as follows :—

I. January 1890	1 Influenza to 4·75	Excess of deaths from diseases of respiratory organs above average of corresponding weeks in previous ten years.
II. May and June 1891.	1 Influenza to 2·1	
III. January and February 1892.	1 Influenza to 2·8	

This explanation, however, will not account for the greater mortality from all causes and from diseases of the respiratory organs in the later epidemics than in the first one.

Influence of accompanying weather upon the fatality of Influenza epidemics.

The influence of weather upon the mortality has also to be considered. In my previous report I have shown evidence, which appears to me conclusive, that the outbreak of an epidemic of Influenza is not the effect of any particular kind of weather, but it is possible that the kind of weather accompanying the epidemic may have some influence upon its course and fatality.

In 1890 a frost which had prevailed for the previous week broke up on January 3rd, shortly after the commencement of the epidemic, and the remainder of January was mild, with frequent S.W. gales and a considerable amount of sunshine for the time of year. These conditions, ordinarily in winter conducive to good health, may have rendered the epidemic milder than it would otherwise have been. On the other hand, if there had been no epidemic then, the mortality would under such weather conditions have probably been below the average, so that the increase due to the epidemic of January 1890 was probably greater than at first sight appears.

In 1891, February was remarkable for being entirely without rain, though with frequent frosts and fogs in London. The first part of March was warm and pleasant, but on March 9th a gale and heavy snowstorm occurred, most severe in the S.W. of England. The remainder of March and April were fine and dry, but with a prevalence of cold easterly winds, which made the spring very backward. The first half of May was very fine and warm, with a dry air and easterly and northerly winds. On May 15th a very cold and wet period suddenly set in; the mean daily temperature at Greenwich, which on May 13th was 65·2° or 13·1° above the average fell on May 16th to 40·2°, or as much below the average, and on May 18th to only 39·3°, or 14·8° below the average. This cold period lasted till May 30th. The week June 6th–12th was cold and dry, with N.E. winds; the remainder of the month was variable. It may be that the sudden change in the middle of May from fine dry and warm to cold and wet weather increased the fatality, and prolonged the duration of the Influenza

* The following remarks taken from the annual report for 1891 of Mr. May, M.O.H. to the Aston Manor Urban District, to some extent bear out this idea :—"In "the Influenza epidemic of 1890 the medical practitioners recognised its presence, "but did not acknowledge it in their certificates, ascribing the deaths to the chest "complications which supervened upon it; but it has been acknowledged very fully "this year (1891) as a primary cause of death, and there were 30 deaths certified "as caused by Influenza (in 1890 there were only 3 so certified). The Influenza "epidemic of 1889–90 was responsible for about 100 deaths in Aston Manor, and I "estimate that the epidemic of the present year has been directly and indirectly "responsible for 114 deaths, chiefly among adults and elderly persons."

epidemic; but it is clear that the epidemic, which had commenced in the last days of April, had already almost attained its height before the change in the weather occurred, especially when we take into consideration the intervals of time which necessarily take place between the commencement of illness and death, and between death and registration.

In the winter of 1891–2 the early part of December was mild. On December 19th a week of severe frost set in, with calm stagnant air and dense black frozen fogs. This broke up on Christmas Day, and was followed by a week of mild, damp weather. On January 4th a period of frost again set in, with little wind and absence of sun, and lasted nearly to the end of the month. The first half of February was variable, but in the third week a period of frost again occurred. Influenza had been present in London, as it would appear all through the early part of December 1891 (if indeed it had ever been really absent since the previous epidemic); the number of cases under treatment began to rise about December 29th, and the epidemic continued to prevail all through January, and declined about the middle of February. It will be noted that it commenced, as two years before, just after Christmas, but under meteorological conditions in exact antithesis to those of 1889–90. It is possible that the severe weather in January 1892, as compared with that in 1890, may have had to do with the higher mortality in the later year, but the frosty weather in the third week of February 1892 did not interrupt the decline of the epidemic.

Epidemic Influenza does not check other epidemics.

It has sometimes been stated by observers in former times that other epidemic diseases which may have previously prevailed, subside at the onset of an epidemic of Influenza, to reappear at its close. The returns of the Registrar-General do not support this view. An examination of the weekly returns shows that during the Influenza epidemic period of 1890 there were high death-rates, sustained for several weeks, from whooping cough at London, Bristol, Brighton, Bolton, and Salford, from measles at Liverpool, and from scarlet-fever at Sheffield.

During April, May, and June 1891 there was, concurrently with the Influenza epidemic, a sustained high mortality from whooping cough at Bradford, Manchester, Leicester, Oldham, Liverpool, and Preston; from scarlet fever at Halifax, and from measles at Portsmouth.

In the winter of 1891–2 the Influenza epidemic was accompanied by a sustained high mortality from whooping cough at Newcastle-on-Tyne, Wolverhampton, Portsmouth, London, West Ham, Croydon, and Blackburn, from measles at Newcastle-on-Tyne, London, Norwich, and Liverpool, and from diarrhœa at Preston.

Increased mortality during Influenza epidemics of 1891 and 1892 in large towns of England and Wales.

In the large towns of England and Wales the progress of the Influenza epidemic of 1891 and 1892, so far as indicated by the death-rates, may be seen in the annexed tables (pp. 18–21), compiled from the Registrar-General's weekly reports and annual summary. They may be compared with a similar table for the first epidemic on p. 115 of my previous report.

Town.	Recorded Date of Influenza.	Death-rate per 1,000 Population per Annum in 1891 in Weeks ending																		Average of 10 years, 1881-90.
		March 7.	March 14.	March 21.	March 28.	April 4.	April 11.	April 18.	April 25.	May 2.	May 9.	May 16.	May 23.	May 30.	June 6.	June 13.	June 20.	June 27.	July 4.	
London	1st record, April 14; epidemic, beginning of May.	24·9	22·0	21·6	20·6	22·2	21·3	21·8	22·3	24·8	25·6	27·7	27·6	28·9	27·0	23·3	23·0	19·0	16·8	20·5
Brighton	-	18·0	22·1	18·5	22·6	15·8	19·8	19·4	12·6	21·2	20·7	20·7	18·5	21·7	18·0	14·0	15·3	19·4	15·8	18·5
Portsmouth	Epidemic, May 14	21·8	24·1	26·4	27·0	27·7	31·0	23·1	25·4	24·8	26·1	31·3	25·4	21·5	25·7	18·6	17·3	17·6	14·7	19·0
Norwich	April	25·2	14·9	20·1	22·1	18·5	18·5	21·6	23·7	16·0	26·8	23·2	21·6	16·5	16·5	20·1	19·0	21·1	16·5	20·0
Plymouth	May 12	27·2	22·8	22·8	24·7	29·6	23·5	17·9	25·9	27·8	22·8	27·2	24·7	21·0	19·1	16·0	14·2	24·1	19·1	21·4
Bristol	Latter half of May	24·2	26·8	21·6	26·1	23·5	22·8	21·1	19·0	18·3	19·0	26·1	22·1	22·5	17·4	18·5	18·8	20·0	20·0	19·3
Wolverhampton	Epidemic, May 16	17·6	19·5	27·7	23·3	19·5	22·0	17·0	25·2	27·7	25·8	29·6	29·6	27·7	35·9	27·7	30·9	28·3	24·6	21·7
Birmingham	1st record, April 8; epidemic, April 30.	26·6	22·4	21·3	21·5	19·8	24·4	18·9	26·2	21·6	26·8	36·4	34·3	38·1	38·2	30·9	26·9	23·5	17·3	20·8
Leicester	Beginning of May to July	27·1	27·4	22·7	24·1	28·2	22·3	28·2	24·5	24·9	32·6	44·6	31·8	28·2	30·4	21·2	22·3	27·8	15·7	20·6
Nottingham	Epidemic May 2	20·6	23·3	20·6	19·9	20·8	23·5	25·7	23·3	25·0	28·7	34·8	24·5	36·0	24·0	27·0	20·8	17·9	13·2	21·5
Derby	Beginning of May to June	19·3	19·3	13·8	15·5	15·5	25·9	19·9	17·7	27·0	26·5	40·3	34·8	38·6	22·6	17·1	16·0	19·3	16·0	18·6
Birkenhead	Beginning of May	21·5	23·0	17·8	19·4	29·3	20·9	20·9	23·0	16·2	27·7	26·7	24·1	28·8	26·2	19·4	22·5	16·8	13·1	19·9
Liverpool	End of April	25·6	26·8	27·0	28·1	27·5	28·7	26·3	31·7	32·7	37·3	35·8	39·1	38·7	35·5	31·4	29·3	26·8	20·2	26·3
Bolton	May 2	19·5	24·0	21·7	17·6	19·5	26·2	20·8	21·3	27·6	19·5	29·0	23·5	33·0	28·5	22·6	30·8	20·8	22·6	22·4
Manchester	1st cases, end of March; epidemic, beginning of May.	27·5	28·1	28·1	25·9	26·9	29·7	27·4	29·8	35·1	43·6	41·9	44·1	44·1	39·8	29·1	27·0	23·8	23·1	26·6
Salford	Present all through early months till June.	28·1	30·4	28·6	26·5	32·3	27·0	26·0	22·3	28·9	39·1	45·7	42·0	45·9	39·4	23·1	24·7	23·1	17·3	24·5

Figures underlined mark periods of high mortality probably due to Influenza epidemic.

Town.	Recorded Date of Influenza.	Death-rate per 1,000 Population per Annum in 1891 in Weeks ending																	Average 10 years 1881 to 1890.
		March 7.	March 14.	March 21.	March 28.	April 4.	April 11.	April 18.	April 25.	May 2.	May 9.	May 16.	May 23.	May 30.	June 6.	June 13.	June 20.	June 27.	July 4.	
Oldham - -	Epidemic in May and June -	24·9	31·6	33·2	30·4	25·3	24·5	27·3	29·6	34·8	43·8	45·0	57·7	39·9	35·9	24·9	27·2	23·7	18·6	23·9
Blackburn -	Middle of May to end of June -	30·3	32·5	37·7	30·3	30·3	28·6	23·4	25·1	40·2	32·0	33·3	35·5	39·8	48·5	37·6	27·7	21·6	22·9	24·8
Preston - -	Beginning of May to June -	24·2	25·1	29·0	29·0	28·5	24·2	26·1	29·5	32·4	36·7	31·4	27·5	35·8	33·3	30·9	27·5	21·3	21·7	26·5
Huddersfield -	End of April to June - -	34·4	31·6	35·4	36·5	22·4	28·9	21·3	26·2	26·7	39·8	54·5	36·0	43·6	34·4	21·7	26·2	20·2	18·5	20·8
Halifax - -	Epidemic, April 25 - -	32·0	23·2	27·6	26·3	28·2	19·4	27·0	33·9	40·8	35·1	32·6	43·3	33·9	33·9	31·4	23·8	23·8	21·3	21·2
Bradford -	Epidemic, April 25 to end of June	25·5	21·9	23·1	22·8	20·7	26·0	20·7	29·1	36·3	45·4	56·7	48·8	34·6	29·1	17·5	20·4	17·8	16·1	20·4
Leeds - -	Epidemic, April 21 - -	25·6	22·5	24·7	22·9	27·3	26·0	24·0	28·0	36·2	48·5	43·7	35·2	29·7	24·9	19·5	15·1	18·8	17·7	22·1
Sheffield -	1st cases, beginning of April; epidemic, April 13.	25·6	22·6	20·7	22·0	22·6	25·8	35·3	60·1	73·4	61·7	35·9	27·4	23·7	18·9	22·9	21·3	18·1	17·5	22·0
Hull - -	1st cases, end of February; epidemic, middle of March.	19·2	21·3	22·8	25·2	40·0	46·4	52·4	40·0	31·9	27·2	17·6	22·8	19·0	17·8	17·6	19·2	20·0	17·1	21·0
Sunderland -	May 7 - - - - -	29·8	21·8	21·5	21·5	23·4	25·0	27·8	24·2	23·4	24·6	24·6	26·6	24·6	21·8	22·6	17·9	19·5	20·3	25·0
Newcastle -	Slight epidemic in April and May.	27·8	23·4	24·5	21·7	25·9	30·0	27·8	23·4	27·5	27·8	23·9	15·0	21·7	21·4	19·7	23·1	21·7	17·8	23·8
Cardiff -	Epidemic, May 9 - -	30·8	25·6	24·4	18·4	21·2	24·8	23·2	23·6	20·8	24·4	22·0	25·6	30·4	24·8	21·6	24·4	16·0	14·8	22·1

Figures underlined mark periods of high mortality probably due to Influenza epidemic.

Town.	Recorded Date of Influenza.	Death-rate per 1,000 Population per Annum in Weeks ending																			
		November 1891.				December 1891.				January 1892.					February 1892.				March 1892.			
		7.	14.	21.	28.	5.	12.	19.	26.	2.	9.	16.	23.	30.	6.	13.	20.	27.	5.	12.	19.	26.
London	End of Dec.	17·9	19·6	20·1	19·9	21·2	18·1	17·9	21·9	42·0	*32·8*	*40·0*	*46·0*	*41·0*	*30·6*	*24·6*	20·7	22·4	18·9	22·1	24·3	21·3
Croydon	January	—	—	—	—	—	—	—	—	—	15·7	*33·4*	*39·3*	*47·2*	*28·6*	*20·1*	18·2	14·2	15·7	21·1	13·8	12·8
West Ham	January	—	—	—	—	—	—	—	—	—	*29·5*	*33·6*	*30·5*	*28·8*	*23·3*	*20·2*	19·2	16·6	19·5	17·8	15·6	19·9
Brighton	Epidemic in Jan.	15·8	15·8	21·2	14·4	18·0	25·7	22·1	18·0	*38·3*	*37·2*	*51·5*	*60·9*	*24·6*	*23·7*	*24·6*	17·5	17·9	24·2	17·9	15·7	9·9
Portsmouth	January	16·0	15·6	14·7	13·4	17·3	16·3	11·7	17·9	*27·0*	*36·0*	*57·0*	*44·3*	*32·5*	*30·6*	*24·2*	*22·0*	*25·2*	15·3	19·1	21·3	17·8
Norwich	Cases in beginning of Dec.; epidemic, beginning of Jan.	14·9	23·2	28·8	17·0	22·1	12·4	18·0	20·1	*33·5*	*31·0*	*40·1*	*44·7*	*39·1*	*39·6*	21·8	22·3	24·4	19·8	21·8	23·9	21·8
Plymouth	November	19·1	21·0	*24·7*	*38·3*	*25·9*	*45·1*	19·8	24·7	35·8	32·3	21·9	26·2	26·2	17·7	21·9	18·9	23·1	15·8	21·9	18·9	17·1
Bristol	Began middle of Nov.	23·7	*26·8*	*24·4*	*25·1*	*33·3*	22·3	22·1	25·1	29·8	24·7	24·0	25·0	27·5	21·2	19·6	22·2	30·5	18·7	21·7	25·2	21·0
Wolverhampton	Epidemic in Jan.	25·2	22·7	24·6	29·0	34·0	40·9	31·5	26·4	*45·3*	*35·0*	*48·1*	*39·3*	*30·0*	26·8	23·1	16·9	25·0	25·6	28·1	32·5	25·0
Birmingham	Epidemic, beginning of Jan.	17·3	22·8	19·5	20·1	19·0	18·8	16·5	16·5	29·1	18·8	20·0	27·2	19·8	21·7	20·9	22·0	23·6	20·7	20·5	23·4	22·4
Leicester	End of Dec. to Feb., but mild.	16·8	18·7	16·8	19·0	16·5	12·8	17·6	13·2	20·5	16·2	17·1	15·6	21·2	16·2	22·9	23·5	21·5	21·2	19·7	21·8	20·6
Nottingham	End of Jan.; epidemic, Feb.	15·9	18·1	15·9	17·7	18·9	21·6	10·8	20·6	26·2	19·9	22·5	19·4	*26·6*	*26·4*	*32·0*	*23·2*	*27·4*	16·7	25·7	20·8	23·0
Derby	Epidemic, beginning of Jan.	18·8	18·2	24·8	16·0	18·2	18·8	19·9	17·7	35·9	22·8	29·4	23·4	25·0	19·6	20·1	13·0	29·9	21·2	26·1	22·3	13·6
Birkenhead	- - - -	24·6	16·8	21·5	16·2	20·4	19·4	14·7	17·3	22·0	22·1	27·8	26·3	34·0	21·6	17·0	23·2	27·8	19·6	21·1	23·2	22·7
Liverpool	January	26·1	24·8	27·1	24·6	29·7	25·0	24·1	28·1	42·2	*36·3*	*42·0*	*40·0*	*33·1*	27·1	24·1	25·1	29·6	27·8	30·0	33·1	26·8
Bolton	- - - -	19·9	16·7	22·2	15·8	20·8	14·9	19·5	26·2	29·2	22·0	30·5	25·6	27·8	27·4	20·6	17·9	25·6	20·2	22·4	32·7	22·0

Figures underlined mark periods of high mortality probably due to Influenza epidemic.

Town.	Recorded Date of Influenza.	Death-rate per 1,000 Population per Annum in Weeks ending																			
		November 1891.				December 1891.				January 1892.					February 1892.				March 1892.			
		7.	14.	21.	28.	5.	12.	19.	26.	2.	9.	16.	23.	30.	6.	13.	20.	27.	5.	12.	19.	26.
Manchester	1st case beginning of Dec.	22·5	27·9	22·4	21·8	24·1	19·5	21·6	27·1	35·6	24·0	26·6	23·9	27·6	24·2	24·2	23·6	25·0	26·2	27·2	29·2	29·2
Salford	"A slight return in Nov. and Dec."	25·2	20·5	19·9	21·8	24·1	16·8	17·3	25·2	32·5	23·1	24·4	29·0	19·5	24·6	21·0	24·4	26·5	21·8	25·2	33·2	27·5
Oldham	Apparently absent	17·4	26·9	19·0	20·5	22·5	23·7	16·6	24·1	28·4	24·5	26·4	23·3	22·1	25·3	25·6	26·4	26·4	21·8	25·6	33·4	35·0
Blackburn	Slight outbreak in Dec.	25·1	19·0	22·9	14·3	21·6	21·6	16·9	20·3	30·7	26·9	20·9	*29·4*	*37·5*	*27·7*	*24·7*	*27·7*	*26·9*	19·2	28·2	26·0	18·8
Preston	Middle of Jan.	24·2	23·7	22·7	25·1	29·0	16·4	19·8	25·1	33·8	24·9	22·5	*29·6*	*23·0*	*31·1*	*30·6*	*28·2*	*27·8*	*32·5*	20·6	26·3	29·6
Burnley	—	—	—	—	—	—	—	—	—	—	20·1	21·9	21·3	21·3	20·7	19·6	26·5	34·0	30·5	27·6	33·4	23·0
Huddersfield	—	16·4	16·9	11·5	14·7	20·9	15·8	13·6	16·4	21·8	17·8	14·6	17·3	20·5	18·4	17·3	20·0	17·8	26·4	24·3	27·5	37·2
Halifax	—	13·2	17·6	13·2	20·1	20·1	15·2	12·5	16·9	19·4	18·6	19·2	19·8	19·2	17·4	18·0	24·2	32·9	26·7	31·0	29·8	31·0
Bradford	—	16·8	16·3	18·7	16·8	18·5	17·5	16·6	22·6	32·0	16·6	15·7	20·5	20·7	13·8	16·6	15·5	19·7	19·0	24·7	24·7	19·7
Leeds	1st cases beginning of Nov.	19·1	15·1	16·8	18·6	20·6	20·6	20·1	24·2	35·5	26·4	25·1	20·3	21·8	17·1	17·9	17·4	21·4	18·9	23·6	24·7	22·6
Sheffield	Cases beginning of Dec. No epidemic.	17·2	17·0	18·1	21·8	19·9	18·1	21·5	26·0	26·3	25·2	20·3	21·8	20·1	20·9	19·8	20·3	21·7	22·9	19·9	24·8	22·8
Hull	Cases beginning of Dec. No epidemic.	17·1	17·9	18·9	14·5	18·9	13·5	17·1	16·9	26·7	20·6	25·7	19·1	23·9	22·7	19·9	19·4	18·3	20·9	19·9	21·1	16·6
Sunderland	—	28·6	33·0	31·8	32·2	38·9	31·4	30·6	27·0	25·0	25·1	25·5	23·6	18·4	20·8	20·8	15·7	19·6	19·2	25·9	25·9	19·6
Gateshead	—	—	—	—	—	—	—	—	—	—	27·1	35·3	21·2	28·8	21·2	18·8	18·2	20·6	17·7	23·5	21·8	19·4
Newcastle	Nov. to Jan.	23·4	24·7	25·9	*31·1*	*35·0*	*36·4*	*32·8*	*45·1*	*37·0*	*43·7*	*31·5*	*31·2*	22·8	20·6	27·1	17·9	25·8	15·5	16·5	17·4	17·9
Cardiff	—	25·2	21·2	26·0	24·8	37·2	33·2	28·8	22·4	34·8	28·3	23·4	24·5	22·6	25·3	21·8	23·0	21·8	20·3	16·8	22·2	24·5
Swansea	January	—	—	—	—	—	—	—	—	—	32·8	38·4	29·4	26·5	23·7	22·0	24·8	23·7	28·8	32·8	27·1	26·5

Figures underlined mark periods of high mortality probably due to Influenza epidemic.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

The following table, derived from one in the "British Medical Journal" of 1892, shows the number of deaths ascribed to Influenza in the several metropolitan sanitary districts during the epidemic of January and February 1892.

The results are discussed on page 56 of this report.

Mortality from Influenza in London districts, January and February 1892.

DEATHS from INFLUENZA in METROPOLITAN DISTRICTS. 1892.

	District.	Population, 1891.	In Week ending January 9.	January 16.	January 23.	January 30.	February 6.	February 13.	February 20.	February 27.	Total in Eight Weeks.	Rate per 1,000 in Eight Weeks.
Western.	Paddington	117,838	5	8	13	17	18	5	4	1	62	·53
	Kensington	166,321	5	18	48	33	20	11	1	2	138	·84
	Hammersmith	97,237	6	8	13	5	4	5	1	2	44	·45
	Fulham	91,640	4	5	13	5	7	2	1	1	38	·42
	Chelsea	96,272	3	13	16	16	8	6	1	2	65	·68
	St. George, Hanover Square.	78,362	7	6	11	24	8	8	2	2	68	·87
	Westminster	55,525	1	2	2	2	3	—	2	—	12	·22
	St. James, Westminster.	24,993	—	1	2	5	1	1	1	1	12	·48
Northern.	Marylebone	142,381	3	12	21	9	10	4	2	2	63	·45
	Hampstead	68,425	2	4	5	9	3	3	—	2	28	·41
	St. Pancras	234,437	4	15	43	19	17	6	5	5	114	·49
	Islington	319,433	12	23	44	37	15	13	5	7	147	·46
	Hackney	229,531	3	15	24	19	13	5	3	4	86	·37
Central.	St. Giles	59,778	—	1	8	4	4	7	—	?	24?	·60?
	St. Martin-in-Fields.	14,574	—	—	1	1	—	1	—	—	3	·21
	Strand	25,107	1	—	—	—	2	1	1	—	5	·20
	Holborn	33,503	—	—	4	4	—	5	2	?	15?	·45?
	Clerkenwell	65,885	1	5	8	9	3	1	—	1	28	·43
	St. Luke	42,411	—	1	6	3	1	2	1	—	14	·33
	City of London	37,504	3	2	6	5	3	2	—	1	22	·59
Eastern.	Shoreditch	124,009	—	3	7	8	4	6	1	1	30	·24
	Bethnal Green	129,134	1	7	14	8	11	6	5	1	53	·41
	Whitechapel	74,420	7	9	8	12	3	1	—	1	41	·56
	St. George-in-East.	45,546	—	2	4	5	3	2	—	1	17	·37
	Limehouse	57,599	1	5	3	5	3	2	1	1	21	·36
	Mile End Old Town.	107,565	1	3	13	2	4	3	5	1	32	·30
	Poplar	166,697	2	7	11	7	8	2	—	2	39	·23
St. Saviour, Southwark.		27,162	—	2	4	2	2	1	—	1	12	·45
St. George, Southwark.		59,712	—	3	2	2	—	2	1	1	11	·18
Newington		115,663	2	4	7	5	4	5	2	—	29	·25
St. Olave, Southwark		12,694	1	—	3	—	—	—	—	?	4?	·31
Bermondsey		84,688	1	6	11	4	7	4	1	3	37	·44
Rotherhithe		39,074	—	1	2	5	5	4	2	2	21	·54
Lamboth		275,202	5	13	24	26	30	8	6	7	119	·43
Battersea		150,458	4	10	12	17	17	14	3	—	77	·51
Wandsworth		156,931	3	10	22	17	12	7	4	1	82	·52
Camberwell		235,312	—	15	26	28	15	11	9	1	105	·45
Greenwich		165,417	2	9	16	12	6	4	4	—	53	·32
Lewisham		92,647	33			16	11	1	1	—	62	·67
Woolwich		40,848	1	4	1	1	1	—	1	—	9	·22
Plumstead		88,539	2	8	12	16	10	3	1	2	45	·51

The following table is based upon the Registrar-General's annual report for 1890 and 1891.

Deaths from Influenza in England and Wales in 1890 and 1891, arranged according to Registration Counties.

	Registration Counties.	Population, 1891.	Deaths from Influenza.		Rate per 1,000.	
			1890.	1891.	1890.	1891.
London		4,211,056	624	2,302	0·15	0·55
South-Eastern.	Surrey (extra-metrop.)	572,060	91	300	0·16	0·52
	Kent (extra-metrop.)	806,287	151	261	0·19	0·32
	Sussex	554,538	94	214	0·17	0·39
	Hants	666,239	94	183	0·14	0·27
	Berks	268,352	65	114	0·24	0·41
South Midland.	Middlesex (extra-metrop.)	575,254	91	250	0·16	0·43
	Herts (extra-metrop.)	215,160	43	89	0·20	0·41
	Bucks	164,325	41	103	0·25	0·63
	Oxford	188,225	40	79	0·21	0·42
	Northampton	308,149	66	291	0·21	0·95
	Huntingdon	50,290	3	34	0 06	0·68
	Bedford	165,997	27	97	8·16	0·58
	Cambridge	196,266	40	91	0·20	0·46
Eastern.	Essex	761,172	108	319	0·14	0·42
	Suffolk	365,479	49	100	0·14	0·27
	Norfolk	448,609	69	254	0·15	0·57
South-Western.	Wilts	255,120	72	132	0·28	0·52
	Dorset	188,965	48	31	0·25	0·16
	Devon	636,184	118	208	0·19	0·33
	Cornwall	318,601	47	181	0·15	0·57
	Somerset	510,064	131	215	0·26	0·42
West Midland.	Gloucester	548,901	114	235	0·21	0·43
	Hereford	113,391	32	111	0·28	0·98
	Salop	254,745	72	238	0·28	0·94
	Stafford	1,103,322	164	472	0·15	0·43
	Worcester	422,515	74	182	0·18	0·43
	Warwick	801,760	72	520	0·09	0·65
North Midland.	Leicester	379,214	44	199	0·12	0·53
	Rutland	22,123	3	30	0·13	1·36
	Lincoln	467,184	78	555	0·17	1·19
	Nottingham	505,154	82	315	0·16	0·62
	Derby	432,414	52	389	0·12	0·90
North-Western.	Cheshire	707,962	93	383	0·13	0·54
	Lancashire	3,957,954	401	2,394	0·10	0·61
York-shire.	West Riding	2,464,379	326	1,930	0·13	0·79
	East Riding	399,961	96	393	0·24	0·98
	North Riding	354,407	45	277	0·13	0·78
Northern.	Durham	1,024,259	140	411	0·14	0·40
	Northumberland	506,096	76	214	0·15	0·42
	Cumberland	266,550	93	151	0·35	0·56
	Westmoreland	66,215	13	67	0·20	1·02
Welsh.	Monmouth	275,086	49	274	0·18	1·00
	South Wales	1,049,960	163	614	0·16	0·59
	North Wales	451,074	129	489	0·28	1·09
	Total	29,001,018	4,523	16,686	0·157	0·572

In the following table the several counties are arranged in the order of their relative mortality from Influenza in the years 1890 and 1891:—

Comparison of local mortality in 1890 and 1891.

1890.				1891.			
Below Average.		Above Average.		Below Average.		Above Average.	
Huntingdon	·06	Surrey*	·16	Dorset	·16	Bedford	·58
Warwick	·09	Middlesex*	·16	Hants	·27	S. Wales	·59
Lancashire	·10	Bedford	·16	Suffolk	·27	Lancashire	·61
Leicester	·12	Nottingham	·16	Kent*	·32	Nottingham	·62
Derby	·12	S. Wales	·16	Devon	·33	Bucks	·63
Rutland	·13	Sussex	·17	Sussex	·39	Warwick	·65
Cheshire	·13	Lincoln	·17	Durham	·40	Huntingdon	·68
W. Riding	·13	Worcester	·18	Berks	·41	N. Riding	·78
N. Riding	·13	Monmouth	·18	Herts*	·41	W. Riding	·79
Hants	·14	Kent*	·19	Oxford	·42	Derby	·90
Essex	·14	Devon	·19	Essex	·42	Salop	·94
Suffolk	·14	Herts*	·20	Somerset	·42	Northampton	·95
Durham	·14	Cambridge	·20	Northumberland	·42	Hereford	·98
London	·15	Westmoreland	·20	Middlesex*	·43	E. Riding	·98
Norfolk	·15	Oxford	·21	Gloucester	·43	Monmouth	1·00
Cornwall	·15	Northampton	·21	Stafford	·43	Westmoreland	1·02
Stafford	·15	Gloucester	·21	Worcester	·43	N. Wales	1·09
Northumberland	·15	Berks	·24	Cambridge	·46	Lincoln	1·19
		E. Riding	·24	Surrey*	·52	Rutland	1·36
		Bucks	·25	Wilts	·52		
		Dorset	·25	Leicester	·53		
		Somerset	·26	Cheshire	·54		
		Wilts	·28	London	·55		
		Hereford	·28	Cumberland	·56		
		Salop	·28	Norfolk	·57		
		N. Wales	·28	Cornwall	·57		
		Cumberland	·35				
England and Wales	·157			England and Wales	·572		

* Extra-Metropolitan portions of these counties.

From an inspection of this table we may make the following observations:—

1 That in almost every county the death-rate from Influenza was much higher in 1891 than in 1890, Dorset being the only exception.*

2. That the relative position of individual counties, in many cases, is different in the two years.

   Thus the populous manufacturing and mining counties of the North-Western, North Midland, and Yorkshire divisions, which occupied a low place in the scale of Influenza mortality in 1890 were high up in 1891. (Owing to this, the list of counties in which the death-rate from Influenza in 1890 was below the average of the whole kingdom is shorter than that of counties in which it was above the average, whereas the contrary was the case in 1891, the shorter list in each year comprising the populous counties of Lancashire and the West Riding.)

   On the other hand, the south-western counties of England, which occupied a high position on the list in 1890, were low down in 1891, except Cornwall, which occupied a middle position in each year.

3. That in certain counties as Hants, Essex, Suffolk, and Durham the Influenza death-rate was low in each year.

4. That in London, Surrey, Norfolk, South Wales, and Cornwall the Influenza mortality in each year was about the mean for the whole Kingdom.

5 That in certain other counties the mortality from Influenza was notably above the average of the whole kingdom in both years.

* Dorset seems to have suffered severely in the early months of 1892.

This was the case in North Wales, Shropshire, and Herefordshire—counties with a declining population, and therefore presumably containing a large proportion of elderly persons; also in Monmouthshire, Northamptonshire, Bucks, Lincolnshire, and the East Riding of Yorkshire.

6. The mountainous regions of North Wales, Westmoreland, and Cumberland have experienced a specially heavy mortality.

Mortality at different ages and in two sexes in 1891.

The following table shows the number of deaths in England and Wales attributed to Influenza in 1891 in persons of different ages and sexes :—

Sex.	Ages at Death. All Ages.	0-3 Months.	3-6 Months.	6-12 Months.	Total, —1 Year.	1—	2—	3—	4—	Total under 5 Years.
Males - -	8,621	164	132	257	553	211	76	52	65	957
Females - -	8,065	77	89	142	308	188	77	57	58	688
Both sexes -	16,686	241	221	399	861	399	153	109	123	1,645

Sex.	5—	10—	15—	20—	25—	35—	45—	55—	65—	75—	85—
Males - -	127	108	246	252	668	1,000	1,321	1,471	1,526	832	113
Females -	150	101	203	242	597	729	1,035	1,373	1,721	1,007	219
Both sexes -	277	209	449	494	1,265	1,729	2,356	2,844	3,247	1,839	332

The following gives the proportional mortality from Influenza per 1,000 persons living of each sex at different ages :—

Sex.	All Ages.	Ages. 0-5.	5-10.	10-15.	15-20.	20-25.	25-35.	35-45.	45-55.	55-65.	65-75.	75-85.	85+
Males -	·61	·54	·07	·07	·17	·20	·32	·62	1·11	1·91	3·43	5·71	6·93
Females -	·54	·38	·09	·09	·14	·17	·26	·42	·79	1·54	3·12	5·17	7·91
Both sexes	·57	·46	·08	·08	·15	·19	·29	·52	·94	1·73	3·27	5·40	7·60

Mortality from Influenza in different classes of districts compared.

Some opportunities for a further study of the distribution of Influenza mortality are afforded by annual reports of medical officers of health. Influenza is not one of the diseases required to be entered in the official tables of sickness and mortality, but a good many medical officers of health in compiling their reports have mentioned the number of deaths which have been ascribed to Influenza. More, however, have done so for 1891 than for 1890. From the reports which have come under my notice, in which the figures for both years are given, I have compiled the

following table, the figures for London, as given by the Registrar-General, being also added for comparison:—

District.	Aggregate Population, 1891.	Deaths from Influenza.		Rate per 1,000 Inhabitants.	
		1890.	1891.	1890.	1891.
London - - -	4,211,056	624	2,302	·15	0·55
24 great towns of over 80,000 population.	4,045,528	439	2,417	·11	·59
35 large towns of between 20,000 and 80,000 population.	1,464,113	186	765	·13	·52
21 towns of between 10,000 and 20,000 population.	312,161	46	196	·15	·63
60 small towns of under 10,000 population.	298,121	62	196	·21	·54
85 rural sanitary districts -	1,376,997	317	841	·23	·61

These figures show the much greater mortality ascribed to Influenza in 1891 than in 1890, but, in reference to this, it has to be observed that in some of the provincial towns the deaths from Influenza in 1891 comprise a portion of those which occurred in the epidemic of the winter of 1891–92. In London the mortality from this later epidemic was almost entirely comprised in 1892.

The table also shows that the mortality from Influenza has been somewhat greater proportionally in rural districts than in towns, though this is more fully brought out when a larger series of districts is taken for which figures for 1891 are given, many of which could not be included in the foregoing table on account of the number of deaths from Influenza in 1890 not having been recorded.

1891.

Districts.	Aggregate Population.	Deaths from Influenza in 1891.	
		Number.	Rate per 1,000 Population.
London - - - -	4,211,056	2,302	0·55
26 great towns of over 80,000 inhabitants.	4,661,044	2,697	0·58
64 large towns of between 20,000 and 80,000 inhabitants.	2,475,184	1,277	0·52
63 towns of between 10,000 and 20,000 inhabitants.	899,354	524	0·58
177 small towns of under 10,000 inhabitants.	822,420	524	0·64
192 rural sanitary districts -	2,937,885	2,154	0·73

The following table gives the mortality from Influenza in the great cities and towns of England and Wales, so far as it is contained in reports sent to the Board. From Norwich and Burnley the Board do not receive reports, and the reports of the medical officers of health for West Ham, Preston, and Gateshead do not state the number of deaths ascribed to Influenza :—

Mortality from Influenza in great towns :

Town.	Population, 1891.	Deaths from Influenza.		Rate per 1,000 Population.	
		1890.	1891.	1890.	1891.
Croydon - -	102,697	13	74	·11	·73
Brighton - -	115,402	23	71	·20	·62
Portsmouth -	159,255	15	48	·09	·30
Plymouth - -	84,179	?	44	?	·52
Bristol - -	221,665	54	89	·24	·40
Cardiff - -	128,849	12	78	·09	·61
Swansea - -	90,423	8	20	·09	·22
Wolverhampton -	82,620	2	13	·02	·16
Birmingham -	429,171	Not given.	214	?	·50
Leicester - -	142,051	7	56	·05	·39
Nottingham -	211,984	27	106	·13	·50
Derby - -	94,146	4	75	·04	·80
Birkenhead -	99,184	2	48	·02	·48
Liverpool - -	517,951	8	247	·015	·48
Bolton - -	115,002	17	50	·15	·43
Manchester -	505,343	50	347	·10	·75
Salford - -	198,136	21	100	·10	·50
Oldham - -	131,463	28	157	·21	1·20
Blackburn - -	120,064	0 (?)	72	—	·59
Huddersfield -	95,422	10	111	·11	1·18
Halifax - -	82,864	1	30*	·01	·37
Bradford - -	216,361	14	135	·06	·62
Leeds - -	367,506	19	194	·05	·53
Sheffield - -	324,243	96	399	·30	1·23
Hull - -	199,991	49†	166†	·25	·83
Sunderland -	130,921	0	30	—	·23
Newcastle - -	186,345	Not stated.	66	?	·35

* Primary cases only. † Including cases complicated with inflammation of lungs.

No very clear general conclusions are deducible from this table. The mortality from Influenza was lowest in Wolverhampton, Swansea, and Sunderland, towns not ordinarily distinguished for a specially low death-rate. It was highest in Sheffield, Oldham, and Huddersfield, and was high in several other towns in Yorkshire and the adjoining counties, but in those further north it was low. A high mortality from Influenza was observed in the usually healthy towns of Croydon, Brighton, and Derby, and, as already mentioned, a low one in certain towns not accounted amongst the most healthy ; but no general rule seems to hold good, for Oldham, which has a high general death-rate, was in each year among those towns most severely affected with Influenza.

In some instances neighbouring and otherwise comparable towns experienced a similar mortality from Influenza, *e.g.*, Liverpool and Birkenhead ; Salford and Bolton ; Leeds and Bradford ; but in other instances this was not so. Cardiff* had a much higher Influenza mor-

* In Newport (Mon.) the Influenza death-rate was still higher than in Cardiff, viz., ·91 per 1,000 inhabitants.

tality than Swansea; Derby than Leicester; Birmingham than Wolverhampton, and Huddersfield than Halifax. These variations may probably partly depend upon differences in certification, or in classification of cases in which Influenza complicated or was complicated other diseases, as the weekly death-rates in a table on pp. 18 and 19 exhibit no differences on a similar scale between neighbouring towns in the general mortality during the epidemic period. Thus, in Halifax only 30 deaths in 1891 are attributed to Influenza, and in Huddersfield 111, or, in proportion to the population, more than three times as many, but the death-rate from all causes in the five weeks ending May 30th, 1891, the period of the epidemic averaged 37·1 in Halifax and 40·0 in Huddersfield. The 30 deaths in Halifax are stated to include only those primarily attributed to Influenza; there being besides at the same time a very large mortality from respiratory diseases, which was probably attributable to the effect of the epidemic, as the season was not an unusually cold one. In Hull the deaths in 1891 include 41 from Influenza and 125 from Influenza accompanied by diseases of the respiratory organs.

in Sheffield;

The experience of Sheffield in the first epidemic of 1891 is noteworthy, not only because of all the great towns, it exhibited the largest proportional mortality from Influenza, and the highest mortality from all causes during the epidemic period—the deaths during the week ending May 2nd, 1891, being at the rate of 73·4 per 1,000 inhabitants per annum—but also because, contrary to the experience of London, the course of epidemic Influenza in Sheffield was lingering and fluctuating in 1890, and short and abrupt, both in its rise and fall, in 1891.* In reference to this Dr. Harvey Littlejohn, medical officer of health for Sheffield, makes the following remarks in his Annual Report for 1891:—

"In 1890 we have the first death from Influenza reported during the week ending February 8th, after which deaths are registered from the disease during each consecutive week up till June 20th; but in only two weeks during this period do the number of deaths exceed 10. The deaths from bronchitis and pneumonia, however, during the greater part of the time were abnormally high, and if we keep in mind the fact that Influenza at this time was not so well known and recognised as it is now through greater experience, it may reasonably be supposed that some at least of the deaths from these two diseases may be more truly debited to Influenza. "Even supposing this to be so, the mildness of the invasion, compared with that of the following year, and yet its great effect on the general death-rate for the year, its protracted and lingering presence, are remarkable facts, and difficult to explain satisfactorily when compared with the characteristics of the more recent invasion.

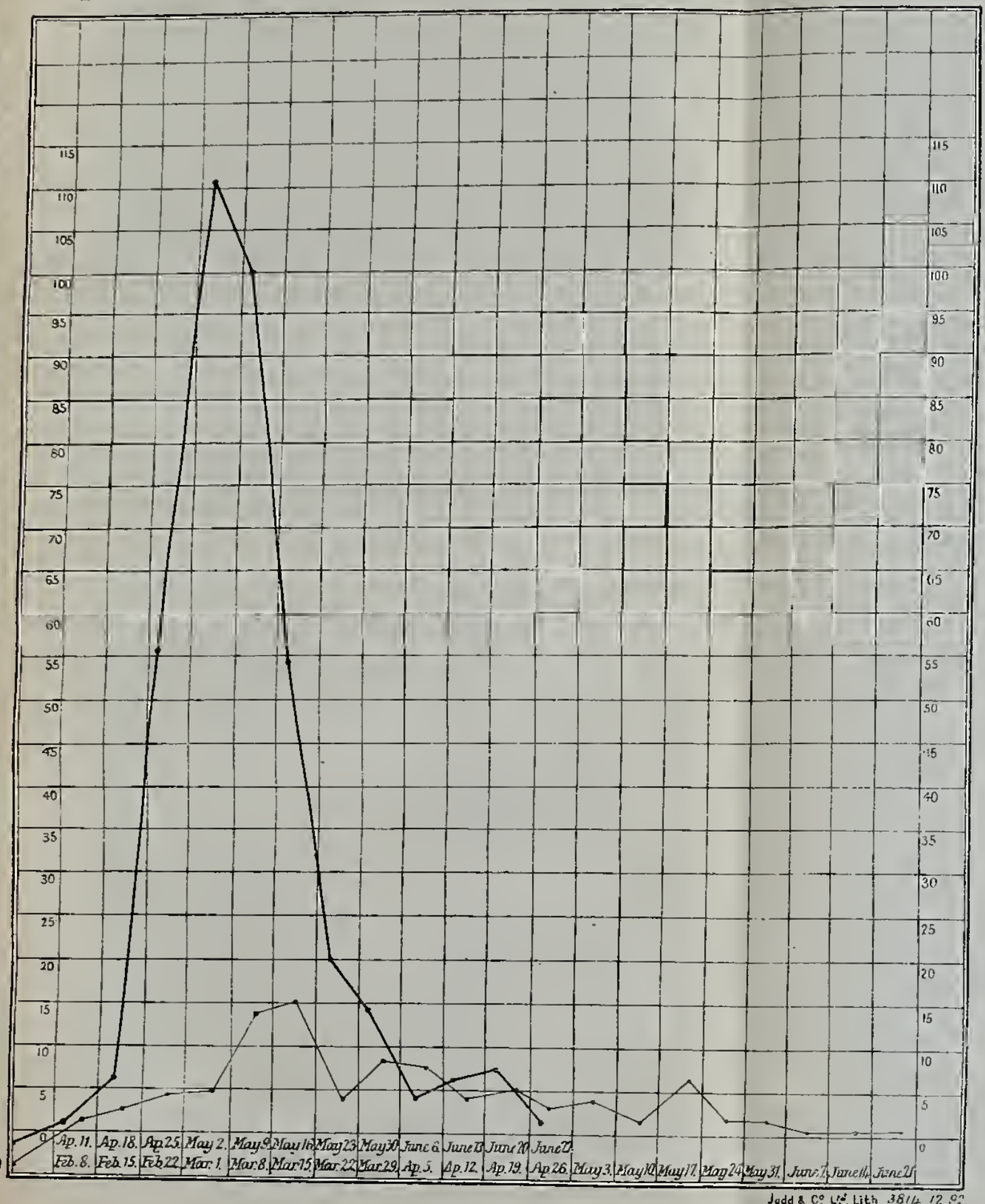
The epidemic of 1891 presented, on the other hand, totally different features. It came on suddenly; spread with great rapidity over the whole borough; and affected large numbers simultaneously. It attained a maximum in four weeks, and in another month had practically disappeared, leaving behind it a death-roll of 399, as compared with only 96 in 1890. A glance at the following chart represents vividly the absence of any premonitory warning and the suddenness of onset, the acuteness of attack and equally rapid decline as the wave of infection passed onwards. There is no relapse or recrudescence of the epidemic.

* A similar contrast between the behaviour of Influenza in 1890 and in the spring of 1891 is noticed by the medical officer of health for Wath-on-Dearne, in a neighbouring part of Yorkshire. In that district a very widespread and severe epidemic began suddenly over the whole district between April 11th and 13th, 1891; it attained its maximum in 10 days, and continued high for 20 days longer, after which it rapidly declined, after having attacked, as estimated, nearly a fourth of the inhabitants.

To face page 28.

