MAP OF NEW ZEALAND.
Auckland to Sydney

MAP

NEW ZEALAND

RAILWAY, STEAMER, AND COACH ROUTES.

STATISTICS.

Area of Dominion, square miles...

Population (Census for 1911)...

Chief Cities.

Auckland, 40,536; including Suburbs...

Wellington, 64,372;

Christchurch, 53,116;

Dunedin, 41,529;

GAME SEASONS.

DEER

Approximately, 24th February to 31st May. (Opening and closing dates vary slightly according to locality.)

FISHING:

Auckland Acclimatization District, 1st November to 15th May; Rotorua, 1st November to 31st May; other districts, 1st October to 30th April.

FEATHERED GAME:

1st May to 31st July, except for geese (which is from 1st February to 30th April). The year will be a Close Season every third year thereafter.

SPORTSMEN are recommended to peruse the Tourist Department's "Itinerary of Travel" (issued free of charge) for further particulars regarding Game Seasons, Deer Preserves, Trout-fishing Streams, &c.
A STUDY of the INFLUENZA EPIDEMIC in NEW ZEALAND 1918.

EPIDEMIOLOGY, ADMINISTRATION, and BACTERIOLOGY.

with a

REPORT on the PATHOLOGY of TWENTY FIVE FATAL CASES.


by

S.T. Champtaloup, M.B. Ch.B. (1st. Class Honours) 1906.

B.Sc. (Public Health) 1909.
THE INFLUENZA EPIDEMIC IN NEW ZEALAND 1918.

CONTENTS.

Part 1.

Epidemiology.

4. Influenza in New Zealand prior to 1918. Page 11.

Part 11.

Administration and Prophylaxis.

12. Administrative measures adopted to combat the Epidemic Page 35.
   (1) General measures Page 35.
   (2) Special measures Page 43.
      a. Inhalation chambers Page 43.
      b. Face Masks Page 50.
      c. Prophylactic vaccination Page 54.

Part 111.

Pathology and Bacteriology.

## Contents. (continued.)

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>14. A summary of ante-mortem and post-mortem Bacteriology of hospital cases.</td>
<td>82.</td>
</tr>
<tr>
<td>16. Investigation of Pneumococcal strains, Inulin fermentation-Bile solubility, Agglutination with Type sera.</td>
<td>97.</td>
</tr>
<tr>
<td>17. Discussion of results--the Influenza Bacillus--a Filterable Virus,--the Staphylococcus Aureus</td>
<td>100.</td>
</tr>
<tr>
<td>18. General Summary</td>
<td>105.</td>
</tr>
<tr>
<td>19. References</td>
<td>110.</td>
</tr>
<tr>
<td>20. Description of plates and photomicrographs</td>
<td>116.</td>
</tr>
<tr>
<td>22. Box microscope slides</td>
<td></td>
</tr>
</tbody>
</table>
TABLES, CHARTS, ILLUSTRATIONS.

Map.

Map of New Zealand. Inside front cover.

Table I.

| II. | Comparative Table Temperature & Rainfall, New Zealand. |
| III. | Census returns Population N.Z. in age groups. |
| IV. | Crude Death Rates, New Zealand. |
| V. | Infantile Mortality Rate. |
| VI. | Incidence of Principal Infectious Diseases. |
| VII. | Deaths from Influenza-Pneumonia, 1913-1917. |
| VIII. | Incidence of Influenza N.Z. Military Camps. |
| IX. | Deaths from all Catarrhal Diseases, Aug-Dec. 1917 and 1918. |
| X. | Notifications of Influenza 1918-1919. |
| XI. | Death rate from Influenza in the cities. |
| XII. | Deaths from Influenza Jan. - Dec. 1918. |
| XIII. | Death rates from Influenza, Europeans and Natives. |
| XIV. | Death rates in age groups and in both sexes from Influenza. |
| XV. | Vaccines in Prophylaxis, composition of. |
| XVI. | Incidence of Influenza in the inoculated and non-inoculated. |
| XVII. | Clinical, Pathological and Bacteriological Survey 25 fatal cases. |
| XVIII. | Bacteriological results Sputum examinations. |
| XIX. | Blood cultures. |
| XX. | Secondary Complications. |
| XXI. | Summary 25 fatal cases. |
| XXII. | Pneumococcal strains investigated - sources. |

Chart I.

| II. | Deaths from Influenza in London and New Zealand. |
| III. | Deaths from Influenza daily during the Epidemic. |
| IV. | Death rates per 10000 of population in age groups. |
| V. | Admissions to hospital following inoculation - negative phase. |
Tables, Charts, Illustrations. (Contd.).

Plates ... ... ... ... Inside back cover.


" 2. " " " " Trachea & Bronchi. Haemorrhagic oedema type.


" 6. " " " 686 " 676

" 7. " " " 693 " 691.

Microscope Slides. A set of Pathological and Bacteriological slides in separate case.

Note. The bacteriological slides were made during routine work, and in the interval since the epidemic several of the best preparations have so faded that it is difficult to present as satisfactory and representative a collection as desirable.
A STUDY OF THE INFLUENZA EPIDEMIC IN NEW ZEALAND.

1918.

Part I.

EPIDEMIOLOGY.