CHART

Showing weekly numbers of deaths from Influenza in Sheffield during its prevalence in 1890 and 1891.



Judd & Co Ltd, Lith 3814 12.90

it will be noticed; the general death-rate remains low throughout the rest of the year, and the mortality, both from respiratory and zymotic diseases, is considerably less than usual."

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

Of English towns Brighton seems to have experienced the highest mortality in the Influenza epidemic of 1891–2, the death-rate which was 18·0 in the week ending December 26th, 1891, having risen to 60·9 in that ending January 23rd, 1892. Dr. Newsholme, medical officer of health, attributes the excessive mortality in Brighton, which began in the week ending January 2nd, 1892, to the large inrush of visitors for Christmas, and the sickness which they brought with them. In reply to a suggestion that the apparently large death-rate in January might be due to the deaths being those in a temporarily increased population, he tells me that there are generally only a small number of visitors in January, but it is likely that many who had come down for Christmas would be unable to return on account of attacks of Influenza. He attributes the large mortality to the fact that Brighton receives a large number of convalescents from Influenza who form centres of infection. It also contains many convalescents from other diseases, many chronic invalids, especially phthisical persons, and others with weak chests, and many aged persons—classes of people among whom Influenza is notoriously most fatal. It is to be remarked, however, that Brighton suffered comparatively lightly in the two former epidemics, although the same circumstances would then be equally in operation, and it is possible that the severe character of the later epidemic at Brighton may be partly due to the small degree of protection left by the previous epidemics.

in Brighton.

Districts with high Influenza mortality in 1891.

In the following districts the mortality ascribed to Influenza exceeded one death per 1,000 inhabitants, some specially high rates being noted :—

Urban Districts.

East Grinstead.
Waltham Holy Cross.
Eton.
Woodford.
Downham Market.
Wiveliscomb.
Taunton (2·3).
Wellington (Somerset).
Kington.
Sutton Coldfield.
Coventry.
Ashby Woulds.
Market Rasen.
Boston.
Louth.
Whittington (Derby) (2·5).
Brampton and Walton.
Alvaston and Boulton.
Barmouth.
Neston and Parkgate.
Oldham.
Haslingden.
Withington.
Haydock.
Pontefract.
Greetland (3·05).
Todmorden.
Sheffield.
Huddersfield.
Grasmere.
Cleator Moor.
Egremont.
Abertillery.
Monmouth
Pontypridd.
Llangefni (3·7).
St. Neots.
Tenby.
Kings Lynn.
Burnham (Somerset).
Droitwich.
Grantham.
Stamford.
Dronfield.
Bollington.
Garton.
Rotherham.
Thornton.
Denby.
Pickering (3·0).
Bedwellty.
Pontypool.
Bridgend.

Rural Districts.

Kingston-on-Thames.	Chesterfield.
Farnham.	Barton-on-Irwell.
Newbury.	Clitheroe.
Basingstoke.	Scarborough.
Whitchurch, (Hants.)	Kirby Moorside.
Stockbridge.	Easingwold.
Newport Pagnell.	Helmsley (3·7).
Hardingstone.	East Ward.
Malmesbury.	Bootle (Cumberland).
Marlborough.	Monmouth.
Launceston.	Machynlleth (3·2).
Seisdon.	Anglesey.
Stone.	Rye.
Melton Mowbray.	Kettering.
Billesdon.	Potterspury.
Uppingham (2·1).	Brixworth (2·3).
Stamford.	Rugby (2·7).
Grantham.	Huntingdon.
Sleaford.	Woburn.
Boston.	Kington (2·2).
Spilsby.	York (2·3).
Lincoln.	Bedale.
Brigg.	Kendal.
Bingham.	Whitehaven.
Southwell.	Crickhowell (2·6).

Districts with no direct mortality from Influenza in 1891.

In the following districts there appears to have been no mortality directly attributed to Influenza in 1891 :—

Urban.

Folkestone.	Bridport.
Lydd.	Ivybridge.
Romsey.	Dawlish.
Whittlesey.	Teignmouth.
Stowmarket.	Malvern Link.
Aldeburgh.	Felling.
Wells, Norfolk.	Wigton.
Marlborough.	Blackrod.
Tenterden.	Long Sutton.
Sheerness.	Bonsall.
Wokingham.	Swadlincote.
Calne.	Meltham.
Weymouth.	Rishworth.
L. Brixham.	Filey.
Paignton.	Briton Ferry.
South Molton.	Llandovery.
Madron.	Llanfyllin.

Rural.

Thingoe.	Mere.
Bideford.	Wootton Bassett.
Worcester.	Calne.
Beverley.	Bridport.
Alston.	Bourne.
Hursley.	Wrexham (N. Division).
Romsey.	

In Aldeburgh and Dawlish Influenza is stated to have hardly amounted to an epidemic either in 1890 or 1891.* Generally speaking the rural districts and small towns seem to have suffered most heavily, especially in the North Midland counties. There are some curious and inexplicable differences, however, between neighbouring districts. Thus, in Wiltshire there appear to have been no deaths attributed to Influenza in the Wootton Bassett and Calne rural districts, while in the Malmesbury and Marlborough rural districts on either side of these a high mortality was attributed to it. Similarly also in Lincolnshire it is stated that no deaths from Influenza occurred in the Bourne rural district, while in the neighbouring and similarly situated rural sanitary districts of Stamford, Grantham, and Sleaford there was a very high mortality from Influenza.

It seems probable, however, that the varying mortality of Influenza in different districts may be explained to some extent by the different proportions of inhabitants at different ages of life, being low where there is a large proportion of children, as in an increasing artizan population, and high in a community which, like a residential well-to-do neighbourhood or a decreasing agricultural population, contains a large proportion of adults at the middle and later periods of life, and a small proportion of children.

This question, however, has already been considered in the section which contains a statistical study of the mortality from Influenza in the epidemic of 1889–90, as recorded in the Registrar-General's annual report for 1890.

Local figures of prevalence of Influenza in epidemics of 1891-2.

The following are some figures given by medical officers of health as to the number of cases in their respective districts, from which some idea may be gathered of the extent of prevalence of the disease. A wide difference is to be noted between the estimated proportion of the inhabitants attacked in some districts and the proportion of the known attacks to the population in other districts. Probably in the former districts such estimates are liable to overstatement, while in the latter districts the proportion is understated owing to many of the cases, especially the mild ones which do not come under medical treatment, not being included among those of which the medical officer has know ledge.

Scarborough Rural District. Population 9,792.—Influenza epidemic in April and May 1891. The medical officer of health obtained from medical men information of 1,602 cases coming under treatment, and there were 11 deaths.

Attack-rate = 164 per 1,000 population.
Death-rate = 1·1 „ „
Case mortality = 6·9 per 100 cases.

Hull. Population 199,991.—Influenza epidemic in March, April, and May 1891. 60,000 estimated cases, or 300 per 1,000 inhabitants. Of men at large engineering works one fifth were attacked, and 24 out of 279 police, or 86 per 1,000. The deaths due to Influenza, directly or through complications affecting the respiratory organs, were 166 = ·83 per 1,000 inhabitants, or ·28 per 100 cases. The epidemic is estimated to have cost the Friendly Societies in Hull 4,000*l.*

South Shields Rural District. Population 12,682.—Influenza epidemic from March 1891 to the end of the year, especially in August. The medical officer of health reports 771 cases (261 under five years

* Dawlish is said also to have escaped an epidemic in the winter of 1891–2, though a visitor died there of Influenza not acquired locally, and there was some other cases of a mild kind.

old, 510 above)=61 per 1,000 inhabitants. Deaths 4=0·3 per 1,000 inhabitants, or 5·2 per cent. of cases. In 1890 130 cases were reported.

Yeadon. Population 7,396.—Influenza epidemic began in April 1891. The medical officer thinks that probably three-fourths of the inhabitants were attacked = 5,547 cases. Only two deaths were directly attributed to Influenza (= 0·4 per cent. of the estimated cases), but there was a high mortality from lung diseases not directly ascribed to Influenza.

Kirkby Moorside Rural District. Population 5,093.—Influenza epidemic in April and May 1891. The medical officer of health estimates that half the population was attacked=2,546 cases. There were 9 deaths=1·8 per 1,000 inhabitants, or 3·5 per cent. of estimated cases.

Newport Pagnell Rural District. Population 25,613.—The medical officer of health estimates that one-fourth of the inhabitants were attacked by Influenza in 1891. 30 deaths were ascribed to it (=0·47 per cent. estimated cases), but many more were probably due to it.

Thornton, Yorks. Population 5,680.—More than 1,000 cases of Influenza in March to June 1891, and 11 deaths. These figures give—

Attack-rate about 176 per 1,000 inhabitants.
Death-rate 1·9 „ „
Case mortality about 1·1 per cent.

Willenhall. Population 16,851.—The medical officer of health reports 421 cases of Influenza in 1891, viz., 223 in second quarter, 58 in third quarter, and 140 in fourth quarter=25 per 1,000 inhabitants. Deaths 9=·54 per 1,000 inhabitants, or 2·1 per 100 reported cases.

Barton-on-Humber. Population 8,140.—Influenza epidemic in March, April, and May 1891. One fifth of inhabitants estimated to have been attacked. 7 deaths=·86 per 1,000 inhabitants, or 4·3 per cent. of estimated cases.

Northam, Devon. Population 5,031.—About 100 cases of Influenza =40 per 1,000 of the inhabitants, and 3 deaths.

Market Rasen. Population 2,947.—Epidemic in April and May 1891. About 500 cases of Influenza=170 per 1,000 inhabitants. 6 deaths= 2·05 per 1,000, or 1·2 per cent. of cases.

Sleaford Rural District. Population 17,916.—Influenza epidemic in April, May, and June. 75 per cent. of the inhabitants in some villages said to have been attacked. 33 deaths.

Belper Rural District. First Division. Population 11,132.—368 cases in first epidemic of 1891=33 per 1,000. Apparently no death.

Pudsey. Population 13,444.—Three-fourths of inhabitants estimated to have been attacked in the first epidemic of 1891. Deaths 5=·37 per 1,000 inhabitants, or 0·5 per cent. of estimated cases.

Weardale Rural District. Wolsingham Division. Population 3,169. —There were two outbreaks of Influenza in 1891, the first in July, about 50 or 60 cases; the second, more severe, began October 18th, and lasted to the end of December; about 700 cases. The proportion of inhabitants attacked was about 16 per 1,000 in the first epidemic and 220 in the second.

Truro. Population 11,131.—Influenza was prevalent in the spring of 1891, but much more so in October, November, and December, when a very large per-centage of inhabitants suffered. Of 600 Oddfellows and Foresters in Truro, 64 were on the sick list through Influenza between October 23rd and December 21st, 1891=nearly 11 per cent. 3 deaths.

Bury St. Edmunds. Population 16,630.—Half the inhabitants are estimated to have suffered in December 1891 and January 1892. 3 deaths.

Stockbridge Rural District.—The medical officer of health gives for certain villages the following figures of cases and deaths from Influenza in 1891 :—

Broughton (population 842).—43 cases, 1 death.
East and West Tytherley (population 901).—32 cases, 1 death.
Over and Lower Wallop (population 1,286).—220 cases, 4 deaths.
Total, 295 cases and 6 deaths in a population of 3,029.
Attack-rate, 98 per 1,000 inhabitants.
Death-rate, 2 „ „
Case mortality, 2 per cent.

Wilton. Population 9,894.—A few cases of Influenza in February, March, and April 1891, extremely prevalent from May to July, and again in December. 195 cases came under the medical officer of health's observation, and there were 3 deaths.

In 1890, 203 cases came under the medical officer of health's observation in January, February, and March, and there was 1 death.

Panteg. Population 5,763.—Influenza broke out in October 1891, almost every house being affected. The medical officer of health reports 420 cases and 4 deaths.

Nantyglo and Blaina. Population 12,627.—Influenza was at no time entirely absent during 1891, but the greater number of cases were in October and November. 20 per cent. of the inhabitants are estimated to have been attacked and 11 deaths were ascribed to it. In 1890 also 20 per cent. of the inhabitants are estimated to have been attacked in the first two months of the year, but there were only 2 deaths.

Part III.—On the History of Influenza Abroad in the years 1891 and 1892.

Influenza abroad in interval between epidemics in England and Wales.

In Part II. of my former report I have traced the recent history of epidemic Influenza in different parts of the globe up to the end of 1890. The epidemic of 1889–90 seems to have spent itself in Europe by the end of March, with the exception of Iceland, which was attacked in June and July. In March, April, and May India, New Zealand, and Australia were attacked, and in the latter half of 1890 local epidemics were recorded in remote parts of Africa* and Asia, as St. Helena, Mauritius, Abyssinia, Yunnan, and Kashmir.

There seems also to have been a smouldering on of the disease both in this country and elsewhere with some local outbreaks, as at Killarney in Ireland, in Wurtemburg and Australia.

Recurrence in America in March 1891.

The earliest intimation of the recurrence of Influenza abroad in 1891, came from the United States, where it appeared early in March, *i.e.*, nearly at the same time as at Hull—in Chicago, Pittsburg, Cleveland, and other towns in Ohio, and Iowa, and spread thence eastwards, reaching New York about the end of March and becoming epidemic in

---

* A paper by Dr. Bowie in the "Lancet" of July 11th, 1891, gives an interesting account of the Influenza epidemic at Blantyre in the Shiré Highlands, Central Africa, the introduction of which is distinctly traced to personal communication.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

the beginning of April. In the middle of April it was reported to be very prevalent among the Indian tribes in Washington territory in the north-west of the United States, and about the same time at Vancouver's Island, and on May 5th to be epidemic at Mexico. On April 25th it is reported to be declining in the United States.

This epidemic, *i.e.*, the second of recent years, though very severe in the United States and in the north of England, seems to have spared the continent of Europe to a great extent.

In Scandinavia.

In April Influenza was stated to be epidemic in Christiania, and at Gothenberg, and in May 23rd to be abating in Christiania, but spreading in Copenhagen. It was still present to some extent in the latter city in August.

But Continental Europe mostly escaped Influenza in spring and summer of 1891.

On April 24th Influenza was reported to be spreading in the towns of Southern Russia, and on May 15th in Russian Poland. It is reported as epidemic towards the end of April in Alsace and the Rhine district, and at Hamburg on June 3rd. The death-rate in Berlin and the other large cities of the German Empire does not, however, according to a return issued on January 14th, 1892, by the Imperial Health Office, show any increase in the general death-rate at a time corresponding to this epidemic. I learn also from Mr. A. H. Smee that no recurrence was observed in Austria, Hungary, or Bavaria.

France seems also to have escaped any epidemic at this time, though a few cases of "influenza" in Paris are mentioned in newspapers of May 16th, in connexion with the inclement weather at that time.

Cases of Influenza of a severe type are reported in April to have occurred in the interior of Portugal.

At Gibraltar Influenza was introduced in May by a warship from England, and appears to have spread to some extent.

On May 19th Influenza was announced to have broken out in Cairo, and to be especially prevalent among the natives.

The second epidemic was experienced in Scotland and Ireland, but not to any great extent. Edinburgh, Glasgow, Aberdeen, and Dundee were all affected; in Edinburgh 19 deaths being attributed to Influenza in May and June. In Dundee the disease is stated to have been present continually from the second week in July to the beginning of October, when it commenced to spread rapidly.

During August and September 1891 local epidemics are reported in France, in the north of Portugal, and in certain provinces in the south and west of Spain.

Epidemic in Australia in September and October 1891.

At this time—*i.e.*, during the interval between the second and third epidemics in this country—a severe epidemic of Influenza occurred in Australia. It is reported to have been very severe in Melbourne in the beginning of September, and to have spread from thence to the up country parts of Victoria. In New South Wales this epidemic formed the subject of a valuable report by Dr. Ashburton Thompson, chief medical inspector to the Board of Health; this report, like his previous one on the epidemic of 1890, being based on the answers to a collective inquiry from medical men practising in the colony. As a result of the first inquiry Dr. Thompson had reached the conclusion that the spread of Influenza, instead of being the sudden invasion described by earlier writers, took place by slow degrees, both in the several towns and over the country as a whole, and was in every respect accordant with what was known to be the habit of the communicable diseases. This conclusion, however, through a caution which Dr. Thompson considered to be reasonable at the time of writing, but which afterwards appeared to him to have been superfluous, was re-stated by him in the interrogative form, as follows:—"Is not human intercourse necessary to transport the

"contagion of the disease? Is not some stage intermediate between "man and man necessary to its spread in epidemic form? Is the con- "dition, which for convenience has been called aërial, of more con- "sequence in production of epidemics of Influenza than it is in produc- "tion of other diseases which at times do, and at others do not, "assume an epidemic form?" As a result of his later inquiries, Dr. Thompson considers that the first of these questions can be answered unhesitatingly in the affirmative, and the third in the negative. As to the second, there appears to him good reason to think that it was ill-founded, and that Influenza spreads only by direct communication from the sick to the healthy, and that the secretions of the mouth and lungs furnish the means of such communication.

The epidemic of 1890 appears to have ceased soon after the end of May in Sydney, and in the country districts at a later date, between June and the end of the year. During the inter-epidemic period there appears to have been a scattered succession of cases, more numerous during the six or seven months after the end of the first epidemic and during the three months before the commencement of the second epidemic, than in the middle of the interval. This suggests a continuous smouldering of the disease and that the second epidemic was due to a recrudescence and not to a reimportation. In this connexion it is noticed that whereas in 1890 the capital was attacked before the country districts, in 1891 some places in the country began to suffer before the capital, but when the capital had been invaded the extension to other parts of the country soon followed. [The experience of this country has been similar in the two last epidemics, both of which appear to have commenced in distant parts of the country, and to have spread thence comparatively slowly to London; but London having been invaded, the disease soon became general throughout the country.] In New South Wales the date of the commencement of the second epidemic was by a few observers placed before June and July, but by the majority placed between the end of August and the middle of October in Sydney, and about a fortnight later in country places. As in this country, the second visitation was more serious than the first. The proportion of the population who were medically attended on account of Influenza is roughly estimated at one eleventh; but the total proportion who suffered was no doubt larger. Environment seemed to have little influence on the spread of the disease, the most important factor being exposure to the sick at close quarters. Little information was obtained as to spread by fomites. As regards protection by former attack, it is concluded that if Influenza protect at all against itself, it does so uncertainly for the one part and temporarily for the other.

New Zealand was visited by the epidemic about October 1891, after the chief cities of Australia. ("Lancet," December 12th, 1891.)

From Sydney the disease seems to have been carried to the Samoan Islands, where an epidemic was just over on January 4th, 1892. ("British Medical Journal," February 13th, 1892.)

Revival in October 1891 in British Isles;

October 1891 witnessed a revival of Influenza in an epidemic form in various parts of Europe. In the British Islands recrudescences commenced in the early part of the month in Scotland (Dundee), and in the north of Ireland (Londonderry), and somewhat later towards the end of October in the west of Cornwall, and in Kent. London, however, was not seriously attacked until after Christmas. This epidemic appears to have been severe in Scotland and the extreme north of England (Northumberland and Durham), and especially so in the south and west of England and South Wales, but Yorkshire, the Midlands, and North Wales, which suffered most severely in the earlier epidemic of 1891,

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

escaped lightly. In Scotland, and the places in Cornwall and South Wales first attacked, the epidemic was declining at the end of 1891, and in England generally it subsided in the course of February.

in France,

In France, which had escaped the earlier epidemic of 1891, but where local outbreaks had occurred in the provinces earlier in the autumn, Influenza became epidemic in October. In Paris it is said to have broken out in the week ending October 14th, but the mortality did not begin to rise until the middle of December, and did not attain its maximum until the third week in January 1892. Influenza was epidemic in October and November, in the departments of Charente and Dordogne, in the S.W. of France; it was reported at Havre on December 5th, and by January was general over France, being especially severe in the northern departments on the borders of Belgium. The latter country and Holland also suffered from the epidemic in December and January. The highest death-rate was reached in Brussels in the second and in Amsterdam in the fourth week of January.

Low Countries and Peninsula.

In Spain and Portugal also local epidemics seem to have developed into a general one. Influenza is reported as epidemic in the northern parts of Portugal in August and September 1891, and there were then a good many cases in Lisbon. On October 1st it was prevalent at Coimbra, and in the end of November was general in Portugal, being especially severe at Thomar and Evora, in the centre and south of that kingdom. At Lisbon it was less severe than in 1890, and was on the decline at the beginning of January. In November it had reached the Azores. In September 1891 Influenza was epidemic in the provinces of Caceres, Jaen, and Cordova in the south and west of Spain. In the beginning of January it is reported as epidemic alike at Santander in the north, at Granada in the south, and at Barcelona in the east of Spain, and on February 8th as increasing at Madrid.

At Gibraltar Influenza had appeared on December 14th among troops just arrived from England, but an epidemic does not seem to have immediately followed. The disease is reported as prevalent there on February 7th.

Revival also in Russia and bordering countries in autumn of 1891.

Another focus of renewed activity of Influenza in the autumn of 1891 was in Eastern Europe, in Russia and the countries bordering on it. It will be remembered that of European countries Russia was the first to be attacked in 1889, and in the earlier part of 1891 Influenza also prevailed there to some extent. In St. Petersburg the number of cases of Influenza reported in August was 23, in September 190, and in the first fortnight of October 213. ("Laneet," November 14th.)

It is reported in newspapers of October 23rd that Influenza had reappeared in Austria, having begun in Galicia, into which province it had been imported from Russia and Russian Poland, and where it had been extensively prevalent in Lemberg and other towns. It had also reached Vienna, where the first cases had occurred about three weeks before.

On October 24th it was announced that an epidemic of Influenza of a malignant nature had broken out at Münsterberg, and on October 29th, in the town and district of Neisse, in Silesia; it spread rapidly in this province during November, and also in the provinces of Posen and West Prussia, all of these being frontier provinces bordering on Russian Poland.

In Roumania Influenza, which had been raging for weeks in Moldavia, the province nearest Russia, was announced on October 31st to have reached Bucharest.

In the middle of November Influenza had become epidemic in St. Petersburg, and in the large towns and famine-stricken districts of Southern Russia, and continued so during December.

We hear of it in January in Greece, where, however, it was stated to have been of mild type, and at Constantinople, where it was declining on February 1st.

On February 3rd it was epidemic at Sophia (Bulgaria), where it had existed in a sporadic form for two months.

Epidemic in S.E. Europe;

At Vienna, where cases of Influenza were observed early in October, the epidemic seems to have run a comparatively slow course, increasing through November, being developed in December and January, and declining at the end of the latter month. The death-rate reached its highest point in the first and second weeks in January, and in Buda-Pesth in the last week of December. In Germany the epidemic spread westward from the Russian frontier, where, as we have seen, it had begun in October. It was present early in the month at Lübeck and Rostock, and towards the end in Hamburg and Schleswig-Holstein. In Berlin the earliest cases were noticed in the beginning of November, and the disease was on the increase during that month, the highest death-rate being attained in the first week of December. By the end of January Influenza had subsided in Berlin, but was still prevalent in the provinces of Germany.

in Austria and Germany;

In Copenhagen, where Influenza had been epidemic in May, a few cases were still occurring in August, and in December it again became epidemic, the highest death-rate being reached in the last week of that month. On December 14th it was spreading in the country districts of Denmark. It had previously, in November, been prevalent in Schleswig-Holstein.

in Scandinavian kingdoms.

Stockholm and Gothenburg in Swedeu were attacked in December, but Christiania and Bergen in Norway not until late in January.

In Italy, according to newspapers, cases of Influenza were observed in Genoa by December 10th, and the epidemic reached its height there in the third week of December. By the end of December it was epidemic in Milan, Turin, Florence, Bergamo, and other parts of Italy.

in Italy;

The official "Bollettino Sanitario" gives the number of cases of Influenza notified throughout Italy as follows:—

1891, January to October - - -	0
,, November - - - -	30
,, December - - - -	6,461
1892, January - - - -	84,543
,, February - - - -	55,352
,, March - - - -	28,046
,, April - - - - -	7,962
,, May - - - - -	1,468
,, June - - - -	223

It is not clear whether the notification of Influenza is compulsory in Italy, or with what degree of completeness it is carried out. The numbers given of the cases of Influenza are made up of figures, often round numbers, for certain districts, while for many other districts none are given. From a report issued by the German Imperial Health Office it appears that of all the large European cities Venice suffered the most from this epidemic of 1891–2, the death-rate having been as high as 93·3 in the second week of January 1892; but according to the "Bollettino Sanitario" only 254 cases of Influenza in that city were notified in December 1891, and none in January 1892.

On the whole the epidemic would appear to have been more general in northern than in southern Italy.

It was said to be showing signs of recrudescence at Milan in April 1892.

At Malta the crews of warships were attacked with Influenza in January.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

In North America;

Out of Europe, Influenza is recorded in December at Cuba and the United States, where it was epidemic in the middle of the month at Denver, St. Louis, and Philadelphia, and a few cases had occurred in New York. In the beginning of January it was epidemic in New York, Boston, Chicago, and many other cities in the United States; and in British North America at Montreal, Ottawa, and other places, from St. John's in New Brunswick to Vancouver Island.

in West Indies.

Dr. Law, medical officer to Her Majesty's penal settlement, Massaruni, British Guiana, states ("British Guiana Medical Annual," 1892), that that colony was visited by Influenza for the first time in the middle of 1890, being one of the last countries in the world in which it made its appearance. At the penal settlement the first case came under notice on August 16th, 1890, and from that date to November 14th, 21 cases in all were treated. From that date to January 20th, 1891, there were no fresh cases. In the end of January 3 cases occurred, in February 2, in March 2, and in April 3. In May there were none, but towards the end of June 2 cases occurred, and in July and August 8 and 6 cases respectively. September, October, November, and the first half of December were free. On December 19th, 1891, a warder developed symptoms of Influenza a few days after his return from George Town. Five days later a boy living close to him was attacked, and the disease then spread over the whole settlement, attacking alike convicts, warders, officers, and their families. From December 19th to the end of the month there were 7 cases, in January 1892, 138, in February 21, and in March, up to the middle of the month, 8. Only 3 cases had been previously attacked. There was no death. The disease was of the cerebro-spinal type, and appeared to be modified by malarial influence, the feverishness being in many cases markedly intermittent, with a tendency to congestion of the liver and spleen. With few exceptions there was a marked absence of catarrhal symptoms and of pulmonary complications. These differences from Influenza as observed in Europe Dr. Law attributes to the difference of climate.

In the island of St. Kitt's, West Indies, Influenza was introduced on December 14th, 1891, by a gentleman and his wife who were suffering from Influenza, and had left Southampton a fortnight before. Within a week or two the majority of the friends who had been to visit them contracted the disease, and afterwards it spread rapidly in the town of Basseterre (of which one third of the inhabitants were estimated to have suffered), and later to the country villages near the town.

At St. Martin's and Crab Island Influenza is said to have been brought by a gentleman from Paris, who had suffered on board ship.—(A. P. Boon, F.R.C.S., in "British Medical Journal," March 26th, 1892.)

In January 1892 Influenza was epidemic at Tunis, Lower Egypt, Cape Town, Teheran, and Ispahan, Tonquin, and China, in February in Upper Egypt, and in March in the Soudan.

later outbreaks.

In August 1892 Influenza was reported to be epidemic in Peru.

In December 1892 and January 1893 a certain degree of prevalence of Influenza is reported to have occurred in some of the large towns of Germany and Russia, and in Ghent Madrid and New Orleans and as already mentioned there have been local recrudescences in this country.

Comparison of later history of Influenza with conclusions drawn in former report.

The later history of Influenza confirms on the whole the conclusions which on pp. 51–53 of my former report were drawn from the history of the epidemic of 1889–90, viz., that the spread of the disease has followed the lines of human intercourse; that it has not travelled faster than human beings could travel, and that it has not commenced by a large

number of persons being simultaneously attacked in a place heretofore free, but that a succession of scattered cases has preceded and led up to the epidemic.

In the later history of the disease no definite general direction of progress from east to west has been noticeable. This course indeed seems to have been followed by the epidemic which spread from Russia to Germany in the later months of 1891, but in the recurrences in this country, in Western Europe, and in the United States, the direction of progress of the epidemic, so far as it can be stated in general terms, appears to have been sometimes from west to east, and sometimes from north to south. The later history of Influenza in fact points, 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. It would appear that the contagium of the disease, scattered broadcast in the first epidemic, retained its vitality, but in a suspended or inconspicuous form—perhaps by transmission from one human being to another in a succession of mild sporadic cases, perhaps in some medium external to the human body—and that under the influence of some circumstances of a widely diffused character it awoke to renewed life and vigour. What these circumstances are can only be matter of conjecture. As shown in my previous report, Influenza may prevail as an epidemic in any place, irrespective of season, climate, or weather, and our later experience confirms this so far as invasion by an epidemic already developed elsewhere is concerned. But the recrudescence of the disease in epidemic form in a country, as distinguished from its introduction ab extra, has appeared to take place chiefly at one or other of two seasons of the year, viz., early spring and autumn. Thus, in 1891, it began to be epidemic in March in Yorkshire, Wales, and the United States, and in September (the corresponding season in the southern hemisphere) in Australia. In autumn, again, about October, it became epidemic in Scotland, Cornwall, France, and Russia, developing apparently independently in these different centres, and spreading thence to other districts. (It may be noted in this connexion that, as has been already mentioned, there was a tendency to increase in the number of deaths ascribed to "influenza" in London in November 1890.)

Apparent independent revival of activity at about the same time in distant quarters of the world;

generally in spring or autumn.

The conclusion that the progress of an Influenza epidemic from place to place and its development in a given place are gradual, and not, as was formerly asserted, instantaneous, or so nearly so as to be incompatible with propagation from person to person, has been arrived at by observers who have studied the recent epidemics in various parts of the world. So far as I am aware, the only evidence in favour of the older view comes from Canada in reply to the circular from the Colonial Office mentioned on page 14 of my former report. This reply had not been received when that report was published. The replies from several provinces of that Dominion express the opinion that its appearance in 1889–90 over the Dominion was too simultaneous, and the multiplication of cases too rapid to be explicable by personal communication, and it is accordingly held to be propagated by atmospheric agency. Nevertheless it is by most reporters allowed to be also communicable from person to person, and on examining the reports more closely it is found that the occurrence of the epidemic was not really so simultaneous as is broadly stated, and that circumstances are recorded which point to personal communication being after all the agent by which it was spread.

Epidemic of 1889–90 in Canada asserted in have been too sudden and simultaneous to have been spread by human agency;

In Prince Edward Island it is stated by Dr. Johnson, health officer for the city of Charlotte Town, that two epidemics of Influenza occurred in successive years. In the first of these the first cases were

previous epidemic in 1889–9 in Prince Edward Island,

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

observed in December 1888, and the epidemic prevalence lasted from January to April 1889. According to a writer in the "Maritime Medical News" of May 1889, an epidemic of pneumonia, complicating generally, in the case of young children and of the aged, a severe epidemic of Influenza was very prevalent in Prince Edward Island during February and March 1889, and was at the time of writing (April 13th, 1889) still in full career in many districts. 546 cases of such pneumonia with 41 deaths are recorded as having occurred in the practice of 26 medical men, not half the number practising on the island, the population of which is about 130,000. One medical man says, "The pneu- "monias were here as elsewhere an accompaniment to an acute catarrhal "fever or Influenza, which is now on the decline apparently for lack "of new material, as not a child in the whole city seems to have "escaped." An epidemic of whooping-cough occurred at the same time. This outbreak is the earliest in recent years of which I have information, preceding those in May 1889, in Athabasca, Greenland, and Bokhara, mentioned in my first report. Dr. Johnson states that, so far as known to him, no similar epidemic existed at the same time in the other maritime provinces.

Epidemic of 1889-90 in Prince Edward Island;

Of the second epidemic, the date of the earliest cases is given as December 1889, and of epidemic prevalence January to April 1890. Dr. Johnson considers himself in a position to state:—

1. That the occurrence of the disease was practically simultaneous in all parts of the island.
2. Its incidence on particular localities was scarcely known before its epidemic prevalence in the same localities was also discovered.
3. It was not unusual for only one member of a household to be attacked.
4. Its invasion of the island was practically simultaneous with its invasion of the nearer Continental provinces.
5. It occurred at the season when the ground was generally sealed and covered with frost and snow.

"These facts," says Dr. Johnson, "seem to make it quite improbable "that the causation of the disease in the province was by malaria of "local origin, or that its dissemination was effected solely by contagion "as usually understood. The mass of evidence strongly supports the "opinion that the disease is a contagious fever of a virulent type, and "that its incubation is exceedingly brief.

"Promptly upon the seizure there would seem to be a prolific repro- "duction of the contagion, which is not confined within the ordinary "limits of aerial conduction in order to retain its infectivity, but which "rather appears to be generated or reproduced in such condensed and "virulent form as to admit of large dilution (or if microbic, of further "reproduction) in the air, and of being thus conveyed to great, and as "yet unmeasured, distances with retained activity. Atmospheric "waves thus surcharged with the contagious element, as they reach "new centres, and encompass large areas of population, may cause the "epidemic prevalence of the disease to be actually simultaneous with "its first appearance.

"It is estimated that the disease attacked, with varying degrees of "virulence from 40 to 50 per cent. of the population of this pro- "vince."

The mortality directly attributable to uncomplicated Influenza was however almost nil, and fatal complications during the second epidemic were rare. In this respect it differed from the first epidemic (1888–9), in which the mortality from Influenza, complicated with pneumonia, was about 8 per cent. of the cases thus complicated.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

In Ontario the date of first occurrence of Influenza is given as December 4th, 1889, and of epidemic appearance as about December 20th, its decline occurring about the end of February 1890. According to the secretary of the Provincial Board of Health, "it was in "the exact sense of the term a pandemic, and was infectious." It prevailed very generally in Manitoba and the North-West Territories during a winter severe even for that climate, the mercury being for days together from 15° to 20° below zero F. In Ontario, on the other hand, the temperature was greatly above the average,—in December 1889 as much so as 8° F. above the average,—with prevalence of W. and S.W. winds, excessive rainfall and humidity, and extreme daily range of temperature. "Under these circumstances the epidemic was developed— in Ontario;

"(*a.*) When the resistance of the physical system to disease was lessened, and when the mucous membrane of the respiratory tract congested by the chill damp atmosphere presented a fertile soil for the inhaled germs of the disease.

"(*b.*) When, through the absence of sunlight, house atmospheres became more than ordinarily loaded with fungoid and bacterial impurities.

"(*c.*) When, through the excessively wet weather, the majority of the people were kept housed, and exposed to sewer or other emanations in an unusual degree, and to an extent incompatible with health.

"(*d.*) And when exposure to the exhalations of persons already attacked, in the infected air of houses, factories, schools, churches, &c., notably increased the opportunities for the spread of the disease."

In Quebec, Influenza is stated to have been first observed about November 20th, 1889, and to have become epidemic about the middle of December 1889, declining about the end of January 1890. No information was gathered as to its method of origin or spread, except that it appeared to have extended from the shipping portion to the interior of the country. in Quebec;

In Manitoba, Dr. James Patterson, of Winnipeg, states that the first noted occurrence of Influenza was on December 23rd, 1889, and that it was epidemic from that date to March 1st, 1890, with occasional cases up to May 1st, 1890. He thinks it must have been due to some peculiar condition of the atmosphere, on account of its rapid and almost simultaneous development over nearly the whole of North America. There was no exemption of localities or races, Indians, half-breeds, and white people all suffering alike. in Manitoba.

In the earlier months of 1891 a milder and less wide-spread epidemic prevailed, without the acute characteristic symptoms of the former, but with a more marked tendency to bronchitis and other inflammatory conditions of the pulmonary tissues. Recurrence in 1891;

In New Brunswick, Dr. Currie, secretary to the Provincial Board of Health, writing March 28th, 1891, states that as no record is kept of epidemic Influenza, his answers to the form of queries are necessarily imperfect. He states that epidemic Influenza had been very prevalent throughout the whole province during the past 18 months, beginning about October or November 1889. It gradually spread over the country in an irregular way, became less frequent in the spring (1890), but returned about December, and had continued with greater or less severity in different parts ever since. He believed it to be probably contagious, successive cases occurring in households at intervals of four or five days. in New Brunswick;

In Vancouver Island, according to Dr. Harrington, of Victoria, Influenza was recorded first on December 22nd, 1889, nearly simul- in Vancouver Island;

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

in North-Western Territories.

taneously with telegraphic news of its appearance in Eastern Canada. It prevailed as an epidemic from about January 1st to May 1st, 1890.

From the North-Western Territories, Dr. Jukes, senior surgeon to the North-Western Mounted Police, forwards a series of reports relating to the appearence of epidemic Influenza among that force, and also extracts from monthly reports of the Indian agents, referring to its appearance among the Indian bands located in that territory. He says:—

"The Territory, to various districts of which the enclosed reports apply, every portion of which is under the constant supervision of the North-West Mounted Police posts, outposts, and patrols, covers an expanse of territory extending from the western boundary of Manitoba in about 101° 20′ west longtitude, to Banff in the Rocky Mountains, somewhere in the neighbourhood of the 116th degree of west longitude, *i.e.*, upwards of 700 miles from east to west, and from Fort Saskatchewan on the Great Northern river of that name, in about 53½° north latitude to the International Boundary line (*i.e.* 49° north latitude, about 310 miles from north to south), embracing an area of about 170,000 square miles.

"The North-West Mounted Police Force stationed throughout these extensive territories, and numbering about 1,000, are young and exceptionally vigorous men in the prime of life, who have undergone individually a rigid medical examination before admission to the force, and are not accepted unless absolutely free from disease, and, so far as can be ascertained, from any special tendency to disease.

"The epidemic among all ranks was, at Regina, almost universal, but, so far as regards the men themselves, was of a comparatively light and favourable character, rarely unfitting them for service for more than three or four days, often for a less period, many coming up on "sick parade" being returned on the daily sick report to "medicine and duty;" but among some of those in advanced life, both among the officers and their families attached to the force at head-quarters, and also among civilians in the neighbouring town of Regina, severe bronchial and laryngeal complications occasionally were met with, accompanied by very obstinate affections of the eye and internal ear, attended in the latter with deep-seated suppuration, in both of which cases recovery was protracted. More or less severe frontal headache accompanied by nervous depression, bronchitis, and coryza were common symptoms during the earlier stages of the disease, and apply equally to the epidemic prevailing at every post throughout the North-West Territories.

"At some of these, and especially those situated along the course of the North Saskatchewan River, the epidemic was almost universal, a very large proportion of the divisions (numbering 100 men each) occupying 'Forts Saskatchewan' and 'Prince Albert' being attacked almost simultaneously at the former, 75 per cent. of the whole division 'G' of one hundred men, exclusive of officers, appearing on the sick report with 'Influenza' during the month of January, and a like proportion of 'F' Division (also 100 strong) at Prince Albert during the same period. At Fort MacLeod, where two divisions of 100 men each were stationed at that time, 20 per cent. of the men on the sick report during the month of January 1890 with Influenza, and in 'K' Division at Lethbridge 27 per cent. were similarly affected during the same period. These members do not include the officers or their families, a very large proportion of whom suffered from the prevailing epidemic. No single fatality from Influenza occurred in the North-West Mounted Police from the prevailing epidemic during its continuance. Bronchial complications were common throughout,

laryngeal occurring but rarely, and no single case of pneumonia was recorded anywhere in this force during the prevalence of the epidemic. I may mention here that true pneumonia is one of the rarest diseases in the North-West Territories, the only case which I find recorded as occurring in the force during the last nine years having appeared in a division stationed in the Bootenay Valley, in the Rocky Mountains, about 100 miles to the south-eastward of Banff, in 1887. Acute inflammation of the serous membranes, except as the result of injury, is almost if not quite as rare.

"The invasion or outbreak of Influenza throughout these territories occurred *almost simultaneously* over very wide expanses, and its rapid diffusion cannot well be accounted for by the theory of direct contagion alone, the rate of its progress over wide uninhabited plains having far exceeded that of the travelling medium, the appearance of the epidemic being almost simultaneous at points so widely separated as Fort MacLeod, Fort Saskatchewan, and Prince Albert, between which posts no railway communication existed at the period referred to, and at each of which large numbers were simultaneously affected. My impression is, that the disease was both epidemic and contagious, and that the extraordinary rapidity of its diffusion here over immense expanses of uninhabited country can only be accounted for by atmospheric influences, but that its subsequent and rapid propagation locally may have been due in some measure to direct contagion also.

"The native population, that is, the 'Indians' of the North-West Territories, are now, and have for some years been, collected together in settled but widely separated bands and communities on reserves specially set aside for their use and occupation in various remote portions of the North-West Territories and British Columbia, very little intercourse existing between them. The appearance of Influenza among those scattered tribes and communities extending from the western boundary of Manitoba to the Pacific Ocean was almost simultaneous, so far as I can discover, and could not possibly be accounted for by directly transmitted contagion, but was, in my opinion, essentially due to a widely prevailing atmospheric and epidemic influence.

"Through the courtesy of the Indian Department at Regina, I am enabled to forward herewith a number of extracts taken from the reports of agents in charge of various Indian reserves and communities throughout the North-West Territories and have added thereto some extracts respecting the epidemic referred to as it affected the Indians on the Pacific slope of the Rocky Mountains, in British Columbia, which, though not strictly within the bounds of my own special domain, I have thought might assist in the proposed investigation.

"The Honorable the Minister of the Interior, in his annual report for 1890, says: 'The epidemic of Influenza, popularly known as "La 'Grippe," prevailed very generally among the Indians last winter and 'spring, almost every band from the Atlantic to the Pacific, and as far 'north as the Department has had reports from, was attacked to a 'greater or less degree by this disease, and in the case of many old 'persons, and of those who were suffering from diseases of a pulmonary 'or other chronic character, or who were of a delicate constitution, the 'end was precipitated owing to the complications caused by this 'catarrhal affection.'

"An examination of the public reports of Indian inspectors throughout the three great districts or territories of Saskatchewan, Assiniboine, and Alberta, and also of those throughout British Columbia generally

and on the Pacific coast, at least as far north as Alberta, shows everywhere, during the prevalence of this epidemic, a very largely increased mortality at the period referred to, due unquestionably to its presence alone.

"I have touched upon this subject as it has affected the native races throughout the western portions of the Dominion, as it is possible the sources of information from which my knowledge is derived may not be within reach of the Medical Officer of the Local Government Board."

With regard to the rapid spread of Influenza among the mounted police, it is to be noted that they appear to be men in association one with another, living in barracks.

The following are the dates of the epidemic as returned from the different divisions of the police:—

F Division, Prince Albert.—First observed about the beginning of January 1889; epidemic declined about middle of March 1889 (? 1890).

E Division, Calgary.—Influenza epidemic during the months of November and December 1889 and January 1890. (The reporter disclaims accuracy.)

A Division, Maple Creek.—First case, December 18th, 1889. Epidemic at its height for the following 12 days in barracks. Cases among civilians cropping up later into the middle of February.

D and H Divisions, Fort MacLeod.—First case, December 28th, 1889. Epidemic from December 29th, 1889, to January 1890. "It occurred almost simultaneously at the different towns in this "district, and a large number were attacked at once. I had an "excellent opportunity of observing this among the men in and "out of barracks."

B Division, Regina.—First observed January 1st, 1890, when epidemic began; declined steadily through March, but occasional cases continued to appear in diminishing numbers till April 1st.

C Division, Battleford.—First cases observed January 4th, 1890, when epidemic began. There appeared to be no more new cases the end of that month.

K Division, Lethbridge.—Epidemic, January 7th to 21st.

As regards the Indians, reports are quoted referring to 19 settlements, of which 6 are mentioned as attacked in December, 12 in January, and 1 in February.

Recapitulation of conclusions of former report respecting method of spread of Influenza.

Part IV.—Further Considerations respecting the Etiology of Influenza.

In my previous report on the Influenza epidemic of 1889–90 it was shown that certain traditional beliefs as to the behaviour of epidemic Influenza upon which was based the then commonly received view of its being due to an atmospheric cause, had not been confirmed during that epidemic. These beliefs were, 1st, that the epidemic, in invading in succession different places, travelled with a speed outstripping human communication; 2nd, that it commenced in any given place by the simultaneous attack of a large number of the inhabitants; and 3rd, that it attacked persons who were so placed as to be physically debarred from the possibility of contracting the disease from other persons. On

tho contrary, it was shown that multiple cases in a household frequently occurred in succession; that in numerous instances the first case in a household or neighbourhood could be traced to exposure to infection from a previous case, or to a visit to an infected place; that the special incidence of the disease fell at first upon persons liable to come into contact with infection, and that persons living under circumstances in which the possibility of infection could be excluded had escaped Influenza. The conclusion was drawn that the spread of the disease took place directly or mediately from person to person; its rapid development as an epidemic being explicable by the shortness of the incubation period, the early stage at which infectiousness is developed, the large number of persons who are susceptible and the existence of numerous mild cases, the subjects of which, though not rendered unable to go about, are capable of conveying infection. The experience of the two more recent epidemics has served to confirm these conclusions. Similar conclusions have also been arrived at independently by numerous observers in this and other countries, and the communicability of Influenza may be said to be now very generally, though not quite universally, accepted.

Illustrations of spread by contagion in later epidemics.