During the latter half of 1918 the Dominion of New Zealand, in common with many other countries, was visited by an unusually severe epidemic of Influenza. A primary wave of moderate severity but with a low case mortality was succeeded in November and December by a secondary wave of unusual severity, the incidence and case mortality being far in excess of anything which had been previously experienced in the Dominion.

My official duties brought me into close touch with the administrative measures which were adopted in combating the epidemic, while the intimate connection of my department with the Hospital enabled me to see at first hand the clinical and pathological features in a large number of cases both in the primary and secondary waves.

In the following pages I have attempted to outline the epidemiology of the epidemic as a whole, the general and special administrative measures adopted locally to combat it, and have summarised the pathological and bacteriological findings in 25 fatal cases during the secondary severe wave of the epidemic of which I was able to make a preliminary study at the time. Certain details, more particularly in connection with the bacteriological laboratory findings, have since been more fully worked out than was possible during the almost unprecedented conditions of life which prevailed while the epidemic was at its height. So much has been written recently on different aspects of this world wide pandemic that I have as far as possible confined my remarks to the position in New Zealand, about which little has been published other than a short preliminary communication by several colleagues and myself to the local Medical Journal.\(^{(1)}\)
GEOPGRAPHICAL, CLIMATE, AND METEOROLOGY.

Before discussing the general features of influenzal epidemics in New Zealand, a short account of the geographical position, area, climate and meteorology, and vital statistics of the Dominion, which relate to the matter under discussion, will serve to show that, whereas conditions of overcrowding, poverty, lack of ventilation, and severity of climate which are frequently associated with outbreaks of infectious diseases, do not prevail here, we cannot look to these except in a very minor way as being responsible for the severity of the Epidemic in New Zealand.

BOUNDARIES & AREA. Reference to the accompanying map will show that the Dominion of New Zealand, which forms an outpost of the great British Empire, is bounded on all sides by the Pacific Ocean, and lies within latitude 34° - 48° and longitude 166° - 179°E. Like Great Britain it runs roughly North and South, but being in the Southern hemisphere the Northern part of the Dominion has a semitropical to temperate climate, while the climate of the Southern part is more like that of the South of England.

The Dominion consists of three main islands with several groups of small islands. The main islands known as the North, South and Stewart Islands, have a coast-line 4,330 miles in length. The total area of the Dominion proper is 66,292,232 acres or 103,581 sq. miles.

According to our geologists, the New Zealand area was probably the foreshore of a great continent, but after the Trias-Jura sediments were deposited, far reaching changes involving the breaking up and disappearance of the continental land took place. The New Zealand area was necessarily involved in these earth movements, and, as the result, the existing strata were folded, broken, and raised above sea level. After extensive denudations of its surface had taken place, New Zealand was again probably several times depressed and elevated, either in whole or in part.

FLORA & FAUNA. While there are several plants possessing poisonous properties, there are no native animals or insects which are in any way harmful to man, except a small spider, (3) the Katipo, which is found on some of the sea beaches, and whose bite is followed by severe symptoms of poisoning. (4)

TEMPERATURE & RAINFALL. The following chart prepared from particulars supplied by the Government Meteorologist for the year 1918 shows, in comparison with an average of previous years, that the
## Comparative Table, 1918

<table>
<thead>
<tr>
<th>Stations</th>
<th>Temperature in Shade</th>
<th>Rainfall</th>
<th>Prevailing Winds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Highest &amp; Date.</td>
<td>Lowest &amp; Date.</td>
<td>Mean Max. Temp. for Year</td>
</tr>
<tr>
<td>Auckland</td>
<td>°Fahr. 78.0 Jan. 13</td>
<td>°Fahr. 35.0 July 26</td>
<td>°Fahr. 64.6</td>
</tr>
<tr>
<td>Wellington</td>
<td>°Fahr. 79.3 Feb. 6</td>
<td>°Fahr. 30.1 July 27</td>
<td>°Fahr. 61.3</td>
</tr>
<tr>
<td>Christchurch</td>
<td>°Fahr. 83.9 Jan. 18</td>
<td>°Fahr. 27.2 July 17 Aug. 16</td>
<td>°Fahr. 60.5</td>
</tr>
<tr>
<td>Dunedin</td>
<td>°Fahr. 81.0 Feb. 13</td>
<td>°Fahr. 28.0</td>
<td>°Fahr. 57.8</td>
</tr>
</tbody>
</table>

Average Rainfall 12 stations:

- 1915: 37.85 in.
- 1916: 44.28 in.
- 1917: 49.58 in.
- 1918: 50.49 in.

The mean temperature was lower and the total rainfall higher in that year. The figures for Auckland in the North and Dunedin in the South, and Wellington situate about midway between these two are given.
VITAL STATISTICS.

The population of the Dominion. (2)

The estimated population of the Dominion including the Maoris and residents of the Cook and other Pacific Islands at the end of 1918 was 1,175,325. The Maori population in the census year 1916 was 49,776.

With the exception of the last two years of the war the population of New Zealand has shown a continuous, though not regular, increase in each year since 1855, the first year in which accurate records of births and deaths were obtained and used with the returns of immigration and emigration.

The population of the four principal towns including suburban areas as at the census of 1916 was as follows:-

Auckland. 133,712.
Wellington. 95,235.
Christchurch. 92,733.
Dunedin. 68,716.

The average rate of natural increase for the period 1908-17 was 16.86 per 1000.