The following history given through Dr. Bruce Low by Dr. Broster, of Wirksworth, well illustrates the mode of spread of Influenza:—

> "Mr. X., a teacher of music, went from North Derbyshire on April 6th, 1891, to Sheffield to see his two sisters then ill with Influenza. He returned on the morning of April 7th. He felt very ill on the morning of April 9th, but struggled through his work, and was completely exhausted by night. On April 10th his temperature was 104° F., and he passed through a typical attack of Influenza, with a relapse on getting up too soon. On the morning of April 9th he gave a music lesson to some pupils at Miss A.'s school; none of these contracted Influenza. Later in the day he gave a lesson to Miss B. who began with Influenza on April 11th. At another house just afterwards he gave a lesson to Miss C. who began on April 11th; to Miss D. at another house, who also began on April 11th, and to three girls at Mrs. D.'s house, all of whom began on April 11th with Influenza. In the evening of April 9th he gave a lesson to a small choral society in a village schoolroom. Five of the members of this class began to be ill with Influenza on April 11th. Of the five at least four had stayed behind after the class in conversation with Mr. X. So far as known the above were all the places or houses visited by Mr. X. on April 9th, and no fewer than 10 persons developed the disease on April 11th. The only condition common to all was contact with Mr. X. suffering from the malady and who had previously been in contact with his sick relations at Sheffield. On April 12th the married couple with whom Mr. X. lodged were both taken ill with Influenza; both had waited on Mr. X. on the 10th. Up to April 9th no cases of Influenza to Dr. Broster's knowledge had occurred in the locality. After April 11th the disease spread rapidly, each infected family being a centre of infection."

Dr. Wills gives a similar instance in the Worksop district. A gentleman went home unwell from an office in Sheffield on a Saturday during April 1891. It was not understood what he was suffering from until his mother, who had nursed him, was seized with a severe attack of Influenza on the Wednesday following, and his father on the Friday. It spread through the whole family of 14 persons, and from this house-

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

hold to the village, causing the death of two persons; it was also caught by the medical attendant, who had it severely.

In the Stockton rural district two epidemics of Influenza occurred in 1891, in June and December. The medical officer of health states that the first one had seemed to him to be due partly to an air-borne infection, but in the second, which was more carefully inquired into by him, he found that all the cases had been directly or indirectly exposed to infection.

I will give one other instance in illustration of the mode of spread of Influenza, and this time from Ireland, inasmuch as the communicable nature of Influenza has not been generally recognised by medical men in that country. I am indebted for the particulars to Dr. Blakeley, of Fivemiletown, county Tyrone.

A gentleman residing in Manchester, who had had a slight attack of Influenza a week before, left on Friday, May 15, 1891, with his wife and two children on a visit to his parents in Ireland. On the following Monday his eldest child was taken ill with Influenza, and two days later the younger child. Within a week his wife, father, mother, and two sisters were all down with the Influenza, the only person in the house who escaped being a female servant, who had had the disease in the spring of 1890. Two other members of the family who went to stay at a house about 200 yards distant, and were kept away from those who were ill, also escaped. There were no other cases in the village at that time. Dr. Blakely says:—"I am aware that there are many medical "men in this country who do not believe that Influenza is contagious, "but from my experience of the epidemic of last year and what "occurred in this family this year, I am of opinion that it is contagious. "The fact that it did not spread is due, I believe, to the precautions "taken to prevent anyone coming in contact with those who were ill, "and disinfection was carried out to the utmost extent."

It having been publicly stated as evidence of the atmospheric origin of Influenza that the keepers of the Caskets Lighthouse, (which is situated on a rock in the Channel Islands, distant from land,) had been attacked without previous communication with the shore, I wrote for particulars to the Secretary of the Trinity House, and received the following reply:—

"With reference to your letter on the subject of the cases of Influenza which recently occurred among the keepers of the Caskets Lighthouse, I am directed to inform you that one keeper was first attacked on January 10th, 1892, and on January 16th two more keepers were also attacked. The last communication, before their illness, which these men had with the shore was on January 1st. Influenza had previously been prevalent in Guernsey, and there were also cases of it at Alderney."

An incubation period of nine days is longer than usual in Influenza, but it cannot be said that the possibility of infection from a previous case is excluded.

Further questions respecting etiology of Influenza.

The following are among the principal epidemiological questions concerning Influenza, which in my former report were left unsettled, and on which further investigation is still desirable:—

1. Is there any medium in which the contagium of Influenza can live or multiply outside the human body?
2. Granting that Influenza is propagated by infection from person to person, what are the circumstances which conduce to its epidemic spread at one time (often simultaneously in widely distant places) and not at another?

3. In what degree (if at all) and, if so, for how long, does one attack of Influenza confer immunity upon *the individual?*
4. Does an epidemic of Influenza in any degree protect a *community* against a recurrence?
5. Are there any circumstances, as of locality or of occupation, which favour severe incidence of Influenza?
6. Or, on the other hand, are there any circumstances, as of locality or occupation, which tend to exemption from Influenza?
7. Is the concurrent pneumonia to which a large proportion of the mortality from Influenza is due, a part or symptom of the original disease (*e.g.*, as nephritis of scarlet fever), or is it something superadded (*e.g.*, as diphtheria may be upon scarlet fever)?
8. How far are pneumonia and other lung complications of Influenza due
   (*a*) to exposure to cold or fatigue before recovery;
   (*b*) to influence of drain air or other unsanitary conditions?
9. How soon does the period of infectiousness begin in a case of Influenza, and how long does it last?

1. *Is there any medium in which the contagium of Influenza* (*a*) *can live, or* (*b*) *can multiply, outside the human body.*

Can contagium live or multiply outside the human body.

(*a.*) In my former report I mentioned instances tending to show that the contagium of Influenza might attach itself to articles of clothing and letters, and that persons coming into contact with such infected articles might contract the disease, and some similar instances seem to have been met with in the later epidemics.

Thus Sir Peter Eade ("British Medical Journal," August 8, 1891) mentions the case of a gentleman who was taken ill of Influenza 48 hours after moistening with his tongue the gum of an envelope sent for return in a letter from a correspondent who wrote that he was himself suffering severely from the disease.

The accounts given in a later part of this report of the outbreaks of Influenza in the Milnthorpe and Houghton-le-Spring Workhouses point to the probability that the infection can be conveyed by soiled linen, and a similar instance is mentioned by Mr. H. B. Collins, "British Medical Journal," March 5, 1899, p. 510.

(*b.*) In my previous report (p. 102) I suggested* the possibility that the contagium of Influenza once imported into a locality might multiply outside the human body in some appropriate medium, such as perhaps damp ground, or air contaminated with organic exhalations, or laden with dust. I cannot say that the experience of the later epidemics has given any additional reason for supposing the existence of such a medium, and still less has it pointed to what the medium may be.

On this point we may hope for enlightenment from bacteriological research.

The question of the relationship of human Influenza to the "influenza" or "pink-eye" of horses, as also that of the transference of the disease to or from human beings and other animals, has been studied by Dr. Klein.

---

* A similar suggestion has been made by others. Thus Dr. Ashburton Thompson, in his report on Influenza in New South Wales in 1890 (quoted on page 45 of my previous report), thinks that while human intercourse may be necessary to transport the contagion of the disease, some stage intermediate between man and man may be necessary to its epidemic spread. In his later report, as will be seen on p. 35, he considers this view to be unfounded.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

Dr. Caldwell Smith, Lecturer on Hygiene and Public Health at Anderson's College, Glasgow, in some valuable notes which he has given me, under date March 7th, 1892, makes the following remarks on this and allied points :—

"Etiology.—I have not the slightest doubt that this disease is caused by the bacillus discovered by Pfeiffer. In the spring of last year I made some examinations of the blood of Influenza patients and found in it a small diplococcus as I considered, but on reading Pfeiffer's paper I concluded that this must have been the bacillus as described by him. I tried to cultivate the organism but failed, as I used ordinary agar and nutrient gelatine. In one case in which several relapses occurred, these diplococci were found in large numbers.

"It is to the life history of Pfeiffer's bacillus that we must direct our attention if we wish to understand the seemingly strange vagaries of the disease. I believe strongly that it is very infectious, and that it is so even in the prefebrile or incubation period. An individual is infected by breathing at once the expired air from a person suffering from the disease, and I believe this to be the only method of infection. In this respect it resembles typhus fever. I cannot see my way to believe in mediate infection, as the germ cannot, according to Kitasato, grow at a temperature lower than 28° C. From my own clinical observation I have rarely met with a case which could not have arisen from a previously existing one, and even in those few rare cases, if strict inquiries had been made, I believe that the source of infection would have been discovered. Again, the bacillus is, I think, easily destroyed by free ventilation. From my own observation I would say that drying does not preserve, but destroys it, and that, like the cholera spirillum, its life history is, though difficult to explain, a very short one. In a suitable medium, that is, in the human body, it must multiply enormously, and in overcrowded apartments, it plays havoc with the inmates.

"It may be that some peculiar hygrometric condition of the atmosphere prevents the drying and consequent death of the bacillus, but on this I would not insist. Other climatic conditions have no effect on the disease. Rain, hail, snow, frost, &c. have been common enough here this winter, but none of these had any influence on the epidemic. Any other mode of propagation than that of direct infection is to me utterly untenable, and I would suggest that careful experiments with the bacillus should be made before any very decided opinion is given as to the prevention of the disease. That the bacillus enters the system by the respiratory tract there is little doubt, and I cannot agree with Dr. Bezly Thorne's idea that the materies morbi enters the system through the conjunctiva, although I believe with him that the cerebro-spinal nerve centres are principally affected."

2. *Granting that Influenza is propagated by infection from person to person, what are the circumstances which conduce to its epidemic spread at one time (often simultaneously in widely distant places) and not at another?*

What are the circumstances under which Influenza assumes epidemic character?

In this question we have the *crux* of the whole matter. It seems clear, to me at least, that the disease is transported by human agency, and spreads by infection from person to person, mainly or solely. Yet how is it that we find the disease at one time smouldering on or altogether extinct, and at another time blazing up into an epidemic? Thus, as it seems probable, genuine Influenza was present in this country in the form of sporadic cases or local outbreaks in the latter part of 1889 for

several weeks or months before the epidemic which began shortly after Christmas in that year. Again there is reason to think that since the first epidemic this country has never been wholly free from the disease; sporadic cases with occasional local prevalences being reported in various places, both during the interval of a year which elapsed between the cessation of the epidemic of 1889–90 and the commencement of that of the spring of 1891, and during the shorter interval between the epidemic last mentioned and that of 1891–2.

Again there are instances like those quoted on pp. 99 and 100 of my former report in which Influenza imported into a district has not spread apparently further than the first patient or his household, but an epidemic has broken out later and seemingly from a different origin.

It may be said that in this we have only what we are accustomed to witness with other diseases admittedly spread by infection from person to person, as small-pox, scarlet fever, and measles. It cannot probably be fully explained why small-pox assumed in 1870–72 such an exceptionally severe character and world-wide spread; nor, on the other hand, why in 1887–8 it should have developed into a formidable epidemic at Sheffield at a time when the rest of the kingdom was comparatively free. In the case of a local outbreak the explanation may be suggested that circumstances which we are accustomed to call accidental, such as assemblages or migrations of persons, or the presence among other people of an individual having the disease in an unrecognised form, may favour the spread of the infection, which, failing such favouring circumstances, would die out, and cease with the early cases. This explanation, however, does not meet the case of a disease which re-asserts itself in an epidemic form independently and at the same time, or nearly so, not only in different parts of the same country, but also in widely distant parts of the world. Thus the second epidemic of recent years seems to have commenced in March 1891 in Yorkshire, Wales, and the United States, and was prevalent in April and May at the same time in England, Northern Europe, and Egypt. The third epidemic began in October 1891 in Scotland and the S.W. of England, and in Paris, on the Russian frontier, and other parts of Europe; though the disease had been present in an epidemic form in other localities on the Continent, as parts of Portugal, Spain, and France, and also in Australia, in the months intervening between the second and third epidemics in this country. This last epidemic raged at the same time in the British Isles, most parts of the Continent of Europe, Egypt, China, and parts of the United States, and Canada. These nearly simultaneous independent recrudescences must seemingly be due to some common cause acting over a wide part of the earth's surface; but what this cause may be it is impossible, with our present knowledge, to say. For the reasons given in my former report, and borne out by later experience, season, climate, and weather may be set aside; as also may be, I believe, any hypothetical atmospheric miasm or cosmical agency. Does some phase in the life history of a parasitic micro-organism cause it to assume periodically increased virulence? If so, why do these periods occur so irregularly and independently of season? Does the recrudescence of Influenza depend upon external circumstances favouring the multiplication of the micro-organism, or upon diminished powers of resistance on the part of human beings exposed to its attacks?

One condition which there is good reason to believe favours the epidemic spread of Influenza is the assemblage together of large numbers of people. Instances were given in my first report in the epidemic of

1889–90, and, in the postscript, others in the earlier epidemic of 1891. Other examples may be given in that and the last epidemic.

At St. David's Influenza broke out quite suddenly during the week ending December 26th, 1891. The medical officer of health considered it to have been brought thither by visitors who had come to spend their holidays, and he observed, too, that a great number of persons who had attended a large public meeting at St. David's on Christmas night took the disease.

At Eccleshall Influenza appeared quite suddenly, a large majority of cases following two or three days after an Oddfellows' fête.

The medical officer of health for the Kington rural district is of opinion that the May fairs of 1891 were the cause of the wide-spread outbreak in that district. His first case, seen on May 22nd, had attended Hay fair on the 18th; the same evening a policeman living 6 miles distant, but who had also attended the fair, was down with the disease, and within 24 hours there were several other cases.

It is also to be observed that both the first and the third of the recent epidemics commenced in London and in many places in the country shortly after the Christmas season, with its increased travelling and opportunities of meeting.

The concourse of people is favourable to the spread of Influenza in two ways. 1st. It affords increased opportunities for persons who are in a condition to impart the disease to come in contact with those who are in a condition to receive it. 2nd. Where such concourse takes place in a confined space the poison is likely to be present in a more concentrated form, while the powers of resistance may be lowered by the vitiated air.

Dr. Rouse, medical officer of health for Northam, Devon, in speaking of the value of fresh air as a preventive, gives the following instance, as tending to show that the spread of Influenza is promoted by breathing foul air:—"A party of seven persons came to Northam to a concert "from a neighbouring town in which there was little or no Influenza. "There were six persons inside the carriage and one on the box-seat. The "night was very cold, so on the return journey the windows were shut. "Within three days all the six who had ridden inside the carriage were "laid down with Influenza, but the one who was outside escaped."

As I have suggested in my former report there is reason to suppose that the epidemic spread of Influenza largely depends upon the degree of concentration of the specific poison. I have likened its spread to a fire lighted in greenwood; if the fire be small it will die out, but if large it will set fire to the wood, which will continue to burn. Or we may compare the resisting forces of the human body (be they "phagocytes" or something else) to a strong man who can vanquish a number of foes one at a time, but would be overpowered by them if they attacked him all at once. A person of ordinary powers of resistance may escape serious harm from a small dose of the Influenza poison (whether microbe or its product), but will succumb to a large dose or to a prolonged exposure. The feelings of malaise so commonly felt during an Influenza epidemic by persons not actually themselves the subjects of the disease are, we may suppose, the effects of minor doses of the poison.

3. *In what degree, if at all, and if so, for how long, does an attack of Influenza confer immunity upon the individual?*

An attack of Influenza protects at most but imperfectly against another attack.

It seem clear that an attack of Influenza does not confer upon the patient any such degree of protection against another, as is afforded, say, by an attack of small-pox, scarlet fever, measles, or whooping-cough;

but there is a difference of opinion among observers as to whether any protection at all is afforded, some considering that a certain degree of protection is afforded, though it may be an incomplete or transitory one; while others, on the other hand, consider that one attack rather predisposes than otherwise to another.

Relapses.

In this matter we have to distinguish between relapses occurring shortly after the primary attack, and second attacks occurring in a subsequent epidemic. The frequency of relapses is admitted, as mentioned in my previous report (p. 68). Dr. William Squire has published ("Lancet," August 16th, 1890), some cases of relapse occurring, where every care was taken to prevent fatigue and exposure during convalescence.

Second attacks.

As regards second attacks in a subsequent epidemic, Dr. Squire says, "Not only is one attack not protective against a recurrence, but it "seems to me rather to predispose to it."

The medical officer of health of Ulverston states that one attack of Influenza is not protective against another. Those who had it in 1890 were the first to be seized in 1891. He himself, who had had Influenza in 1857, had it again in 1890, and three times in 1891.

The medical officer of health for the Kirby Moorside rural district (who estimates that half of the population of his district were attacked during the epidemic in the spring of 1891) says that those attacked in 1890 (when the district on the whole was lightly affected) did not escape in 1891; thus one family that had three or four well-marked cases in 1890, had three of the same members again attacked in 1891, proving that an attack does not confer immunity.

The medical officer for the Haresfield division of the Wheatenhurst rural district says that some persons have been attacked four times in two years (apparently including relapses).

Dr. Davidson, of Congleton, says that one attack seems to confer no protection against another, and believes that not only may a person be attacked after three months' interval, if living in one place, but that he may after an interval of only a fortnight, if he remove into a district in which the disease has recently become epidemic; showing, he thinks, that the interval of immunity depends not upon any quality of the individual, but upon that of the poison necessary to infect him. A lady suffered from Influenza in Congleton, early in May 1891. At the end of a fortnight she went to London, where the epidemic was more recent, and immediately became infected again. When well enough to travel she went to Scarborough where Influenza had just broken out, and contracted it again, thus having three severe attacks in less than six weeks.

[It is possible that in this history other medical men would look upon the later attacks as relapses, perhaps brought on by exposure and fatigue in travelling, rather than as the result of repeated contracting of infection. Moreover, the dates given do not accord with my information. Influenza began to be epidemic in London at the end of April or the beginning of May 1891, and, according to Dr. Cuff, of Scarborough, it prevailed as an epidemic in the neighbourhood of Scarborough from the beginning of April to the end of May.]

On the other hand, Dr. Caldwell Smith says:—

"One attack does not confer complete immunity on all individuals; but looking over my cases, I find that in the last epidemic, viz., that in November and December 1891, and January 1892, 75 per cent. had never suffered in the two previous epidemics. The other 25 per cent., viz., those who did suffer before, may be divided into four classes:—

"(1.) Those suffering from any chronic lung affection, as bronchitis and phthisis.

"(2.) Those in whom a previous attack had left some permanent mischief, *e.g.*, endocardial affections.

"(3.) Those who suffered from organic diseases other than lung disease, and whose resisting power was consequently small.

"(4.) Those in whom there existed nothing that could be held as predisposing to a second attack, this latter class forming about 15 per cent.

"In my own case, having suffered from it in February 1891, although day after day breathing directly the expired air of Influenza patients, both experimentally and accidentally, no second attack occurred.

"I should say that one attack confers immunity on the large majority of healthy individuals for some time; but it would be impossible with the means at our disposal to fix a numerical limit.

"I have had several cases in old people who have suffered from it in 1847, and one lady of 75 also suffered in all the last three epidemics, viz., in January 1890, January 1891, and January 1892.

"One attack does not, except in the cases mentioned above, in classes (1), (2), and (3) conduce to a second.

"Dr. Parsons, in his report, states that probably the immunity is not true immunity, but that individuals acquire a certain tolerating power similar to that obtained by persons working in rag factories in a dust-laden atmosphere. I cannot agree with him in this, as we are not dealing with inanimate particles suspended in the atmosphere, but with a living organism as the causal agent of the disease. The immunity of the majority of individuals is for the time being a true immunity, but it is only a partial and probably a short-lived one."*

Dr. Niven, as the result of inquiries among medical men in Oldham, states that with few exceptions, those persons who suffered from Influenza in 1891 had escaped in 1890, while those who had suffered in 1890, escaped in 1891.

He says in a letter: "It seems to me difficult to resist the general "impression that people who had Influenza last year do not take it "this year (June 1891), with rare exceptions. For instance, I receive "to-day a letter from a medical man in large practice who remembers "only one such case.

"The one fact in my own knowledge is that several medical men of "my acquaintance, along with myself, were attacked last year and none "of us this, and I believe that the same is true of others. Considering "the intimate exposure to infection both years, this seems striking."

The medical officer of health for the Monmouth rural district (who states that Influenza was never quite absent in his district during the earlier half of 1891, and began to prove fatal in June, though December was by far the most fatal month), says: "In a few cases people were "attacked more than once during the year; but, as a rule, one attack "seems to give immunity for at least a year."

The medical officer of health for Newport, Monmouthshire, states that the post office staff and police, numbering together some 250 persons, who suffered severely at the first outset of Influenza in 1890, have since almost escaped, pointing, he thinks, to the acquisition of some degree of immunity.

The experience of the London Post Office seems to have been similar. Mr. Steet, Chief Medical Officer, informs me (May 15, 1891): "There

* In the passage alluded to the distinction which I had in view was not that between an inanimate matter and a living organism, but between the toleration acquired for an irritant during its presence but lost on the cessation of exposure to it, and the immunity which persists after exposure to the irritant has ceased. The difference, however, may be looked upon as one of degree only.—H. F. P.

"has been a recurrence of Influenza among the Post Office staff this "spring, it has, however, been neither so extensive nor so serious in "character as that of last year."

In his annual report for 1891 on the health of the Customs officers of the Port of London, Dr. W. Dickson, medical officer, says: "In regard "to the question of relapse, or recurrence of the disease a second time, "only 5 per cent. of the 112 cases under observation (? 6 cases) were "found to be so affected." As 11 per cent. of the Customs officers in the Port of London suffered from Influenza in 1890, this would indicate, so far as such small figures go, a certain amount of protection conferred by a previous attack.

The question of the amount of protection is one which it is very desirable to bring to numerical test on a large scale. Probably this could most readily be done by the experience of large institutions. Taking the individuals who were inmates of the same institution during two successive epidemics of Influenza, the comparison of the respective proportions of those who, having suffered on the first occasion, were attacked or escaped on the second, should, if the numbers were sufficiently large and other circumstances were equal, throw light upon this question. I am able to give one such instance through the courtesy of Mr. O'Grady, medical officer to the Industrial Schools at Swinton, near Manchester. These schools were severely affected in March 1890, 171 out of 589 children having suffered, or 29 per cent. (*see* page 237 of my former report). In the first epidemic of 1891 they were again affected, but to a less extent, only 35 cases occurring. At that time there were in the schools 449 children who had been there at the time of the former epidemic. Of these 150 had had Influenza in 1890, and 4 of them had it again, or 2·6 per cent. 299 had escaped Influenza in 1890, and 17 of these had it now, or 5·7 per cent. Thus, so far as these figures go, an attack of Influenza confers a degree of protection which after the lapse of a year diminishes by one half the liability to contract the disease.

4. *Does an epidemic of Influenza in any degree protect a community against a recurrence?*

Does an epidemic at all protect a community against another?

Here again it is clear that any such protection can only be of a partial and relative kind. London has, as we see, suffered in each of the three recent epidemics,* and with increasing severity; and most places in England and Wales appear to have suffered more or less in each of these epidemics. If the protection afforded to individuals by an attack of Influenza be but at most a partial one, it is not to be expected that the community of which they are components will be completely protected against the recurrence of an epidemic. Besides, in any epidemic it is only a fraction—rarely, I am inclined to think, more than a minority—of the community who contract the disease, and so acquire even this partial protection. Moreover between one epidemic and another there is necessarily more or less change in the component items of the population, through births, deaths, and migrations.

Nevertheless I think that there is some evidence that a community which has suffered severely from an epidemic of Influenza has acquired a certain amount of protection, and is less likely to suffer severely on a return of the disease.

---

* This is true of London as a whole, but it may not be true of individual parts of London. Possibly different parts may have suffered unequally in different epidemics; but on this point I have no information.

Thus the large towns of the Midlands, Yorkshire, and the north of England, which, judging from the mortality returns, escaped comparatively lightly in the epidemic of 1890, suffered very severely in that of the spring of 1891, but again escaped lightly in the winter of 1891–2. Newcastle-on-Tyne, however, which escaped lightly in the two first epidemics, suffered heavily in the third, and, less markedly, the same seems to have been the case at Liverpool and Glasgow.

On the other hand, in the south and south-western counties of England the second epidemic was comparatively mild in type, but the third was very severe, occasioning a high mortality. Thus in Brighton the weekly death-rate from all causes, which did not reach 25 (calculated per 1,000 inhabitants per annum) during the epidemic period of 1890, nor 22 during that of the first half-year of 1891, rose to 51·5 in the second and 60·9 in the third weeks of 1892. Similarly in Portsmouth and Plymouth the weekly death-rates did not exhibit any distinct periods of high mortality corresponding with the first and second epidemics, but in the third the death-rate rose in Portsmouth to 57·0 in the second week of 1892, and in Plymouth to 45·1 in the 49th week of 1891. Further testimony as to the comparative mildness of the second epidemic and the severity of the third in the south-west of England is given by many medical officers of health.

The different local incidence of successive epidemics of Influenza has been observed on a smaller scale in the different parts of a single district, those villages which were most severely affected in one epidemic escaping lightly in another, and *vice versâ*.

Thus Sir Peter Eade ("British Medical Journal," August 8th, 1891) says that "It has been observed by several most competent practitioners "in this district (Norfolk) that although one attack has not appeared "absolutely to protect those who suffered last year from illness again "this season, yet that in the most remarkable way villages in which "the Influenza prevailed extensively in 1890 have been almost or quite "free from the disease in this present year, while adjacent villages "scarcely affected before have recently been generally and severely "attacked."

Mr. F. Broadbent, medical officer of health in the Newark Rural District, says: "I have come to the conclusion that Influenza is highly "contagious, and also that in most people one attack is protective, as is "the case with most of the zymotic diseases. I formed these opinions "from watching the epidemic in my district; villages that were overwhelmed with it in 1890 did not have it in 1891."

Mr. Williams, medical officer of health for the Richmond (Yorks) rural district, says in his annual report for 1891: "The inhabitants of "those villages that suffered severely in 1890 escaped almost entirely "in 1891, in the face of the disease being again introduced amongst "them; while inhabitants of those villages that escaped in the former "year suffered severely during the last epidemic." He renounces an opinion which he had formerly expressed, that one attack conferred no immunity against subsequent infection, and has now no doubt that the disease does confer a certain degree of immunity against a future attack, though some susceptible persons take it two or three times.

Mr. Gosse, medical officer of health for the Eccleshall division of the Stone rural district, states as his experience that "One attack seems to "give a certain amount of immunity; a small proportion of those suffering in 1892 having had a previous attack, and localities chiefly "affected in 1892 being those which escaped in 1891."

In some localities it is stated that whereas in 1890 Influenza was most prevalent in the towns, in 1891 the disease attacked especially the

country villages, the towns comparatively escaping. This is stated to be the case by medical officers of health at Epping, Horncastle, Mansfield, and Stockton.

At Driffield the reverse was the case, the market town, which had escaped lightly in 1889–90, suffered severely in the spring of 1891, while the neighbouring villages, *e.g.*, Langtoft, which suffered early and severely in the epidemic of 1889–90, got off lightly in 1891.

Dr. Cuff, in his annual report for 1892 on the Scarborough Rural District—a district which had been severely affected by Influenza in the summer of 1891, says, "During the spring of 1892 there were a "few cases of Influenza in each of the sub-districts; it was most "severe, however, in Hackness, which village had escaped on previous "occasions. The population of Hackness at the last census was 188, "and the number of cases of Influenza of which I received information "was 56, with one death."

On this point Dr. Caldwell Smith gives me the following observations:—

"Does an epidemic of Influenza in any degree protect a community against another?

"It does to some extent, but there will always remain a large number of individuals in any community who have not suffered from it, and others who are peculiarly susceptible on account of reasons given in the last answer.

"If a community suffers badly and generally, I do not think it will suffer so badly in any future epidemic, occurring shortly after the first. In this district we have had three epidemics. The first two were light, and pneumonia and other complications were rare, I may say almost absent, while the last epidemic was very severe and pneumonia was common.

"I believe, as regards the community, immunity is partial, but not absolute. The fact of London suffering badly could, I think, be explained if one were carefully examining the returns from the separate districts, when it would probably be found that, although in the aggregate the metropolis had suffered heavily in the three epidemics, the districts suffered unequally.

"This has decidedly been the case in Scotland, one parish suffering badly in one epidemic and lightly in the next, while the very opposite was the case in neighbouring parishes."

5. *Are there any circumstances, as of locality or of occupation, which tend to produce severe incidence of Influenza?*

Circumstances conducing to severe incidence of Influenza.

Topography.

I have seen no reason to think that the prevalence or fatality of Influenza are in any way modified by the circumstances of different localities in regard of such matters as situation, soil, or altitude. Statements indeed are sometimes met with that Influenza was most prevalent or severe in places in certain situations, but these statements are so contradictory that they seem to neutralize one another. Thus, within the limits of a single county, Lincolnshire, two observers, viz., Dr. E. W. Barton, medical officer of health for Market Rasen, and Mr. Eminson, of Scotter ("British Medical Journal," June 13th, 1891), state that the disease seemed to select high and exposed places, as those on the Wolds, in preference to those in more sheltered situations; while the medical officer of health for the Stamford rural district states that it was more severe in low-lying parishes than in those on higher ground; and two other medical officers of health, viz., those for the Spilsby and Sleaford rural districts, state that they could observe no connexion between the prevalence of the disease and the topography, the portions of the population dwelling on high ground and in the fens being alike attacked.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92 by Dr. Parsons.

In the Orsett rural district, Essex, the medical officer states that in the epidemic of 1891 the villages on the high ground were especially attacked, in contrast to 1890, where the epidemic was most severe in the low-lying villages.

Nor, as already remarked in referring to the statistics of 1890 and 1891, does the severity of the incidence of Influenza, at any rate as regards its mortality, appear to be determined by those unfavourable sanitary and social conditions which foster other diseases of the zymotic class, and cause the total mortality in certain of the larger towns of England to be habitually excessive. Some usually healthy towns have suffered severely, as Croydon, Brighton, Portsmouth, and Norwich in January 1892, and Derby and Huddersfield in May 1891; while other towns in which the death-rate is notoriously high, as Liverpool and Preston, have not in either of the recent epidemics had their mortality increased to a proportionate extent.

and sanitary condition are without effect.

As regards the parts of towns again, it has been found that the quarters in which the mortality from Influenza have been highest have been often those inhabited by the well-to-do classes and not the crowded and unhealthy quarters inhabited by the poor.

Thus in London (see table on p. 22) the deaths from Influenza in January and February 1892 were most numerous, relatively to the population, in the sanitary areas of St. George (Hanover Square), Kensington, Chelsea and Lewisham, quarters containing a large proportion of good houses, and wealthy or well-to-do people; the proportion was also above the average in St. Giles, the City of London, Whitechapel, Rotherhithe, Paddington, Wandsworth, Battersea and Plumstead, districts differing much one from another in character. The proportion was lowest in St. George (Southwark), Westminster, Woolwich, Poplar, Shoreditch, and Newington, districts for the most part inhabited by the working class.

In explanation of this anomaly the medical of health for Shoreditch states that in the poorer districts persons attacked by disease do not keep indoors until absolutely compelled to do so, and do not consult a medical man until the early and distinctive stages of Influenza have passed, and bronchitis or pneumonia has resulted. If death occurs it is thus certified as due to the latter cause. This, he says, may give the impression that Influenza did not affect the eastern district of the metropolis so much as the western or suburban ones.

In his health return for the second quarter of 1891 Dr. Tatham, medical officer of health for the city of Manchester, says: "It is remarkable that epidemic Influenza was relatively most fatal, not, as might have been expected, in the poorest and most unhealthy districts of the city, but in the comparatively salubrious locality of Blackley, where presumably the patients would be well nursed and cared for."

Similarly Dr. Alfred Hill, in his report on the health of Birmingham for the quarter ending July 4th, 1891, says: "Influenza has been most fatal in St. Martin, All Saints, Duddeston, and Edgbaston sub-districts. It is singular that Edgbaston should have suffered so heavily, as, generally speaking, this part of the town has a very low death-rate, particularly from infectious diseases."

The medical officer of health for West Hartlepool makes a similar remark.

Age-constitution will to some extent explain difference in mortality.

The explanation of the anomaly is probably, as already remarked, partly to be found in the different proportions of the population at different ages in "residential" and in working-class localities. Influenza, while it attacks persons of all ages, and often a considerable proportion of the inhabitants of a locality, is especially fatal to old persons and

those in middle life. On the other hand, children, after the first year, who are the principal sufferers from other infectious disorders, are commonly mildly affected by Influenza and comparatively rarely die of it. Hence, *cæteris paribus*, in a community containing many children and young persons the death-rate from Influenza, will be lower than in one containing a smaller proportion of children, and a larger one of elderly persons. Now in the wealthier districts there are, for obvious reasons, a large number of elderly persons and of unmarried adults, especially domestic servants, and therefore, on the other hand, a relatively small proportion of children. In the part of this report dealing with the statistics of 1890, I have shown that, as a rule, the mortality from Influenza was high in those counties where the birth-rate was low, and *vice versâ*.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

As regards occupation, I have in my former report shown reason for thinking that fatigue and exposure render persons more liable to contract Influenza, or cause them to have it in a severer form if they do contract it. The danger to the patient of going about while suffering from the disease or at too early a stage of convalescence is generally recognised.

Exposure to great heat, to night air, and to the inhalation of dust-laden air have all been suggested as conducing to Influenza, but I have not seen any attempt to prove this numerically. As I have previously shown, the frequently-observed earlier incidence on persons whose occupations take them out of doors may be explained by their more frequent opportunities of coming in contact with infection.

Occupation of doubtful influence.

Dr. Caldwell Smith gives me the following notes as the results of his experience on these points:—

"I cannot say that, in my experience, either locality or occupation had any effect on the spread of Influenza.

"In this district (Motherwell, Lanarkshire) all sorts and conditions suffered equally, colliers, iron and steelworkers in every department, both in Siemens and Thomson-Gilchrist processes, joiners, masons, labourers, shopkeepers, all suffered.

"The stablemen and grooms at two large hiring establishments here were not exempt, although it is worthy of note that in one yard, where several horses took influenzal pneumonia, the two grooms who were in immediate attendance were the only two who did not take the disease, while other five grooms, &c., who had nothing to do with the care of the affected animals, took it.

"That exposure to dust-laden air did not render the disease more severe, was seen in the fact that although in this district colliers suffered severely, yet in a neighbouring parish where the coal mines are no better ventilated, and where the methods of working are much the same, they escaped almost altogether.

"Policemen in this district, although exposed much to night air, did not suffer more severely than others.

"Puerperal women suffered very severely, and were very prone to infection. Three cases came under my own observation, and all gave me great anxiety for many weeks. Convalescence was also slow."

6. *Are there any circumstances, as of locality or occupation, which tend to exemption from Influenza?*

Few places in this country appear to have escaped Influenza entirely in the last three years, though a few are said to have been only lightly affected.

The village of Rookhope, Durham, is said to have remained almost free from Influenza in each of the three recent epidemics, although other

Circumstances exempting from Influenza.

places in the neighbourhood have been severely affected. Rookhope is a village of 438 inhabitants, containing lead-miners, smelters, and farmers, and is in an elevated situation at the head of a branch dale out of Weardale. Its escape may perhaps be due to its secluded position.*

Of occupations, it has been asserted that dyers working with bichromate of potash have enjoyed a marked immunity from Influenza. Mr. Grant, of Galashields, states as the result of his inquiries, which extended to all the principal dyeworks in Scotland and to some in England and the United States, that in the epidemic of 1890, in all cases where the dyehouse workers were exclusively employed in the dyehouse, and not connected with the washing or scouring department, there was an almost absolute immunity from attack, while in cases where the dyehouse and scouring department were combined there was a marked decrease in the proportion of cases as against the general community. For the epidemic of 1891, he gives me figures respecting two cloth factories in Walkerburn, Scotland, from which it appears that of 25 dyers only 3, or 12 per cent. were attacked with Influenza, while of 591 persons employed in other departments, 178, or 30 per cent., were attacked. The figures, however, are too small to carry much weight in a question of this kind.

Dr. Caldwell Smith says: "No occupation, in my experience, has "been exempt. With regard to dyers working with bichromate of potash, "I communicated with Dr. Simpson, medical officer of health for Perth, in "whose district are many large dyeworks, and he states that, without "making strict inquiry, which he had no time to do, generally dyers "were probably least of all trades affected, but he could not say to what "extent the bichromate workers escaped more than the others."

Mr. Fletcher, the Board's chief inspector under the Alkali Acts, in his annual report for 1891, has collected some information, which he thinks seems to show that men engaged in chemical manufactures are to some extent protected against attacks of Influenza, the men employed in alkali works, tar, gas, and sulphate of ammonia works escaping best The following table is modified and abbreviated from one given on page 22 of his report:—

Works.	Men employed.	Cases of Influenza.	Per cent. attacked.
Alkali	3,100	144	4·6
Sulphuric acid	975	116	11·9
Chemical manures	2,114	129	6·1
Cement	3,122	248	7·9
Tar	1,076	56	5·2
Gas	3,016	264	8·7
Sulphate of ammonia	590	38	6·4
Salt	640	66	10·3
Other processes	917	63	6·9
Total chemical workers	15,550	1,124	7·2
Labourers and artizans (outdoor) at chemical works	3,208	521	16·2
Boiler men at ditto	70	15	21·5

He says that the comparative immunity from Influenza of men employed in sulphate of ammonia manufacture has been noticed by

* At Southend, Essex, two convent establishments containing respectively 130 and 100 inmates, mostly children, are stated to have entirely escaped during each of the three recent epidemics.

many; in several works there was complete freedom. The average, however, was brought up by one or two returns which tended to contradict this. This shows the care necessary in generalising from such data.

7. *Is pneumonia an integral part or symptom of Influenza, or is it something superadded?*

Relation of pneumonia to Influenza.

The answer to this must be given by those who have had more clinical experience of the disease. Judging from the periods in the case at which pneumonia may commence, I should say that both views might be correct.* In some cases as those on the "Shaftesbury," spoken of by Dr. Male on p. 76 of this report, pneumonia comes on so early that it would appear as if the disease fell primarily upon the lungs. In other cases, again, when pneumonia comes on late in the case, or during convalescence, it may be regarded as a complication or sequela, rather than a part of the original disease. The importance of the question is that the mortality from Influenza is brought about in large part by pneumonia and other inflammations of the lungs, and there would appear to be more hope of guarding against these if they may be regarded as complications due to secondary, and perhaps avoidable, causes, than if they are a part of the original action of the Influenza poison.

8. *How far are pneumonia and other lung complications of Influenza due (a.) To exposure to cold or fatigue before recovery; (b.) To influence of drain air or other insanitary conditions?*

On these points Dr. Caldwell Smith says:—"Exposure to cold at the "beginning of the disease was, in my opinion, the exciting cause of all "the cases of influenzal pneumonia which came under my observation.

"In five cases out of 30 the pneumonia seemed to arise coincidently "with the Influenza, but there can be little doubt that the latter preceded "the former, although possibly only for 12 hours.

"I could not find in any of my 30 cases of Influenzal pneumonia, "any insanitary condition which might originate the pneumonic con- "dition.

"Influenzal pneumonia differs in so many respects from ordinary "pneumonia, that one can hardly think it is, like ordinary pneumonia, "superadded to Influenza.

"I communicated with Dr. Russell, pathologist to the Royal Infirmary, "Edinburgh, and he corroborated the above expression of opinion.

"If pneumonia had been a distinct disease, and caused by the pneumo- "coccus, one would have expected several cases in the same house. "This was not my experience, as although frequently attending half-a- "dozen individuals in the same room suffering from Influenza, in no case "did the pneumonia extend to more than one individual. With respect "to this question Dr. Russell states: 'Any of the factors mentioned may "'probably determine the pneumonia, but in others it comes on with the "'onset of the Influenza, and in these, whether the poison is purely "'influenzal, or something added, it is, I should say, impossible to "'absolutely determine without very elaborate laboratory investigation; "'short of that we can only form an opinion.'"

9. *How soon does the period of infectiousness begin in a case of Influenza, and how long does it last?*

Duration of period of infectivity.

As to this Dr. Caldwell Smith says:—"In from 12 to 24 hours in "many cases I believe the disease is infectious. The poison is most

* *See* Mr. Campbell's experience on p. 66 of this report

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

"virulent when the temperature is at its highest, and the power of "infection lasts for eight days after the temperature has fallen to normal.

"If pneumonia occurs, Influenza is still liable to be caused if a "susceptible individual breathes the expired air of the person suffering "from the pneumonia, and if relapses occur with rises of temperature the "Influenza becomes again infectious."

Dr. Cuff, in his annual report on the Scarborough rural district, in the course of some thoughtful observations on Influenza says :—"It seems "that the disease is infectious during the first 24 hours of its course, and "that the infection lasts at least five or six days."

Dr. Jackson medical officer of health for Barnstaple records a case in which Influenza seemed to have been imported by a convalescent from London five weeks after her recovery. She came on Saturday and left on the following Tuesday, and during the course of the week following her coming three inmates of the house to which she came were taken ill of Influenza.

The history recorded on p. 45 of this report seems to show that Influenza is communicable within a few hours of the commencement of the attack, though not from the time of the earliest symptoms.

Part IV.—Clinical Features of the later Epidemics.

Clinical features and sequelæ. Prolonged debility;

On clinical points I am not able to add much from my own observation to what has been already said.

A characteristic feature of Influenza not sufficiently noticed in my previous report is the prolonged weakness and nervous depression by which it is often followed. Persons previously robust are frequently so pulled down by an attack as not to have regained after many months their previous health and vigour. This prolonged debility seems to be the result of some profound disturbance of nutrition of the nervous system, and in persons of neurotic tendency the malnutrition may result in various affections of that system, such as neuralgia, neuritis, paralysis, epilepsy, and insanity.

The debility left by Influenza has also been thought to predispose the patient to other diseases, as phthisis and the infectious fevers.

lung complications;

There appears to be no doubt that the two later epidemics of Influenza have been marked by a greater tendency to lung complications than was observed in that of 1889–90, and especially to a low and insidious form of pneumonia, to which the mortality from Influenza was in large part due, but in other respects there seems to have been considerable diversity in the experience of medical practitioners in different parts of the country as to the relative features of different epidemics. Thus Sir Peter Eade ("British Medical Journal," August 8th, 1891), remarking on the clinical differences between the epidemic of the spring of 1891 and that of the previous year, states that nasal catarrh was even less prevalent in 1891 than in 1890, but that bronchial irritation was more so, and was often followed by pneumonia and pleurisy. Acute nerve pains at the commencement of the attack and nervous sequelæ were less prominent than in 1890, and such sequelæ as did occur were almost exclusively affections of the sensory and not of the motor nerves. The disease, though more diffused, was milder in 1891 than in 1890. On the other hand, Dr. Robert Simon ("Birmingham Medical Review," January 1892), states that while muscular pains were less marked in 1891 than in 1890, catarrhal symptoms were more prominent, and the disease was, as a rule, much more severe in 1891, and frequently accompanied by pneumonia.

Some practitioners in the eastern counties have observed Influenza to have been accompanied or followed in the later epidemics by symptoms resembling those of cerebro-spinal meningitis.

cerebro-spinal meningitis;

In some cases the outbreak of Influenza has been preceded or accompanied by a prevalence of diarrhœa. Thus Dr. Young, medical officer of health for the Okehampton rural district, in his annual report for 1891, states that during June a large number of cases of gastro-intestinal catarrh occurred. These cases were commonly, and perhaps justly, set down to Influenza. The symptoms present were usually shivering, feverishness, vomiting, diarrhœa, and great prostration of strength. In many cases there was transient cases of jaundice. The disease was too widely spread to be accounted for by any local unsanitary surroundings. A similar prevalence of diarrhœa accompanying Influenza occurred in the Penrith rural district, and recently at Great Waltham, Essex.

diarrhœa;

resemblance to scarlet fever.

The simulation of scarlet fever by cases of Influenza attended with a red rash was noted by some observers in the epidemic of 1889–90 (*see* p. 273 of my former report), and has been observed also in the later epidemics. Thus Dr. Niven, of Oldham, says: "In several "instances I had considerable difficulty in diagnosing between scarlet "fever and Influenza; the erythema, the desquamation, and the straw-"berry tongue of scarlet fever being strikingly reproduced in Influenza, "as well as the course of the fever."

My colleague, Dr. Bruce Low, gives me the following notes collected from medical practitioners in Derbyshire respecting the clinical features of the epidemic of May 1891:—

1. Desquamation was noticed in a proportion of cases either with or without previous rash: the rash when present being variously described as measly or scarlatinal, but generally patchy and mostly on hands and face.
2. Flushing of the face was common, some medical men spoke of their patient's face being like a "boiled lobster." I do not think this was very frequently noticed in the epidemic of 1889–90.
3. Delirium occurred in some cases, and was occasionally of a maniacal kind; at times there were delusions, and a few cases ended in suicide.
4. Earache, with or without resulting discharge, was frequent.
5. Very marked intermissions of pain and other symptoms were noted. Patients could often foretell the hour when they would feel worse on succeeding days. Several medical men, who had been abroad and themselves suffered from malarial fever, said the above fact suggested to their minds the possibility of a connexion between malaria and Influenza, and they treated their cases successfully with quinine.
6. Tonsillitis was a common complication in the recent epidemic, but was of less frequent occurrence in the outbreak of 1889–90.
7. Herpes zoster was seen in a number of cases, as well as herpes labialis in others.
8. A peculiar pungent odour was remarked as coming from the sweat of Influenza cases. This odour caused the medical man to sneeze on entering the room in special instances. The smell was variously described as "peppery," or as "mousey," "fusty," or "mouldy."

Dr. Wood, of Colchester, sends the following notes under date February 14th, 1892:—

"In these cases the cerebro-spinal affection has been very clear; the pain at the back of the head and upper part of spinal cord being very

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

severe. Complications of various kinds have arisen; the peculiar pneumonia, or, as I fancy in old days we would have called it, capillary bronchitis, is undoubtedly very infectious. At a farm about seven miles from this town, the farmer fell ill; pneumonia came on and death took place; his wife fell ill and also his son, both had pneumonia. The wife died the day before the farmer's funeral and the son the day after. Now I hear the two daughters are suffering from pneumonia.

"A fine eruption like that of scarlet fever has been common, and last week I was called to see a lad of 19 on whose body there was a distinct eruption like that of measles. His mother and sister are ill to-day and the same eruption is on them.

"I saw a very bad case at Great Claeton a fortnight ago in a boy of 9 (he was born in Africa), and during his illness severe meningitis came on, with intense sickness. He tried many things to reduce the temperature and sickness, but nothing succeeded until we gave him cinchonidine.

"There were some cases in this locality last year terminating in the same infectious pneumonia.

"In some patients various complications took place, one after another, but I cannot help thinking these were caused by carelessness. A nervous syncope has been very common whenever the upright position was attempted.

"The spleen in some instances has been enlarged."

Dr. Niven, from his own observation and from information received from other medical men in Oldham, gives the following as sequelæ of Influenza which have been noted:—

1. Great prostration, with confusion and an approach to mental and physical paralysis.
2. Neuralgia.
3. Persistent dyspepsia.
4. Abdominal pains.
5. Inflamed throat.
6. Rheumatism.
7. Phlebitis.
8. Meningitis.
9. Deafness.
10. Peritonitis.
11. Pericarditis.
12. Excessive prolongation of subnormal temperature, as sometimes observed after scarlet fever.
13. Diarrhœa.
14. Desquamation of tongue and fauces as in scarlet fever.

Diagnosis between catarrh and Influenza.

Dr. E. W. Barton, of Market Rasen, gives the following as diagnostic marks between ordinary catarrh and mild cases of Influenza:—

1. Ordinary catarrh is not infectious; it does not spread through a household as Influenza does. (This is opposed to popular belief.)
2. It commences with dryness of the nostrils or throat on one or both sides.
3. The discharge from the nostrils is acrid, often markedly so, and tends to excoriate the skin.
4. There is not the same tendency in ordinary catarrh as in Influenza to pains in the back and in the course of the spinal nerves; the dental and frontal nerves are most affected.
5. There is a tendency in ordinary catarrh for the fulness and congestion about the head to increase towards night.

6. Ordinary catarrh is often accompanied by muscular rheumatism, which makes the diagnosis between the two diseases more difficult.
7. In ordinary catarrh the patient feels more malaise before the discharge commences; running from the eyes and nose comes as a relief. In Influenza the reverse is the case.
8. Influenza is followed by greater and more persistent prostration.

Part VI.—Reports on Outbreaks of Influenza, in 1891, in certain Institutions and Establishments.

Influenza in workhouses and other establishments.

In this place I give a series of reports on outbreaks of Influenza in Workhouses, obtained through my colleague in the Poor Law Department of the Board, Dr. Downes. This class of institutions had seemed to have been comparatively little liable to suffer from Influenza in the the epidemic of 1890, but whether this were so or not, many outbreaks at workhouses have occurred during the two later epidemics. It will be seen that the experience at workhouses is quite consistent with the views set forth in my first report as to the method of spread of Influenza: there has been in many cases a history of introduction from outside, the workhouse often not having been attacked until after the disease had been prevalent for some time in the outside population. The epidemic outburst was preceded by a succession of preliminary cases. The progress of the epidemic in workhouses seems to have been often comparatively slow, as might be expected from the circumstance that a workhouse consists of a number of buildings containing inmates of different classes; one class having comparatively little intercourse with another. In some instances a number of inmates of one class have been taken ill in a batch at about the same time.

I add also a report by myself on an outbreak of Influenza, with unusual and grave clinical characters, which occurred at an Industrial School in Kent, early in the epidemic of 1891–2; some notes, by Dr. Male, of Grays, on an outbreak of Influenza on the training ship "Shaftesbury," in the spring of 1891; and an extract from the annual report of Dr. Walter Dickson, R.N., late Medical Inspector to Her Majesty's Customs, on Influenza in that force at the Port of London in 1891.

Report on Influenza in the Sculcoates Union Workhouse, Hull, 1891; by J. Dix, Medical Officer.

Hull, June 22, 1891.

In Sculcoates Union Workhouse.

The origin of the attack was, as far as I know, spontaneous; and it began in the workhouse about the same time as in the other parts of the town. It pervaded all parts of the house, but was most severe and most prevalent in the bed-riddden wards.

A few of the children in the schools, a detached building, were also attacked. Many of the sick in the infirmary, suffering from various other complaints, had also Influenza, but as their names and diseases were already entered in the "Medical Relief Book," and no special note was made at the time of the supervening Influenza, this return, so far as as they are concerned, is necessarily but a rough calculation, and the dates of the attack are not recorded, but the estimate here given is fairly accurate.

Dates of Attack.

March	24	-	1
„	28	-	1
„	30	-	2
„	31	-	1
April	1	-	1
„	2	-	2
„	3	-	4
„	4	-	2
„	7	-	4
„	8	-	5
„	9	-	3
„	10	-	4
„	11	-	4
„	12	-	3
„	13	-	1
„	14	-	3
„	15	-	7
„	16	-	3
„	18	-	2
„	20	-	1
April	21	-	2
„	22	-	3
„	23	-	3
„	24	-	4
„	25	-	1
„	26	-	3
„	27	-	1
„	28	-	1
„	29	-	3
„	30	-	4
May	1	-	3
„	3	-	2
„	4	-	3
„	5	-	1
„	6	-	1
„	10	-	1
„	13	-	1
		Total -	91

The number of people in the hospital at the time of the outbreak was 81, and about two-thirds of these, *i.e.*, 54, also had the Influenza, making a total of 145 persons attacked.

Report on Epidemic Influenza of 1891, in the Nottingham Union Workhouse; by H. G. Ashwell, Medical Officer.

The epidemic had been prevalent in the town some little time before it became so in the workhouse.

The first cases that were under treatment came into the hospital from the town while suffering from the disease.

The large majority occurred during the month of May.

In all 125 individuals (excluding 3 officers) were attacked, the proportions being as follows:—

56 men:	Average number in the house				338	- 16·6	per cent.
61 women:	„	„	„	„	273	- 22·4	„
8 children:	„	„	„	„	50	- 16·0	„
125					661	19·0	„

Of the resident officers, the master, the labour master, and the porter had attacks followed by bronchitis. No case occurred amongst female officers.

No. of male officers - - - - -	9
„ female „ - - - - -	11

In all there were 10 deaths; the average being 60 years.* All cases in which death occurred, except one, were complicated by some pulmonary disorder.

* Excluding the fatal case of a woman of 29, who aborted on the second day of attack, the average age of the patients who died was 64 years; the youngest being 45 years old.

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

With regard to the spread of the disease, it appears to be infectious in its character. When one case occurred in a ward, several others almost invariably followed. Against this, however, is the fact that all were exposed to the same atmospheric surroundings.

No isolation was possible on account of the limited accommodation.

The numbers of inmates attacked on the several dates, as extracted from a table accompanying this report, were as follows:—

Date.	Men.	Women.	Children.	Total.
March 27 - -	—	1	—	1
April 10 - -	—	1	—	1
„ 19 - -	2	—	—	2
„ 21 - -	2	—	—	2
„ 25 - -	—	1	—	1
„ 27 - -	1	—	—	1
„ 28 - -	2	—	—	2
„ 30 - -	2	—	—	2
May 2 - -	3	—	1	4
„ 4 - -	3	—	—	3
„ 5 - -	2	—	—	2
„ 6 - -	2	1	—	3
„ 7 - -	—	1	1	2
„ 8 - -	4	4	—	8
„ 9 - -	2	3	—	5
„ 10 - -	1	7	1	9
„ 11 - -	—	1	—	1
„ 12 - -	3	5	—	8
„ 13 - -	2	3	—	5
„ 14 - -	1	4	—	5
„ 15 - -	2	4	—	6
„ 16 - -	2	1	—	3
„ 17 - -	3	—	—	3
„ 18 - -	1	1	—	2
„ 19 - -	1	2	—	3
„ 20 - -	4	2	—	6
„ 21 - -	1	2	—	3
„ 22 - -	1	1	—	2
„ 23 - -	—	2	—	2
„ 24 - -	1	3	—	4
„ 25 - -	4	1	—	5
„ 26 - -	1	1	—	2
„ 27 - -	1	—	—	1
„ 28 - -	1	4	—	5
„ 29 - -	—	1	—	1
„ 30 - -	—	3	—	3
June 2 - -	1	—	—	1
„ 5 - -	—	1	1	2
„ 6 - -	—	—	4	4
	56	61	8	125

The progress of the epidemic seems to have been comparatively gradual, and its stress fell upon the men before the women, and last of all on the children.

Report on Influenza in the Saddleworth Union Workhouse; by Colin G. Campbell, Medical Officer.

In Saddleworth Union Workhouse.

The first person attacked in the house, April 15th, was the matron. She had not been out of the house for many weeks, but three days before her illness commenced a number of people came to the work-

house in connexion with counting the votes for the guardians' election. I am unable to state whether any of these came from infected houses for the epidemic had only commenced in the district. Three days later three of the inmates were attacked, the next day there were six on the books. In a week we had 27 cases, and in the course of another week 35 inmates were attacked.

I herewith append their ages:—

Ages.	Over 5 yrs & under 10 yrs.	Over 10 yrs. & under 25.	Over 25 yrs. & under 50.	Over 50 yrs. & under 60.	Over 60 yrs. & under 70.	Over 70 yrs. & under 80.	Over 80.	—
No. attacked	2	3	7	6	9	5	3	Total 35.

Equal to 58 per cent. of inmates.

One of the remarkable facts about the epidemic in the workhouse was that all the cases—old and young—presented the same initial symptoms, *i.e.*, sickness and vomiting; whereas the early symptoms of this disease generally are varied in an extraordinary manner. All the cases had accompanying bronchitis, whilst six developed pneumonia in the later stages.

Pneumonia and pleuro-pneumonia are amongst the most fatal complications of this terrible disease. My experience distinctly indicates two forms, the one commencing *with* the disease, generally fatal, the other commencing when the Influenza has apparently run its course. Our six cases all were in the latter category, and all recovered.

The fact to which I desire to draw your official attention is that we have not had *one death* either directly or indirectly due to the disease. This is the more remarkable inasmuch as there are no "able-bodied" patients; they were all feeble, either mentally or bodily, and many had exceeded the "three score years and ten."

This remarkable immunity from death is due, in my opinion, in a great measure to the indefatigable care and attention given by the matron, Mrs. Wood, to the inmates. I cannot speak too highly of her nursing, at a time when she sorely needed nursing herself.

Report on Influenza in the Louth Union Workhouse; by C. J. Myers, Medical Officer.

Louth, June 13th, 1891.

In reply to the questions of the Local Government Board respecting the outbreak of Influenza in the workhouse:—

1. The following is the proportionate number of persons attacked of each class, including officers, viz.:—

Class 1	2 to 17	Class 6	4 to 15
„ 2	4 to 23	„ 7	3 to 9
„ 3	3 to 8	„ 8	1 to 3
„ 4	1 to 6	Officers	4 to 6
„ 5	7 to 20		

2. The circumstances attending the origin of the outbreak and its spread:—

The date of the first recognised cases of Influenza in Louth was March 19th, 1891. Odd cases kept coming under treatment till about April 21st, when it became epidemic in the town and neighbourhood.

The first case in the workhouse occurred on April 15 in class 7, and was a girl attending the National School; the next case was that of the portress, two days later, and on the 20th the industrial trainer, and two more girls in class 7, attending the National School. It was two days later still, or a week after the first case, before any males were attacked, so that it was apparently introduced by the girls in class 7 attending the National School. This was the time when Influenza became epidemic in the town.

3. The following are the dates of the several attacks:—

April 15	- - 1	April 28	- - 3
„ 17	- - 1	„ 30	- - 3
„ 20	- - 3	May 2	- - 3
„ 22	- - 2	„ 8	- - 2
„ 23	- - 1	„ 9	- - 1
„ 24	- - 2	„ 13	- - 2
„ 25	- - 2	„ 20	- - 1
„ 26	- - 4	„ 27	- - 1

4. The result of each attack:—

All the cases recovered except one, a delicate woman of 55, who succumbed to bronchitis following the Influenza.

Most of the cases commenced with frontal headache, or frontal neuralgia, accompanied with shivering and a high temperature. There have been a few cases with intense abdominal pain and diarrhœa, and a few with temporary paraplegia.

Report on Influenza in the Wakefield Union Workhouse; by W. Roulston, M.D., Medical Officer.

Wakefield, June 12th, 1891.

The first case of Influenza in the Wakefield Union Workhouse occurred in a woman—a "worker" in the hospital. She was attacked on April 10th, 1891, at which time the epidemic was prevalent in the town of Wakefield, and was rapidly increasing. Later on the same day an old woman, a patient, was attacked, and on the morning of the following day one of the male patients was down with it.

On April 14th the master began to suffer, and was immediately followed by his wife. These two being the first cases from the "body of the house."

On the 16th April were 10 fresh cases.
„ „ 17th, 18th April were 7 fresh cases.
„ „ 20th April „ 13 „ „
„ „ 23rd „ „ 23 „ „
„ „ 27th „ „ 9 „ „
„ „ 29th „ „ 5 „ „

Those who suffered were—

37 males.
23 females.
7 children.

Most of those were patients confined in "hospital," only nine of the above-enumerated cases occurring in the "house." Several isolated cases have been met with since the epidemic, those being almost all found in "casuals" who presented themselves at the gate, and were discharged as soon as better.

Report on Influenza in the Kendal Union Workhouse, Milnthorpe; by J. C. Carden, Medical Officer.

Milnthorpe, June 28th, 1891.

On the 3rd May 1891 an epidemic of Influenza broke out in this workhouse.

The disease was first contracted by the porter, about the last week in April, and after him, by his wife and child.

In my opinion the porter contracted it from the vagrants, and from the porter's lodge it was introduced to the house.

On May 3rd seven of the young women were simultaneously seized with rigors, pain in the back, headache, and fever, the temperature varying from 103° to 104°·5, and with that exception, the symptoms in each case were remarkable in their similarity.

On the 5th those symptoms had disappeared, leaving the patients very weak. They were all convalescent on 10th, except one who contracted pneumonia, and ultimately recovered. The older women were then attacked, but the prostration seemed more severe, and six, who developed bronchitis, died from that cause. The matron and the nurse for the imbeciles were then smitten, and a few more cases occurred on the men's side. The master and the other nurse were also ill. It is remarkable that the women suffered more than the men, not only in the number attacked, but also in the severity of the symptoms.

The men's rooms are situated with a southerly aspect, and are warm and sunny.

The women's rooms are situated on the opposite side, with more of a northerly aspect.

The epidemic lasted about three weeks, only a few mild cases occurring after that time.

The following information is also furnished by Mr. Carden:—

The inmates of the workhouse at the time of the epidemic were 101 in number, besides the master, matron, two nurses, and the porter and his family. Of the inmates only nine were children. Among the inmates 41 cases of Influenza occurred. The dates of commencement of the cases were as follows:—

Dates.	Inmates attacked.	Staff attacked.
April 24 - -	—	Porter.
May 1 - -	—	Porter's wife.
,, 3 - -	7	Porter's child.
,, 4 - -	3	—
,, 5 - -	3	—
,, 6 - -	5	—
,, 7 - -	6	Matron and nurse.
,, 8 - -	1	Master and nurse.
,, 10 - -	1	—
,, 14 - -	2	—
,, 16 - -	7	—
,, 17 - -	3	—
,, 18 - -	2	—
,, 19 - -	1	—
	41	7

During the epidemic 10 deaths occurred; all from diseases of the respiratory organs. Of these, five were certified as from Influenza and bronchitis; one from Influenza and asthma; one, of an infant, from bronchitis; two from phthisis, and one from pneumonia.

The porter's lodge is separated from the workhouse by a large yard. In Mr. Carden's opinion the disease was communicated to the porter by a vagrant. The porter has to go to and from the house for the vagrants' food. The porter's linen is not washed at the workhouse laundry; but the bedding and night clothing, &c. used by the vagrants are washed in the laundry in the workhouse, and Mr. Carden believes that by this means the Influenza was introduced to the house.

The fact that the earliest cases were young women who would probably be employed in the laundry seems in favour of this view, and the circumstance that the outbreak began by seven of them being taken ill on one day points to infection received at one and the same time from a common source.

Report on Influenza in the Boston Union Workhouse; by W. H. Smith, Medical Officer.

Boston, July 4th, 1891.

Influenza first made its appearance this year in the Boston Workhouse on the 19th day of April, but until the 9th of May only four cases were admitted into the infirmary. About this time several of the schoolboys fell ill, and some ten days later many of the schoolgirls began to suffer. The epidemic now became general throughout the house. All the boys and girls, numbering 78, suffered more or less, with two exceptions, a brother and sister. After a few days illness most of these patients were convalescent, and all eventually recovered.

Altogether about 78 patients* were prostrated by the pestilence, and about 40 more suffered in a minor degree, but, though taking medicine, were never absolutely incapacitated. There were four deaths: in three, the immediate cause of death was bronchitis, and in one, general debility.

Out of the seven resident officers four were attacked. On May 7th, when travelling on business connected with the union, the matron developed the disease, and was confined to her bed in Derby for a fortnight. The master was ill for about 10 days, but never entirely gave up work. The nurse was confined to her room May 13th to June 8th, and one of her substitutes was incapacitated for a few days. The sewing maid was also unable to leave her room from May 13th to June 8th.

The epidemic appeared to be malarial in character, but, from the rapidity with which it spread through a section of the building when once it gained admission, I was led to suspect that it was infectious.

Appended was a list of 78 cases with some details. The following table shows the daily numbers of new cases:—

* The inmates of the workhouse on June 4th numbered 241; so that the proportion who suffered from Influenza must have been altogether nearly one half.

Date.	Cases originating in Workhouse.	Cases admitted from Outside.
April 18	1	1
„ 27	1	—
May 6	—	1
„ 9	1	1
„ 10	5	—
„ 11	1	—
„ 13	1	—
„ 14	—	1
„ 17	8	—
„ 19	1	—
„ 20	5	—
„ 21	5	—
„ 24	42*	—
„ 26	2	—
„ 27	2	1
„ 30	1	1
Total	71	6

Influenza in the Frodsham Union Workhouse.

In Frodsham Union Workhouse.

Dr. Robinson, medical officer, states that Influenza had its origin in the workhouse, in a tramp admitted ill on May 4th. The next patient, a woman of 64, was taken ill on May 10th; how she contracted it he was unable to ascertain. Then followed the master of the workhouse, and three other men were admitted to the hospital from outside, and on May 18th, 19th, and 20th it broke out all over the hospital, and a little later among the inmates of other parts of the house. The total number of cases was 35, and of deaths, 7.

The following are the number of attacks on the several dates:—

Date.	Cases admitted from Outside.	Cases originating in Workhouse.
May 4	1	—
„ 10	—	1
„ 11	1	1
„ 13	1	—
„ 16	1	1
„ 18	1	2
„ 19	—	2
„ 20	—	4
„ 22	1	6
„ 27	1	—
„ 29	—	2
„ 30	—	1
June 2	—	1
„ 3	—	3
„ 8	—	1
„ 14	—	1
„ 18	1	—
„ 29	1	—

* 38 of these were children under 16 years.

Reports on Influenza in the Houghton-le-Spring Union Workhouse, Durham; by D. S. Park, Medical Officer.

I.

Houghton-le-Spring, November 5th, 1891.

Ann Bolam, aged 37 years, an inmate of Houghton-le-Spring Workhouse, was removed to the infectious hospital of that institution on the 29th October, suffering from Influenza. On the 3rd of the present month the disease broke out all over the workhouse Some of the officials—including the matron and nurse—are also suffering from the disease. The complications are chiefly bronchitis and pleurisy, which greatly aggravates the disease in the young and old. In the town and district the disease broke out about the end of April, and still continues with little abatement, and is not infrequently accompanied with pneumonia.

II.

Houghton-le-Spring, November 1891.

The total number of cases of Influenza in the workhouse has been 39. As was to be expected, the incidence of the disease fell with greatest severity upon those under treatment at the time, especially the old and debilitated. Ann Bolam, the first case that came under my notice, was removed to the infectious hospital on the 29th of October, the day on which she took ill, and thus the case was isolated from the rest of the ordinary patients. Next day there did not appear to be any unusual symptoms among the other patients, and I detected nothing which might have led me to anticipate the epidemic which has since appeared. But on the 1st November a batch of cases, 18 in number, were visibly affected with the usual symptoms of Influenza. On November 2nd, 11 other cases occurred, and so on, as a glance at the table will show. The outbreak was so sudden, that it was found impossible to remove the cases to the infectious hospital, and, as the disease broke out in various wards simultaneously, no good could have been obtained. It is, however, singular and worthy of remark that no cases occurred in the old men's wards, although these enjoy no privileges which are not also extended to the inmates of the aged female wards.

On the 29th October the number of inmates was as follows:—

Men		38
Women		36
		74
Boys		13
Girls		12
		25
Total number of inmates		99

Thus the total number of inmates on 29th October, being 99, the proportion of seizures up to the present date is 39·3 per cent. of the whole number of inmates. But although the number of cases may be received as 39, I have no doubt that other cases occurred of so mild a character and showing symptoms so obscure that I did not feel justified in classing them as cases of Influenza, and among the number stated

(39) a great proportion were cases of a very mild character. Though some of the officials, including the nurse and matron, were seized, they were still able to discharge their duties. The nurse alone was obliged to remain off duty for one day, but the portress very ably supplied her place during that short interval. The mortality connected with the epidemic has been heavy, as four deaths have occurred; but this must by no means be taken as any criterion of the severity of the epidemic, for all the fatal cases occurred to aged persons who were already under treatment for other diseases, and who in fact were in a dying state before they were seized with the disease. How the disease was first introduced into the wards there is no evidence to show, as the inmate, Ann Bolam, who was the first seized, was employed at the time in the laundry, which is not in direct communication with any of the wards, and a good distance from them. But there is more reason to be surprised that the institution has been so long free from the disease, for I can find no record of its presence in any of the books, nor in the medical journal, and neither the master nor any of the officials has any knowledge of its ever having existed within the walls. As an evidence of the extent to which the disease prevails among the inhabitants of the district, I may mention that in my own private practice there are at the present moment over 200 cases of Influenza on my books, and, as I said before, the only ground for surprise is that the workhouse has escaped so long. The dates of attack cannot be accepted as absolutely correct, but I have filled up this part of the table to the best of my ability and judgment. There have been three cases of relapse, and two of them, Robert Wilson, wardsman, and Ann Bolam, have again been confined to bed.

With regard to the disease itself, the more severe cases complained of headache, and a feeling of giddiness with sore throat, and pains in the back and legs. The tongue was coated and of a yellowish-brown colour, but usually resumed its natural appearance in three or four days, by which time the headache had disappeared, and generally also the pains in the back and legs. Heats and chills also alternated in the earlier stages of the disease. There was in every case the liquid or suffused eye, and in children vomiting was not uncommon. At first there was little or no cough, except in cases complicated with bronchitis or pleurisy, but in a few days the usual symptoms of a severe cold supervened. The temperature rose rapidly, and remained high until the disappearance of the headache. There was also a corresponding acceleration of pulse, with prostration and general weakness. These were the general symptoms of the disease.

The following are the numbers attacked day by day:—

Date.	New Cases of Influenza.
October 29	1
November 1	18
„ 2	11
„ 4	4
„ 8	3
„ 13	2
	39

Only four of the cases were in children.

Dr. Prior, medical officer of health for the Biggleswade rural district, states that Influenza was introduced into the Biggleswade Union Workhouse by a known channel, and that among about 133 inmates, there were 120 cases and 9 deaths. The chaplain and the medical officer also took the disease, and the former died. The workhouse is healthily situated outside the town.

Influenza at the St. Vincent's Industrial Schools, Dartford, Kent.

The aid of the Local Government Board having been requested by the Home Office in investigating and advising as to a serious epidemic, suspected by the medical officer of the school to be Influenza of a severe type, which had broken out at the St. Vincent's Industrial School, in accordance with instructions, I visited the school on October 28th, 1891, when I found 35 boys, out of a total of 200, ill in bed and undoubtedly suffering from epidemic Influenza. Two other boys had recently died after short illnesses, but it seemed doubtful whether their cases were of the same nature. The dates of commencement of the earlier cases, as taken from the school infirmary register, were as follows:—

Date.	Initials.	Nature of Case as entered.	——
October 20 - -	R. M.	Acute hepatitis -	Died October 20.
„ 21 - -	D. C.	? -	Died October 21.
„ 22 - -	J. J.	Sickness.	
„ 22 - -	J. H.	„	
„ 23 - -	J. R.	„	
„ 23 - -	W. McD.	Pneumonia.	
„ 23 - -	E. L.	Sickness.	
„ 23 - -	S. G.	„	
„ 25 - -	G. B.	„	
„ 26 - -	W. A.	„	
„ 26 - -	C. B.	„	
„ 26 - -	G. M.	„	
„ 26 - -	T. McC.	„	

The remaining cases, about 24 in number, had been taken ill on the 27th and 28th. One of the "brothers" who had assisted in the nursing was also ill.

The symptoms were of a very severe type, and except as regards the first two cases, had been very similar. The illness began suddenly with a feeling of chilliness, frontal headache, nausea or vomiting, and great prostration. High fever rapidly came on; in one case a temperature of 104·8° was observed within three hours after the commencement of the attack. A boy seen by me, who had been taken ill only 16 hours before, had a temperature of 103° and a pulse of 132; he had a dry brown-coated tongue, and sordes on the lips, and lay in a state of great prostration, closely resembling a patient in the second or third week of typhoid fever. I subsequently learn that he had for about eight days continuous delirium, sometimes very violent, but eventually recovered, though he remained long in a weak anæmic condition. Many of the patients had earache; several were delirious. Nausea was present in every case and vomiting (sometimes almost uncontrollable) in most,

but not one had diarrhœa. Coryza was absent; there was a slight cough in some cases; and two showed signs of pneumonia, which in one was eventually fatal. Herpes labialis occurred in many cases, of which all were severe and one of them was fatal, but no other eruption on the skin was noticed. Muscular pains were not prominent. One case had severe epistaxis; and hæmaturia was a frequent symptom, being noted in about 40 of the 102 cases which in all occurred up to the end of the outbreak. On a post-mortem examination of one of the early cases the kidneys were found much enlarged and filled with blood.

The first supposed case, R. M., aged 14, was a weakly boy who had been in the school for six years; he worked in the tailor's shop, not being strong enough for out-of-door work. He often complained of abdominal pain and had been in the infirmary on that account in August. He was taken ill in getting up at 6 a.m. on October 20th, and admitted to the infirmary at once. He then complained of pain in the head and in the right side over the liver, and he vomited matter tinged with bile. There was slight discolouration of the skin, hardly amounting to jaundice. There was great prostration; the pulse was very rapid and thready, almost imperceptible; the respiration quick and shallow, and the temperature subnormal (95° F.), and the voice very weak. The pupils were normal. There was no diarrhœa. His legs were not drawn up as he lay in bed. He was sensible up to about 6 p.m., but passed gradually into a state of stupor, from which he was roused with difficulty, and he was unconscious before his death, which took place at 8.30 p.m. after only 14 hours' illness. (This account of his symptoms is given me by Dr. Hamilton, medical officer to the school.) Although this case began, like the subsequent ones, with vomiting and headache, the absence of elevation of temperature is unusual in Influenza, though it is recorded to have been observed in other cases.* The history of previous abdominal pain and the sudden collapse suggest perforating ulcer of the stomach, but the ordinary symptoms of peritonitis do not seem to have been present. I inquired about poisonous berries or toadstools, but could only learn that R. M. had been seen to eat some haws on October 18th. It was stated that when out for a walk that day he had passed a heap of sewage sludge, but this, which I saw, was too old to have been then offensive, being overgrown with weeds.

The second boy, D. C., age 11, was taken ill on October 21st at noon with pain in the head and vomiting. An aperient powder was given him, but he was not seen during life by a medical man, and no clear history of his case was obtained. The vomiting continued, and he sank and died quietly at 8.30 p.m. on the day of attack. A post-mortem examination was made by Dr. Hamilton, but nothing was found to account for death. The liver was somewhat enlarged, the kidneys congested, and the spleen somewhat so. No abnormal contents were found in the stomach or intestines except in the latter a small loose chip of wood. A coroner's inquest was held, but at the time of my visit stood adjourned to November 13th. Professor Pepper, to whom the contents of the stomach had been sent for analysis, at the adjourned inquest gave it as his opinion that death was due to blood poisoning from some acute specific disease, probably Influenza.

Another case, with symptoms similar to the above, occurred at the end of the epidemic. The boy was taken ill on November 22nd, and

---

* "Influenza with severe abdominal pain and collapse," by R. M. Simon, M.D., "Brit. Med. Journal," June 13, 1891. "Apyrexial Influenza," by H. W. Godfrey, M.B., *ibid.*, July 18th, 1891.

died after an illness of 16 hours. No post-mortem examination was obtained.

Although the nature of the first two cases appeared obscure at the time, yet upon subsequent consideration, and in view of the history of the outbreak, I am inclined to consider them to have been Influenza of an unusually severe "fulminating" type; analogous to the cases sometimes met with in an epidemic of scarlet fever or measles in which the patient is struck down and dies before the typical symptoms of the disease have had time to develop.

No history of the introduction of Influenza was obtained, but Dr. Hamilton had seen some cases, which he believed to be of that disease, in Dartford within the few days preceding my visit. Only four newcomers had been received into the school during October, the last of these being on October 14th; this boy had been in a workhouse for a fortnight before his admission. The last visiting day before the outbreak was so far back as October 4th. The boys go out for a country walk every Sunday afternoon, the latest occasion before the outbreak having been on October 18th; they are also sent on occasional errands to Dartford, &c.

The staff, who are members of a religious order, reside on the premises; there are also four or five artizan instructors who live outside. Bread, meat, groceries, butter, &c. are obtained from Dartford, but milk from cows kept on the premises. Washing is done at home.

The cases of Influenza occurred at random throughout the school, with no special incidence on any group of boys. The whole of the boys, however, are much mixed up together; they all use the same schoolroom, workroom, and dining hall, and they sleep in two large dormitories containing one 130, and the other 70 beds. The infirmary wards communicate with the rest of the building, so that nothing like isolation was possible.

The epidemic continued to spread through the school up to the middle of November, in all 102 boys out of 200 being attacked, or more than half, and four died. Two of the "brothers" also, who had attended on the sick, were attacked, and one of them died of pneumonia. The dates of the cases, as given me by Dr. Hamilton, were as follows:—

Date.	Attacks.	Date.	Attacks.
October 20	1*	November 1	6
„ 21	1*	„ 2	9†
„ 22	2	„ 3	4
„ 23	4	„ 4	6
„ 25	2	„ 5	6
„ 26	10	„ 6	1
„ 27	14	„ 7	1
„ 28	12	„ 8	5
„ 29	6	„ 19	1
„ 30	5	„ 22	1‡
„ 31	5		
			102

* Died. † One of these died November 10. ‡ Died November 23.

The school is in an open situation, overlooking the Thames valley, about a mile from Dartford; the soil is gravel over chalk. The health of the school has generally been good, although the boys are of a low class. Only five deaths had previously occurred there since it was opened in

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

1879. Influenza was prevalent there in January 1890; 22 cases are entered in the infirmary register, but the disease was of a mild type. 14 cases of Influenza also occurred at various dates between March 27th and June 16th, 1891. A good many of the boys who had the disease before suffered again in October 1891, and one boy had had it on all three occasions.

No explanation can be given of the severity of the later epidemic as compared with the mild character of the previous ones.

Influenza on training ship "Shaftesbury."

EXTRACT from MEDICAL NOTE BOOK of TRAINING SHIP "SHAFTESBURY," Grays, Essex, Re Outbreak of Influenza, March and April 1891; by H. MALE, M.D., Medical Officer.

Date.	Number of Boys affected.	Disease.
March 20	1	"Catarrh."
„ 21	1	"Catarrh."
„ 27	1	Pneumonia.
April 3	2	Pneumonia.
„ 3	1	"Catarrh" (Influenza).
„ 4	1	Influenza.
„ 5	1	Pneumonia.
„ 5	1	Influenza.
„ 7	1	Pneumonia.
„ 7	1	Influenza.
„ 8	1	Pneumonia.
„ 8	1	Influenza.
„ 12	2	„
„ 13	4	„
„ 14	2	„
„ 15	1	„
„ 16	13	„
„ 17	5	„
„ 19	5	„
„ 20	3*	„
„ 21	2	„
„ 23	1	„
	4	

* One of these had slight pneumonia.

After April 23rd no serious cases occurred among the boys, and consequently none were admitted to the infirmary. A good many mild cases, however, occurred, but these were treated on board in a part of the ship screened off for the purpose. No special note was taken of these cases, but they continued to crop up till about the end of the first week in May.

In all about 80 boys were affected out of 409 boys on board at the time.

A large number of the boys complained at first of "pain in the side" of the chest, so much so that it was thought to be from imitation. There was an absence of catarrhal symptoms. Many complained of abdominal pain at the commencement, and vomited.

In the majority of cases the illness only lasted two or three days, the more serious cases from four to eight days.

The cases of pneumonia commenced as such, and were not preceded by ordinary symptoms of Influenza. The onset as regards symptoms

was rapid, but the physical signs in some cases seemed slow in developing.

Several cases were suspected to be commencing pneumonia, but did not develop.

One boy died of pneumonia on the ninth day. On the third day of his illness he had symptoms of acute suppuration of left ankle joint. (He had also old-standing disease of the aortic valves.) On post-mortem examination were found greatly thickened pleura of the left lung, with deposit of lymph on surface; pneumonia of the lower two-thirds; and contracted aortic valves. The left ankle joint contained pus, and there was a small patch of necrosis in front of articular surface of astragalus.

In another boy (admitted on the same day as the boy last mentioned) lung did not clear up and showed evidences of phthisis. He recovered, however, after prolonged illness.

This outbreak was more gradual in its development and lasted over a longer period than that of 1890. The earlier cases were not recognised as Influenza. Though fewer boys were affected, the disease was of a more severe type, and seemed to have a special tendency to develop chest complications.

In the epidemic of 1890 no case of pneumonia or other complication occurred, though 204 boys had the disease out of 394 then on board.

The outbreak in 1891 occurred in the ship about the same time as it began to be prevalent on shore. There is frequent communication between the ship and shore, but there was no special circumstance to favour its development.

Dr. Male states that many of the boys who had Influenza were also affected last year, but is unable to give the proportions of those then attacked who suffered and escaped on the later occasion.

Extract from the Annual Report on the Health of the Inferior Officers of Her Majesty's Customs in the Port of London in the Year 1891; by Walter Dickson, M.D., R.N., Medical Inspector.

Both years (viz., 1890 and 1891) are notable for the visitation of Influenza, which fell on the Customs force in nearly the same proportion in both epidemics: and this ratio is probably a fair measure of the incidence of this remarkable form of fever on the general population. The year 1891 witnessed two epidemics, one in May and June, the other, the third invasion of the malady, being in December (with eight cases in that month) and increasing in the new year to as formidable proportions as its predecessors.

In all these, with slight variety, the main features were alike. The same acute fever and prostration at the onset, the same proclivity to chest complications, whether from exposure to cold, or from a morbid bias of the essence of the malady, and the same persistent debility retarding convalescence. It is this last peculiarity that renders it so unwelcome an illness in the public service, necessitating a longer absence than most other slight forms of disease. In this force no death could be attributed directly to it, and only one indirectly, in which the bronchitis that followed, developing into consumption, proved fatal in five months. The simultaneous or speedy attacks in families of various members lent force to the opinion of its infectious nature which has lately gained ground. So far as could be ascertained, the instances in which other members of the household of the affected officers also suffered were about 10 per cent. of the whole. In regard to the question

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

of relapse or occurrence of the disease a second time, only 5 per cent. of the 112 cases under observation were found to be so affected.

The first case recorded in the London division of the force was on April 20th, the last on June 18th; in all 52 cases came under treatment, and in the Gravesend division 22 others about the same time were also entered. The proportion of officers affected was therefore 11 per cent. The weather in May being cold and wintry, pulmonary complications were frequent, and other troubles arose to intensify the epidemic, and it is matter for congratulation that, although the working power of the force was much crippled for a quarter of the year, the rate of mortality and superannuation was not affected, and that if the recoveries were protracted, they were complete. The disappearance of the disease in July and its recrudescence in the beginning of December, and its continuance through the exceptionally severe winter of 1891–92 are noteworthy incidents of this scourge, which has proved so disastrous in many sections of the community, but has passed comparatively lightly over this Department.

Part VII.—Prophylaxis.

Measures of prevention:

It is to be regretted that at present our knowledge of the pathology of Influenza does not enable us to advise any measures of precaution further than those contained in the Provisional Memorandum issued by the Board on January 23rd, 1892, and which is appended to this report.

isolation;

Of the possibility, by timely isolation and other precautions, of preventing Influenza from spreading, instances may be given.

Dr. Prior, medical officer of health for Bedford, states that the Bedford Union Workhouse, containing 200 inmates, remained free while Influenza was epidemic in the town. A man came in with symptoms of Influenza, but was isolated and the disease did not spread.

Dr. Cuff, medical officer of health for the Scarborough rural district, says: "I have seen in several instances the disease undoubtedly confined "to one member of a household by isolation; twice this happened in "my own house, nearly a year clapsing between the cases."

Dr. Newsholme, medical officer of health for Brighton, states that the Borough Sanatorium, being very strictly isolated in every respect, escaped during the first two outbreaks of Influenza, and in the third until a servant who had been absent on leave for a few days returned to the Sanatorium and almost immediately developed Influenza. Her sister caught the disease from her, but these two patients having been strictly isolated, there was no further spread of the disease in the establishment.

Mr. Miller, medical officer of health for the Bridgend and Cowbridge district, says: "In proof of the apparently satisfactory manner in "which the disease may be prevented from spreading by suitable "means being taken to ensure disinfection and isolation, I can only "say that I have not seen it to my recollection spread to the other "members of a household where strict precautions were adopted."

The instance given by Dr. Blakely on p. 46 of this report may be noted in this connexion, and other instances of successful limitation of Influenza by isolation are recorded by Dr. R. Sisley in a paper read before the Society of Medical Officers of Health, January 1892.

limitations to its practicability;

A limit to the possibility of stamping out Influenza by isolation has to be pointed out, viz., that such isolation as is practicable cannot be

complete. A sick person with an infectious disease must have attendants to supply his needs, and if these attendants be not protected in some way the disease will spread among them, and from them perhaps to other persons outside the place of isolation. Possibly Influenza is less liable to spread in the spacious and well-ventilated wards of a well-kept hospital than in an ordinary dwelling-house, but so far as we know, there is no means of personal protection; even one attack of the disease affording but a very partial protection against another.

Other difficulties in the way of limiting the spread of Influenza by isolation are that the disease is probably infectious in an early stage before its nature is fully declared, and that as many of the patients are adult bread-winners it would be difficult to detain them sufficiently long to ensure that infectiousness had ceased.

Mr. H. B. Collins, however ("British Medical Journal," March 5th, 1892), from inquiries into the behaviour of Influenza in various model dwellings and old houses let in lodgings, infers that even incomplete isolation with cleanliness readily checks the spread of the disease; whereas in old houses let in single rooms the disease in most instances spread through the house.

notification.

As a corollary of the recognition of Influenza as a "dangerous infectious disorder" it has been urged, more especially by Dr. R. Sisley, that it should be included among the diseases of which the notification is compulsory under the Infectious Diseases Notification Act, 1889.

It appears to me, however, and the same seems to be the opinion of many able and thoughtful medical officers of health, that the advantages to be gained from the compulsory notification of Influenza would not in most districts be commensurate with its cost, for the following reasons:—

The difficulty of distinguishing between true Influenza and ordinary catarrhal and feverish attacks has to be taken into account. Until the pathology of Influenza is better known and its nomenclature more generally agreed upon, it is certain that, if it were notifiable, a great many cases would be returned as "influenza" which were not so; and, on the other hand, that many actual cases of the disease would not be returned. The symptoms of a typical attack of Influenza are well marked, but when the disease has been long absent as an epidemic they may fail to be recognised, as seems often to have happened in the latter part of 1889, when the disease first returned after an absence of 40 years. Yet it is these early cases which must be notified, if notification is to prevent another epidemic. Again, as pointed out in my previous report (p. 97), the typical cases of Influenza, as of other diseases of the same class, are accompanied by numerous mild ones, which, though not presenting characteristic symptoms and not compelling the patient to depart from his usual habits of life, are nevertheless essentially Influenza, and capable, as we have reason to believe, of spreading the disease. If such cases, many of which do not come under the notice of a medical man, were not notified, notification of known cases would often fail of its purpose.

On the other hand, the inclusion of Influenza among notifiable diseases would probably lead to many cases being notified which were not true Influenza, especially in view of the difficulty of diagnosis, and of the habit which has prevailed of applying the name "influenza" to severe cases of common catarrh. But even if none but genuine cases of Influenza were notified, the payments which would have to be made under the Infectious Diseases Notification Act would, during an

epidemic, entail a serious expense.* This might not be a reason for not requiring the notification of Influenza if there were sufficient ground for expecting that the notification would materially help to check its spread, but I do not see that there is any such prospect. Notification of infectious diseases is only of use as a means to an end, that end being to enable effectual means to be taken to prevent their spread, or to enable us to trace their origin with a view to removing their causes. In diseases which are propagated direct from person to person, isolation of the patient, combined with disinfection of infectious matters, affords the best means of limiting their spread; but no amount of hospital accommodation which it would be practicable to provide would suffice to receive the patients in an epidemic of Influenza.

Other difficulties in the way of checking the spread of Influenza by isolation on a large scale have been already referred to.

For these reasons I do not think that the compulsory notification of Influenza affords much prospect of utility, though there may be districts, *e.g.*, small and comparatively isolated health resorts, in which it might be worth while to make trial of it; more especially if means could be devised by which fees should be payable only for the first cases in a house, or the earlier ones in a locality.

In any case, however, compulsory notification of Influenza to be of any use should be adopted in the interval before another epidemic. For any benefit to arise from the compulsory notification of Influenza it must be in force so as to give information of the earliest cases that may occur; it would be vain to expect any useful results from it if deferred until an epidemic was in full force. Not only so, but the legal formalities necessary under section 7 of the Infectious Diseases Notification Act for the inclusion of Influenza under its provisions would occupy a still further period, at least 10 days in a case of emergency, and more than six weeks in an ordinary case, so that if the resolution to include Influenza among compulsorily notifiable diseases were deferred until an epidemic was present, it would probably not come into force until the epidemic was over.

School closing.

The closing of schools in presence of Influenza is another precaution of which mention may be made. The mere exclusion of scholars from households known to be infected would probably fail, for the reason that many of the cases are not heard of and that the disease is infectious in an early and often unrecognised stage. In many instances schools have had to be closed during the prevalence of an epidemic of Influenza owing to a large number of the teachers and scholars being prostrated with it. But in some few instances schools have been closed as a precautionary measure at the outset of an epidemic, and in one case in which an appeal was made the Board expressed the view that the order to close the school under these circumstances was not unreasonable.

The Rev. J. E. Vize, vicar of Forden, a scientific observer, tells me that he became convinced during the epidemic of 1890 of the infectious nature of Influenza, and that it was largely disseminated through his parish, a wide and scattered one, by means of children attending the village school, who, though lightly affected themselves, gave it to their parents in a more severe form. Accordingly on the reappearance of the disease in 1891 he immediately closed the school, with the sequel that his parish, which had suffered severely in 1890, remained comparatively free, while those around were severely attacked. The many instances

* Dr. Newsholme estimates that the fees for notification of Influenza in Brighton during the last three epidemics would have amounted to 6,000*l.*

however, already noted, in which villages which have suffered severely in one epidemic, have escaped lightly in another, prevent much weight being attached to a solitary experiment of this kind.

Ventilation.

The importance of free ventilation, especially of rooms occupied by crowded assemblages of people, as a precaution against the spread of Influenza has to be pointed out. This is illustrated by the outbreak at Llanfyllin given on p. 90 of my former report. Influenza, introduced from London, spread among the English congregation at the morning service, but the air of the church having been thoroughly changed to get rid of an escape of gas, the Welsh congregation at the evening service did not contract it.