AGE & SEX CONSTITUTION OF THE PEOPLE. The withdrawal of a large body of men between the ages of 20 and 45 for military service overseas has materially altered the age and sex constitution of the population as estimated at the 1916 census. Now most of these men, except those who paid the supreme sacrifice, have returned, but during the period under discussion, i.e. July - December 1918, they were still absent from the Dominion.

The following table compiled from the census returns of 1896, 1906, and 1916 show the numbers of males and females in age groups, 0-5, 5-20, 20-45 (military age), above 45, at each census.
TABLE II.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 5 years</td>
<td>42,448</td>
<td>41,211</td>
<td>52,499</td>
<td>50,246</td>
<td>67,320</td>
<td>64,735</td>
</tr>
<tr>
<td>5 years &amp; under 20 years</td>
<td>126,969</td>
<td>125,257</td>
<td>133,902</td>
<td>130,261</td>
<td>162,884</td>
<td>160,563</td>
</tr>
<tr>
<td>20 years &amp; under 45 years</td>
<td>131,121</td>
<td>117,874</td>
<td>193,256</td>
<td>169,723</td>
<td>199,499</td>
<td>220,908</td>
</tr>
<tr>
<td>45 years &amp; over</td>
<td>70,292</td>
<td>47,376</td>
<td>90,860</td>
<td>66,888</td>
<td>121,381</td>
<td>100,893</td>
</tr>
</tbody>
</table>

A calculation of the proportion per cent at each age group to the total of males and females shows the effect of a declining birth rate on the ages under 15, the proportion of males at these ages being 30.19 per cent in 1911, against 34.31 per cent in 1896, and of females 32.58 per cent against 38.02 per cent respectively. When the proportions for 1916 are considered, it will be seen that the figures in all age groups for the male portion of the population have been materially affected by the withdrawal of men between the ages of 20 and 45 for service overseas.

The increased proportion at the higher ages is due to the advanced age of the then mostly adult immigrants introduced during the early stages of settlement. These form the greater portion of the groups 60 years and over.

There has been a gradual equalisation of the sexes since 1861, the numbers of females to 1000 males having risen from 622 in 1861 to 903 in 1901. The proportion was slightly lower in 1906 and 1911 but has risen to 993 in 1916 mainly on account of the absence of so many men at the war.

VITAL STATISTICS IN BRIEF. The Birth rate has fallen from 35.40 per 1000 of the population in 1886 to 26.34 in 1901 and 25.69 in 1917. The proportion of births of males to every 1000 females in 1917 was 1030.
The Death Rate shows a very gradual decline, and is one of the lowest on record, the rate per 1000 of the population being as follows:

**TABLE III.**

**Crude Death Rates per 1000 Population.**

<table>
<thead>
<tr>
<th>Year</th>
<th>New Zealand</th>
<th>Commonwealth of Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1887</td>
<td>10.29</td>
<td>-</td>
</tr>
<tr>
<td>1901</td>
<td>9.81</td>
<td>-</td>
</tr>
<tr>
<td>1911</td>
<td>9.39</td>
<td>-</td>
</tr>
<tr>
<td>1917</td>
<td>9.58</td>
<td>-</td>
</tr>
<tr>
<td>1909-13</td>
<td>9.33</td>
<td>10.70</td>
</tr>
</tbody>
</table>

Male deaths to every 100 female deaths were 143 in 1908 and 134 in 1917.

**INFANTILE MORTALITY** rate, i.e. deaths under one year of age per 1000 births.

New Zealand has a remarkably low infantile mortality rate, and this has shown a progressive decrease since 1908. The rate in 1917 (48.16) was the lowest recorded in New Zealand.

**TABLE IV.**

<table>
<thead>
<tr>
<th>Year</th>
<th>Infantile Mortality rate per 1000 births</th>
</tr>
</thead>
<tbody>
<tr>
<td>1908</td>
<td>67.89</td>
</tr>
<tr>
<td>1909</td>
<td>61.60</td>
</tr>
<tr>
<td>1910</td>
<td>67.73</td>
</tr>
<tr>
<td>1911</td>
<td>56.31</td>
</tr>
<tr>
<td>1912</td>
<td>51.22</td>
</tr>
<tr>
<td>1913</td>
<td>59.17</td>
</tr>
<tr>
<td>1914</td>
<td>51.38</td>
</tr>
<tr>
<td>1915</td>
<td>50.05</td>
</tr>
<tr>
<td>1916</td>
<td>50.70</td>
</tr>
<tr>
<td>1917</td>
<td>48.16</td>
</tr>
</tbody>
</table>
Besides advantages of climate, New Zealand possesses a population younger in age constitution than that of most other countries - conditions favourable to a low rate of mortality. The extremely low rate of infantile mortality is remarkable, and depends partly on climatic conditions, partly on the better housing conditions and rates of pay prevailing here, and partly on an active educational propaganda by the Royal New Zealand Society for the Health of Women and Children since 1907, under the able and enthusiastic direction of its founder, Dr. Truby King, C.M.G.

THE DENSITY OF POPULATION. The number of persons (excluding Maoris) to a square mile at the last census was 10.64.
THE PEOPLE OF NEW ZEALAND.

Our people are mostly of English, Scotch and Irish ancestry, except for a few thousand natives. Possibly because of our remotesness from the thickly populated centres of the Old World, the foreign element is small, and our ancestry, conditions of life, and temperate climate have given rise to a freedom-loving, democratic, and for the most part healthy-living and loyal people.

If we consider the combined populations of Glasgow and Edinburgh spread out over the whole of Great Britain we get some idea of the density of population for the Dominion as a whole. Even in the cities the people are well-housed, well-fed, and well-educated, and there is an absence of that poverty and overcrowding which is the curse of many of the larger cities in the Old World. Our industries are mainly connected with produce—wool, mutton, hides, butter, cheese, timber, and subsidiary industries depending on these. Our workmen are well paid, and consequently the conditions of work, and living and housing, are such that epidemic diseases should not obtain as favourable a nidus as is the case in countries less favourably situated.

Yet with all these initial advantages, we are by no means free from infectious disease, as a glance at the following table will show. (5) Tuberculosis (principally pulmonary) is all too rife, and Diphtheria, Scarlet Fever, and Measles are common. Typhoid fever is relatively uncommon, except amongst the Native race, where it is impossible to maintain the sanitary standard and conditions of living found amongst the white population.

TABLE V.

Number of cases for four years 1915-18, and rate of incidence per 10,000 of the population for 1917 of the principal infectious diseases notified.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Number of Cases</th>
<th>Incidence per 10,000 of population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scarlet Fever</td>
<td>2301</td>
<td>4278</td>
</tr>
<tr>
<td>Diphtheria</td>
<td>1393</td>
<td>2376</td>
</tr>
<tr>
<td>Enteric Fever</td>
<td>821</td>
<td>806</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>998</td>
<td>950</td>
</tr>
<tr>
<td>Poliomyelitis</td>
<td>10</td>
<td>1018</td>
</tr>
<tr>
<td>Meningococcal</td>
<td>82</td>
<td>135</td>
</tr>
<tr>
<td>Meningitis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The returns for Enteric fever are chiefly due to cases amongst the native population.

There was an epidemic of this disease in 1916 when 1018 cases were notified, as against 54 in 1917.

There was a marked drop in the number of cases of Scarlet Fever reported as compared with the previous years but there has been a great increase in the returns for diphtheria.

The Colonial is said to be less resistant to respiratory and catarrhal diseases than those of the same age-groups in Great Britain, and there is some evidence to support this view in the greater prevalence of these diseases amongst colonial troops in Great Britain than amongst the British troops. Two reasons may be advanced to explain this difference: first and of less importance is the fact that we live under considerably better climatic conditions than do those at Home, so that in the sudden translation to the severe winters there, our troops were less resistant to those diseases upon which climate may be said to have some influence. Secondly, and I think more important, is the difference in density of population. Given a population, the bulk of which is spread over a wide area in villages, on farms, sheep stations etc., there are less opportunities for intimate intercourse, and therefore less opportunity for contracting respiratory diseases. A race exposed to and frequently suffering individually from catarrhal diseases must develop some immunity or a greater immunity on the whole, than people living as they do in New Zealand.

Our young people to whom this specially applies have therefore less immunity, and, given the opportunity for mass infection such as prevails in military camps, they are likely to show a higher incidence for respiratory and catarrhal diseases.

This is well borne out by the experience of military medical officers when at Home, and, to quote a specific instance, it was definitely proved that the country recruit was much more susceptible to meningococcus infection shortly after coming into camp than was the town recruit. So much so that special temporary camps were set up to receive country recruits before they were drafted to the main camps. Our experience in this respect is, I believe, similar to that of medical officers in Great Britain and America.
I have indicated sufficiently how favourable the conditions prevailing in New Zealand as affecting the health and well-being of the people as a whole, but how our isolation and, in consequence, our relative susceptibility to certain diseases provided a soil upon which the Influenza virus played as much, if not more, havoc than in almost any other country excepting perhaps South Africa.
In New Zealand, as in all other countries, Influenza has been reported each year, generally in the winter months, and a few deaths are attributed to this cause, generally from secondary complications such as pneumonia. From time to time epidemics of more than average severity arise constituting a pandemic of world wide significance.

No accurate historical record of any value is available, but Cumpston (7) has recently traced the history of the disease in Australia - the neighbouring state - as far back as 1820.

Particulars of a general epidemic in 1860 are supplied, a limited outbreak in 1885, and again in 1890, the year of a great outbreak. In 1889 there were 21 deaths in the State of Victoria from Influenza. In 1890 the number was 154, and in 1891 it reached 1035. Again, in 1899, no less than 963 persons died of influenza. The outbreaks occurred in the winter months between March and October.

The pandemic of 1890-91 affected New Zealand markedly, but the British epidemics of 1895 and 1900 passed almost unnoticed here. Prior to November 1918, Influenza was not a notifiable disease in New Zealand, and the only returns available are those of the Registrar-General showing the annual deaths from Influenza and Pneumonia. The following table is compiled from these returns:

<table>
<thead>
<tr>
<th>Table VI.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deaths from Influenza.</td>
</tr>
<tr>
<td>1913.</td>
</tr>
<tr>
<td>1914.</td>
</tr>
<tr>
<td>1915.</td>
</tr>
<tr>
<td>1916.</td>
</tr>
<tr>
<td>1917.</td>
</tr>
</tbody>
</table>
Influenza in New Zealand in 1918.

In common with the rest of the civilised world, New Zealand suffered very severely from the pandemic of influenza in 1918, and, as in most other countries, it showed itself in two distinct waves, the first of which reached its maximum in August and September, and the second wave about the middle of November.