Dr. Caldwell Smith remarks:—"The opinion that a certain degree of "concentration of the Influenza poison is necessary for its epidemic spread "would explain some points which are difficult to understand. This "concentration takes place in crowded assemblies, and the greater the "facilities for these crowded assemblies, and the more they are taken "advantage of, the more rapidly will Influenza spread. Repeated obser- "vations made by me confirm this beyond any reasonable doubt. One "reason, I believe, why this district suffered so badly and so frequently "is that these fatalities exist here very markedly. There is not the "slightest doubt that the disease is largely spread in crowded theatres, "churches, and halls, simply by personal infection, and one reason why in "some districts the rich suffered more than the poor is that the former "frequented overcrowded and overheated rooms much oftener than the "latter. If introduced into an overcrowded house, especially if this con- "sisted of a single apartment, it spread rapidly, and infected almost all "the occupants. Free ventilation is the best preventive of Influenza, and "this, combined with isolation of individual cases, is at present the only "means at our disposal for its prevention. It would be a practical "impossibility to disinfect the sputa, except of those who are confined to "bed (? or to the house), but it is possible for many to live in well-venti- "lated apartments, to avoid overcrowded rooms, and to avoid direct "contact with those who are suffering from the disease."

Disinfection of sputa.

As regards disinfection, if the essential cause of Influenza be, as seems probable, a micro-organism inhabiting the mucus of the air passages, it is for the discharges from the bronchial tubes and nostrils that measures of disinfection are indicated; the most convenient and safest being probably to use, instead of a handkerchief, pieces of rag or paper which are immediately burnt. There may be a difficulty in doing this when persons are going about out of doors; but then persons suffering from Influenza should not go about out of doors, both for their own sake and for that of others.

The recognition of Influenza as a serious disease of an infectious nature will, it is to be hoped, lead to its being reckoned in public estimation as a disease of which it is worth while to take some pains to prevent the spread. To this end various sanitary authorities have issued placards calling attention to the infectious nature of the disease. The Society of Medical Officers of Health on January 18th, 1892, passed a resolution "that in the opinion of this Society, Influenza is a dangerous infectious disease."

On the Influenza Epidemics of 1889-90, 1891, and 1891-92; by Dr. Parsons.

Provisional Memorandum upon Precautions advisable at times when Epidemic Influenza threatens, or is prevalent.

Copy of Board's Provisional Memorandum on precautions during Influenza epidemic.

In July 1891 the Board issued a Report by Dr. H. F. Parsons "On the Influenza Epidemic of 1889–90," together with an Introduction by their Medical Officer.*

It was then pointed out that "action for the prevention of disease, in "order to be effectual, must be based on a knowledge of its causation," and since our knowledge of the natural history of Influenza, and especially of the circumstances of time and place under which it spreads, remains most imperfect, any advice which can be given as to the precautions to be taken for its prevention or mitigation can only be correspondingly incomplete.

But, in view of the recurrence and maintained prevalence of the disease, the Board feel that there may be advantage in setting out certain points as to which some definite knowledge has been obtained.

I. *Influenza is spread by infection from person to person.*

On this point the Medical Officer of the Board wrote:—"The disease has "long been regarded as of the 'miasmatic' group; of that group, namely, "wherein pathologists and statisticians comprise the common infectious "diseases of our own and other countries" . . . . "In its epidemic "form Influenza is an eminently infectious complaint, communicable in "the ordinary personal relations of individuals one with another. It "appears to me that there can henceforth be no doubt about the fact."

"In some circumstances it would seem that infectiveness of Influenza through the atmosphere shows itself over a wider area than the limits of household life. Probably also there are other less direct ways by which the infection of the disease can travel; and ways, moreover, by which the infection can be retained for a time in a state of suspended activity . . . . But we have, no doubt, much to learn about the dissemination of Influenza and particularly of the stage when the complaint acquires its epidemic power."

[Since the above was written, evidence has accumulated to indicate that Influenza is infectious at quite an early stage of the illness, and may remain so as late as at least the eighth day from attack.]

"By having established a place for this Influenza among infectious diseases, we assert a position for the disorder within a class of diseases over which we habitually exercise a measure of control. But from what we have thus far seen of the specialities of Influenza, we cannot feel particularly confident of our ability, under the existing conditions of society, to successfully defend ourselves against a further outbreak. A disease that can be absent in an epidemic form for 30 years together cannot, even if a first attack confer immunity, avail to give the protection of a first attack to any large part of a population.† Early isolation precautions, applicable perhaps to children suspected to have measles, cannot well be applied to persons suspected of Influenza among the bread-winners of a community; and the singular ability possessed by Influenza to disperse itself over a population, owing to its brief incubation period, must add to the difficulties of dealing with an infection that finds the bulk of the population susceptible to its attack. Having, as would seem, something like a third part of the incubation time proper to small-pox, measles, or typhus, Influenza has correspondingly rapid ability to reproduce itself; can, that is, give rise to some thousand attacks in the time that small-pox

---

* Report on the Influenza Epidemic of 1889–90, by Dr. Parsons, with an introduction by the Medical Officer of the Local Government Board, [C.—6387], pp. 324. Eyre and Spottiswoode, East Harding Street, E.C.

† Abundant evidence has now accumulated to show that Influenza does not, in any marked degree, or for any considerable length of time, confer immunity against another attack.

or typhus had taken to produce ten; each of the thousand cases being ready to infect other susceptible people, and the difficulty of applying principles of isolation and disinfection being in like measure enormously enhanced."

A further difficulty in applying the process of "stamping out" by means of isolation and disinfection at the commencement of a threatened epidemic of Influenza, when alone success is likely to be attainable, arises from the circumstance that the disease does not possess any definite and easily recognisable feature like the rash of some of the other infectious diseases; so that the first cases may not be discriminated from ordinary catarrhs, transient febrile attacks, &c. The consequence is that such preventive measures as are available are delayed until obscure cases have multiplied, and the disease already prevails.

In view of the difficulties referred to, it is not practicable to devise any restrictive measures for the prevention of the spread of Influenza which shall be universally applicable.

But, under some circumstances and for certain classes of persons, some such measures should be resorted to, and this notably:—

(*a.*) For persons in whom an attack of Influenza would be specially dangerous by reason of age or infirmity;

(*b.*) For the inmates of institutions, the mode of life in which can be regulated and controlled;

(*c.*) For the first cases of Influenza in a locality or a household, where these are early recognised.

In such cases:—

1°. Separation between the sick and the healthy should, as far as practicable, be carried out. Measures to this end have in some instances been adopted with marked success.

2°. With isolation, should be combined disinfection of infected articles and rooms.

Persons suffering from Influenza should not expose themselves in public places.

Since the propagation of Influenza is known to be promoted by the assemblage of large numbers of persons in a confined atmosphere, it is advisable that when an epidemic threatens or is present, unnecessary assemblages should be studiously avoided.

The ventilation and cleanly keeping of any building in which many people are necessarily collected together, should receive special attention when Influenza threatens or is present, with a view to secure that the air of the building shall be frequently changed, at any rate during the intervals of its occupation, and to avoid accumulation of dust and dirt.

II. *The liability to contract Influenza, and the danger of an attack, if contracted, are increased by depressing conditions, such as exposure to cold, or to fatigue, whether mental or physical.*

There is reason to believe that the development of an attack of Influenza in a person exposed to the infection depends very largely upon the receptivity of the individual; and that the power of resistance varies not only in different persons, but also in the same person from time to time; being diminished by any conditions which depress the general bodily vigour. It is therefore important that at the time of an epidemic all persons should, as far as they are able, pay attention to such measures as tend to the maintenance of their health, wearing clothing of suitable warmth, and avoiding unnecessary exposure to cold and fatigue, unwholesome food, and excessive use of alcoholic liquors. Similar principles should be borne in mind by those who, as managers of institutions and establishments, have to make regulations for others.

There is also a very general agreement among medical practitioners that the risk of a relapse, and of the occurrence of those pulmonary complications which constitute a chief danger of the disease, is increased by anything which involves exposure to cold or fatigue before complete recovery.

Persons, therefore, who are attacked by this malady should not attempt to fight against it, but should at once seek rest, warmth, and medical treatment.

The nature of such treatment does not fall within the scope of this Memorandum.

R. Thorne Thorne,
Assistant Medical Officer.

Local Government Board,
January 23rd, 1892.

Report on Influenza, in its Clinical and Pathological Aspects; by Dr. Klein, F.R.S.

INTRODUCTORY.

The investigations to be here recorded were carried out during February and the following months of 1892. The materials for the investigations were chiefly supplied by patients admitted into the Kensington Infirmary, under the care of the Medical Superintendent, Dr. H. P. Potter; and partly by a few cases observed by Dr. Andrewes, Dr. Cautley, and myself, at St. Bartholomew's Hospital. It is principally to Dr. Potter that this report is indebted for careful clinical histories of the several cases of influenza.

The bacteriological investigation of the blood and sputum was carried out on a considerable number of cases in the Kensington Infirmary, and on some also in the out-patients' room of St. Bartholomew's Hospital. To these may be added several cases of the disease which during the first influenza epidemic of 1889–1890 had been observed at Dr. Barnardo's Home. The present inquiry unfortunately did not commence until the epidemic was on the wane; and by the end of March and beginning of April there were only few cases available for observation; yet the number of observations on the living have been sufficient, as will be seen in the sequel, to allow of definite conclusions being drawn as to the disease.

I should add that during my enforced absence, owing to influenza pneumonia, Dr. F. W. Andrewes conducted the work of bacteriological and experimental investigation for this report, particularly in certain experiments on monkeys, to be recorded in the following pages. Dr. Cautley and Dr. Schorstein also rendered valuable services in the bacteriological portion of the work.

PART I.

The Occurrence of Micro-organisms in the Blood *in* Influenza.

By the comprehensive and detailed inquiries of Dr. Franklin Parsons (Report on the Influenza Epidemic of 1889–1890, Local Government Board, 1891), it is definitely established that Influenza is a highly infectious disease; and his report suggests that the spread of the contagium as well as the reception of infection may take place by the respiratory organs.

The fact that the initial febrile disturbance of influenza is a prominent symptom of the disease, antecedent to any diseased condition of the respiratory organs, has been noticed by many observers, and has also been invariably noticed in our own cases. From this fact it would *a priori* seem probable that the contagium or virus of influenza enters the blood, there first manifests its activity, and only afterwards finds its nidus in other tissues—principally the respiratory, bronchial, and pulmonary tract—and this course would appear readily to account for the phænomena of the disease and the distribution of its contagium by the exhaled air.

Accordingly, in the first instance, we proceeded to investigate bacteriologically the blood *of acute cases of influenza;* they were all pure

cases of influenza, commencing suddenly with rigors, pains in the back and limbs, and frontal headache.

In all cases the last phalanx of the middle finger was well brushed with soap and warm water, then well rubbed with carbolic lotion and dried with a clean cloth. With a piece of tape, a venous congestion was established on that phalanx, and the volar side of it was pricked with a sterile needle; then, as the blood oozed out of the prick, it was allowed, first, to run up a capillary glass pipette, and this was used for the cultivations (to be recorded below); secondly, to the remaining drop of blood at the site of the prick a clean cover-glass was applied, and this was slid over another clean cover-glass; so that, on separating the two glasses, each was covered on one side with a thin film of blood; then, again, with a third clean cover-glass a further drop, oozing out from the prick, was touched, and this was in the same way slid over a fourth clean cover-glass; in this manner in each case at least four cover-glass films of blood were obtained. They were slowly dried over a spirit flame, and taken to the laboratory for further manipulation.

The above capillary pipette of blood was used for inoculating nutritive mediā: (*a*) faintly alkaline beef broth peptone, (*b*) faintly alkaline glycerine Agar broth peptone set with slanting surface, and (*c*) faintly alkaline Agar broth peptone also set with slanting surface; each culture received a droplet of blood, some a full drop, others only the quantity adhering to a platinum loop. In every case at least two broth and two glycerine Agar cultures were made, in the great majority of instances as many as six and eight (half broth half Agar) tubes were inoculated; they were all incubated at 37° C. We may be confident that in these cultivations made by us no contamination with extraneous organisms occurred,* except in a few instances (three in all and omitted in the record) where the blood had been taken from the patient at a time when the wards had just been cleaned and swept.

The cover-glass specimens were, after carefully heating over the flame of a Bunsen burner, placed in absolute alcohol for five minutes and then in the stain. This stain was either Carbol methylblue such as we ourselves usually employ, or more commonly it was that used by Canon, a noteworthy investigator of influenza bacteriology. This is a mixture of Eosin and methylblue made as follows: Czenzynke's mixture of concentrated watery methylblue 40 grammes, with the addition of 20 grammes of a $\frac{1}{2}$ per cent. solution of Eosin (70 per cent.) alcohol, and of 40 grammes of distilled water.

In one or other of these stains the cover-glass specimens remained for several hours at 37° C., in most instances they were then left in the dye over night at the temperature of the room, after this they were well washed in water, dried and mounted in Canada balsam. It may be here stated that when bacteria could be demonstrated by the Eosin methylblue staining, they could be equally well brought out by the Carbol methylblue.

In the following cases the blood was subjected to examination by cover-glass specimens and by culture; the history and clinical notes of these cases will be given in detail in Part II. The reader who notices that the subjoined results were principally obtained in February, will observe that that month was by far the most abundant in opportunities for investigation.

* That in this method of cultivation no appreciable risk of accidental contamination with extraneous bacteria need be apprehended, I have shown in my report in 1887 on my experiments on cultivation with blood of cases of scarlet fever.—(E. K.)

Case 1.—F. B., male, was taken ill at 10 a.m. on February 2nd; admitted into Kensington Infirmary at 4.30 same day; temperature 100·8° Fahr.: blood taken.

No bacteria of any kind in the cover-glass specimens, no growth of any kind in any of the culture tubes.

On February 3rd, in the afternoon, the temperature had gone down to 97°, blood was again taken and used for cover-glass specimens and for cultivation, and the results were the same as above, viz., negative.

Case 4.—E. J., female, was taken ill on January 24th. Admitted on the same day. Blood was taken on February 2nd. The temperature had been normal since January 30th. No result in cover-glass specimens or cultivation.

Case 6.—F. N., male, was taken ill on January 31st. Admitted on February 2nd. Blood was taken on February 4th in the afternoon, the temperature being then 100°. No bacteria in cover-glass specimens, no growth in cultivation.

Case 7.—S. P., male, was taken ill on January 31st; admitted on February 3rd. On February 4th in the afternoon, temperature was 103·4°: took blood. Result negative. On February 5th temperature in the afternoon 102·8°, took blood again. Results negative.

Case 9.—E. R., female, was taken ill on February 6th; admitted on same day. On February 8th the temperature in the afternoon was 99°; took blood. Results negative.

Case 12.—E. Th., female. Was taken ill in the evening of February 9th; admitted on February 12th. Temperature was then 101°. Blood was taken. Results negative.

Case 13.—J. P., male, was taken ill on February 13th. On February 14th temperature 102·2°: took blood. Results negative. On February 15th temperature 98·8°: took blood again. Results negative.

Case 14.—C. C., female, had relapse of acute influenza on February 13th at 5 p.m. During February 14th temperature had gone down from 101·4° in the morning to 98·4° in the afternoon; at about 11 a.m. it was 99·2°: took blood. Result negative. On February 15th the temperature in the afternoon was subnormal 97°: took blood again. Results negative.

Case 15.—F. H., female, was taken ill on February 12th. On February 15th temperature in the morning had fallen (from 101° on February 14th) to 99·8°, but rose again in the afternoon to 101·2°. Took blood in the morning. Results negative.

Case 16.—M. T., female. Second attack of influenza on February 17th. Blood was taken on February 18th, temperature 102°. Results negative.

Case 17.—A. W., female, was taken ill on February 17th. Admitted February 18th. Temperature on February 18th at 7 p.m. 102·2°, on February 19th in the afternoon 98·4°. Blood was taken. Results negative.

Case 18.—G. G., male, was taken ill in the night from February 18th–19th. On February 19th blood was taken. Results negative.

Case 20.—C. H., female, was taken ill suddenly on February 24th. Admitted on February 25th, temperature 100·2°. Blood was then taken. Result of cultivation negative. One of four cover-glass specimens shows fairly numerous bacilli; their character to be described below. *See Case* 47.

Case 21.—H. G., female, was taken ill on February 20th at 11 a.m. Admitted February 23rd. On February 25th temperature had gone up in the afternoon at 2 p.m. to 101°, and blood was then taken. Results negative as regards cultivation; in all four cover-glass specimens made of the blood there were found altogether about a dozen rod-like bodies which looked like bacilli.

Case 22.—Nurse Carson, female, was taken ill on February 25th. On February 27th temperature had fallen to just below 100°. Blood taken. Results negative.

Case 23.—B. B., female, was taken ill on February 25th. On February 26th temperature had risen to 103·2°. Blood taken. Results negative.

Case 24.—E. C., female, was taken ill on the morning of February 26th. On February 29th temperature had slightly risen at 2 p.m. from 98° to 100°. Took blood. Results negative.

Case 25.—T. D., male, taken ill on March 29th. Admitted on April 2nd. Temperature at 4 p.m. was 103·4. Took blood. Results negative.

Case 26.—S. P., female, was taken ill on February 26th. Admitted on February 27th. On February 29th took blood. Results negative.

Case 27.—J. F., male, was taken ill on March 25th. Admitted on April 1st. On April 2nd temperature in the afternoon at 2 p.m. was 101·2°. Took blood. Result negative as regards cultivation; in one out of four cover-glass specimens several small short straight bacilli with rounded ends; their characters the same as of Case 20, and will be further described below.

Case 28.—W. H., male, was taken ill on February 18th at 4 p.m. Admitted on February 19th. Temperature at 2 p.m 102·4°. Took blood. Results negative.

Case 29.—E. Q., female, was taken ill on March 27th. On April 1st temperature in the afternoon was 100°. Took blood. Results negative.

Case 30.—J. G., male, was taken ill on March 26th. On March 28th temperature had fallen to 97·4°. Took blood. Result of cultivation negative. Of four cover-glass specimens one shows a few rods of the same character as Case 27. On March 31st the temperature had again risen in the afternoon to 100°. On April 1st it again fell to 97° in the afternoon. Took blood. Results negative.

Case 31.—J. S., male, was taken ill on March 27th. On March 31st temperature at 10 a.m. was 103·2°. Took blood. Results negative.

Case 32.—J. W., male, was taken ill on April 5th at 10.30 p.m. Was admitted on April 6th, temperature was 103.6° at 7 p.m. On April 7th at 10 a.m. it had fallen to 96·6°. Took blood. Results negative.

Case 33.—Myself. Was suddenly taken ill on February 17th, at 11 a.m., temperature at 12 noon 100·2°, when blood was taken. Cultures negative, cover-glass specimens showed no bacilli. At 4.30 p.m., temperature 99·8°. Blood was taken. Cultures negative, cover-glass specimens show a few bacilli. On February 18th temperature in the afternoon had risen to 102·8° (pneumonia of left lung was commencing). Blood was taken. Cultures negative, cover-glass specimens show fairly numerous bacilli, of which hereafter. *See Case* 47.

Case 34.—E. S., male, at St. Bartholomew's Hospital, was taken ill with influenza on February 14th. On February 15th temperature 101·8°. Blood was taken. Results negative.

Case 37.—F. B., male, St. Bartholomew's Hospital, was taken ill on February 2nd with influenza. Temperature on February 3rd, 100·1°. Blood was taken. Results negative.

Case 38.—D. C., male, St. Bartholomew's Hospital, was taken ill on January 31st. On February 3rd temperature 101·5°. Blood was taken. Results negative.

Case 39.—J. H., male, St. Bartholomew's Hospital, was taken ill on February 1st. On February. 3rd temperature 99°. Blood was taken. Results negative.

Case 40.—C. G., male, was taken ill on February 5. Temperature on February 6th 100°. Blood was taken. Results negative. Patient was seen again on February 8th, temperature 99°. Blood was taken. Result of cover-glass specimens negative; three Agar cultures remained sterile; out of three broth cultures, one showed a growth after several days, *this growth proved to be the same organism as was constantly found in the bronchial sputum of influenza* and as will be described in detail in Part III.

Case 41.—C. T., male, St. Bartholomew's Hospital, was taken ill on February 10th at 7 a.m. Temperature 102·8°. Blood was taken, Result negative. The patient was seen again on February 11th, temperature 99·4°. Blood was taken. Results negative.

Case 42.—W. C. H., male, St. Bartholomew's Hospital, was taken ill at 1 p.m. on February 12th. On February 13th temperature at 10.30 a.m. 100·4°. Blood was taken. Results negative. The patient was seen again on February 15th, temperature normal. Blood was taken. Results negative.

Case 43.—A. G., male, St. Bartholomew's Hospital, was taken ill in the morning on February 14th. Temperature on February 15th 102·4° at 3 p.m., at 7 p.m. 104·2°. Blood was taken at 5.45 p.m. Cultivations negative, in one out of four cover-glass specimens few bacilli, same as in former cases, and as will be described below. On February 16th temperature 102·6°. Blood was again taken. Results negative both as to cover-glass specimens and cultures.

Case 44.—A. C., male, was taken ill about noon on February 23rd. On February 24th temperature 101°. Blood was taken. Results negative. The patient was seen again on February 25th, temperature normal. Blood was again taken. Results negative.

Case 45.—H. P., male, was taken ill in the night of February 23rd—24th. On February 24th at 2.30 p.m. temperature 99·7°. Blood was taken. Results negative.

Case 46.—A. B., male, St. Bartholomew's Hospital, was taken ill with influenza on January 22nd On January 23rd temperature at 10.30 a.m. 100°. Blood was taken. Results negative.

Case 47.*—Boy, aged 12, at Dr. Barnardo's Home. Had been ill 24 hours with influenza. Blood was taken when temperature was 100·2°. Result of cultivation negative. In two out of four cover-glass specimens

* This and the following cases were in boys attacked during the 1889–90 epidemic. It is to be observed that throughout this report the designation of the several cases by numbers does not signify any chronological sequence of the cases.

there were present a fair number of bacilli of the same kind, in one specimen particularly they were in some fields abundant. Figures 1 and 2 show this.

These bacilli are in size, aspect, and staining the same as were mentioned of my own blood (Case 33, Figures 3 and 4) and of Cases 20, 27, 30, and 43. The bacilli are minute rods, generally straight, a few slightly curved; they measure in thickness 0·4 to 0·5 μ (micromillimetres) in length they are generally 1·6 μ, few are shorter 1·2 μ, and a few are longer up to 2·4 μ; their ends are rounded; they do not stain uniformly, since most of them show (either centrally or more towards one end) a less stained almost clear spot; most of the bacilli are single, a few are in dumbbells, they occur isolated, they do not form aggregated masses; occasionally though rarely two or even three are found closely side by side.

Cases 48, 49, 50, 51, and 52, were boys at the same establishment, they were all affected with typical influenza of from a few hours to 24 hours, their temperature was various. Blood was taken, but the results of cultivation and of cover-glass specimens was in all cases negative.

The result of the examination of the blood of the preceding cases was this:—

Of 43 cases of blood examination, no bacterial forms could be discovered in 37*. In the other six cases, cover-glass specimens revealed the presence of one and the same kind of minute bacillus; in one (Case 47) the bacilli were numerous, in two (myself Case 33, and Case 20) they were fairly numerous, and in the other three they were very sparse.

In the positive cases the temperature at the time the blood was taken was: (1) Case 20, 100·2°; (2) Case 27, 101·2°; (3) Case 33, 102·8°; (4) Case 30, 97·4°; (5) Case 43, between 102·4° and 104·2°; (6) Case 47, 100·2°. In the 37 cases the temperature in the majority was higher than normal, in a minority it was normal or sub-normal. In some cases blood was taken at two different periods:—during and after the fever; or both times during the period of raised temperature; or both times when the temperature had again fallen:—but in these respects no definite relation as to the presence or absence of the bacilli could be made out. Thus, in my own case, the bacilli were found on two out of three examinations, both affirmative instances being associated with raised temperature; while in Case 30 few bacilli were present at temperature 97·4°, absent at 97°. In Case 41 result was negative both at temperature 102·8 and at 99·4; in Case 42 the result was negative at 100·4, and also at normal temperature; in Case 43 the result was positive at temperature 102·4°–104·2°, negative at 102·6°; in Case 44 negative both at 101° and at normal temperature.

It is then clear from these observations that neither during the febrile stage nor after the temperature has again fallen do the bacilli occur in the blood with anything like constancy, considering that in 37 out of 43 cases no bacilli could be found, in each case at least four cover-glass specimens, in some six and even eight, being made, stained with the appropriate methods, that is to say, by methods by which they are readily shown in the positive cases. But also the extremely varying number in which the bacilli occurred in the six positive cases indicates that their presence in the blood cannot be of the same essential value for the disease as is the case in the typical acute infectious diseases—"blood-diseases," of

* Case 21 must be either omitted or put down as very doubtful.

which the various known septicæmias, anthrax, and fowl cholera are types. This view is strongly borne out by the consideration that in all six cases in which the bacilli were found in the blood in the cover-glass specimens they could not be demonstrated in culture; the media used for these cultures being (as will presently be shown) perfectly suitable for the living bacilli of the bronchial sputum. This would appear to indicate that the bacilli found in the six affirmative cases were not living. Thus, in my own blood, taken on February 17th, and still more in that of February 18th, a fair number of bacilli were to be demonstrated in cover-glass specimens, but no bacillary growth could be obtained by the method of culture. And in this connexion it is to be noted that the quantity of blood employed in the various culture experiments was at least as large as that used in cover-glass preparations.

Unless the experience of Case 40, where a successful cultivation was obtained, must be regarded as invalidating the general conclusion, there would seem to be only one explanation of the observed facts, viz., *that any bacilli of influenza that may gain access to the circulation, lose here their vitality and are present in the blood only as dead bacilli.* And Case No. 40 cannot claim to be regarded as hostile to this conclusion. For in the case of this successful culture, we have only to believe, as regards particular bacilli, that a certain interval has elapsed between their admission to the blood stream and the destruction of their vitality, and Case No. 40 comes to be comprised at once within the general law. The one successful culture in this case gave altogether the characters of cultivations from sputum bacilli, to be described further on.

Previous Investigations of Influenza Blood.

The observations on the results of the bacteriological examination of the blood in influenza are very numerous. Dr. Friedrich in his memoir on influenza in the Arbeiten aus dem K. Gesundheitsamte VI., p. 254, has given an exhaustive account, and his own results, as also those of most other observers, can be summarised in one word, negative; neither in cover-glass specimens, nor in culture, were any definite bacterial species to be demonstrated. The first observer who in this respect made the statement that in acute influenza there occurs constantly in the blood a definite species of bacillus, pathognomonic of the disease was Canon (Deutsche Med. Wochenschr. No. 2, 1892); this observer states that he found in all cover-glass films of blood of influenza by the method mentioned on a former page [dried, placed in absolute alcohol for five minutes, then in methylblue eosin (Czenzynke's mixture) for several hours at 37° C.] a particular kind of bacillus present in numbers varying from 5 to 20; the bacilli and the nuclei of the white cells being stained blue, the blood discs and the body of the leucocytes pink. Now, our observations do not bear out this statement of Canon, since by the same methods (we stained the specimens for even a longer time than Canon) we failed to find bacilli in 37 out of 43 cases, and we therefore, in opposition to Canon, do not consider the presence of these bacilli as of pathognomonic value, or their absence as of diagnostic importance.

Canon further (ibidem, No. 3) makes the statement that he succeeded constantly in successfully cultivating, in sugar-agar, the bacilli from the blood of cases of influenza by using for each plate cultivation 8 to 12 drops of blood. Now to every reader of his first memoir on the subject this result must have come rather as a surprise; for if in every cover-glass specimen of influenza-blood there occur (according to Canon) 5–20 bacilli, and since a cover-glass film

represents only a portion of one drop of blood, it does not appear from Canon's account of his experiments why, in order to obtain a successful culture, it was necessary to add so large an amount of blood to his culture medium; seeing that a single bacillus should be sufficient to start a colony. The reason, however, would seem to be given by our own experiments as recorded above. We do not doubt that Canon succeeded in obtaining, by the use of 8–12 drops of blood, colonies of the same bacilli as those seen by him in cover-glass specimens; for we ourselves (from case No. 40) succeeded in obtaining a successful culture after inoculation with only a portion of a drop of blood. But what seems to us to be evident from his observations is that the bacilli which Canon found in so many of his blood cover-glass specimens were not living but dead bacilli: and now we may see the reason for major success by Canon's culture processes; viz., that he multiplied the chances of including some bacilli that had not yet lost their vitality after entry to the blood stream. Canon's experiences are therefore reconcileable with those of ourselves and many other observers who have obtained by ordinary cultivation processes, results that are mostly negative; and (looking always to such diseases as fowl cholera and anthrax for our standard) we may believe of Canon's experiments that they too agree with our conclusion that the blood cannot in any sense be regarded as the primary nidus of the microbe of influenza.*

Pfuhl (Centralbl. f. Bacteriologie und Parasitenk. XI., No. 13) also made observations which are in agreement with our own; he states that in about 100 cover-glass specimens stained after Canon's methods, he only in one single specimen of the blood of one patient found six bacilli, and he therefore does not attach any great importance to the microscopic examination of the influenza blood. But Pfuhl using (like Canon) a large quantity of blood from two cases (8–10 drops) for culture on Glycerin-Agar did not succeed in getting the bacillus of Canon in culture. Pfuhl is inclined to think that of one case he did get it, but looking at his description of the morphological characters of the bacilli that he got in the culture (his bacillus No. 2) it must be evident that the two are different species. In the second case Pfuhl failed to obtain the bacillus of Canon, but he got colonies of an admittedly different species.*

In a further and more recent publication by Pfeiffer and Beek (Deutsche Med. Woch., May 26, 1892) these authors confirm Pfeiffer's first observation as to the constant and abundant occurrence of the influenza bacilli in the bronchial secretion. They further state that notwithstanding many experiments they have not been able to find the bacilli in the blood, and according to them "it appears to be proved "that the morbid process of influenza is accomplished within the "bronchial territory, and that an infection of the blood may, as a "regular occurrence, be with certainty excluded" (Br. Med. Journal, June 25, 1892, p. 104). Their conclusions seem in harmony with our own detailed above, in so far as pathological manifestations are concerned.

---

* In reading of the method of cultivation used by Canon and also by Pfuhl:—8–10 or 12 drops of blood are placed on the surface of solidified Agar contained in a Petris's plate cultivation dish, then with a platinum loop the blood is smeared all over the surface of the Agar—one cannot wonder at their getting various colonies on the surface of the Agar after their cultivation processes were completed. The method seems to us open to considerable risks of accidental contaminations.

PART II.

Account of the Cases of Influenza subjected to Examination.

In this section the clinical notes are given of the cases which served for material for the investigation of the blood (Part I.) and the bronchial secretion to be described in Part III. The first 32 cases are recorded by Dr. Potter and were patients of the Kensington Infirmary.

Cases 34, 35, and 36 were seen by Dr. Andrewes at St. Bartholomew's Hospital, cases 37, 38, 39, 40, 41, 42, 43, 44, 45, by Dr. Cautley also at St. Bartholomew's Hospital. Of the history of these, as also of the others seen by myself, only so much is given as was thought essential in regard to the bacteriological examination of the blood or the sputum or both.*

Dr. Potter supplies me with a brief general account of the behaviour of Influenza in the Kensington Workhouse and Infirmary during 1892, as follows :—

During the recent epidemic which extended over a period of three months—from the middle of January to the middle of March —150 cases were under treatment; of this number 55 were complicated or were of such a severe nature that the patients were detained in the infirmary for a period of about three weeks.

The incidence of Influenza appeared to be independent of the previous state of the health of the individual attacked. In our experience, Influenza has been found to be rare in children, and more common in men than in women.

A typical case shows the following symptoms:—The onset is sudden without definite rigor, but there is a feeling of chilliness and intense *malaise*. Pain is complained of in the head and lumbar region and pain frequently runs around the chest following the course of the intercostal nerves. Pain also occurs at the back of the orbits and in the calves of the legs. The patient vomits, is constipated and passes high-coloured urine with a deposit of lithates. The tongue is moist but coated. Solid food is not tolerated and there is intense thirst. The temperature is about 102° F., pulse 90, respiration 25. The acute attack lasts for two or perhaps three days, and as the fever subsides the pains disappear, the tongue cleans and a more generous diet is appreciated.

The most frequent complication was Bronchitis attacking both lungs, but Pleuritis and Pneumonia were not infrequent. These inflammatory conditions so long as they last cause a sustained elevation of temperature, and convalescence is more tardy when these diseases supervene upon Influenza, as contrasted with the occurrence of similar lung mischief appearing in a fairly healthy person.

A not uncommon symptom is a general diffused eruption of a crimson colour, which is perhaps more intense on the flexor surfaces of the limbs and the anterior part of the trunk. This is followed by branning or even peeling of the skin. Both during and after the eruption there is absence of albumen in the urine. This eruption subsides with the fall of temperature.

It appears that the muscular and nervous prostration which follows Influenza is in direct proportion to the amount of sweating which accompanies the fever and has no relation to the temperature.

---

* I recall that the numbers indicating the cases have no relation to chronological order. Four cases recorded here are without index numbers and are designated as cases A, B, C, and D.

As regards the infectious character of the disease it deserves to be recorded that, in these wards, Influenza frequently attacked patients who had been in close contact with the sufferers admitted for the complaint in the adjoining bed to them.

The following are the clinical histories of Dr. Potter's 32 cases of Influenza. The temperatures (T.) are shown in degrees of Fahrenheit; P. and R. show the pulse and respiration :—

Case 1.—Frank Bennett, 22, coster. Complexion fair. Health always good. Coryza and general catarrh for three weeks.

February 2nd, 1892. At 10 a.m. shivering, frontal headache, general pains and malaise ; no vomiting. On admission, tongue much coated, white. Admitted to Infirmary 4.30 p.m.

February 3rd. Sweating profusely at 2 a.m. Rhonchi all over chest, few moist sounds left posteriorly, expectoration muco-purulent. Feels better, but now has occipital pains.

February 4th. Pains in the head, no pains in the limbs, vomiting.

—	2 Feb.			3 Feb.						4 Feb.		
	4.30p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.
T. -	100·8	101·5	102·2	99	97·6	97	97	97	96	95·4	95	97·2
P. -	—	92	98	84	74	76	72	68	56	85	50	68
R. -	—	28	23	21	18	18	18	20	17	17	17	16

Case 2.—Walter Hall, 18, butcher's assistant. Health always good, but has had inflammation of the bowels three times, never hæmoptysis. Family history good.

On January 19th, 1892, had sudden and intense pains in the head at 7 p.m. ; at his work till 10 p.m.

January 20th. Worse, shivered in the evening, no vomiting, no thirst, stayed at home.

January 25th. Went to work, but during the day had a relapse of the previous symptoms with pains across the back.

January 27th. In bed all day, only able to take cocoa and toast.

,, 28th. Admitted to the Infirmary :—Antipyrin gr. x. every four hours.

January 29th. Much improved.

February 3rd. Chest resonant, few large râles posterior right, rhonchi, back and front, expectoration muco-purulent nummular. Patient looks thinner and anxious.

—	Jan 28.		Jan. 29.		Jan. 30.	
	M.	E.	M.	E.	M.	E.
T. - -	—	101·2	101·2	99·6	98·4	98

Case 3.—Charles Joyce, 70, railway guard. Very abstemious habits; health always good.

January 18th, 1892. Felt ill, commenced suddenly with shivering and pains in the head.

January 25th. Admitted. Coughs. Rhonchi general, expectoration muco-purulent, moaned much in sleep. Poultices to chest back and front. Takes nourishment well. Delirious.

February 2nd. Flushed, no rigor, more cough. Expectoration rusty.

February 3rd. Worse; sputum rusty and offensive. Takes but little nourishment, sputum swallowed. Râles coarse and fine all over chest. Dulness right posterior base. Effusion in pleura. Died.

February 4th. Post-mortem 15 hours after death :—

Slight effusion left side; no consolidation. Right side, 10 ounces of effusion with patch of consolidation and recent adhesions. Specimens of lung and fluid taken by Dr. Klein. The consolidated portion of lung sinks in water and is without crepitation on section.

This is a case of bronchitis following Influenza, then the formation of a pneumonic patch around a large dilatation of the bronchial tube.

The temperatures in this case were as follows :—

—	Jan. 25.		Jan. 26.		Jan. 27.		Jan. 28.		Jan. 29.		Jan. 30.	
	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.
T. -	—	101·5	98·2	101	99·8	101	99·8	100	99	100·6	99	101
P. -	—	—	—	—	—	—	—	—	—	—	—	—
R. -	—	—	—	—	—	—	—	—	—	—	—	—

—	Jan. 31.		Feb. 1.		Feb. 2.		Feb. 3.			
	M.	E.	M.	E.	M.	E.	M.	8 a.m.	12 noon.	4 p.m.
T. -	99·5	99·8	99·2	99·6	103	101	105	104·8	105	103
P. -	—	—	—	—	—	—	—	120	124	120
R. -	—	—	—	—	—	—	—	52	60	40

Case 4.—Elizabeth Jordan, 28, laundress. Health good. No grave illnesses. Subject to "cold." Family history good. Was well up to 24th January.

January 24th. Felt faint. General pains, much headache, and constant vomiting, no diarrhœa. 3 p.m. shivering, could not get warm; sleeps fairly well.

January 25th. Much worse, shivering all day, fainted once, taking no food.

January 26th. Walked about 1 mile; a little better.

„ 27th. Admitted to Infirmary. Delirious; a few hours later, does not remember coming into Infirmary. Torpid and lethargic. Skin very pungent. Expectoration rusty.

January 28th. Still unconscious as to surroundings. Fine crepitations left lung posteriorly and slight dulness at the base. Taking beef tea, milk, and eggs.

January 29th. Better. Taking antipyrin and quinine.

February 2nd. Improving. Feels stronger. Rhonchi all over right lung. Pneumonic patch left clearing up. Expectoration slightly rusty.

February 3rd. Expectoration less.

—	Jan. 27.		Jan. 28.		Jan. 29.		Jan. 30.		Jan. 31.	
	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.
T. - -	—	104·6	102·4	103	100	99·8	99	98·4	99	98·6

Case 5.—John Nash, 36, labourer. Third attack. (Pneumonia).

Well up to February 3rd, then shivering, headache, pains in back, vomiting, loss of appetite, thirst. In Infirmary last Easter for pneumonia.

February 4th, p.m. Cough, rigor.

„ 5th. Sputum tinged with blood on admission at 7 p.m.; crepitation in lung (left) posterior.

February 6th. Perspirations, rusty sputum, pains in left side, cephalalgia.

February 7th. Perspiring, rusty sputum. Tongue coated, moist.

„ 8th. Sputum still rusty, still a few crepitations left posterior.

—	Feb. 5th.		Feb. 6th.					
	7 p.m.	11 p.m.	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.
T. - - - -	103·4	102·8	100·8	99·6	101	102·4	103·2	102·4
P. - - - -	—	94	80	74	80	89	85	84
R. - - - -	—	30	26	24	25	26	29	34

—	Feb. 7th.						Feb. 8th.					
	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.
T. -	101·4	103·2	101·2	100·5	101	102	100·2	97·6	100	99·2	99·2	98·8
P. -	88	92	80	76	76	84	80	74	60	80	52	52
R. -	33	36	30	30	29	30	28	28	24	22	22	22

—	Feb. 9th.						Feb. 10th.		
	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.	3 a.m.	7 a.m.	11 a.m.
T. - - -	98·6	98	98·6	98·4	98·4	97·8	97·6	98	98
P. - - -	50	60	55	59	49	56	54	56	59
R. - - -	18	17	23	20	22	21	19	20	17

Case 6.—Frank Neill, 38. First attack, subject to bronchitis.

January 31st. Shivering, pains in head, general pains, lumbago, no vomiting, feels very cold, cough.

February 2nd. Delirium in evening, slept much, eyes red. Admitted 6.30 p.m.

February 3rd. Constipation, cephalalgia, pains in eyeballs. Tongue coated white, cough.

February 4th. Still intense headache, torpid.

„ 5th. Pains in joints, headache, and pains in eyeballs.

„ Pain left chest on coughing, occasional sweats.

—	Feb. 2nd.		Feb. 3rd.			Feb. 4th.				
	6.30p.m.	12.	4 a.m.	6 a.m.	8 p.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.
T. - -	100	100·8	100	102	101	99·8	99	100	99·6	100
P. - -	—	84	85	—	—	87	74	80	80	78
R. - -	—	22	18	—	—	22	20	20	22	19

—	Feb. 5th.						Feb. 6th.	
	4 a.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.	8 a.m.	12.
T. - - - -	99·8	98·4	98·4	97·4	98·4	98·6	98·4	97·4
P. - - - -	80	76	55	66	64	66	64	60
R. - - - -	18	20	18	19	17	19	17	20

Case 7.—Sidney Pinnell, 22, 'bus conductor. First attack.

January 31st. General pains, cephalalgia, feeling of malaise, spinal pain, violent delirium, tore up shirt, cough, to bed.

February 1st. No better, no solids taken.

„ 2nd. In bed still, no vomiting.

„ 3rd. Sent for doctor. Bowels confined since 31st. Cough. Tongue coated, red at tip and edges. Middle lobe left lung dull, few indistinct crepitations posteriorly.

February 4th. Dulness left posterior, but no crepitations or bronchophony, no expectoration.

—	Feb. 3rd.	Feb. 4th.					
	11 p.m	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.
T. - - - -	103·6	104·6	104	103	103·4	103·8	103·6
P. - - - -	104	100	—	100	100	100	98
R. - - - -	28	28	—	26	26	24	27

—	Feb. 5th.						Feb. 6th.					
	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.
T. -	103	103·6	102·8	102·8	103	103·4	101·6	102·2	100·4	101·4	102·2	100·4
P. -	96	97	94	94	94	92	90	92	84	84	84	82
R. -	26	26	24	24	26	24	24	26	24	24	24	22

—	Feb. 7th.						Feb. 8th.					
	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.
T. -	102	100·4	100·4	102	102	102·2	100·8	98·4	100	101·6	100·6	100·2
P. -	86	84	84	86	86	90	86	80	80	84	84	84
R. -	4	24	24	26	26	28	26	24	24	24	24	24

—	Feb. 9th.				
	3 a.m.	7 a.m.	11 a.m.	3 a.m.	7 p.m.
T. - -	99	97·8	98	98·2	98·4
P. - -	80	70	68	68	70
R. - -	24	20	18	18	18

Case 8.—Amy Walter, 24, single, servant.

January 28th. All the signs of influenza. Admitted to Infirmary. Has mucoid expectoration, crepitation of left base.

—	Jan. 28.	Jan. 29.		Jan. 31st.		Feb. 1st.		Feb. 2nd.		Feb. 3rd.	
	E.	M.	E.	7 p.m.	11 p.m.	3 a.m.	7 p.m.	8 a.m.	8 p.m.	M.	E.
T. - -	99·8	99·4	100	103·4	102·8	102·4	101·6	98·8	101	101·8	102·8

—	Feb. 4th.		5th.		6th.		7th.		8th.		9th.	
	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.
T. -	98	103	99·8	102·6	99	101·6	101	101·8	100	102·2	99·8	100

—	Feb. 10th.		11th.		12th.		13th.		14th.		15th.	
	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.
T. -	99·2	100	99	100·4	97·2	100	98	106·6	99	100	100	97·4

Case 9.—Emma Rollinson, 22, single.

February 6th. Lumbago, hacking cough, shivering morning of 6th with vomiting. Respiration quick, ? neurotic.

February 7th. No pain, slight cough, no expectoration, no lung trouble. Tongue clean. Costive.

February 8th. Better; respiration still quick. No cough, no pain.

—	Feb. 6th.		Feb. 7th.						Feb. 8th.	
	7 p.m.	11 p.m.	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.	3 a.m.	7 a.m.
T. - - -	103·2	103	100	96·2	97·6	99	99·2	98·4	98	96
P. - - -	105	120	80	70	71	73	80	70	60	70
R. - - -	60	60	55	50	54	56	70	60	37	30

Case 10.—Charles Sparkes, crossing-sweeper, aged 55. Has paralysis agitans.

February 9th. 10.30 p.m. all the symptoms of Influenza.
,, 11th. Cough.
,, 13th. Sleeps badly. Cough.
,, 14th. Much pain in head, back, and limbs.
,, 18th. Sputum purulent.

—	Feb. 10th.		11th.		12th.		13th.		14th.		
	a.m.	p.m.	M.	E.	M.	E.	M.	E.	M.	E.	12 p.m.
T. - -	104·4	99	102·1	98·4	98·4	98·4	98	101·4	100·3	102·2	102
P. - -	—	—	—	—	—	—	—	—	—	—	104
R. - -	—	—	—	—	—	—	—	—	—	—	26

—	Feb. 15th.						Feb. 16th.					
	4 a.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.	4 a.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.
T. -	100	98·6	101	100	100	102	98·8	98·8	98·4	100	99	100
P. -	80	78	—	—	—	70	64	76	76	79	70	70
R. -	24	24	—	—	—	23	17	20	20	17	21	20

	Feb. 17th.						Feb. 18th.	
	4 a.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.	4 a.m.	8 a.m.
T.	98·4	98·6	98	98·6	99	98	98·4	99·2
P.	64	80	70	70	74	64	64	68
R.	22	20	17	17	19	23	23	18

Case 11.—John Innis, 63.

February 9th. Suddenly attacked with pains in the head and limbs as well as in the lumbar region.

February 12th. Feels better. Temperature normal. Expectoration consists of frothy mucus.

Case 12.—Elizabeth Thorn, aged 14.

February 9th. In the evening felt generally ill.

February 10th. Worse, cephalalgia, shivering and pains in the back.

February 11th. Still feeling very ill. Pains in chest, no cough.

February 12th. Admitted 5 p.m. Running at eyes. Tongue coated, white. Flushed. Pupils very dilated, preeordial pain.