The mortality due to the first wave, though abnormal for New Zealand, was not alarming, but the virulence of the second wave was far in excess of anything which has hitherto been experienced with influenza.

Two facts of interest to the epidemiologist are, firstly, that the same division into first and second waves with undue morbidity of the second wave was experienced here, and that the greatest intensity of the second wave was almost synchronous in countries so widely separated as Britain and New Zealand; secondly the brief interval between the August and November peaks is even more striking than the 16 week interval between the peaks in Great Britain, which Newsholme (9) states is exceptionally short.

Newsholme (9) further remarks that he has not found another instance of a widespread epidemic culminating in the summer months - the London curves with this single exception showing that since 1881 epidemic peaks have always occurred in winter and spring. In this respect our peaks roughly correspond in point of time with those experienced in Great Britain but they conform to previous epidemics in that they occurred in our winter and spring.

The following chart (10) illustrates the first point - the red line shows the deaths from Influenza in London during the height of the second wave, and the black line, the deaths in New Zealand during the second wave. It is interesting to note that the greatest number of deaths in any one week in London was 1705, whereas in New Zealand with a total population one-fifth that of London, the greatest number of deaths for any one week was 1860.
Comparing the two curves it is seen that the second wave is equally an explosive one of great intensity, but there is the remarkable fact that the two waves in London and New Zealand so nearly coincide. The crest was reached in the one case in the weeks ending November 2nd and 9th., and in the other during the weeks November 16th and 23rd., about two weeks later. This coincidence cannot be explained by the movement of troops and shipping, for the interval be-
Between London and New Zealand by the ordinary routes of travel is at least six weeks.

The only other countries with which we were in direct communication at this time were the United States, Canada and Australia.

In Boston the peak of the second wave was reached on October 5th, New York on October 26th, and San Francisco on November 2nd., or about the same time as London.

In Australia the secondary wave did not develop until well on in January 1919. Australia differs from other countries in that the secondary wave was delayed and did not show the explosive character so marked elsewhere. Certainly the people there suffered much less than we did, and for their good fortune the Australians point to the very rigid quarantine set up when, in October 1918, New Zealand was in the throes of the second wave.
THE PRIMARY WAVE OF THE EPIDEMIC.

Since Influenza was not a notifiable disease prior to November, 1918, the records of this phase of the epidemic must be incomplete. There are two sources of definite information (a) the returns of the military training camps, (b) the Registrar-General's return of deaths. Further evidence has been collected by the Public Health Department from Medical Officers, which shows that during July, August, and September, 1918, Influenza was more prevalent than in the same months of the preceding years. Not only was the disease more prevalent, but the cases were of a more severe type, and presented several new clinical features.

Examining these three sources of information we find the following:-

(a) Statistics from military camps. Table VII represents the admission of Influenza cases to hospital in the larger training camps.

<table>
<thead>
<tr>
<th>Month</th>
<th>Cases</th>
<th>Deaths</th>
<th>Month</th>
<th>Cases</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan. 1918</td>
<td>22</td>
<td>-</td>
<td>July, 1918</td>
<td>145</td>
<td>--</td>
</tr>
<tr>
<td>Feb.</td>
<td>30</td>
<td>-</td>
<td>Aug.</td>
<td>571</td>
<td>2</td>
</tr>
<tr>
<td>March</td>
<td>16</td>
<td>-</td>
<td>Sept.</td>
<td>1216</td>
<td>--</td>
</tr>
<tr>
<td>April</td>
<td>13</td>
<td>-</td>
<td>Oct.</td>
<td>1116</td>
<td>2</td>
</tr>
<tr>
<td>May</td>
<td>32</td>
<td>-</td>
<td>Nov.</td>
<td>4369</td>
<td>280</td>
</tr>
<tr>
<td>June</td>
<td>16</td>
<td>-</td>
<td>Dec.</td>
<td>15</td>
<td></td>
</tr>
</tbody>
</table>

We may take this return as a fair indication of the incidence of Influenza throughout New Zealand. The primary wave began in July, (about 6 weeks later than in Great Britain) and reached its crest in September, waning slightly in October. The second wave broke with explosive violence and great mortality in November.
(b) The Registrar-General's returns of Deaths.

The following table supplied by the Registrar-General shows the deaths from all catarrhal diseases including influenza, pneumonia, and bronchitis in the Dominion between the months of August and December, 1917 and 1918.

**TABLE VIII.**

<table>
<thead>
<tr>
<th>Month</th>
<th>1917</th>
<th>1918</th>
</tr>
</thead>
<tbody>
<tr>
<td>August</td>
<td>129</td>
<td>206</td>
</tr>
<tr>
<td>September</td>
<td>99</td>
<td>148</td>
</tr>
<tr>
<td>October</td>
<td>85</td>
<td>158</td>
</tr>
<tr>
<td>November</td>
<td>82</td>
<td>3574</td>
</tr>
<tr>
<td>December</td>
<td>46</td>
<td>2316</td>
</tr>
</tbody>
</table>

It will be seen that during August and September, when catarrhal diseases are usually at a maximum in the Dominion, there were 228 deaths from the above causes in 1917, and 354 in 1918. In the latter year not only was there a considerable increase of deaths during the period covering the primary wave, but the secondary wave showed an alarming increase in deaths as compared with the falling rate for the corresponding months in 1917.

(c). Returns from Medical Practitioners.

Details of some hundreds of cases were collected from medical practitioners, the main features being the unusual incidence and severity of the attacks.

Three cases which I saw in Dunedin at this time were colleagues, all of whom were ill with purulent bronchitis, and in whose sputum I found numerous influenza bacilli and pneumococci.

A localised outbreak of marked severity occurred in July in the North Auckland district, where, in a small settlement, thirty-one persons were attacked with Influenza, of whom eleven had pneumonic complications. Six cases occurred in one household, four of whom had pulmonary complications.
Dr. Davis, Medical Officer to the natives in the Waipiro district, Hawke's Bay, reporting on the primary wave to the Chief Health Officer (12) states "The Influenza occurring in August and September was of the ordinary seasonal type diffused over these months and those immediately preceding and following. But besides the ordinary catarrhal symptoms there were frequently present symptoms not hitherto met with by me, although I worked through the 1889 epidemic in a busy practice in London. The pulmonary complications included spitting of blood, expectoration of almost pure pus, with prolonged symptoms and a tendency to relapse. Deaths were chiefly in those of intemperate habits, or in those suffering from chronic bronchitis and asthma. The incidence was largely on the Natives although Europeans were by no means exempt. The total number of cases reported was 81, with 4 deaths."

This localised outbreak was exceptionally severe and widespread in a small community, and it is interesting to note that this district escaped with a few mild cases and no deaths during the secondary wave.

As far as one can judge from the data available relating to the primary influenza wave, it appears to have been universally present in all districts during July, August and September.

It was characterised by localised explosions of violence such as those mentioned in the Auckland and Hawke's Bay district.

A temporary lull followed in October before the explosive outbreak of the secondary wave in November.
THE SECONDARY WAVE OF THE INFLUENZA EPIDEMIC.

In the previous section a brief outline has been given of the position in New Zealand up to, and immediately preceding November, 1918. Had no secondary wave followed, the general impression left in the minds of the public would have been practically nil. This partly explains our almost total unpreparedness for the severe form of the epidemic, which, starting with alarming suddenness and spreading with great rapidity, so completely disorganised the whole life of the people that it is never likely to be forgotten by the present generation.

That there was some excuse for the apparent lack of foresight of our Health Department is borne out by Newsholmes (8) who remarks "It is evident that we are ignorant of the causes of pandemics of Influenza at irregular intervals, and their occurrence has never, as far as I know, been anticipated". In this respect we do not seem to have progressed greatly in our knowledge since the days when Sydenham expounded his Epidemic Constitutions.

The official attitude at this time, October, 1918, is well reflected in the following extract from the Official Journal of the Government Public Health Department. (13).

"We have evidence that the disease has been present in more or less epidemic form in New Zealand for at least two months. A vessel from overseas which arrived recently at Auckland was reported by wireless as having a severe form of "Spanish Influenza" amongst the passengers and crew. Before the ship was given its clearance, the Minister (of Public Health) required an assurance that this so-called Spanish Influenza was of the same type of disease which at present was prevalent in New Zealand. This was ascertained to be the case, and passengers were allowed to land after they had been treated in a chamber wherein was fitted up an apparatus of the type extensively used in camps and elsewhere for administering inhalation of various antiseptics, thus ensuring disinfection of the throat and nasal passages.

"The Department has been urged to quarantine and disinfect ships arriving with Influenza, but any steps further than those outlined above would not produce any useful results. The quarantining of ships coming from overseas would be a drastic course of action in view of the world wide prevalence of the disease, and would afford no protection to New
"Zealand inasmuch as the disease is already widespread "in the Dominion."

In view of later experience these views have no doubt been considerably modified. The "vessel from overseas" mentioned was the mail steamer "Niagara", which arrived at Auckland from Vancouver and way ports on October 12th. Popular opinion now attributes the whole outbreak to the arrival of this vessel. This is not surprising, since this vessel had many cases of Influenza on board, some of them of severe type, and she arrived just before the outbreak of the second wave.

The steamer carried the Prime Minister and the Leader of the Opposition - at the time a member of the National Cabinet - who were returning from an Imperial Conference in connection with the war, and it is unfortunate that the situation has been clouded in the public mind by political considerations. Even the Royal Commission which was set up to apportion the blame - the three Commissioners being laymen, who could not possibly understand or fully appreciate all the facts laid before them - came to the conclusion that this vessel was responsible. It is easy to blame after the event, and there are many reasons for believing that the ship's arrival was but an incident in a train of events unforeseen but inevitable. This epidemic appeared with the same element of surprise in all countries, and the specific virus must have been carried in a form other than a virulent epidemic wave, and doubtless the same thing occurred in New Zealand. During September five vessels from Europe or America arrived in Auckland, and six in October, carrying convalescents from European Military Hospitals, and among the crews other potential carriers doubtless existed. Probably these eleven vessels coming to Auckland all added their quota to the massing infection, the "Niagara" adding her share with the rest. Newsholme(8) in Great Britain, Cumpston(7) in Australia, are both of this opinion, the former remarking that "during the Spring and throughout the Summer of this year (1918) the usual endemic influenza was being almost constantly replenished by more virulent infection on a very large scale from overseas. That October should have been a particularly cold and wet month at Auckland, with a rainfall of over 8 inches, doubtless added a very powerful influence making for prevalence of catarrhal infection.