February 13th. Better. Sleeps. No pain.

	Feb. 12th.		Feb. 13th.					
	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T.	101·4	102·4	99·4	98·8	100	99·8	100·8	99·6
P.	—	120	100	89	96	100	106	102
R.	—	25	20	19	20	22	25	25

	Feb. 14th.						Feb. 15th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T.	100·2	99·2	99·4	101·2	101·2	101·2	101	99	98·4	101·2	101·6	100·2
P.	120	112	94	102	110	100	98	100	96	100	94	100
R.	22	28	29	29	28	24	20	23	25	25	25	26

	Feb. 16th.						Feb. 17th.		
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.
T.	98·2	98·8	99·2	101·4	98·6	101·4	99	98·2	98·6
P.	96	96	98	96	94	96	102	87	85
R.	26	24	25	26	22	26	25	22	20

Case A.—Mary Drew, 14.

February 10th. Disease commenced during the morning with shivering, headache, lumbago, and general pains in limbs.

February 11th. Flushed on admission, coryza. Bowels costive since 8th.

February 12th. Better. Pains in left side, precordial pain.

February 13th. Still better, sleeps well.

—	Feb. 12th.		Feb. 13th.				
	6 p.m.	10 p.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - -	100	99·4	98·8	99·2	100·4	100·2	98·6
T. - - -	—	130	102	108	108	115	120
R. - - -	—	25	25	22	22	29	25

—	Feb. 14th.						Feb. 15th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	99·8	98·4	99·4	101·8	100	99	98·8	99·8	98·2	99·6	98·6	98·2
P. -	125	108	104	120	106	102	100	101	102	105	110	104
R. -	29	25	24	26	29	20	22	25	25	25	25	25

—	Feb. 16th.					Feb. 17th.		
	2 a.m.	6 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.
T. - - - -	98·8	98·6	97·2	98·4	99·8	99·8	98·8	98
P. - - - -	104	100	100	98	96	94	106	90
R. - - - -	25	28	28	28	24	24	26	24

Case 13.—James Perry, 64, labourer.

January 6th. Admitted with bronchitis.

February 13th. Intense pains in head and back, coldness extending down the spine.

No physical signs of chest trouble, no increase of expectoration.

This case contracted probably from that of Charles Sparks.

January 14th. Sleeping well. Pains in calves. Taking antipyrin gr x quartis horis.

—	Feb. 13th.		Feb. 14th.					
	7 p.m.	11 p.m.	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.
T. - - - -	102·2	101	101·4	100·4	101·4	102·2	102·2	100·6
P. - - - -	—	90	100	98	90	100	100	100
R. - - - -	—	—	26	23	19	22	24	24

—	Feb. 15th.						Feb. 16th.					
	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.
T. -	96	98·4	98·4	98·8	98·2	97·2	97·4	97·4	97	98	98	97
P. -	89	70	82	82	75	84	68	64	82	82	74	70
R. -	23	23	18	19	18	18	17	20	19	19	18	18

—	Feb. 17th.						Feb. 18th.	
	3 a.m.	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.	3 a.m.	7 a.m.
T. - - -	96·6	98	99	99	99·2	97	97	97
P. - - -	64	80	69	80	70	64	60	63
R. - - -	17	20	17	19	17	18	18	18

Case 14.—Caroline Coulter, 27, married. Pregnant 8 months. Relapse of Influenza.

February 2nd. Admitted. Three weeks ago had Influenza. Now complains of pains in the head, back, and limbs.

February 7th. All pains gone except that in left groin extending down front of thigh. These probably due to pressure of pregnancy.

February 13th, 5 p.m. Pains in head, lumbago, shivering, pains in limbs.

February 14th. Restless night. Tongue much coated. Headache more acute. Countenance anxious. Respiration quicker, this (?) due to pregnancy. During the evening had more sleep. Tongue cleaner. No cough, no expectoration.

February 15th. Better. Headache gone.

„ 16th. Still improving, took food well.

—	Feb. 14th.					Feb. 15th.				Feb. 16th.	
	7 a.m.	11 a.m.	3 p.m.	7 p.m.	11 p.m.	3 a.m.	7 a.m.	3 p.m.	11 p.m.	8 a.m.	8 p.m.
T. - -	101·4	92·2	98·4	98	95·6	97	95·6	97	96·8	96·8	96·8
P. - -	—	100	94	80	70	60	60	—	—	—	—
R. - -	—	30	30	32	28	26	24	—	—	—	—

Case B.—Alice Smith, 53. Brought in by police.

February 13th. Admitted. Taken suddenly with weakness and faintness and loss of power in lower extremities, headache.

February 14th. Much headache, pains in back, "cold water running down spine," shivering, cannot get warm. The nurse stated that patient had repeated rigors. Flushed. Tongue rather coated. Hacking cough. 10.30 p.m. some glairy mucus. Sleeping, still flushed.

February 18th. Some muco-purulent expectoration.

Dr. Andrewes saw the patient, but did not take either expectoration or blood.

—	Feb. 13th.	Feb. 14th.				Feb. 15th.					
	11 p.m.	9 a.m.	1 p.m.	5 p.m.	9 p.m.	1 a.m.	5 a.m.	9 a.m.	1 p.m.	5 p.m.	9 p.m.
T. -	97·4	100·2	100·4	100·4	99·8	98·4	97·6	98·4	99·2	98·4	98·2
P. -	—	96	96	94	79	73	73	62	66	67	66
R. -	—	—	19	19	16	16	16	15	16	17	16

—	Feb. 16th.						Feb. 17th.					
	1 a.m.	5 a.m.	9 a.m.	1 p.m.	5 p.m.	9 p.m.	1 a.m.	5 a.m.	9 a.m.	1 p.m.	5 p.m.	9 p.m.
T. -	97·2	97	97	98·2	99·2	98	97·6	98·4	98·4	98·4	98·4	97
P. -	87	70	66	68	68	64	68	62	66	64	64	59
R. -	19	17	16	16	16	19	17	17	17	17	17	16

—	Feb. 18th.		
	1 a.m.	5 a.m.	9 a.m.
T. - - -	97·6	97	97
P. - - -	60	60	60
R. - - -	17	16	16

Case 15.—Frances Hughes, 38.

February 12th. Much headache and diarrhœa.

,, 13th. More headache, pains around loins, shivering.

,, 14th. Shivering, scarlet eruption.

,, 15th. Headache.

,, 16th. No sleep, but better generally.

This case was followed by peeling and branning of the skin of whole body and especially of the scalp. This feature has been noticed in many cases following the scarlet efflorescence.

—	Feb. 14th.	Feb. 15th.		Feb. 16th.		Feb. 17th.
	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.
T. - - - - -	101	99·8	101·2	98·4	99·4	98·4
P. - - - - -	100	—	—	72	—	—
R. - - - - -	—	—	—	—	—	—

Case C.—George Findon, 19. January 21st. Admitted with phthisis.

February 16th, 8.30 p.m. Headache, pains in back, and much shivering.

February 17th. Coughs. Old purulent sputum.

„ 18th. Nummular sputum ; all fever and pains gone.

—	Feb. 16th.		Feb. 27th.						Feb. 18th.	
	8 p.m.	12.	4 a.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.	4 a.m.	8 a.m.
T. - -	101·8	102·6	101·2	98	98	97·8	98·8	98·8	99	99·4
P. - -	96	70	74	—	80	74	98	80	70	88
R. - -	26	20	17	—	18	18	17	28	17	29

Case D.—Albert Snelling, 17.

February 16th. Shivering pains in head, coldness down the back. These symptoms appeared suddenly midday. Has also post-ocular pains.

February 17th. Still pains in head and eyes. Tongue dry not coated, no sore throat, slight cough, coryza.

February 18th. Slight mucо-purulent expectoration.

—	Feb. 16th.		Feb. 17th.						Feb. 18th.	
	7 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	4 p.m.	8 p.m.	12.	4 a.m.	8 a.m.
T. - -	101	100	99·6	98·4	99·6	99·4	99·2	99	97	99·2
P. - -	—	114	100	80	96	100	76	100	76	100
R. - -	—	26	23	23	21	26	24	26	24	24

Case 16.—Margaret Taylor, 48. Second attack.

February 17th. Headache, pains in back, intense shivering.

„ 18th. Headache, loss of appetite, slight cough, no sleep.

—	Feb. 18th.	Feb. 19th.			
	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.
T. -	102·8	101·5	100·6	101·6	101
P. -	—	120	135	105	112
R. -	—	29	35	29	44

Case 17.—Ann Williams, 46.

February 18th. Admitted to the Infirmary. Says she has felt ill since 10th instant, and has kept her bed. On the 17th, pains in eyeballs and all the symptoms of Influenza.

February 18th.—More headache and pains in the back and legs, but the latter were worse yesterday. The tongue is moist, coated. Expectoration mucoid. Right lung, rhonchi anteriorly ; left lung same,

posteriorly: few coarse rales, no dulness. Apathetic, sleeps badly, has pains in the left side of chest, apparently intercostal.

February 19th. Mucoid expectoration.

—	Feb. 18th.		Feb. 19th.		
	7 p.m.	10 p.m.	—	—	—
T. -	102·2	97·8	98·2	97·2	98·4
P. -	109	78	75	71	72
R. -	28	28	34	40	29

Case 18.—George Gray, 26.

February 18th. Restless night.

,, 19th. Headache, pains in loins, sorethroat, pains in eyes and calves of legs. During evening the symptoms the same. Has cough. No expectoration. Apathetic.

—	Feb. 19th.			Feb. 20th.					
	8 a.m.	8 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - -	103·4	103·6	103·4	103	102·8	101·4	102·6	102·2	101·4
P. - - -	108	113	92	94	92	84	82	82	80
R. - - -	22	22	20	22	20	20	20	20	20

—	Feb. 21st.				
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.
T. - - -	100	99·4	99·6	98·8	98·4
P. - - -	78	78	72	70	70
R. - - -	18	18	18	18	18

Case 19.—William Preston, 50, coal-heaver.

February 22nd. Taken suddenly ill with shivering, pain in the lumbar region, which continued till 24th when he was admitted.

February 25th. Feverish, still pain, sputum was muco-purulent.

,, 27th. Much nervous prostration, less cough.

—	Feb. 25th.			Feb. 26th.					
	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - -	101	102·4	101·4	100	99·8	100	99·6	100·4	98·6
P. - - -	—	106	80	80	80	73	82	76	79
R. - - -	—	26	21	21	21	22	22	23	18

—	Feb. 27th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T.	98·4	98·6	98·6	99·2	99·8	98
P.	78	64	62	60	66	66
R.	18	19	22	22	14	24

Case 20.—Charlotte Houghton, 65.

February 24th. Symptoms of influenza came on suddenly with rigors. Much pain in the calves of legs. Tongue not coated.

February 25th. Temperature 100·2, 1.0 p.m.

„ 27th. Convalescent. No bronchial symptoms.

—	Feb. 25th.			Feb. 26th.						Feb. 27th.		
	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.
T.	100·2	102·4	101·4	100	99·8	100	99·6	100·4	98·6	98·4	98·8	99·0
P.	106	106	80	80	80	73	82	76	79	78	64	60
R.	26	26	21	21	21	22	22	23	18	18	19	22

Case 21.—Harriet Griffiths, 40.

February 20th. At 11 a.m. shivering, lumbago.

„ 22nd. Same symptoms still present, with headache and pains in calves.

February 23rd. Admitted to infirmary.

„ 25th. No physical signs in chest.

„ 27th. Much pain in back and intercostal region, no expectoration, but cough present.

Note.—After February 29th developed phthisis, and died March 26th.

—	Feb. 23rd.	Feb. 24th.				Feb. 25th.					
	—	8 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T.	103·2	101·4	104	103·4	103·5	99·8	102·4	101	104	103·4	103·6
P.	—	—	112	108	100	102	104	100	126	102	100
R.	—	—	30	29	28	23	21	28	30	30	25

—	Feb. 26th.						Feb. 27th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T.	100·8	102·4	102·8	102·8	104·2	102·4	102·6	101·8	102·4	102·6	103·2	103·2
P.	100	100	108	120	100	106	100	100	116	118	120	100
R.	25	25	29	30	34	25	28	25	26	30	31	26

—	Feb. 28th.						Feb. 29th.		
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.
T.	100·4	100·6	100·2	103	99·4	100·2	99·4	98·4	97·6
P.	100	98	92	112	97	100	100	100	98
R.	26	26	24	28	27	30	30	28	26

Case 22.—Nurse Carson, 24.

February 25th. General pains, shivering, cough, not much headache, no pains in back, restless.

February 28th. Much nervous prostration, which continued for a week.

—	Feb. 25th.			Feb. 26th.			
	2 p.m.	6 p.m.	10 p.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.
T.	100·8	101	102	101·8	101	101·4	100·8
P.	—	—	—	104	115	105	105
R.	—	—	—	25	22	23	23

Case 23.—Belinda Brown, 43.

Admitted February 15th with hæmatemesis.

February 25th. Much pain in the head, shivering, not much lumbago.

February 27th. Much cephalalgia, much expectoration at night, no vomiting, no cough during the day, very little sleep.

—	Feb. 25th.		Feb. 26th.			Feb. 27th.					
	a.m.	p.m.	a.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T.	99·2	102	102·4	103·2	98·8	101·4	100·4	100·8	102·4	102·4	101
P.	—	—	—	110	86	80	80	100	106	102	100
R.	—	—	—	26	25	28	20	24	25	30	24

—	Feb. 28th.						
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	—
T.	101·6	101·6	100	100·8	100·4	100	Temperature, pulse, respiration normal afterwards.
P.	100	100	94	98	93	82	
R.	24	24	24	24	24	22	

Case 24.—Eleanor Colton, 63.

February 26th. Pain complained of in the early morning in calves, thighs, back; headache, no vomiting.

February 27th. Symptoms continued. Admitted to the Infirmary. Cough. Temperature 100° F. All symptoms of influenza.

February 28th. Intense headache.

—	Feb. 27th.			Feb. 28th.					
	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - -	101·4	103·2	102·2	100·8	99·2	98·8	100·6	101·8	102·8
P. - - -	100	103	100	98	96	70	80	89	109
R. - - -	34	34	27	27	27	30	36	27	35

—	Feb. 29th.						Mar. 1st.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	101·4	100	98	100	100·4	102·2	101	100·4	100·6	102·2	102·4	100·6
P. -	102	84	85	89	90	100	90	84	92	79	89	92
R. -	31	30	31	34	36	36	26	26	33	26	62	33

—	Mar. 2nd.				Mar. 3rd.		Mar. 4th.		Mar. 5th.		Mar. 6th.	
	2 a.m.	6 a.m.	10 a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
T. -	100·2	99·8	100	99·6	98	99·2	98·4	99	98·2	99·4	98	98
P. -	92	80	72	—	—	—	—	—	—	—	—	—
R. -	32	30	20	—	—	—	—	—	—	—	—	—

Case 25.—Thomas Dean, 44.

Admitted April 2nd, 1892. Has worked up to 25th ultimo. Left work at Whiteley's on 2nd. General pains commenced on 29th March with shivering, vomiting, cephalalgia, lumbago. Felt very ill on the 1st April.

April 4th. Sputum rusty. Left pneumonia, lung dull on percussion, with crepitation. Much pain left side of chest, but feels generally better. Tongue coated. Bowels relaxed. Urine dark coloured.

April 7th. Very little expectoration, crepitation of right base appeared, with dulness.

—	April 2nd.			April 3rd.					
	4 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - -	103·4	104	103·2	102·8	103·8	103·8	103·8	104·2	103·2
P. - - -	—	80	90	80	90	80	80	88	90
R. - - -	—	34	30	30	30	28	28	30	36

—	April 4th.						April 5th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	102·6	103·2	102·8	103·2	104·2	103	103	103	103·2	100·4	101·4	101
P. -	90	90	90	96	90	90	90	90	96	90	82	90
R. -	28	36	36	36	36	36	34	36	36	40	26	30

—	April 6th.						April 7th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	101	100·4	101	101·4	100·6	101	101·4	101	102·6	101	102·6	102·8
P. -	90	96	80	86	80	86	86	84	98	88	80	92
R. -	28	26	36	40	38	28	34	30	36	26	28	36

—	April 8th.						April 9th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	103	100·4	102·8	102·4	102·6	103·8	102·8	102·8	102·4	102·2	102·2	102·4
P. -	98	88	96	100	100	94	80	98	96	96	100	100
R. -	36	32	40	32	38	36	34	40	42	34	34	48

—	April 10th.						April 11th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	102	100·6	100	101·2	101·4	101	99·4	100	97·4	99	99·8	99·4
P. -	100	98	96	96	92	94	92	96	92	92	96	90
R. -	46	40	44	42	46	44	40	40	40	36	42	24

—	April 12th.						April 13th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	99·4	99·6	97·4	98·2	98·6	100	100	100	98·4	100	99	99·6
P. -	100	98	98	84	94	94	90	90	100	100	90	93
R. -	44	40	35	28	34	38	36	30	32	34	30	36

—	April 14th.						April 15th.				
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.
T. - -	100	98·6	97·6	99	98·8	98·6	97·6	97·8	98·2	98·4	98·4
P. - -	90	90	76	100	88	100	88	100	96	96	90
R. - -	34	32	32	32	32	30	30	32	30	30	30

Case 26.—Sophia Pearson, 65.

February 26th. Pain in calves, no lumbago, some headache, no vomiting.

February 27th. Admitted to the Infirmary with general pains. No cough. Lies prostrate.

—	Feb. 27th.		
	2 p.m.	6 p.m.	10 p.m.
T. - -	98·8	101·6	101·4
P. - -	86	94	82
R. - -	30	30	26

Case 27.—James Fitt, 27. Admitted April 1st.

Symptoms of influenza began 28th March with shivering, cephalalgia, feeling of cold extending down spine.

Has had cough for one month, this worse since 28th. Took to his bed 30th, in bed since.

April 2nd. Still complains of pains, which are general.

April 4th. Much headache, and pains in back, a little mucoid expectoration, pulse full.

—	Apr. 1st.	Apr. 2nd.				Apr. 3rd.					
	6 p.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	102	100	101·2	100	100·8	101	98·4	100	100·2	101·4	100
P. -	—	—	83	80	70	71	62	80	69	88	65
R. -	—	—	22	27	20	26	21	30	29	26	22

—	Apr. 4th.			
	2 a.m.	6 a.m.	10 a.m.	2 p.m.
T. - - -	97·8	97	97·8	101·2
P. - - -	60	60	60	70
R. - - -	20	22	30	31

Case 28.—Walter Holmes, 29.

February 18th, 4 p.m. Had pain in back, headache, much pain in calves, shivering.

February 19th admitted. Pulse full, temperature 102·2° at 5 p.m. Flushed. Had a rigor at 1.30 p.m. Has had a cough for one week.

February 20th. Better. Coughs still. Expectoration muco-purulent. Flushed. Feels hot.

February 21st. Cold sweat. Cephalalgia, lumbago. Bowels open for first time.

—	Feb. 19th.			Feb. 20th.					
	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - -	102·4	103·4	103·4	100·8	102	100	100·4	99·8	100·4
P. - - -	—	100	94	84	93	90	88	80	80
R. - - -	—	20	24	20	24	26	25	24	22

—	Feb. 21st.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - -	98	97·4	97·6	98·4	99·6	99
P. - - -	64	62	68	68	68	70
R. - - -	16	15	17	18	16	15

Case 29.—Emma Quirk, 19.

March 27th. Attack of influenza commenced with shivering, no lumbago, but headache and much vomiting.

March 30th. In bed ; felt very ill, then lumbago came on, vomiting continued. 31st. Admitted.

April 1st. Has been constipated one week. Bowels open to-day No headache. No expectoration.

—	Mar. 31st.	Apr. 1st.		Apr. 2nd.		Apr. 3rd.	
	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
T. - - - - -	100·4	100	100	98·4	97·4	98·6	97·4

Case 30.—John Gatey, 34. Has been in the workhouse for 10 days. No cases there.

March 26th. Shivering, headache, lumbago, heaviness, no cough. This is his second attack. Not subject to bronchitis. 27th. Better. 28th. No pains now.

March 30th. Not so well, felt hot in head, much headache, feet and legs very cold, no cough.

April 2nd. Headache.

„ 4th. No pains. Got up this evening.

—	Mar. 26th.								
	12.	—	—	—	—	—	—	—	—
T. - - -	102·8	104·4	104	103·8	101	99·6	97·8	98	98
P. - - -	99	110	100	106	88	88	79	79	66
R. - - -	20	22	28	22	16	16	15	16	16

—	Mar. 28th.		Mar. 29th.	Mar. 30th.		Mar. 31st.					
	a.m.	p.m.	a.m.	10 p.m.	12.	4 a.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.
T. -	97·4	97·4	97·4	100	100·8	99·4	99	99·2	100·4	99·4	100
P. -	66	—	—	—	90	72	72	80	86	85	79
R. -	14	—	—	—	14	15	16	18	17	18	16

—	Apr. 1st.						Apr. 2nd.	
	4 a.m.	8 a.m.	12.	4 p.m.	8 p.m.	12.	4 a.m.	8 a.m.
T. - - - - -	99·4	97·4	97·6	97	97	97	97	97
P. - - - - -	90	70	73	66	—	—	—	—
R. - - - - -	16	16	16	14	—	—	—	—

Case 31.—John Summers, 54, shoemaker. Admitted March 30th.

March 27th, 9 p.m. Shivering, feeling very cold, pains in head, back, and chest. Bowels open.

March 28th. Stayed at home.

„ 30th. Headache; lumbago. First time that he has felt warm since attack commenced on 27th. Tongue dry. Expectoration rusty.

April 1st. Has pneumonia of left base, some crepitations, bronchophony, very dull on percussion. In evening sleeping, less cough, but looks worse.

April 2nd. Rather better.

„ 4th. Coughs more, sputum yellowish. Pain of the left side.

„ 7th. Left lung very dull, tubular breathing, few coarse crepitations.

—	Mar. 30th.			Mar. 31st.					
	5 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. - - - -	103·4	103	103·4	102	101·8	103·2	105·2	105·2	104
P. - - - -	—	—	—	70	74	72	72	80	76
R. - - - -	—	—	—	20	22	24	24	26	22

—	Apr. 1st.						Apr. 2nd.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	101·8	102	102	105	103·6	102	101·6	101	103·6	104·2	104·4	103
P. -	70	78	78	88	—	86	84	90	90	80	88	80
R. -	20	25	24	28	—	36	30	40	38	36	40	40

—	Apr. 3rd.						Apr. 4th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	103	103	102·8	102·8	103·2	102·2	102	102·8	101·4	101·6	101	101
P. -	80	90	80	80	90	80	90	90	90	90	86	86
R. -	30	27	38	36	38	34	41	40	40	40	34	34

—	Apr. 5th.						Apr. 6th.					
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.
T. -	101	101	102	101·4	101·2	101·4	101	102·4	102	101·6	100·6	100·6
P. -	90	90	96	96	90	90	90	96	96	80	96	94
R. -	40	40	47	44	34	38	38	48	48	48	—	38

—	Apr. 7th.				
	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.
T. - - -	94·4	99·6	100·4	101·4	99·6
P. - - -	96	80	86	88	84
R. - - -	46	36	32	36	38

From this date until April 16th the temperature pulse and respiration were taken every four hours regularly and it varied between 99·0° F. and 101·0° F.

Case 32.—John Walker, 47. Admitted April 6th.
April 5th, 10.30 p.m. Headache, lumbago, shivering, general pains.

—	Apr. 6th.		Apr. 7th.		
	7 p.m.	11 p.m.	3 a.m.	7 a.m.	10 a.m.
T. - -	103·6	101·4	99	97·4	96·6
P. - - -	84	80	80	78	—
R. - - -	22	23	23	21	—

[*This concludes the record of cases observed at Kensington.*]

Cases at St. Bartholomew's Hospital.

Case 34.—Edwin Smith, age 33, 6, Bartholomew Square, Old Street
Wife taken ill February 10th, took to her bed with symptoms of influenza. Himself taken ill February 14th; headache, shivering, pains all over. Seen on February 15th. Temperature 101·8°. Typical influenza, but no bronchitis and no sputum.
Blood taken on 15th.

Case 35.—Man seen in surgery, January 28th.
Gave history of attack of influenza nine days before; bronchitis beginning four days later.

Case 36.—Man seen in surgery February 1st.
History of attack of influenza nine days before, followed in a few days by bronchitis.

Case 37.—Frederick Bartells, age 35, turner.
Illness began on February 2nd. Frontal headache. "Hair felt sore." Pain in legs and weakness. Feverish. Anorexia. Chilliness during the night. Restless and talked in his sleep. Next day, occasional pains in his limbs. Headache, thirst, and anorexia. Not more weak. No cough. A little running from the nose. Does not know how he got it. T. 100·1. P. 96 soft, fair volume.

Case 38.—Daniel Callaghan, 25, night watchman.
Caught a chill on January 30th and next morning felt general stiffness, thirst, and impaired appetite.
February 1st. Headache. February 2nd. Anorexia. Commencing cough. Small amount of expectoration. Pains more or less all over. Much debility especially in small of back.
February 3rd. Restless night. Tongue slight fur. Cough worse. Examination of chest reveals nothing, save a little emphysema.
Past history. Always fairly healthy except for winter cough. Says he had influenza twice last winter.
On February 3rd temperature 101·5°. Pulse 108, soft, regular.

Case 39.—John Heales, 26, umbrella-frame maker.
Illness began on February 1st with frontal headache. No pain in back or limbs. Has never before had influenza.
February 3rd. Has slept well. Feels rather weak. Slight cough and expectoration. Slight fur on tongue. Anorexia and thirst. Bowels open. Headache continues. Temp. 99°. Pulse 96.

Case 40.—Charles Grigson, 30, carman.
Wife began with influenza on January 26th.
February 5th, at 5 a.m., when he got up he felt bad; had frontal headache, chilliness, and pain in the back. A little thirst. Anorexia. Chilliness on and off all day.
February 6th. Fair night. Less headache. A little chilliness this morning. Tongue furred. Anorexia. Feels weak and has pain in loins. Rather pale. No cough. Pulse soft. Temperature 100°.
February 8th. Still weak and ailing. Sweats readily. Tongue furred. Headache gone. Slight cough. Pulse full and soft. Temperature 99°.

Case 41.—Charles Trotman, 16, tobacconist.
Went to see his brother-in-law who had influenza on February 7th, and left at 10 p.m.
February 10th. At 7 a.m. he had a headache and shivered on getting out of bed. Pain in back and arms feel heavy. 10.30 a.m. Pale and

nervous, feels ill. Slight fur on tongue. Did not eat his breakfast well. Thirst. No nausea. Pulse 144, soft and small. Temperature 102.8.

February 11th. At 3 p.m. much better but still ill and weak. No pain or headache. No cough. Temperature 99·4.

Case 42.—W. C. Hart, 24, printer's labourer.

February 12th. At 1 p.m. he felt ill and could not eat his dinner. Also had a little headache and pains in limbs. Kept on shivering most of the day.

February 13th. Slept heavily. Shivered this morning and still feels chilly. Anæmic and very weak. Frontal headache. Pains all over limbs, but not in back. Tongue furred. Thirst. Anorexia. Bowels open. Slight cough. Scanty expectoration. Pulse very soft. Temperature 100·4° at 10.30 a.m.

February 15th. Sweated freely on 13th. Feels weak and has pains in back but none in the limbs. No headache. Tongue clean. Pulse full and soft. Slight cough. Temperature normal.

Case 43.—Alfred Grey, 20, baker.

February 14th. In morning headache and vomiting. Chilliness. Thirst. Anorexia. Pains all over.

February 15th. Shivering during night. Temperature at 3 p.m. 102·4°, at 7 p.m. 104·2°.

February 16th. Flushed. Tongue a little furred. Pain in right side. Pneumonic sputa. Pulse 116. Respiration 36. Temperature 100·6° at 3 p.m.

Case 44.—Alexander Clark, 22.

February 23rd, 12.30 noon. Unable to eat his dinner. 1.30 p.m. Frontal and occipital headache. Feeling of weakness in limbs, no definite pains. Feverish at night. Thirst. Faintness and nausea at times.

February 24th. Good night. Still faintness and nausea. Anæmic and weak. Slight fur on tongue. Anorexia. Bowels not open, two days. No cough. Pulse full and soft. Temperature 101°.

February 25th. Much better. Weak. Temperature normal.

Case 45.—Henry Penhallow, 23.

February 23rd. Seedy at night. Feverish.

„ 24th. Woke at 4 a.m. with frontal headache. Pain in loins. No nausea. Slight shivering. Ate his breakfast. 2.30 p.m. temperature 99·7°. Still has pain in back and he feels weak and giddy.

PART III.

(*a.*) *The Bacteria of the Bronchial Sputum in Influenza.*

Although the number of observers who have described various species of bacteria in the bronchial sputum in influenza is considerable, the majority have failed to demonstrate any particular species having definite relation to the disease, or as occurring with any constancy in the sputa. Thus Jolles (1) found a capsulated bacillus which in many respects resembled the bacillus of Friedlander and Weichselbaum, others have found (2) the capsulated diplococcus pneumoniæ, Kirchner (3) again has described a different diplococcus also capsulated, Ribbert (4), Finkler (5), Friedrich (6), and others have found the streptococcus pyogenes.

On Influenza in its Clinical and Pathological Aspects; by Dr. Klein.

R. Pfeiffer (Deutsche Med. Wochenschrift No. 2, 1892) was the first who made the announcement that, in all cases of Influenza, there are present in the characteristic grey purulent bronchial secretion enormous numbers of minute non-mobile bacilli. He describes these as occurring only during the acute stages and gradually diminishing in numbers as the disease abates. The bacilli, he tells us, are very minute, about the thickness of the well-known bacilli of Koch's mouse septicæmia but only half their length; they stain with some difficulty in anilin dyes, requiring a somewhat prolonged application of the dye. In stained specimens, these bacilli have a characteristic appearance, inasmuch as their protoplasm is segregated into a stained granule at each end while the middle portion remains unstained and shows only the outline of the sheath. Thus the bacillus looks like a diplococcus, and where two such bacilli are placed end to end they look like a chain (streptococcus) of four spherical cocci. In the sputum these bacilli occur in smaller and larger masses, occasionally almost as a pure culture. In severe cases they form continuous masses in the peribronchial tissue and also in the subpleural lymphatics, and they are also met with inside the leucocytes of the sputum. As the disease passes off, so the bacilli disappear from the sputa. These bacilli are constantly present in influenza, but do not occur in the bronchial secretion of other bronchial or pulmonary affections.

Kitasato, in the same paper, gives his observations on the cultivation of these bacilli of Pfeiffer, and records that they have culture characters by which they can be readily distinguished from other bacilli; that they are, in fact, a definite species not occurring in any disease except in influenza. They do not thrive at temperatures below 28° C., that is to say, at temperatures at which nutrient gelatine still keeps its solid condition. They grow well in broth and on Glycerin Agar at 37° C. or thereabouts. The broth does not become turbid, but remains limpid. The growth in broth appears as whitish small granules and flocculi; on Glycerin Agar the bacilli form minute translucent colonies like droplets, having no tendency to coalesce as growth proceeds. The cultures are also characterised by this fact that they soon die, and therefore subcultures cannot easily be carried on through many generations. In stained specimens grown in cultures, the bacilli retain the same characters observed in the bacilli of sputum, viz., they show the characteristic bipolar staining.

These statements and observations of Pfeiffer and Kitisato are very definite, and if confirmed would afford strong reason for believing that in these bacilli we had found the special microbe of influenza. The life history of this microbe would conform with what we believe to be the facts about the contagium of influenza, its being spread and received by the organs of respiration, and the reception of the infection by the same channel; the presence in most cases of influenza of some kind of bronchial disturbance more or less pronounced, showing itself at the outset of the disease or a few days later, and increasing after the febrile stage of the complaint had been passed.

From our own observations of a large number of cases, including those to be immediately recorded, we find ourselves in a position to confirm the statements of Pfeiffer and Kitisato in all essential points; and accordingly we have arrived at the conclusion that the particular bacilli as described by them ought to be regarded as the specific microbe of influenza.

The cases in which we examined the bronchial sputum were the following; particulars respecting the patients at the time of collecting the sputa being usually given:—

Case 1.—F. B., male. On February 4th (third day of illness) expectoration muco-purulent. Cover-glass specimens and cultivations were made of the sputum. On February 5th sputum copious, muco-purulent. Made again cover-glass specimens and cultivations.

Case 2.—W. H., male, taken ill suddenly on January 19th. On January 25th had relapse. On February 2nd bronchitis, with muco-purulent expectoration. On this day took expectoration and made cover-glass specimens and cultures. On February 3rd sputum is less copious, patient feels better, walks about, and has much less bronchial trouble; sputum used for cover-glass specimens and cultivations; so also on February 6th, on which day there was very little sputum expectorated, the patient feels much improved.

Case 3.—C. J., male. Was taken ill suddenly on January 18th. Admitted January 25th, muco-purulent expectoration. On February 2nd expectoration rusty, pneumonia. Took rusty sputum for cover-glass specimens and cultivation. On February 3rd expectoration copious, rusty, and offensive. Made again cover-glass specimens and cultivation. Patient died on February 3rd, in the evening.

Case 4.—E. J., female. On February 2nd expectoration slightly rusty. Made cover-glass specimens and cultivations.

Case 5.—J. N., male. Third attack of influenza on February 3rd. Pneumonia, with rusty sputum, commenced February 4th. On Feb- 8th sputum still rusty. Made cover-glass specimens and cultivation. On February 9th sputum less copious and less rusty; used for cover-glass specimens.

Case 6.—F. N., male. Illness commenced January 31st. On February 5th muco-purulent sputum taken for cover-glass specimens and cultivations.

Case 7.—S. P., male. On February 5th took a small amount of grey expectoration for cover-glass specimens and cultivation.

Case 8.—A. W., female. On January 28th was admitted to the Kensington Infirmary with all the signs of influenza; has much expectoration; made cover-glass specimens and cultivations.

Case 10.—C. S., male. Was taken ill on February 9th at 10.30 p.m. with all the symptoms of influenza. Cough on the 11th of February. On the 12th slight expectoration, muco-purulent; made cover-glass specimens and cultures.

Case 11.—J. I., male. Was suddenly taken ill on February 9th. On February 12th temperature normal, a small amount of frothy mucoid expectoration, used for cover-glass specimens and cultivations.

Case 17.—A. W., female. Influenza commenced February 17th. Mucoid expectoration on February 19th, used for cover-glass specimens.

Case 19.—W. P., male. Was taken suddenly ill with influenza on February 22nd. On February 25th still fever-temperature, and with muco-purulent sputum, made cover-glass specimens.

Case 25.—T. D., male. On April 4th sputum rusty, pneumonia. On February 8th sputum still rusty, used for cover-glass specimens and cultivation.

Case 31.—J. S., male. Influenza commenced on March 27th. Admitted March 30th with pneumonia, sputum rusty. March 31st sputum rusty; made with it cover-glass specimens and cultivation. April 1st pneumonia with rusty viscid sputum and high temperature still going

on, and now made a new cover-glass specimen and cultivation of rusty sputum.

Case 35.—Man seen on January 28th at St. Bartholomew's Hospital; had attack of influenza on January 19th, bronchitis began January 23rd. Sputum was used for cover-glass specimens.

Case 36.—Man seen on February 1st at St. Bartholomew's Hospital, was attacked with influenza on January 23rd, followed in a few days by bronchitis. Sputum used for cover-glass specimens.

Case 53.—M. F., male, seen at Bartholomew's Hospital on February 29th. Sudden onset of influenza on February 25th. On February 26th cough and expectoration. February 29th much mucuous expectoration. General bronchitis. Sputum used for cover-glass specimens and cultivations.

**Case* 54.—E. F., male. Had an attack of influenza about six days previously. Bronchitis with copious muco-purulent expectoration. Cover-glass specimens and cultivations were made.

**Case* 55.—J. M., male. Had an attack of influenza about 6, 7, days previously; bronchitis, copious muco-purulent expectoration. Cover-glass specimens and cultures were made.

**Case* 56.—A. A., male. Had bronchitis for several days after the febrile attack had subsided, but was getting better. Cover-glass specimens and cultures were made.

In addition to these cases the lung was obtained after post-mortem examination from three cases of influenza-pneumonia. These will be described in connexion with Case 3 which also died from pneumonia.

The bronchial expectoration then was examined in 20 cases from the living patient; of these five were cases of genuine influenza-pneumonia, that is of pneumonia setting in very soon, a few days, after the attack of influenza commenced, and where the history showed that the pneumonia was to be regarded as a part of the disease and not as a secondary complication.

Before collecting the expectoration the patient as a rule rinsed the mouth and gargled the throat twice or thrice successively with warm water; by coughing sputum was brought up and placed in sterile salt solution. In all cases the cover-glass specimens (consisting of a thin film of sputum spread out on a cover-glass and then dried) were stained in carbolmethylblue, whereby all bacteria present were well stained.

The cultivations of the expectoration were made after the manner practised, and described in the reports of the Medical Officer of the Local Government Board, 1889, in making cultivations of diphtheria membrane; namely, a particle of the substance is placed in a few C.C. of sterile salt solution contained in a sterile test-tube and washed by shaking; then it is transferred to a new test-tube containing a few C.C. of sterile salt solution and shaken; from here to a third quantity of sterile solution and here well broken up by shaking. From this last lot by means of the platinum wire or platinum loop a number of broth tubes and Agar tubes are inoculated. This is practically the same method which was also used by Kitisato. The important point to be borne in mind is that the sputum be washed in sterile solution whereby the bacteria added to its surface by its passage through the pharynx and mouth are mostly removed, and further that from the lump of sputum

* These last three cases were lads in Dr. Barnardo's Home, subjects of influenza in January 1890.

after superficial washing in sterile salt solution a muco-purulent looking particle is selected and transferred to a new lot of sterile salt solution. In this way most of the bacteria not belonging to the bronchial secretion itself are removed. The difference between a so-prepared particle of sputum and one examined without any previous washing, &c. is striking; in the latter case the number of different bacteria is considerable, having been added to the bronchial sputum during its passage through the pharynx, fauces or mouth; whereas in the washed sputum we find but a small variety of bacteria, sometimes indeed none except the particular species described by Pfeiffer and now regarded as being the special influenza bacillus.

The examination of the stained cover-glass specimens was generally made antecedent to the cultivation; this was in so far of importance as it gave an insight into the extent of the precautionary processes that had to be taken before inoculating the culture tubes. In some cases in which the sputum examined in cover-glass specimens contained an abundance of Pfeiffer's bacilli almost without admixture, inoculations made with the platinum needle yielded, even from the first washing, pure cultures. If the examination of the stained cover-glass specimens showed the particular species of Pfeiffer in great preponderance there was little need for further special process in order to obtain pure cultures of Pfeiffer's bacillus; inoculation of broth tubes made with the platinum needle from the second or third washing was sure to yield pure cultures in some of the tubes. But there were cases in which the sputum after being well washed, and examined in cover-glass specimens showed nevertheless an abundance of various species of bacteria; the particular bacilli of Pfeiffer could be easily found in larger and smaller masses, but crowds of other bacteria (diplococci, streptococci, thick short bacilli, fine long bacilli) were also present. In such cases we did not succeed in obtaining pure cultures of Pfeiffer's bacilli by the above method of inoculating by the needle. We had to modify the plan of inoculation by first charging a few C.C. of salt solution with a platinum *loop* of the second or third washing and then making from this culture a fresh inoculation of broth or Agar tubes; again with the help of the platinum *loop*.

*Examination of the Cover-Glass Specimens made of Influenza Expectoration.**

Case 1.—In both sets of cover-glass specimens, viz., those made of sputum on February 4th and February 5th, there are present in great numbers small and large masses of Pfeiffer's bacilli, viz., minute bacilli with rounded ends showing the polar-stained granules. The bacilli are easily identified where in masses, but many of them are scattered singly and in twos and threes. Their thickness is $0{\cdot}4\ \mu$, their length $0{\cdot}8\ \mu$. The polar arrangement, as already explained, of the protoplasm is well marked and therefore single or sparse bacilli of this type scattered about the field cannot readily be distinguished from a diplococcus. With oil-immersion ($\frac{1}{12}''$) lens, however, the clear sheath of the bacillus, extending between the polar granules can be definitely made out. In some places the bacilli are found in juxtaposition and then they gave

* In view of the aggregate results obtained in this inquiry, showing the special relation to influenza of the bacillus of Pfeiffer, we propose to use indifferently the words Pfeiffer-bacillus and influenza-bacillus; desiring the reader, however, not to charge us with prejudging this relation before we had become satisfied about it by our own researches.

the appearance of four granules forming a chain, each pair of granules in fact belonging to one bacillus.

The Pfeiffer bacilli were more numerously met with in the sputum taken on February 5th than in that of the preceding day. There are other microbes present, and amongst these in the sputum taken on February 5th strings and clumps of the capsulated bacillus of Friedlander can be identified (Figure 11).

Case 2.—This was in so far an interesting case, as the cover-glass specimens of the sputum taken on February 2nd showed an almost pure culture of the specific bacillus (Figure 8). The sputum is permeated by the bacilli, singly, in dumbbells and in larger and smaller groups. Numerous pus cells are seen in the sputum, and in many of these the bacilli are lodged in the protoplasm; in some cells only two or three bacilli (Figure 12), in others more numerously, in a few the protoplasm is crowded with them. In most of the bacilli, the polar staining is well shown.

The cover-glass specimens made of the sputum on February 3rd showed the same species of bacilli, but their number is distinctly smaller than in the sputum taken on February 2nd. On February 3rd, be it remembered, there was already a distinct improvement noticed in the condition of the patient.

On February 6th (the patient being much improved and very little sputum expectorated) cover-glass specimens were made from the sputum and they showed numerous bacteria of different species (thick diplococci and long bacilli) but it was difficult to find with certainty any but isolated Pfeiffer bacilli.

Case 3.—The sputum of this case, taken on February 2nd after washing, was almost a pure cultivation of the influenza bacillus (Figure 9), many of the bacilli isolated, others in numerous groups; they were contained numerously in the interior of pus cells (Figures 13 and 14). There were very few other bacteria present, here and there a large diplococcus. The cover-glass specimens made with the juice of the inflamed portion of the lung after death contained the influenza bacilli in considerable masses, but there were present everywhere in the specimen numbers of other bacteria, chiefly streptococci, then largish diplococci, and also some cylindrical bacilli.

Case 4.—Few influenza bacilli in the specimens, chiefly in groups, but also other bacteria.

Case 5.—There were present numerous clumps of the Pfeiffer bacilli in the specimens made of sputum of February 8th (Figure 15), but in those of February 9th (when the patient was much better and had less expectoration) the number of these bacilli was distinctly less, few small clumps only to be found. This is a second good illustration of the rapid decrease of the influenza bacilli in the sputum, as the condition of the patient improves.

Case 6.—Few of the influenza bacilli, singly and in small clumps, very few other bacteria present.

Case 7.—Few groups of the influenza bacilli; on the whole the number of bacteria small.

Case 8.—Small groups of influenza bacilli, also some isolated.

Case 10.—Great abundance of the influenza bacilli, isolated, in small and in large clumps (Figure 16). Few other bacteria present.

Case 11.—Few of the influenza bacilli, few of other bacteria.

Case 17.—Few clumps of the influenza bacilli, also here and there an isolated one or groups of two and three.

Case 19.—The number of influenza bacilli considerable, groups of them being found everywhere. Few other bacteria.

Case 25.—Great abundance of the influenza bacilli, isolated in small and in large groups.

Case 31.—Specimens made on March 31st show the influenza bacilli in abundance, also those made on April 1st though in the latter they were not quite so abundant, and were mixed with numerous other bacteria. But the specimens made on March 31st show in some places the influenza bacilli almost like a pure culture (Figure 18).

Case 35.—Bacteria very numerous and amongst them scattered characteristic clumps of the influenza bacilli.

Case 36.—Many influenza bacilli scattered about, but no characteristic clumps of them.

Case 53.—Great abundance of bacteria (Figure 17), amongst them numerous characteristic clumps of the influenza bacilli.

Case 54.—Great abundance of the influenza bacilli, isolated in small groups and in large clumps, in some places almost in pure culture (Figures 5 and 6).

Case 55.—Numerous clumps of the influenza bacilli (Figure 7), pus cells enclose the bacilli but never more than a few bacilli in one pus cell.

Case 56.—Few influenza bacilli only, none in the interior of the pus cells.

The result then of these examinations confirms fully the assertions of Pfeiffer, viz., that the characteristic influenza bacilli are constantly present in the bronchial sputum of influenza cases; that in well-marked cases they occur in great abundance, singly, in small groups and in larger masses, and in some portions of the sputum almost as a pure culture. The results also go to confirm Pfeiffer's statement that as the disease abates, as the patients gets better and as the sputum becomes scantier, the number of the bacilli also rapidly diminishes; this was the case in the sputum from patients having pneumonia of influenza (Cases 3, 5, 25, 31):—before the height of the disease is passed, the number of the characteristic bacilli is very great, after the height of the disease it diminishes (Case 4, second examination of Cases 5 and 31). Also in the cases of bronchitis, the number of the characteristic bacilli is found at first to be considerable, but when the disease abates and the patient improves, as in Case 2, their number becomes greatly diminished.

It deserves notice, as a matter of no small practical importance, that in cases of acute influenza with bronchial expectoration, the *fluids of the mouth* contained abundance of influenza bacili. Thus cover-glass specimens of such bronchial expectoration that had not been washed at all (or at best not well washed) showed scaley epithelical cells, derived from the oral cavity or fauces, literally crowded with masses of what, when duly stained, looked exactly like the typical influenza bacilli. Figure 5, for instance, exhibits a large epithelial scale in this condition.

Dr. Ashburton Thomson, be it noted, in his Report on the Epidemic of Influenza in New South Wales during 1891, strongly affirms, and we think justifiably, the infectious character of the secretions of the mouth in this disease.

Cultivation of the Bronchial Sputum.

The isolation by dilution, and the growth in broth culture, of the influenza bacillus succeeded without difficulty in the following cases:—

(*a*)	Case	1,	sputum of February	4th;
(*b*)	,,	2,	,, ,,	2nd;
(*c*)	,,	3,	,, ,,	2nd;
(*d*)	,,	5,	,, ,,	8th;
(*e*)	,,	6,	,, ,,	5th;
(*f*)	,,	10,	,, ,,	12th;
(*g*)	,,	25,	,, ,,	8th;
(*h*)	,,	31,	,, March	31st;
(*i*)	,,	53,	,, February	29th;
(*j*)	,,	54, and		
(*k*)	,,	55.		

In these 11 cases one or the other broth-tube afforded a pure culture of the influenza bacillus, while one or the other broth-tube remained sterile or was turbid as early as 24–48 hours at 37° C. owing to the rapid development of cocci or mobile bacilli.

1. *Culture in Broth.*—Broth-tubes containing a pure culture of the influenza bacillus remain quite limpid; at the bottom of the fluid there are noticed already after 24 hours, but better after 48 hours, a few whitish-grey irregular granules or flocculi, which during the next two or three days increase in size and number and form at the bottom of the tube greyish-white nebulous fluffy masses; when shaken they break up into whitish-grey granules and floccuti, but soon again settle at the bottom of the fluid, leaving the rest of the broth perfectly limpid. In four or five days (at 37° C.) the growth has reached its maximum. Sub-cultures show the same characters, but we generally noticed that as the number of removes increases, the broth has tendency to show slight turbidity after one, two, or three days' incubation, minute granules sticking to the wall of the tube and showing themselves also in various layers of the fluid.

Furthermore, in successive sub-cultures it is noticed that the amount of growth (flocular masses) at the bottom of the fluid is not invariably the same, being decidedly less in the later than in the earlier sub-cultures.

A point of great interest is the comparatively rapid death of the bacillar elements in the broth cultures. Unless the transmission is carried on within two, three, or four, up to seven days, it will be found that the sub-cultures are sterile; broth cultures from eight to ten days old are very uncertain, broth cultures a fortnight old yield no living organisms to subsequent sub-cultures. But if the sub-cultures are set up every two or three days we did not find a limit to the number of generations to which some of our cultures could be carried on; although in other cases after about a dozen generations in broth no living sub-cultures could be made in broth.

2. *Culture on Agar.*—The cultivations and sub-cultivations were made (*a*) on ordinary nutritive Agar, that is to say, on a mixture of beef broth (not beef infusion), Agar (1 p.c.), peptone (1 p.c.), and salt (1 p.c.); and (*b*) on glycerine Agar, that is the ordinary Agar plus glycerine (6 p.c.). The growth on our ordinary Agar is rather more easily observed than on glycerine Agar, being a little more copious (the colonies being somewhat larger) and a little less translucent, and therefore more readily noticeable.

The colonies on the surface of both these media can be discerned under a glass after 24 hours' incubation at 37° C. They then have the appearance of extremely minute translucent flat droplets, and these during the next day or two, increase somewhat in size, but even at their largest are but small—not exceeding three millimetres in breath, and only just visible to the eye as translucent circular flat droplets—on further incubation becoming flatter (Figures 30, 31, 32). Under a lens their margin is slightly crenated and their centre darker than the rest. The crenated margins show no tendency to coalesce, even when the colonies are thickly planted.

In Agar stab-culture the stab is indicated after two or more days as a grey line, this being made up of granules densely and closely placed (Figures 33, 34); viewed under a glass, minute club-shaped and pear-shaped projections are seen to extend from the dark line of inoculation. In stab-cultures, as in surface growths, the several colonies are a little more copious and less translucent when our ordinary Agar is used for the cultivations than when glycerine has been added.

The condensation water in the Agar tubes (of ordinary as well as of Glycerin-Agar tubes set with slanting surface) show, in the course of one or two days, a copious floccular or granular whitish precipitate, the condensation water itself remaining limpid. The amount of this precipitate increases till about the fifth or seventh day, when it has reached its maximum.

Agar tubes inoculated with the influenza bacillus support life in the organism longer than broth tubes, particularly if the Agar tubes be inoculated by stab-culture. We have successfully carried on sub-cultures from Agar cultures through many generations, in fact we have some cases at present (end of June 1892) reached already the twentieth generation, and we see no reason why there should be any limit placed at all, provided each successive sub-culture be established within a week,—after that time the result becomes uncertain.* But if the culture tube after five or six days' incubation at 37° C. be then kept at the ordinary temperature (capped and protected from drying) the life of the culture can be preserved for much longer time; we have as a matter of fact found it living after two weeks; this would certainly not have been the case if any culture of the series had been kept at 37° C. for a fortnight.

3. *Culture on potato:* No visible growth is to be obtained.

4. *Culture on beef broth* (not beef infusion) *with gelatine.* If this be inoculated with Pfeiffer's bacillus and kept at 20–22° C. practically no growth takes place. After two months the faintest indication of a few greyish round or oblong points can be just detected with a magnifying glass; sub-cultures in broth and on Agar proved them to be the living colonies of the bacilli.

Microscopic Examination of the Cultures.

With the cultures above described cover-glass specimens may be made in the usual way, *i.e.*, a thin film of the fluffy or floccular precipitate from the broth cultures, or of the precipitate from the Agar condensation fluid is prepared by drying and staining; and this is found to exhibit

---

* We add here, while this is passing through the press (November 6), that under the above conditions we have hitherto carried on the subcultures on Agar from the sputum of Case 31 through more than 30 generations.

the bacilli in long twisted chains and threads, aggregated so as to form dense networks and convolutions or frequently forming bundles (Figures 19, 20). Many of the threads are found to measure several millimetres in length, while some are broken up into shorter bits. The threads are formed by the individual bacilli placed end to end; the sheaths of the bacilli forming a continuous sheath for the thread: in the stained specimens each element is marked either as a minute rod or more commonly as a dumbbell of granules, this appearance being due to the polar granules of the individual bacilli being very strongly marked: or, by staining this dried film in carbolmethylblue for about half to one hour, and then washing in water, drying and mounting in balsam, the character of the bacilli in the threads may be very well seen (Figures 22, 23, 24, 27).

In recent cultures the threads are either wholly or partially made up of bacilli which stain at the two poles; such elements as do not show this character appearing as uniform rods about 0·4 μ in thickness, 0·8–1·2 μ in length. Cultures several days old show many of the threads already degenerating; that is to say, shorter or longer portions being empty of protoplasm showing only the faintly stained sheath with here and there indistinct granules in it. But in all specimens made of however recent a culture there are threads, in which here and there a bacillus is swollen up into a spherical or oval ball, many times thicker than the typical element; the number of these enlarged elements is greater in later than in recent cultures, and the largest of them often show a vacuole in their centre, or at one side. From these facts it is probable that these enlarged elements are involution forms (Figures 21, 25, 26, 29).

Preparations made of the colonies grown on the surface of the Agar, or Glycerin-Agar show the bacilli exactly of the same aspect and character as those grown in fluid media, namely as threads or else as large clumps; in these the bipolarly-stained bacilli are very typical and such clumps resemble in every respect the clumps seen in the bronchial sputum (Figure 28).

By staining a cover-glass film of the young colonies first with rubin, and afterwards with methylblue, the sheath of the threads is well differentiated as of pink colour from the polar granules, or the rod-shaped protoplasm in the sheath. The same result is obtained from the growth in broth, but the most satisfactory specimens for microscopic observation were obtained from Agar cultures. We possess specimens from Agar cultures stained double as mentioned above, and the rod-shaped character of the elements of considerable portions of a thread is very strikingly marked by being stained blue in the pink general sheath.

On looking at the threads or clumps of any growth with a moderately high power they are seen to resemble streptococci, but with an oil-immersion lens there is no difficulty in recognizing the elements constituting the threads or clumps as really being bacilli, the protoplasm being either rod-shaped and stained uniformly, or else being segregated as a granule at each end and then receiving the stain at the two poles.

The description which we have here given of the character of the growth in the different media and of their microscopic aspect coincides in every essential with that given by Pfeiffer and Kitisato in their paper already quoted, except that in the paper of these authors sufficient prominence is not given to the thread-like nature of the growth; this however may be entirely owing to their communication having the character of a preliminary short account of their results.

Pneumonia connected with Influenza.

In the foregoing description of our cases we have mentioned several in which pneumonia occurred as a symptom of the influenza attack, such were:—

(*a.*) *Case* 3.—Here the influenza commenced on January 18th; the patient when admitted on January 25th had severe bronchitis with muco-purulent expectoration; and on February 2nd, judging by the rusty sputum the lung affection had developed into pneumonia; this was more pronounced on February 3rd when dulness at right posterior base could be distinctly ascertained. On the post-mortem examination on February 4th (15 hours after death) the diagnosis of pneumonia was confirmed. There was present a considerable amount of pleural effusion on the right side, together with a patch of red hepatised lung-tissue in the right lung. Cover-glass specimens of the rusty sputum taken on February 2nd showed, as mentioned on a previous page, almost a pure culture of the influenza bacillus, also those made with the juice of the red hepatised lung contained an abundance of clumps of the influenza bacilli. But cultivations both of the sputum of February 2nd and of the lung juice after death revealed the presence of streptococcus, and the same thing was very manifest in the pleural exudation. Cover-glass specimens made of this revealed an almost pure culture of the organisms as is shown in photogram (*see* Figure 10); and from the pleural exudation a pure growth of streptococcus was obtained, similar in many respects to the streptococcus pyogenes. It ought to be mentioned that the patient had in the right lung an old focus of bronchiectasis filled with muco-purulent matter, and it was around this focus that the pneumonic patch was found; and it is highly probable that the streptococci are to be referred to this chronic bronchiectasis. In view of the great abundance of the influenza bacilli in the rusty sputum at the onset of the pneumonia it is more probable that this latter was connected with the influenza bacilli and not with the streptococcus, as perhaps otherwise might be inferred from the experience of Ribbert and Finkler. The fact that the rusty sputum on February 2nd, *i.e.*, at the onset of the pneumonia, contained the Pfeiffer bacilli in almost pure culture, whereas the streptococci would readily be accounted for by the existence of an old bronchiectatic focus, leads to the conclusion that the Pfeiffer bacilli were, with much greater probability than the streptococci, concerned in the production of the pneumonia. Sections were made through the hardened pieces of the inflamed lung, and these were stained with carbolmethylblue. Extreme congestion was present, the blood capillaries of the alveoli being filled with and distended by blood; but no great exudation into the cavities of the alveoli was observed except in that part which immediately surrounded the distended bronchiectatic bronchus; here the alveoli surrounding were distended and filled uniformly with leucocytes. For the rest, in the congested portion of the lung capillary vessels were found in many places to be completely blocked by a mass of microbes, so much so that the masses stained blue formed a sort of solid cast of the capillaries (*see* Figures 43 and 44). On examining such casts with an oil-immersion lens they are seen to be entirely composed of the influenza bacilli densely aggregated, and showing well the bipolar staining; and from these capillary plugs isolated or small groups of the bacilli extended also into the surrounding tissue and into the air-cells.

(*b.*) *Case* 4.—Here the influenza commenced on January 24th and the patient was admitted on January 27th with distinct pneumonia with rusty sputum. On February 2nd, the patient having improved since

January 29th, the expectoration was less in amount and less rusty, the pneumonic patch clearing up. Examination of the sputum was not made till February 2nd, but the influenza bacilli were still found present in groups, though their number was small.

(*c.*) *Case* 5.—This was an instructive case, inasmuch as the patient had pneumonia the year before. He was taken ill with influenza on February 3rd, and was admitted on February 5th, when the sputum was rusty and there was crepitation in posterior part of left lung. Large numbers of influenza bacilli in clumps were found in the rusty sputum of February 8th; and on February 9th the temperature having gone down to normal, the number of the bacilli was distinctly decreased.

(*d.*) *Case* 25.—Influenza commenced on March 29th. On April 4th, two days after admission, left pneumonia was first noticed, and the sputum was rusty; the temperature had been high since admission, and it remained so till the 10th of April. The sputum taken on April 8th contained an abundance of clumps of the influenza bacilli.

(*e.*) *Case* 31.—This was also an interesting case. The patient was taken ill with influenza on March 27th and on admission on March 30th pneumonia with rusty sputum was diagnosed. The rusty sputum taken on March 31st showed an abundance of the characteristic clumps of the influenza bacilli.

PART III.

(*b.*)—*Experiments on Animals, with the Bacilli of Pfeiffer.*

1. *Experiments on Rabbits.*

(*a.*) Four full-grown rabbits (Nos. 1, 2, 3, 4), were inoculated on February 3rd with bronchial sputum of Case 2. This sputum contained the influenza bacilli in almost pure culture. For inoculation the sputum was well shaken up in sterile salt solution, this being rendered hereby turbid and full of flocculi. About 0·5 C.C. of this mixture were injected subcutaneously into the groin of each of the four rabbits. The result was totally negative, no rise of temperature, the animals remained lively and fed well.

(*b.*) Two full-grown rabbits, Nos. 5 and 6, were inoculated on February 3rd with salt mixture of the sputum of Case 3. This sputum was also almost a pure culture of the influenza bacilli. The result was negative, no rise of temperature, the animals remained lively and fed well.

(*c.*) Two rabbits, Nos. 7 and 8, were inoculated on February 4th with salt mixture of the inflamed lung of Case 3, on the same day on which the post-mortem examination had been made. The result was negative.

(*d.*) Two rabbits, Nos. 9 and 10, were inoculated subcutaneously with salt mixture of bronchial sputum of Case 1, taken on February 5th Result was negative.

(*e.*) Two rabbits, Nos. 11 and 12, were injected on February 12th directly into the trachea with salt mixture of bronchial sputum of Case 10, taken on February 12th; on this day the sputum contained a great abundance of the influenza bacilli, though also some few other bacteria

were present. The result was that after 16–20 hours both rabbits were found quiet, temperature slightly raised 39·1° C. and 39·9° C. respectively, and they did not feed quite normally. On the day following they had quite recovered.

(*f.*) Two rabbits, Nos. 13 and 14, were inoculated on February 5th subcutaneously with broth culture* of the influenza bacilli of the sputum of Case 2. The culture was a direct culture from the sputum of February 2nd, about one Pravaz syringe-full of the broth culture being injected into each rabbit. The result was negative.

(*g.*) Two rabbits, Nos. 15 and 16, were injected with the same broth culture as in experiment (*f*) into the vein of the ear, one Pravaz syringe-full being injected into each animal. The result was negative, no rise of temperature, the animals were found lively next morning and had eaten all their food.

(*h.*) Two rabbits, Nos. 17 and 18, were injected on February 8th into the vein of the ear ; each with one Pravaz syringe-full of a first broth subculture of sputum taken from Case 6 on February 5th. The result was negative.

(*i.*) Two rabbits, Nos. 19 and 20, were injected on February 9th directly into the trachea with broth sub-culture of same sputum as in experiment (*h*), each animal receiving one Pravaz syringe-full ; one of the rabbits soon (about 15 minutes) after injection was found quiet, and so it remained for some hours, next morning it was found dead ; there was nothing abnormal in the lungs or other viscera, cover-glass specimens of the lung juice and heart's blood were examined, as also cultivations of the lung juice and heart's blood were established, but the result was negative; the second rabbit remained quite well after the injection.

(*j.*) Two rabbits, Nos. 21 and 22, were injected on February 10th directly into the trachea with a third sub-culture in broth made from the sputum of Case 2. The result was negative.

(*k.*) Two rabbits, Nos. 23 and 24, were injected on February 10th subcutaneously with a broth culture established on February 8th from the influenza bacilli of the blood in Case 40. One of these two rabbits was dying on February 13th, it was very emaciated and had a small abscess, about the size of a pea, at the seat of inoculation ; the right lung showed in its middle lobe an oblong deeply congested patch and the posterior part of the middle of the left lung was also much congested. Cover-glass specimens and cultivations were made of the heart's blood and of the juice of the congested portions of the lung ; the result was negative, no bacteria could be demonstrated. The second rabbit remained well.

(*l.*) Two rabbits, Nos. 25 and 26, were injected on February 10th directly into the vein of the ear with same broth culture as in experiment (*k.*), one Pravaz syringe-full being injected into the vein of each rabbit. The result was negative.

(*m.*) Two rabbits, Nos. 27 and 28, were inoculated subcutaneously on February 13th with same broth culture of blood of Case 40 used in experiment (*k.*) ; the result was negative.

(*n.*) Two rabbits, Nos. 29 and 30, were injected on March 28th with broth sub-culture of the influenza bacillus from the blood of Case 40

* In all experiments in which broth culture was used, the culture was well shaken up so as to distribute the growth uniformly in the broth.

directly into the vein of the ear, one Pravaz syringe-full for each animal. The next day; both animals were found quiet, one was killed, and all its organs looked normal; examination was made of its heart's blood in cover-glass specimens and in culture:—no bacteria could be demonstrated.

(*o.*) Four rabbits, Nos. 31, 32, 33, and 34, were injected on April 11th into the vein of the ear with a second broth sub-culture of the influenza bacilli of the sputum of Case 31, one Pravaz syringe-full of the culture being injected into the vein of each animal. The result was negative.

The result of these experiments on rabbits was then negatory, so far as evidence of a pathogenic action of the influenza bacilli upon these animals was concerned. Thus, *ten rabbits* inoculated subcutaneously directly with the sputum which contained an abundance of the influenza bacilli remained perfectly well, while *two rabbits*, Nos. 11 and 12, injected with sputum directly into the trachea were found quiet after 16–20 hours after the injection, but had quite recovered during the second day. It must be remembered, however, that the injection into the trachea could only be done after operation, *i.e.*, after laying bare the trachea, and this might account for the slight rise of temperature and quietness of the animals 16–20 hours afterwards. Equally negatory were the experiments with broth cultures. Two rabbits (13 and 14) injected subcutaneously with the broth culture of the influenza bacilli from sputum remained well; in the case of eight rabbits (15, 16, 17, 18, 31, 32, 33, 34) injected directly into the ear-vein with broth culture of the influenza bacilli from sputum the result was quite negative, and in the case of four rabbits (19, 20, 21, 22) in broth culture of the sputum influenza bacilli from sputum, injection into the trachea, produced no effect upon three out of the four. The fourth rabbit was found dead next morning, having been quiet shortly after the injection, but the post-mortem examination showed no sign of disease, and therefore we are unable to assign any cause for the quietness and for the death of the animal; nor were bacilli found in its blood either by cover-glass specimens or by culture.

As regards the action of the broth culture of the influenza bacilli from the blood of Case 40, the experiments again gave no information. Of four rabbits (23, 24, 27, 28) inoculated subcutaneously three remained well, while in the case of the fourth, which became ill and was dying on the fourth day after the injection, there was congestion of some parts of both lungs. Here again no bacilli could be demonstrated either in the heart's blood or in the congested lungs, either by cover-glass specimens or by culture.—Four rabbits injected with broth culture of the bacillus from the blood of Case 40 directly into the vein of the ear remained perfectly well.

2. *Experiments on Monkeys.*

(*a.*) Two monkeys, Nos. 1 and 2, were inoculated subcutaneously in the afternoon of February 3rd with bronchial sputum of Case 2. The temperature of both remained normal, at all events there certainly was not any conspicuous rise. They remained lively and fed well. The temperatures (centigrade),* taken in the rectum, of the two animals, Nos. 1 and 2, during the week were as follows:—

* Some English readers may be saved trouble by the annexed reminder:—

37·0° C. = 98·6° F.	39·0° C. = 102·2° F.
37·5° C. = 99·5° F.	39·5° C. = 103·1° F.
38·0° C. = 100·4° F.	40·0° C. = 104·0° F.
38·5° C. = 101·3° F.	40·5° C. = 104·9° F.

	No. 1.		No. 2.	
	M.	E.	M.	E.
February 3rd - - -	—	39	—	39·2
„ 4th - - -	39·2	39·6	39·7	39·4
„ 5th - - -	39·0	38·8	39·2	39·0
„ 6th - - -	39·7	39·2	39·0	39·4
„ 8th - - -	38·8	39·2	38·6	38·8
„ 9th - - -	38·6	38·8	39·0	39·0

(*b.*) Two monkeys, Nos. 3 and 4, were inoculated subcutaneously in the afternoon of February 4th with inflamed lung (mashed up in sterile salt solution) of Case 3 after post-mortem examination. The temperature of the two animals during the week was as follows :—

	No. 3.		No. 4.	
	M.	E.	M.	E.
February 5th - - -	39·2	39·8	39·3	40·0
„ 6th - - -	39·6	39·6	39·8	39·6
„ 8th - - -	39·2	40·0	38·8	40·1
„ 9th - - -	39·4	39·2	39·0	39·1
„ 10th - - -	38 8	39·2	39·0	39·4
„ 11th - - -	39·0	39·1	39·2	38·9

The animals remained quite lively and fed well, so that the transitory rise of temperature observed in No. 4 in the evening of February 5th and February 8th, and the rise observed on No. 3 in the evening of February 8th cannot be ascribed to any serious constitutional disturbance. The creatures were something refractory to thermometrical operation, and not improbably some exaltations of temperature ought to be ascribed to physical excitement on their part.

(*c.*) These monkeys, Nos. 3 and 4, were then again injected directly into the trachea with salt mixture made with sputum of Case 10, taken on the evening of February 12th. This sputum contained an abundance of the influenza bacilli and was the one used for injection into the

trachea of rabbits Nos. 11 and 12, previously mentioned. The temperature of these monkeys was as follows :—

	No. 3.		No. 4.	
	M.	E.	M.	E.
February 12th - - -	39·8	39·8	39·6	39·6
„ 13th - - -	39·4	39·4	39·4	39·5
„ 14th - - -	—	40·3	—	40·3
„ 15th - - -	39·4	39·8	39·4	39·8
„ 16th - - -	40·4	39·8	39·4	39·9
„ 17th - - -	39·8	40·1	39·4	40·1
„ 18th - - -	39·9	40·4	39·5	40·2
„ 19th - - -	40·0	40·9	39·6	40·3
„ 20th - - -	40·4	killed.	39·6	40·4
„ 22nd - - -	—	—	39·6	39·8
„ 23rd - - -	—	—	39·2	40·0
„ 24th - - -	—	—	39·2	40·0
„ 25th - - -	—	—	40·0	40·2
„ 26th - - -	—	—	39·0	39·4

Here then we have on several days a rise of temperature. This was striking on February 14th in both animals in the evening, it was equally striking on February 17th, 18th, 19th, and 20th. The notes as to their general condition state :—they were lively and ate well on February 12th and 13th, on February 14th both animals are rather quiet, but would eat; on February 20th in the morning they were quiet. Monkey No. 3 was now killed; No. 4 was in quite good health on February 27th and 29th, and on March 1st, 2nd, 3rd, 4th, 5th, 7th; on Mareh 8th it was disearded from further observation.

From this aceount we might be led to eonelude that the animals were distinetly affeeted by the injection of the sputum into the traehea and that this affeetion showed itself on the third day after; but this eonelusion was not borne out by the post-mortem examination of No. 3, for, on this examination, no viseeral disease of any kind eould be detected. Cover-glass specimens and eultivation from heart's blood and lung yielded no organisms; eultures from the kidney yielded staphylococeus aureus and a streptoeoceus.

(*d.*) Two monkeys, Nos. 9 and 10, were inoculated subcutaneously on February 29th with salt mixture of the bronehial sputum of Case 53, whieh, as stated on a former page, eontained amongst a variety of organisms, numerous elumps of the influenza bacilli.

	No. 9.		No. 10.	
	M.	E.	M.	E.
February 29th - - -	—	39·1	—	39·2
March 1st - - - -	40·0	39·8	39·2	39·8
„ 2nd - - -	39·0	39·8	39·6	39·3
„ 3rd - - -	39·0	39·6	39·0	36·2
„ 4th - - -	38·6	39·6	dying.	—
„ 5th - - -	38·8	39·2	—	—
„ 7th - - -	39·2	39·6	—	—
„ 8th - - -	39·2	40·1	—	—
„ 9th - - -	39·3	40·0	—	—
„ 10th - - -	39·3	39·6	—	—

In both animals the temperature taken just before the inoculation was 39·1 and 39·2 respectively, and though there was no very great change in the temperature of the animals, the general condition was decidedly abnormal, for in the notes it is found :—

Monkey No. 9, was on March 1st (that is day after inoculation) off feed and quiet ; March 2nd, not eating well; March 3rd, eating and lively; March 5th, not eating well but lively. After this it was and remained lively and fed well.

It was killed by chloroform on March 10th. No visceral disease of any kind was found post-mortem. The blood, examined in cover-glass specimens and in cultivation, proved free of bacteria.

Monkey No. 10 was more markedly abnormal. On March 1st (*i.e.*, the day after inoculation) off feed, quiet, has diarrhœa ; March 2nd, off feed, diarrhœa ; March 3rd, eats a little in the morning, but was very ill in the evening. On March 4th, the animal was found dying and was killed by chloroform.

Post-mortem on monkey No. 10.* Extensive lobular pneumonia, chiefly in the centres of the lobes ; most of the pneumonia was on the right side, but some also on the left. The affected portions were in a state of true red hepatisation with hæmorrhagic patches. The spleen was dark and firm; the kidneys excessively pale ; patches of acute injection and swelling in the large intestine. Sections through the hepatised portions of the lung showed the alveoli distended by networks of fibrin and red blood corpuscles, also many leucocytes (Figures 45 and 46).

Cultivations made with the heart's blood yielded, in one out of four broth-tubes, a white scanty granular deposit, which in stained cover-

* Observation of this animal and its pathology and bacteriological examination were made wholly by Dr. Andrewes.

glass specimens was seen to be made up of minute bacilli, and these showed conspicuous bipolar staining. Cultures of the spleen and kidney remained sterile.

Sections through the hepatised lung of this monkey (No. 10), stained in carbolmethylblue, showed continuous masses of minute bacilli present in some capillaries and small veins, and also in the tissue around. These bacilli were of two sorts; the one being exactly like the Pfeiffer bacillus which we have learned to associate specifically with influenza; the other having separate and definite characters of its own, to be presently described. This latter bacillus was present everywhere in the section; it showed itself abundantly in cover-glass specimens of lung-juice (Figure 35), and could easily be grown unmixed with any other kind of bacterium in culture media. It proved to be pathogenic to mice; in this respect as well as in its microscopic characters differing from the organism of influenza.

(*e.*) Four monkeys, Nos. 19, 20, 21, and 22, were inoculated subcutaneously on March 31st, with a salt mixture of the rusty sputum of Case 31. This sputum, as mentioned on a former page, contained on that day an abundance of the influenza bacilli. The temperature just before the inoculation was:—No. 19, 39·4; No. 20, 40·2; No. 21, 40; No. 22, 40; and the record of subsequent dates was as follows:—

	No. 19.		No. 20.		No. 21.		No. 22.	
	M.	E.	M.	E.	M.	E.	M.	E.
April 1st - -	38·9	38·7	40·0	39·0	40·3	40·2	40·0	40·0
„ 2nd - -	39·2	killed.	39·6	39·8	39·7	39·4	39·0	40·0
„ 4th - -	—	—	39·0	killed.	39·1	39·4	39·3	39·4
„ 5th - -	—	—	—	—	39·6	39·4	39·2	38·6
„ 6th - -	—	—	—	—	39·4	40·2	38·9	39·4
„ 7th - -	—	—	—	—	39·3	39·4	39·0	39·3
„ 8th - -	—	—	—	—	39·8	40·2	39·6	39·2
„ 9th - -	—	—	—	—	39·7	killed.	39·7	killed.

As regards the general condition. On April 1st, the day after inoculation, all four animals were lively and were feeding well. On April 2nd all were lively and eating well, and on subsequent days till they were killed were in apparently perfect health.

Monkey No. 19.—On April 2nd in the morning this animal suddenly developed extensive and marked œdema of the skin of the face and neck, and therefore it was killed.

Strange to say one of the two attendants had on that day a similar œdema; this, however, passed off the next day.

On post-mortem examination, the heart-cavities of monkey No. 19, particularly on the right side, were found filled with a dense clot. As

the animal had been killed by chloroform and the post-mortem had been made immediately as the heart stopped, it seemed possible that this clot had been formed before death; and the nature and extent of the clot agreed with this suggestion. All viscera looked normal. Culture of the heart's blood yielded cocci, one of the tubes inoculated with the œdematous fluid of the skin of the face yielded a streptococcus.

Monkey No. 20.—Killed April 4th. All viscera normal. Cultures sterile.

Monkey No. 21.—Killed April 9th. A few petechiæ in lungs, but no organisms in cover-glass specimens or cultures.

Monkey No. 22.—Killed April 9th. Viscera normal, no organisms in cultures.

(*f.*) Two monkeys, Nos. 11 and 12, were injected on February 29th directly into the trachea with salt mixture of bronchial sputum of Case 53, the same as was used on February 29th for subcutaneous inoculation of monkeys 9 and 10. The temperature before injection was 38·9 and 40·5 respectively.

	No. 11.		No. 12.	
	M.	E.	M.	E.
March 1st	39·0	39·8	39·2	39·9
„ 2nd	39·4	40·0	39·6	39·9
„ 3rd	39·5	40·0	39·3	39·6
„ 4th	39·2	40·2	39·0	39·8
„ 5th	39·2	39·8	39·2	39·8
„ 7th	39·2	39·9	39·5	40·2
„ 8th	39·6	40·0	39·5	39·8
„ 9th	39·5	40·0	39·5	39·8
„ 10th	39·4	39·8	39·7	40·3
„ 11th	39·6	40·1	39·6	39·8
„ 12th	39·0	40·0	38·6	39·3
„ 14th	39·0	40·0	39·0	40·2
„ 15th	39·3	40·1	39·6	40·0
„ 16th	39·6	40·4	39·5	40·1
„ 17th	39·8	40·4	40·1	40·4
„ 18th	39·7	killed.	39·5	killed.

As regards the general condition of the animals the note book says the following:—

March 1st., *i.e.*, the day after injection, both animals off feed and quiet.

March 2nd. Both off feed in the morning, but ate a little in the evening.
„ 3rd. Both are lively and eat well.
„ 4th. Both seem rather quiet but eat.
„ 5th. Both rather quiet; eating fairly well.
„ 7th. Both rather quiet, off feed.
„ 8th. Still quiet and off feed.
„ 9th. In the morning a little livelier, but do not eat well; in the evening rather quiet.
March 10th. Still quiet, but eat a little better.
„ 11th. Livelier, have eaten better.
„ 12th. Both off feed and quiet.
„ 14th. Still quiet, but have eaten well.
„ 15th. Have not eaten much but are fairly lively.
„ 16th. Off feed, very quiet.
„ 17th. Off feed, very quiet.
„ 18th. Have not eaten anything and are very quiet. Killed in the afternoon.

Post-mortem examination. In both animals no abnormal condition in any of the viscera; blood cultures remained sterile.

(*g*.) On February 10th six monkeys were injected with broth culture made directly from the blood of Case 40; two, Nos. 5 and 6, each receiving one Pravaz syringe-full directly into the trachea; and four others, Nos. 7 and 8, with Nos. 1 and 2 (discarded from experiment (*a*)) receiving each one Pravaz syringe-full subcutaneously. The temperature of these six monkeys was as follows:—

	No. 1.		No. 2.		No. 5.		No. 6.		No. 7.		No. 8.	
	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.	M.	E.
February 10th -	39·3	39·2	39·0	39·4	39·4	39·2	39·0	38·8	39·2	39·2	38·8	39·2
„ 11th -	29·2	38·9	39·4	39·6	39·2	39·3	39·4	39·2	39·6	39·4	39·2	39·4
„ 12th -	38·8	39·9	38·4	40·0	39·0	39·2	39·4	39·6	39·2	infra	39·4	39·8
„ 13th -	39·2	40·1	38·8	39·7	38·8	39·5	39·3	39·3	—	—	39·2	39·3
„ 14th -	39·1	39·7	38·8	39·7	—	39·7	—	39·5	—	—	—	40·0
„ 15th -	40·0	39·5	40·7 killed		39·8	39·9	39·0	39·6	—	—	38·8	39·2
„ 16th -	39·4	39·6	—	—	39·2	39·6	38·6	39·8	—	—	38·4	39·2
„ 17th -	39·0	39·9	—	—	39·2	40·1	39·2	40·1	—	—	39·2	3·94
„ 18th -	39	39·8	—	—	39·5	39·8	39·0	40·	—	—	38·9	39·0
„ 19th -	39·3	39·8	—	—	39·8	39·9	39·4	39·8	—	—	39·1	39·1

As regards the general condition, there was nothing abnormal noticed on the 11th and 12th February. On February 12th monkey No. 7 was killed by an accident. On the 13th, in the evening, Nos. 1 and 2 were rather quiet; on February 14th, Nos. 1 and 2 were lively, but 5, 6, and 8, were rather quiet, all six of them feeding well. On February 15th, No. 2 appeared ill; quiet, and refusing food, it was therefore killed, and on post-mortem examination, all its viscera were found normal, and cultures made from heart's blood and kidney remained sterile.

Up to February 22nd, Nos 1, 5, 6, and 8, were lively and fed well; but on that date monkey No. 6 was found ill, being quiet, off its feed, and having diarrhœa; it was killed. Upon post-mortem examination

the only lesion found was inflammation and ulceration of the colon. Blood cultures yielded streptococci, also staphylococcus aureus; culture made from lung found to be sterile.

Monkey No. 8 was killed February 26th. All viscera healthy. Nos. 1 and 5 were discarded, being in perfect health.

(*h.*) Two monkeys, Nos. 23 and 24, were injected subcutaneously on April 5th with broth sub-culture of influenza bacillus of blood of Case 40. The result was *nil*. There was nothing abnormal noticed as regards temperature, liveliness, or feeding. The animals were killed on April 11th, when all viscera were found normal; blood cultures remained sterile.

The inferences then to be drawn from these experiments on the monkeys are as follows:—First, the condition of temperature in the various animals before and after inoculation were not such as to allow of any conclusion about their ailments, since in several instances the temperature before the experiment was abnormally raised (probably for the reasons already suggested), whereas, after the experiment the temperatures fluctuated so much and were so irregular that no definite insight could be gained from them as to the health of the animals.

Secondly, we are not justified in concluding from the observed quietness and refusal of food that the animals were really ill, since in several instances of animals in this state post-mortem examination showed no abnormal condition in any of the viscera (Nos. 2, 3, 11, 12, 20, and 23), while the blood was free of any microbes.

Thirdly, as to any inferences derivable from post-mortem observations, in several instances bronchial sputum containing an abundance of the influenza bacilli was used for injection:—Nos. 1, 2, 3, 4, 9, 10, 11, 12, 19, 20, 21, 22. Of these the only animals which showed anything abnormal post-mortem were Nos. 10, 19, and 21. Of these again, No. 19 showed normal viscera, though three days after the injection it developed an œdematous swelling of the skin of the face, from which œdema nothing but a streptococcus was demonstrated by cultivation.

In No. 21 there were found on post-mortem a few petechiæ on the lung, but no organisms could be demonstrated. So there remains one animal only; No. 10, which had well-marked organic disease, in fact pneumonia. We have seen that two kinds of bacilli were present in the inflamed lung of this animal; first, a species which was not the Pfeiffer bacillus, and which will be further considered in the sequel, and secondly, quantities of the Pfeiffer bacilli, which are so habitually present in the sputa of human influenza. In this monkey the pneumonia may have been related to the one or to the other form of bacillus. As regards the monkeys inoculated with broth culture of the influenza bacillus derived from the blood of Case 40, the only one which on post-mortem examination showed an abnormal condition was No. 6, which was affected with inflammation and ulceration of the colon after tracheal injection; but it is impossible to say whether this was produced by the inoculation of the culture. To sum up:—Amongst the twelve monkeys injected with bronchial sputum one only showed definite disease of the lungs, and there the Pfeiffer bacillus was present; and amongst the eight monkeys injected with the broth culture none showed disease of the viscera, except one (No. 6) having ulceration of the colon of doubtful nature.

The bacillus exceptionally observed in the inflamed lung of monkey No. 10 has been the subject of more particular study. It was demonstrated by Dr. Andrewes in the cover-glass specimens, and in the cultures made with the juice of the inflamed parts. The bacilli, which for the sake of

recognition we will at present call the "monkey-pneumonia" bacilli, were cultivated through many sub-cultures by Dr. Andrewes; their morphological and cultural characters are described by him as follows (*see* Figures 36, 37, 38, 39, 40, 41, 42):—

"In sections of the original lung, and cover-glass preparations of the lung juice, the bacillus was present in groups of extremely minute and short rods, with a tendency to bipolar staining (Figure 35). In cover-glass preparations it stained with great difficulty—best in carbol-blue. In sections of the lung the best results were obtained with gentian violet.

"*In broth* at 37° C. In 24 hours the broth becomes hazy, but there is as yet no deposit and no scum on the surface. A peculiar shimmering effect is produced on shaking the broth. At this stage the bacilli are of considerable size, varying from 2·4 to 1·6μ. in length, and about 0·64μ. in breadth (Figure 36). They are often somewhat fusiform, rounded at the ends, or sometimes pointed, and the protoplasm shows not infrequently a tendency to vacuolation, so that sporing is simulated. The bacillus is motile, but not strikingly so; occasional spinning movements are seen in a few bacilli, but the majority exhibit only Brownian movement.

"After the second day at 37° C. the broth shows a little scum round the surface where it is in contact with the glass, and by the third day there is a deposit of whitish flocculent material at the bottom, which gradually increases. The broth becomes increasingly turbid, and eventually a slight scum spreads over the surface.

"After the second or third days specimens of the broth show a number of smaller and shorter bacilli among the rest. The original large forms gradually decrease, and at the end of a week are very few. The large forms sometimes occur joined end to end in short chains (Figures 39, and 40); the smaller forms frequently form long chains of bacilli, in which a tendency to bipolar staining is found. The chains occur chiefly in the deposit, and are more abundant in some cultures than in others, mostly so in those with most deposit. I was at first inclined to believe that I was dealing with a mixture of two different organisms, but numerous sub-cultures, both fractional and by plate cultures, failed to confirm this idea. The chains eventually break up, and after a week or ten days the culture consists mainly of isolated short bacilli.

"*On Agar-Agar* at 37° C. the bacillus grows rapidly. Streak cultures show a semitranslucent growth in 20 hours, whitish in reflected light, which in two days has become opaque, white and somewhat porcellanous in aspect. It does not attain any great thickness, and here and there spreads in a dendritic manner at the edge; but it never spreads far from the original streak. The bacilli grown thus on Agar-Agar differ strikingly from early broth cultures, being far smaller and shorter (Figure 38). Measurements give a length of ·8 to ·4μ, and an average breadth of ·4μ. The larger number are almost coccus-like in form, but with them are a few larger and more deeply stained ones resembling the broth type. Agar-Agar cultures, three weeks old show the same minute forms with a few larger ones. Cultures retain their vitality for at least several months.

"*On Gelatin* at 20°–22° C. the bacillus grows more slowly (Figures 41 and 42). Surface cultures made by rubbing a platinum wire dipped in a dilution of the bacillus material over the surface first show pin-point colonies on the third day, and by the fifth day they attain the size of small pin heads. The colonies are transparent, bluish by reflected, brownish by transmitted light, and resemble droplets of gum. In ten days they reach a diameter of about one-sixth of an inch, and never exceed this. They remain circular and gradually become opaque and whitish; the superficial layer of the gelatin becomes turbid in old cultures. Streak cultures on gelatin show a just perceptible transparent gummy growth in 24 hours. The growth undergoes changes similar to those occurring in isolated colonies, the streak form is preserved, and does not attain a greater breadth than a quarter of an inch. The semi-translucent gummy growth changes in old cultures to an opaque white, while the superficial layer of gelatin becomes turbid.

"Stab-cultures in gelatin grow feebly along the stab and more vigorously at the surface; the organism is distinctly aërobiotic.

"Grown on gelatin the bacilli are small and short, their average length being to their breadth as two or three to one (Figure 37). They are slightly less coccus-like than on Agar-agar, and are not quite so minute. Occasional motility is seen. Long threads are present here and there. In later sub-cultures on gelatin, many of the colonies assumed a more spreading manner of growth. Some doubts were entertained as to whether I was not dealing with two different organisms. Sub-cultures in broth from the more spreading colonies yielded more and larger chains of bacilli than those from the typical colonies. But numerous experiments failed to establish any permanent and essential distinction between the two forms.

"*On potato* the bacillus grows vigorously. When kept at 37° C. a strong brownish growth is present in 24 hours, and in a few days forms a thick spreading moist layer of a brownish pink or flesh colour. When kept at 20° C. the growth is slower, but eventually assumes the same brownish pink tint. The bacilli on potato are of very uniform size and shape, larger than on gelatin, averaging in length as compared to breadth about three to one. But they are never so large as in early broth cultures."

Dr. Andrewes, with his various cultures, carried on a number of inoculation experiments, and reports concerning them as follows:—

(*a.*) Two monkeys, Nos. 13 and 14, were inoculated subcutaneously on March 19th, with broth culture, one syringe-full each of a two days old culture.

No. 13 became ill a day or two after inoculation, diarrhœa, but no fever, then got better. It was killed on March 16th. Post-mortem: Immense hæmorrhage into lungs, chiefly the right lung, looking like pneumonia; but the edges of the lungs were of normal colour. Examined in sections, no hepatisation could be seen, only hæmorrhage. Cover-glass specimens of lung juice showed numerous bacilli exactly like those of the lung of monkey No. 10. Other viscera looked normal, except for a large subperitoneal ecchymosis in the liver. Cultures of the lung and heart's blood remained sterile.

No. 14 remained well. Killed on March 19th. All viscera normal. Cultures of heart's blood remained sterile.

(*b.*) Two monkeys, Nos. 15 and 16, were inoculated subcutaneously on March 9th, with a salt mixture of an Agar culture.

No. 15 was killed on March 19th. Viscera normal. Cultures of heart's blood and lung remained sterile.

No. 16 was quiet and off feed, but no fever. Was killed March 22nd. Lungs showed several (about six) small congested patches, deep red. Cover-glass specimens showed a few bacilli like those of the lung of monkeys Nos. 10 and 13. Other viscera normal. Two Agar cultures made by rubbing over the slanting surface a bit of the monkey lung, yielded each one colony of the typical monkey pneumonia bacilli.

(*c.*) Two monkeys, Nos. 17 and 18, were inoculated subcutaneously on March 16th, with lung tissue of monkey No. 13.

No. 17 remained quiet, and off feed. Killed on March 23rd. Lungs looked normal, but there were in one some firm patches of greyish translucent infiltration. Cover-glass specimens showed bacilli like those of monkeys Nos. 10 and 13. Other viscera normal. Cultivations from blood and lung gave no results.

No. 18 remained well. It was killed on March 21st. All viscera normal.

(*d.*) Two mice, Nos. 1 and 2, inoculated subcutaneously March 5th with an emulsion in salt solution of a one day old Agar culture from the lung of monkey No. 10.

No. 1, white mouse. Ill after two days, found dead March 10th. Heart blood contains many bacilli resembling those in monkey's lung, cultures yielded pure monkey-pneumonia bacilli in great abundance. Lungs intensely engorged; deep red, but not actually hepatised. Lung juice contains very numerous bacilli. Cultures yielded pure monkey-pneumonia bacilli in abundance. General peritonitis; flakes of lymph on gut; numerous bacilli in cover-glass preparations. Spleen large and dark.

No. 2, black and white. Ill after two days, then got rather better. Found dead March 14th, probably died on 13th. Heart blood contains fair numbers of bacilli. Cultures gave two sorts of colonies, (*a*) the monkey pneumonia organism, and (*b*) a spreading growth not unlike the Middlesbrough organism, with much gas production (which monkey pneumonia never gives). Lungs intensely congested but not hepatised. Lung juice contains abundant bacilli. Cultures yielded the two sorts of bacilli, but mainly the spreading Middlesbrough-like form. General opacity of peritoneum, turbid fluid. Guts distended. Spleen large and dark.

(*e.*) Two mice, Nos. 3 and 4, inoculated March 29th with pure broth culture of the monkey pneumonia bacillus from monkey No. 10.

No. 3. Ill on March 31st, found dead April 1st. Heart blood contained numerous bacilli in groups. Cultures gave pure monkey pneumonia colonies in abundance, long strings of bipolar bacilli in broth. Lungs intensely engorged, lung juice swarming with very uniform short bipolar bacilli. Gave pure monkey pneumonia colonies in large numbers. No definite peritonitis, but peritoneum is moist and turbid looking. Stomach full of bubbles.

No. 4 died some days later but was not examined.

(*f.*) Two guinea-pigs, Nos. 5 and 6, inoculated subcutaneously on March 18th, each with a syringe-full of an albumin broth culture of monkey pneumonia. Both remained perfectly well.