There were then at Auckland the necessary ingredients for an explosion - a population of susceptible people, more especially Natives in camps and elsewhere, a catarrhal epidemic and a virus of special potentiality from overseas, and the November outburst
was the result.

I have already pointed out that the low resistance of our troops to pneumonia had been demonstrated in Britain, and have advanced reasons for this susceptibility. It is doubtless to this that we owe the very violent character of the outbreak; but as will be shown later, it is probable that to this very violence we owe our present comparative immunity from a recrudescence of the disease such as visited Britain and America in January. In the words of the Chief Health Officer - "Our vaccination may be said to have produced a severe reaction, but it has been effective".

Table IX, shows the notifications for Influenza since it was made a notifiable disease on November 6th, 1918, and demonstrates the secondary epidemic wave in November and December, and the absence of a third wave such as has been experienced elsewhere.

The notifications for the months of November and December 1918 must necessarily be far below the actual number of cases, for the Department at first asked for notification of cases of a pneumonic type only, or cases which had specially severe complications. Moreover, many cases were never able to obtain medical attention owing to the shortage of medical aid and the great demand on its services.

The returns for January - October, 1919, when conditions had become normal, are probably reasonably correct, so that actually there should be a greater difference between the November - December and the January - October figures.
### TABLE IX. (14)

Notifications of Influenza in New Zealand for one year.

November 1918 - October 1919.

<table>
<thead>
<tr>
<th>Number notified during month.</th>
<th>Comparison with previous month.</th>
<th>Total number notified since commencement of epidemic.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Increase.</td>
<td>Decrease.</td>
</tr>
<tr>
<td>1918.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nov. 2510.</td>
<td>-</td>
<td>2510</td>
</tr>
<tr>
<td>Dec. 2363.</td>
<td>-</td>
<td>4873</td>
</tr>
<tr>
<td>1919.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jany. 366.</td>
<td>-</td>
<td>1997</td>
</tr>
<tr>
<td>Febly. x 76.</td>
<td>-</td>
<td>290</td>
</tr>
<tr>
<td>March x 133.</td>
<td>57</td>
<td>-</td>
</tr>
<tr>
<td>April x 395.</td>
<td>262</td>
<td>-</td>
</tr>
<tr>
<td>May x 284.</td>
<td>-</td>
<td>111</td>
</tr>
<tr>
<td>June x 136.</td>
<td>-</td>
<td>148</td>
</tr>
<tr>
<td>July x 200.</td>
<td>64</td>
<td>-</td>
</tr>
<tr>
<td>Aug. x 316.</td>
<td>116</td>
<td>-</td>
</tr>
<tr>
<td>Sept. x 256.</td>
<td>60</td>
<td>-</td>
</tr>
<tr>
<td>Oct. x 197.</td>
<td>-</td>
<td>59</td>
</tr>
</tbody>
</table>

x = Mostly mild cases.
There is abundant evidence that the second wave spread through New Zealand from Auckland southwards. The returns from the various camps and from medical officers already quoted show that already in September and October there had been localised outbreaks of pneumonic type in several parts of the province. This is further confirmed by the District Health Officer, Auckland, (15) who found, on enquiring from medical practitioners, that many cases of Influenza of the same type that characterised the November outbreak had been under their care. He obtained records of five cases of pneumonic influenza in August, five in September, and eleven in the first twelve days of October, in which period he traced eighteen cases of the haemorrhagic type. That an epidemic of influenza of virulent type was present in Auckland early in October is evident from the newspaper reports of that period. (18) It is interesting to note that this occurred before the arrival of the R.M.S. "Niagara" on October 12th, which is popularly supposed to have brought the new type of infection.

The Registrar-General's returns show that, of the fifty-nine deaths from Influenza in the Dominion during October, thirty-four occurred in the Auckland province, and that in the last three days of the month fourteen deaths occurred there, as against two in the rest of the Dominion. It is evident that this increase in virulence continued in Auckland during October, and that in some manner the infection was so reinforced as to produce an explosive effect in Auckland from October 26th onwards. The epidemic spread with great rapidity after this date, and the crowding due to the news of the Armistice on November 8th produced in Auckland city a marked increase of cases, the crest being reached about November 12th. From Auckland the wave spread southwards to the Military Camps, and then to Wellington, where the first cases occurred about November 1st, and where the crest was reached on November 21st. To other towns and districts the disease was shown to spread by rail and steamer from these centres; thus in the Hawke's Bay Province the secondary wave can be traced to the arrival at Gisborne of a steamer from Auckland on October 27th, this town having no other communication with the outside world than by sea.
In the South Island, which is separated from the North Island by the Cook Straight, the secondary wave does not appear to have established itself in Christchurch till about November 6th, the crest being reached on November 20th. The principal factor in the spread of the epidemic in this district was the race-carnival week, beginning on November 4th, which brought many people from the North Island and produced temporary overcrowding in the hotels, trains, and trams.

In Otago, the southernmost province of the South Island, the second wave was introduced about November 6th by people returning from the Christchurch race carnival. In Dunedin the crest was reached on November 24th. Invercargill, the only town of any size south of Dunedin, was reached on November 9th.

It is evident that the epidemic took the form of a wave which spread from Auckland southwards by rail and steamer, beginning about October 26th and reaching the southernmost districts by November 9th, thus taking about a fortnight to travel through the Dominion.