(*g.*) Three rabbits, Nos. 7, 8, and 9, inoculated subcutaneously March 9th each with a syringe-full of pure broth culture of monkey pneumonia from monkey No. 10. All remained well, but one of them (rabbit 1) was quiet on March 16th and was killed. All organs normal. Cultures gave no result. The others were never ill.

(*h.*) Two rabbits, Nos. 10 and 11, inoculated intravenously with a broth culture of the organism of monkey-pneumonia from heart blood of mouse 1 on March 11th (first day after the mouse had died). One rabbit had a syringe-full into the ear vein, the other half a syringe-full. Both remained healthy.

(*i.*) Two rabbits, Nos. 12 and 13, inoculated on March 28th each with a syringe-full of a broth culture of monkey pneumonia from monkey No. 10. Next day blood was drawn from each, and cultures made. In each case a colony of the bacillus of monkey-pneumonia grew, but only one. After three days one rabbit (12) was killed. Organs healthy. Cultures gave no result. Rabbit 13 remained healthy.

From these experiments it follows then that the bacillus isolated by Dr. Andrewes from the lung of monkey No. 10 has a pathogenic action and produces in monkeys and in mice a hæmorrhagic affection of the lung, which especially in the mouse proved fatal. On guinea-pigs and rabbits it had no effect.

We conclude then from the experiments on the rabbits and monkeys detailed on former pages that by making use either of bronchial sputum of influenza cases containing an abundance of the Pfeiffer's influenza bacilli, or of cultures of these bacilli, and by introducing such materials under the skin, or into the trachea, or by direct injection into the vein (rabbits), it has not been practicable to arrive at any definite production of influenza disease in monkeys or in rabbits. Only in one monkey out of 18 was a definite disease of the lungs produced by such injection, and there (but in company with other bacilli) clumps of influenza bacilli were found; while, out of 30 rabbits injected with like materials, there was no single instance of a disease recognizable as influenza in nature having resulted from the experiment.

Now the question has repeatedly been raised, and indeed has been repeatedly answered in the affirmative, viz., whether the disease influenza," such as prevailed in this country, on the Continent of Europe, and in most other parts of the world in 1889–1890 and in 1891–1892 is a disease to which also the domestic and other animals are subject. It has been particularly asserted that in this country the influenza was

common amongst horses antecedently to and during the prevalence of influenza in man. Our own experiments have not indeed extended to horses. We have not thought ourselves justified in incurring the large expenditure wanted for the purchase of healthy horses, unless there were *primâ facie* grounds for regarding these animals as having a liability more than other animals to suffer under the disease which affects the human race; and we have failed to get any indication of any such probability. The popular notion appears to rest on no better ground than that the name "influenza" has often been given to a contagious febrile catarrhal affection in horses which, owing to its febrile character, the great weakness that follows it, and the congestion of the nasal and conjunctival and bronchial membranes bears a certain resemblance to what we see in the veritable influenza of man. This epidemic disease amongst horses, however, has been frequently known to prevail in years when influenza in man did not prevail. In Germany this febrile disease of horses is well known, and is not confused with the influenza of man.

But though we have not made intentional experiments upon horses or other animals beyond those mentioned in these pages, we have not the less been on the watch during the time that we carried on our inquiry (February to April 1892) for indications of any influenza like disease affecting the lower animals. We could not get evidence of horses being affected with any complaint identical with the influenza in the human, nor as to other animals which live amongst human habitations, are we aware of any evidence proving that amongst them influenza or any similar disease was rife during the periods of the influenza epidemic. Under these circumstances we have made inquiries at the Zoological Gardens in London, and Mr. Beddard has kindly given us the facts as to the condition of illness and deaths amongst the mammals kept there. From his record we learn that the incidence of disease and death at the Zoological Gardens was not unusually heavy during the years of the influenza epidemic in the metropolis. As regards the monkeys in particular, kept at the Zoological Gardens, we also understand from Mr. Beddard that no increased sickness was observed amongst them during these periods. The fact conforms with the results from our experimental observations on monkeys above recorded. It can hardly be supposed that if monkeys were, as a class, susceptible to the infection of human influenza, the creatures living in the monkey-house of the Regent's Park, frequented by many thousands of people a month, while influenza was abundant in the London population, would have kept free from the complaint.* And from the general experience of the Gardens of the Zoological Society, it would appear that few mammalia can share with the human subject a susceptibility to epidemic influenza. At all events, few of them are liable to receive the infection by the method which habitually obtains in man, through the respiratory passages.

Conclusion.—The researches now recorded have afforded certain affirmative results, as to the microphytic elements of influenza: they are such as may be presumed to be constant in influenza; and they are in full agreement with the results obtained by Pfeiffer and Kitisato. Our investigations have shown a bacillus having peculiar morphological and cultural characters, always abundantly present in the bronchial secretion

* At the time when our experiments were made upon monkeys, in the hope that these animals might show the same susceptibility to influenza infection as that exhibited by men, we were not in possession of the experience of the Zoological Gardens.

of patients suffering from influenza, diminishing in their numbers as the disease abates, and disappearing when the disease comes to an end. The same species of bacillus is furthermore found occasionally in the blood of acute influenza; not very often indeed, but often enough to invalidate a suggestion that its appearance there is matter of accident.

The German authorities point to this species of bacillus as being the microbe of influenza; the material by which the disease is communicated from person to person, and without which the disease would not exist. Very probably they are right; but there remain some other facts to be learned before the demonstration of this bacillus as the materies morbi of influenza can be considered as complete. It would be well to have evidence respecting those cases of influenza which are characterised less by disordered secretion of the bronchi than by disordered secretion of the mucous membrane of the intestinal tract or elsewhere; whether the bacillus exhibited in those other mucous secretions the same constancy and abundance during the acute stage of disease, and the same diminution and disappearance as the disease abates, as has been recorded for the bronchial mucus. It goes without saying that what we now most specially want, for the demonstration of this bacillus as the materies of influenza, is evidence that this particular microbe does not occur in any other disease than influenza; and Pfeiffer and Kitisato would go so far as to assert this as a fact. Our own researches have not led us to doubt it; and we have only to give the obvious caution that not sufficient time has yet elapsed since Pfeiffer's discoveryand identification of the microbe to establish their assertion; and perhaps we are as yet hardly safe in denying the presence of his bacillus elsewhere than in influenza.

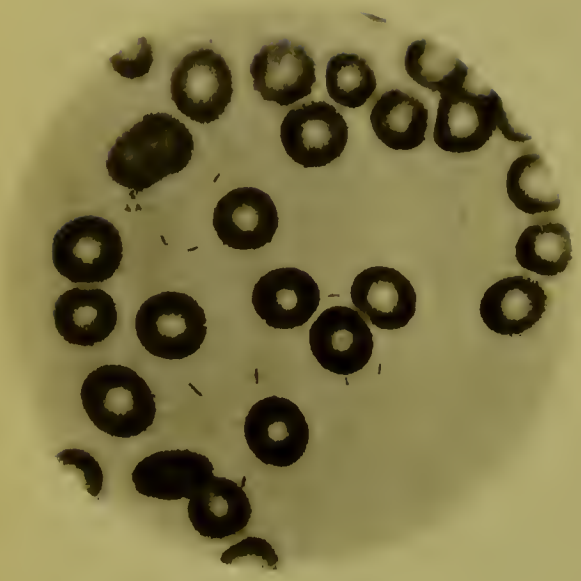


FIG. 1.

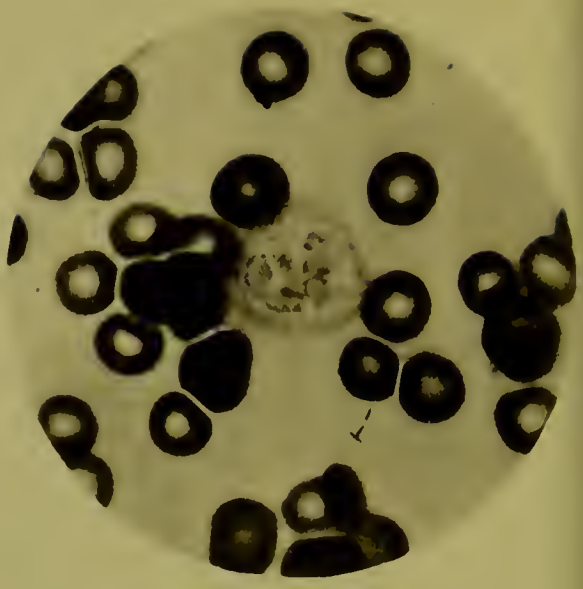


FIG. 2.

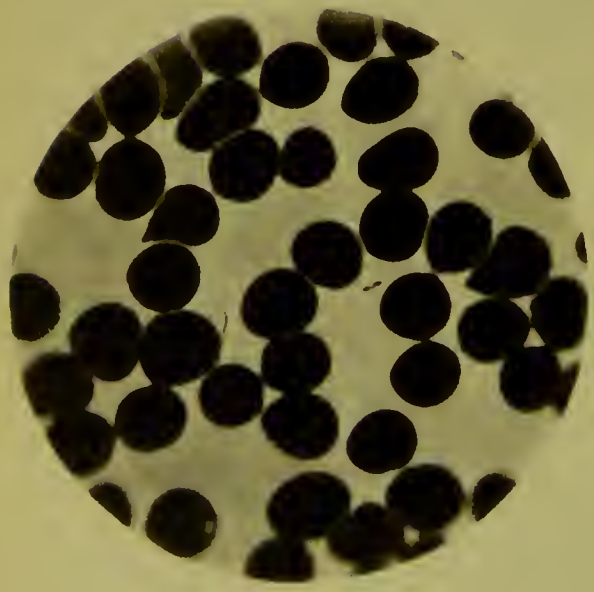


FIG. 3.

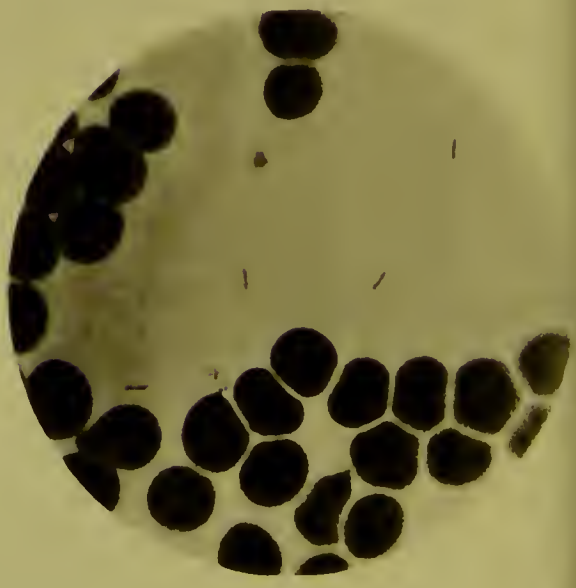


FIG. 4.

ETIOLOGY OF INFLUENZA.

PLATE I.

FIG. 1.

Reproduced from a photograph* of a cover-glass specimen of *blood* from case No. 47. Numerous minute bacilli are to be seen amongst the red blood discs.

FIG. 2.

Reproduced from a photograph of a cover-glass specimen of the same *blood* that is represented in fig. 1.

FIG. 3.

Reproduced from a photograph of a cover-glass specimen of *blood* from case No. 33.

FIG. 4.

Another specimen of blood from case No. 33.

* The photographs illustrating this report are, except when otherwise stated, by Mr. E. C. Bousfield. The magnifying power is, unless specially mentioned, 1,000. All reproductions of photographs are by the Autotpyc Company.

ETIOLOGY OF INFLUENZA.

PLATE II.

FIG. 5.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 54. Large and small clumps of the influenza bacilli are exhibited.

FIG. 6.

Represents another specimen of the same sputum from this case.

FIG. 7.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 55. Several small masses of the influenza bacilli are here seen.

FIG. 8.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 2. Numerous isolated influenza bacilli are shown.

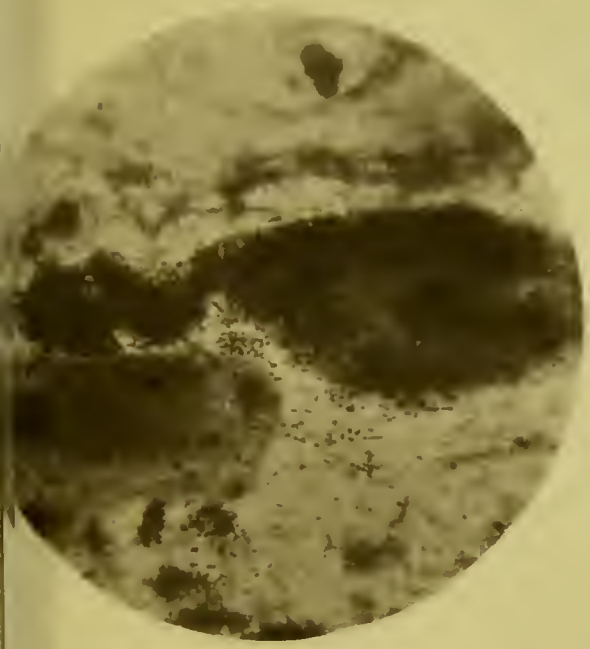


FIG. 5.

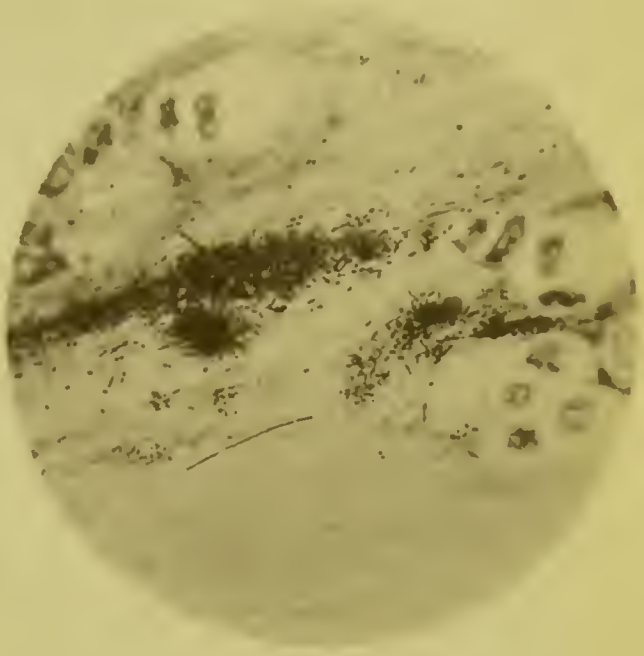


FIG. 6.

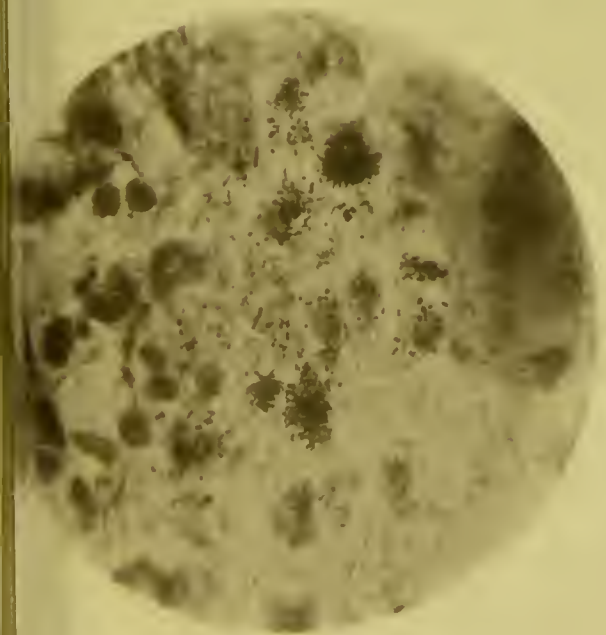


FIG. 7.

FIG. 8.

PLATE III.

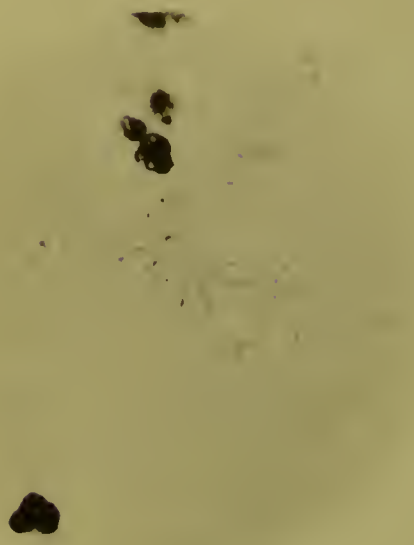


FIG. 9.

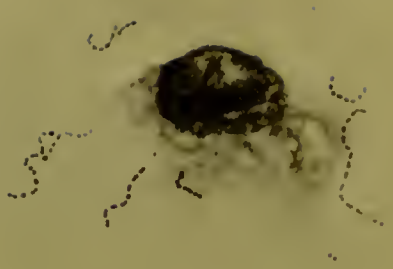


FIG. 10.

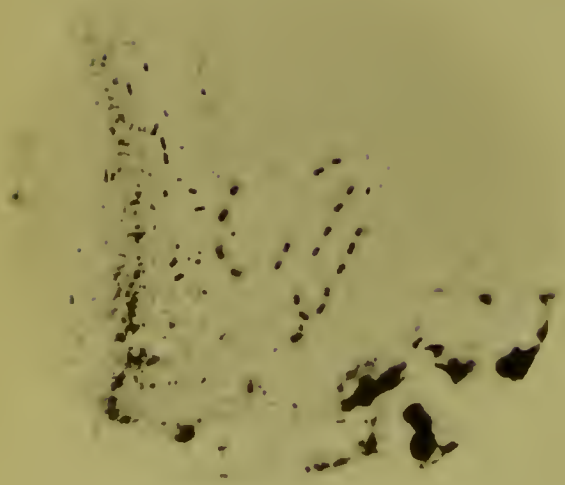


FIG. 11.

FIG. 12.

ETIOLOGY OF INFLUENZA.

PLATE III.

FIG. 9.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 3. The sample exhibited influenza bacilli almost in pure culture.

FIG. 10.

Reproduced from a photograph of a cover-glass specimen of *pleural exudation* from case No. 3. It shows one large endothelial plate, and numerous streptococci.

FIG. 11.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 1. The specimen showed capsulated bacilli of Friedlander; with various other bacteria, amongst them large numbers of the minute influenza bacilli.

FIG. 12.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 2. One leucocyte is here seen inclosing within its protoplasm numerous influenza bacilli.

ETIOLOGY OF INFLUENZA.

PLATE IV.

FIG. 13.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 3. Two leucocytes are depicted, and within their cell substance influenza bacilli are visible.

FIG. 14.

Represents another sample of the same sputum as Fig. 13, and exhibits bacilli within the substance of leucocytes.

FIG. 15.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 5. Masses of influenza bacilli are here exhibited.

FIG. 16.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 10. Again there are shown masses of influenza bacilli.

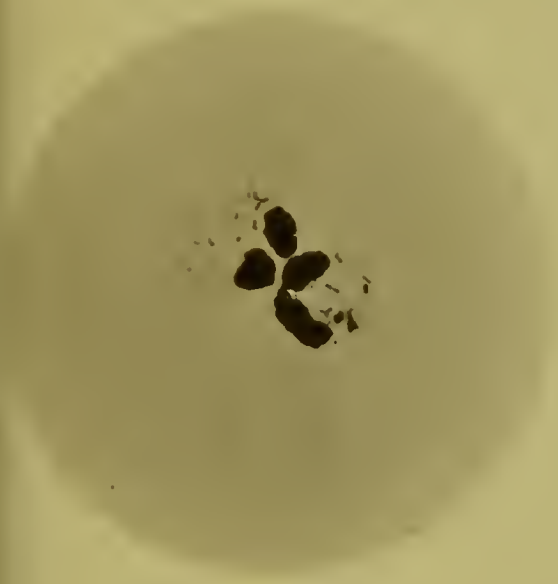


Fig. 13.

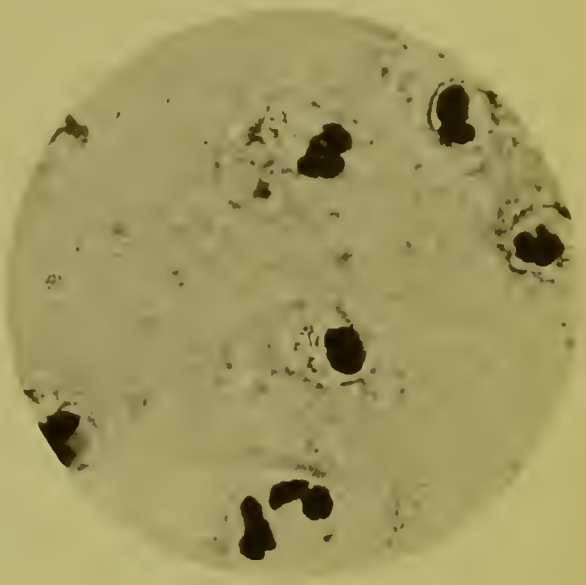


Fig. 14.

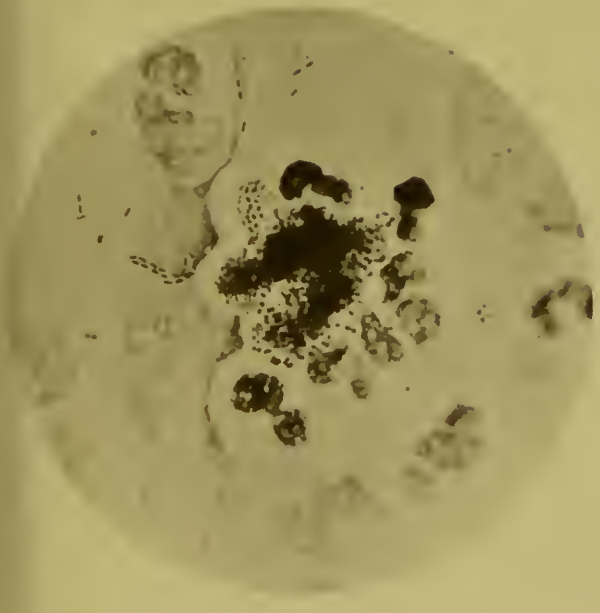


Fig. 15.

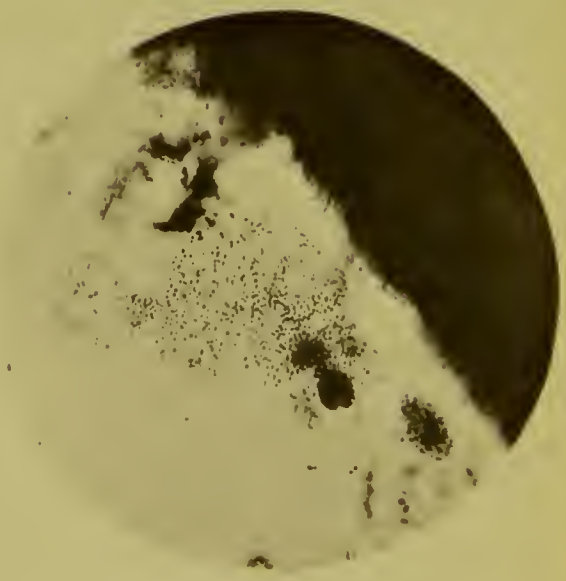


Fig. 16.

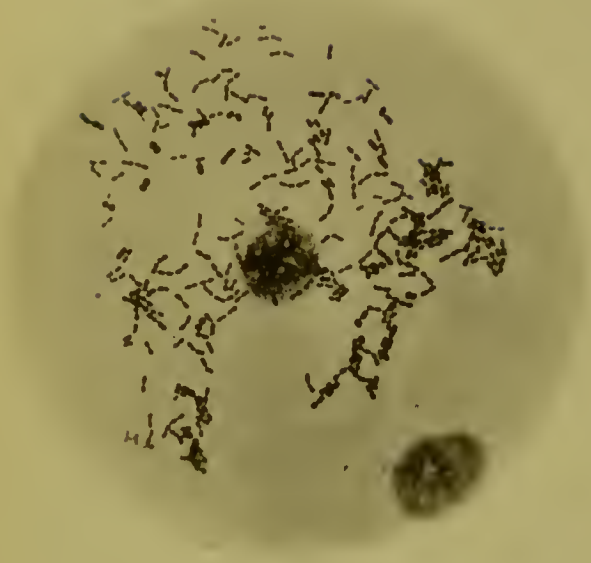


FIG. 17.

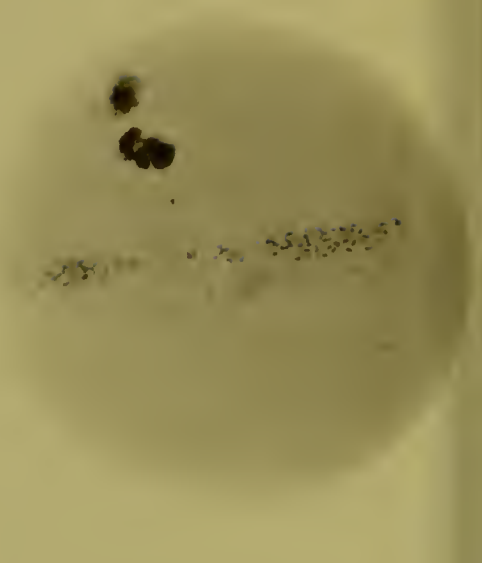


FIG. 18.

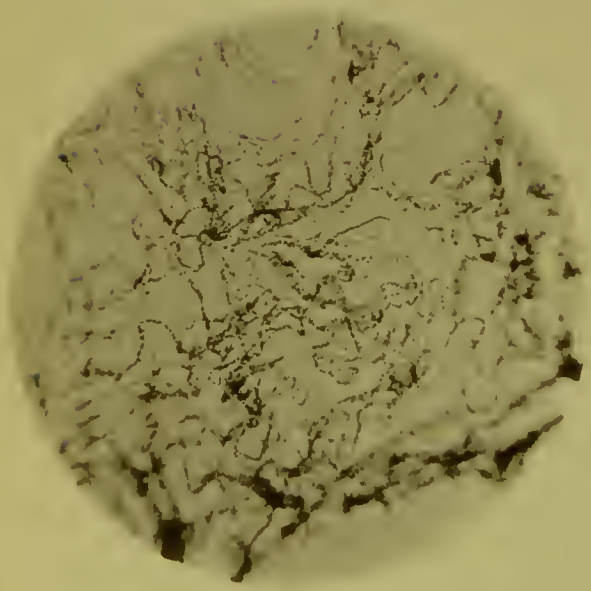


FIG. 19.

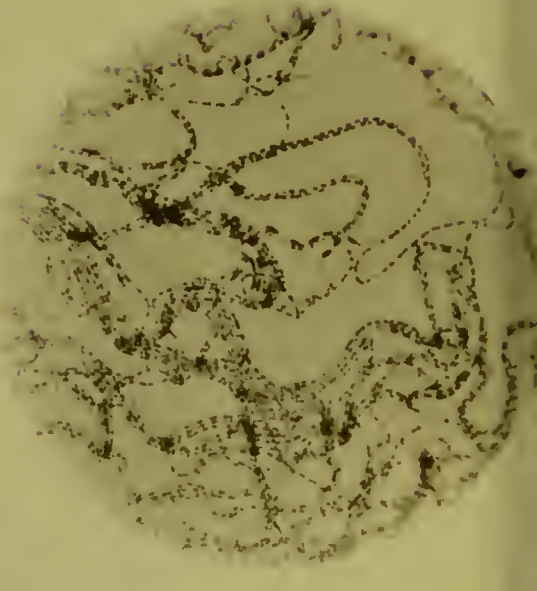


FIG. 20.

ETIOLOGY OF INFLUENZA.

PLATE V.

FIG. 17.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 53. Various species of bacteria are here seen, among them a few influenza bacilli.

FIG. 18.

Reproduced from a photograph of a cover-glass specimen of *bronchial sputum* from case No. 31. The influenza bacilli are here almost in pure culture.

FIG. 19.

Reproduced from a photograph of a cover-glass specimen from an artificial *culture in beef broth* of influenza bacilli derived from the bronchial sputum of case No. 54. The influenza bacilli are here seen forming long chains.

Magnifying power 333.

FIG. 20.

Represents the same specimen more highly magnified (1,000).

ETIOLOGY OF INFLUENZA.

PLATE VI.

FIG. 21.

Reproduced from a photograph of a cover-glass specimen from an *Agar culture* of influenza bacilli derived from the bronchial sputum of case No. 2.

FIG. 22.

Reproduced from a photograph of a cover-glass specimen from a *broth culture* of influenza bacilli derived from the bronchial sputum of case No. 5.

FIG. 23.

Represents another specimen from the same culture as fig. 22.

FIG. 24.

Reproduced from a photograph of a cover-glass specimen from a *broth culture* of influenza bacilli derived from the bronchial sputum of case No. 31.

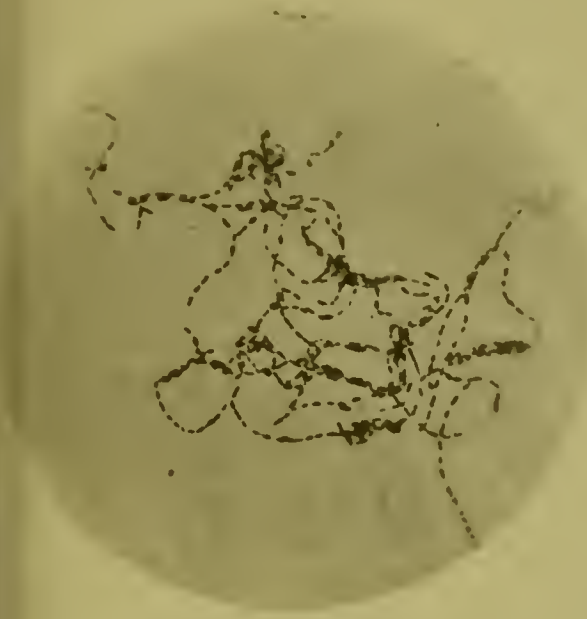


FIG. 21.

FIG. 22.

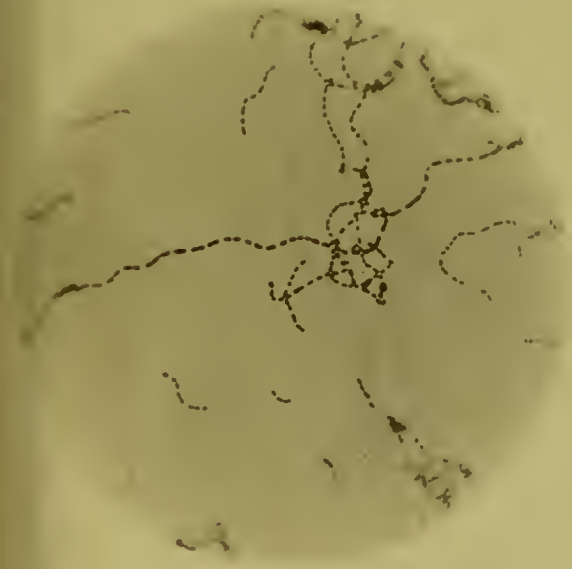


FIG. 23.

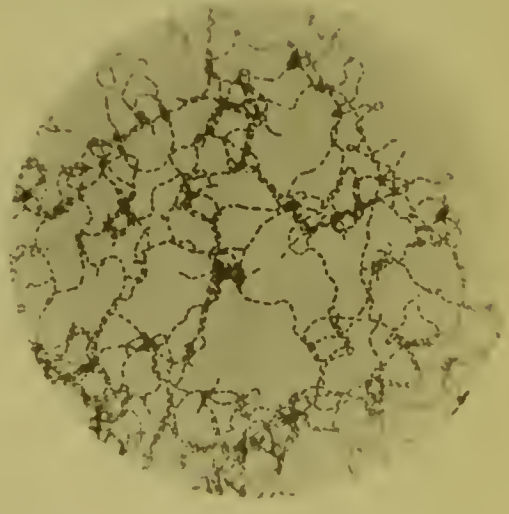


FIG 24.

PLATE VII.

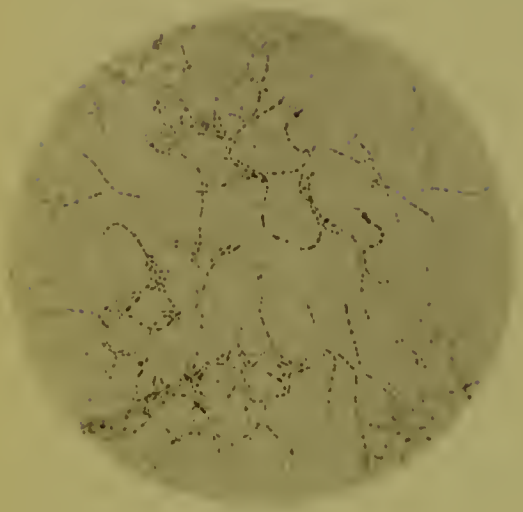


FIG. 25.

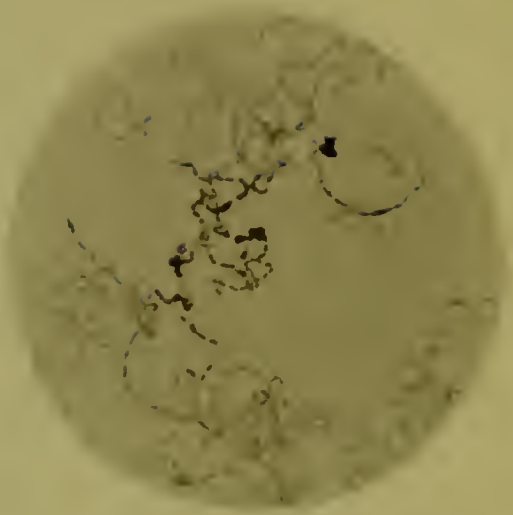


FIG. 26.

FIG. 27.

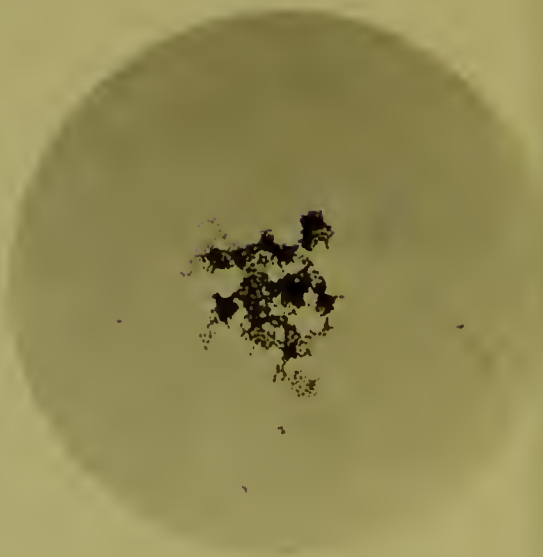


FIG. 28.

ETIOLOGY OF INFLUENZA.

PLATE VII.

FIG. 25.

Reproduced from a photograph of a cover-glass specimen from an *Agar sub-culture* of influenza bacilli derived from the bronchial sputum of case No. 31. A few enlarged involution forms of the bacillus are here seen.

FIG. 26.

Represents another specimen taken at a later date from the Agar sub-culture referred to under fig. 25. Involution forms of the bacillus had, when this specimen was taken, become more numerous.

FIG. 27.

Reproduced from a photograph of a cover-glass specimen from a *broth culture* of influenza bacilli derived from the *blood* of case No. 40. The influenza bacilli are in chains.

FIG. 28.

Represents a specimen from an *Agar sub-culture* of the same blood (csse No. 40).

ETIOLOGY OF INFLUENZA.

PLATE VIII.

FIG. 29.

Reproduced from a photograph* of a cover-glass specimen from a *broth culture* of influenza bacilli derived from the bronchial sputum of case No. 54. The influenza bacilli are forming as usual chains, individuals showing in most instances the characteristic polar staining. There are a few involution forms.

* The photograph in this instance is by Mr. Andrew Pringle.

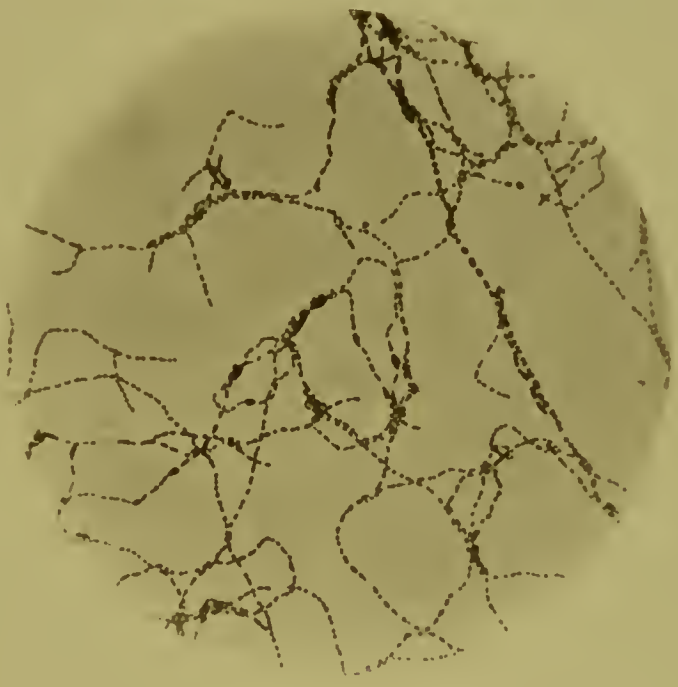


FIG. 29.

PLATE IX.

FIG. 31.

FIG. 30.

ETIOLOGY OF INFLUENZA.

PLATE IX.

FIG. 30.

Reproduced from a photograph of colonies of influenza bacilli on the surface of *Agar* set slantingly in a test tube, as seen by reflected light. The fluid which has gravitated to the bottom of the test tube is seen to be clear, except for a fine flocculer precipitate from the growth.

Magnifying power, 2.

FIG. 31.

Represents the above colonies as seen by transmitted light; the test tube being in this case inverted.

Magnifying power, 4.

ETIOLOGY OF INFLUENZA.

PLATE X.

FIG. 32.

Reproduced from a photograph of colonies of the influenza bacillus *on the surface of Agar*, as seen by reflected light.

Magnifying power, 2.

FIG. 33.

Represents a *stab-culture* in Agar of the influenza bacilli.

Magnifying power, 2.

FIG. 34.

Represents the stab-culture in fig. 33 further magnified.

Magnifying power, 4.

FIG. 32.

FIG. 33.

FIG. 34.

PLATE X.

ETIOLOGY OF INFLUENZA.

PLATE XII.

FIGS. 39 and 40.

Reproduced from a photograph of a cover-glass specimen from a *late sub-culture* of the monkey's pneumonia bacilli.

FIG. 41.

Represents colonies *on gelatine* of monkey-pneumonia bacilli.

FIG. 42.

Represents a *streak-culture on gelatine* of the monkey-pneumonia bacilli.

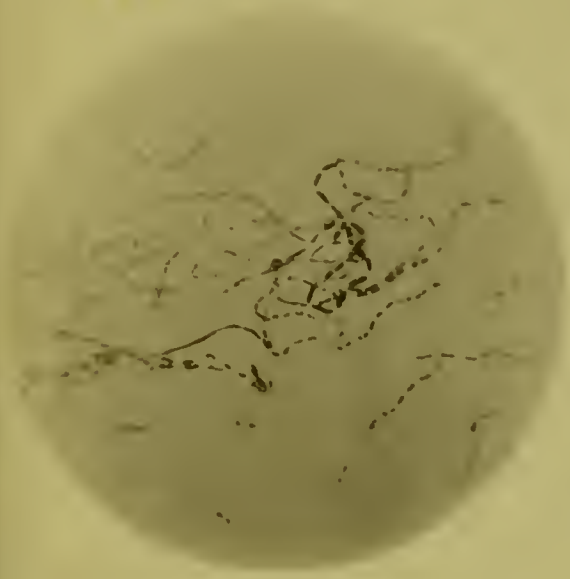


FIG. 39.

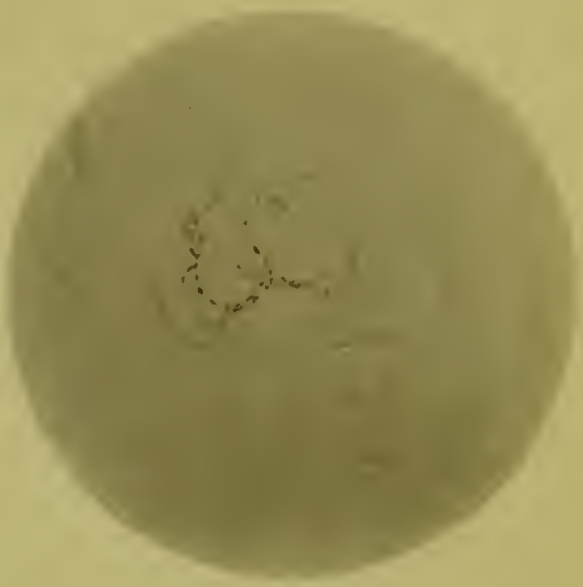
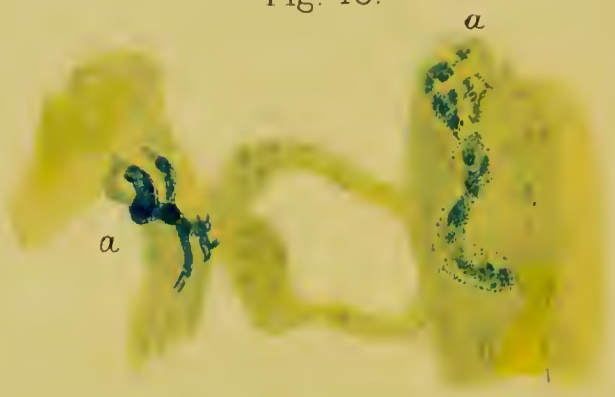


FIG. 40.

FIG. 41.

FIG. 42.

Fig. 43.



× 300

Fig. 44.

× 300

ETIOLOGY OF INFLUENZA.

PLATE XIII.

FIG. 43.

Represents a section through the lung of case No. 3.

a.—Capillary blood vessels plugged with Pfeiffer's bacilli.

FIG 44.

Represents another section from the same lung as fig. 43, and depicts similar conditions.

ETIOLOGY OF INFLUENZA.

PLATE XIV.

Fig. 45.

Represents a section through the superficial portion of the *lung* of monkey No. 10.

a.—Pleura covering the lung; the air vesicles near it filled with fibrin and blood, and appearing deeply stained.

b.—A large patch of hæmorrhage deeper in the lung; the air vessels uniformly filled with and distended by blood and fibrin.

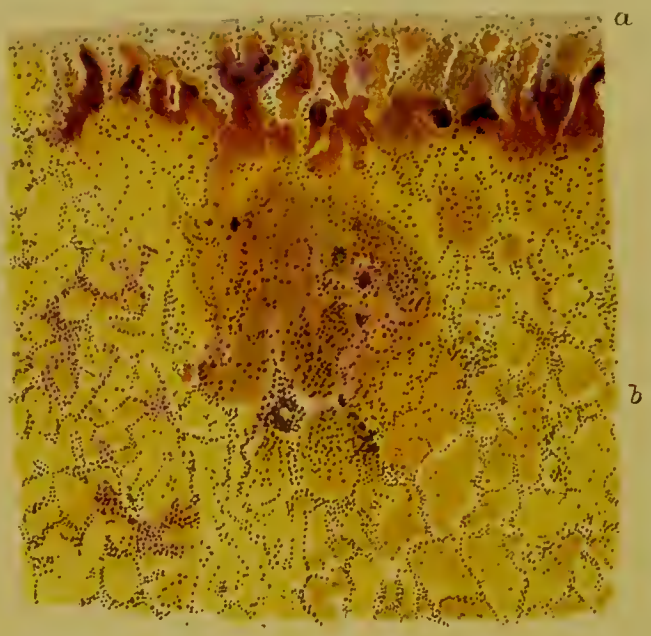
Fig. 46.

Represents a section from the same lung as fig. 45.

a.—Pleural covering.

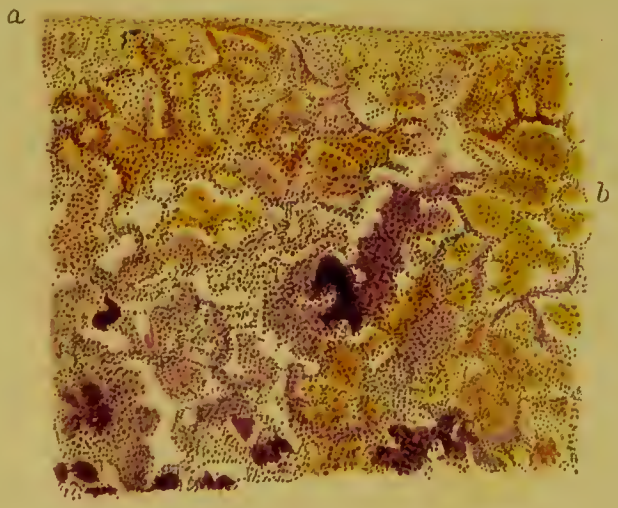
b.—Bronchi, infundibula, and air cells containing blood and fibrin.

Fig 45.



× 50

Fig 46.



× 50

M.H. Lapidge

JUDD AND Co. LTD LITH 63 CARTER LANE DOCTORS COMMONS 1852. 3 32