As the result of a visit which I paid to the chief centres after the epidemic, I obtained the impression, as the result of conversations with medical officers, that the wave lessened in virulence as it spread South. This is borne out by the Government Statistician's returns, which show the death rate per 1000 of the population in the four main towns during 1917 and 1918. Dunedin city, which generally has the highest crude death rate on account of the age distribution of the population, and the greater severity of its climate, has this year, in which all the figures are abnormal by reason of the epidemic, the lowest rate, while that of Auckland is strikingly high.

<table>
<thead>
<tr>
<th>City</th>
<th>1917</th>
<th>1918</th>
</tr>
</thead>
<tbody>
<tr>
<td>Auckland</td>
<td>12.36</td>
<td>21.40</td>
</tr>
<tr>
<td>Wellington</td>
<td>10.05</td>
<td>16.59</td>
</tr>
<tr>
<td>Christchurch</td>
<td>12.13</td>
<td>17.15</td>
</tr>
<tr>
<td>Dunedin</td>
<td>12.23</td>
<td>16.40</td>
</tr>
</tbody>
</table>
Chart II illustrates the incidence of the second wave in which the deaths from Influenza daily during the epidemic are shown. It also indicates that the average peak wave of incidence for the whole Dominion was the week ending November 18th, the greatest number of deaths for any one day (303) being notified on November 21st.

CHART II.

Deaths from Influenza daily during the Epidemic.
From the literature available it appears that, in five or six weeks, this pandemic had covered the whole of the larger countries in the world with the exception of Australia, and that it had not followed any definite geographical course, but burst out almost simultaneously at places so far apart as Boston and Cape-town, and later in Britain and New Zealand.

Its course when once established seems to have been comparatively brief, the wave developing rapidly and receding as quickly.

Although the general appearance of the secondary pandemic did not take the form of a wave advancing from one country to another, such a wave is shown in the countries attacked. Thus in the States the spread was from Boston-West; in Africa the spread was from Capetown to Natal, and in New Zealand from North to South. It is interesting to note in these cases that the commencing point in each case was an important seaport town.
THE POSITION IN AUSTRALIA.

An experiment in Maritime Quarantine.

Although I have confined my remarks to the Dominion of New Zealand as far as possible, the position in the Commonwealth of Australia, the nearest country of any size to New Zealand, is of peculiar interest. The distance by sea is from 1200 to 1300 miles, and there is frequent steamer communication between the various ports of the two countries.

The primary wave appeared there in September and October, 1918, and apparently affected the whole of the Commonwealth; but the secondary wave did not develop in common with the rest of the world, and was delayed until the end of January, 1919, appearing first in Melbourne, where it assumed epidemic proportions early in February. New South Wales was not visited by a fatal form of the disease at this time. A wave of greater intensity, with peaks on April 29th, was experienced in both States. Moreover, it did not follow the rapid course experienced elsewhere, but lingered for several months, varying in intensity, but never reaching the acute virulence experienced in New Zealand in November 1918. New South Wales was proclaimed as infected with Pneumonic Influenza on January 26th, 1919, and Victoria the next day; but, while there was not the definite wave peak seen in this country, the period of greatest intensity was not experienced until as late as the last week in April in both these States.

The Commonwealth of Australia has a very efficient Quarantine Service, and strict quarantine measures were established in October, 1918.

Maritime quarantine of vessels arriving in Australian ports was established if anyone on board showed signs of Influenza. Shortly after, information about the South African epidemic came to hand and the restrictions were tightened. All vessels arriving from South Africa and New Zealand were at that time required to perform a seven days period of quarantine, whether infected or not. All persons detained were subjected to a daily thermometer parade and a zinc sulphate spray inhalation. On ships with cases of influenza on board the contacts were, in addition, inoculated with the Commonwealth vaccine. Dr. Cumpston states that such good results followed the application of this measure that it was considered highly desirable.
to proceed with it. The members of the staffs at the quarantine stations were compelled to wear masks.

These restrictions were reduced after January 2nd as regards South African, and after April 17th as regards New Zealand vessels.

In addition to maritime and interstate quarantine, attempts were made to protect the public by the establishment of vaccine stations for the free administrations of the mixed influenza vaccine, of public inhalation chambers, and the compulsory wearing of face masks. Australia being the only country where these precautions were so thoroughly enforced, and which at first appeared to be successful, it is impossible to avoid the conclusion that the incidence of the epidemic was altered thereby.

Discussing the result of maritime quarantine in Australia in an attempt to exclude a disease like Influenza, the Director of Quarantine admits that it was an experiment which it was freely predicted was bound to fail (7). During the operation of the quarantine from October 1918 to April 1919, 149 uninfected vessels with 7,075 passengers and 7,941 crews, as well as 79 infected vessels with 48,072 passengers, 10,454 crew and 2,795 patients were dealt with.

The failure of quarantine defence could be established by a demonstration that there had been continuity of infection from any one ship to the shore population, or that there had been an epidemic on shore during the period of quarantine operations, and that the characteristics of the epidemic were identical with those encountered on the quarantined vessels. It is claimed that no evidence has been produced of such an escape of infection, for, during the period of extreme danger no infection at all comparable to that existing in South Africa and New Zealand was introduced into Australia. It was not until January 9th, 1919, that the first cases of the Australian epidemic were recorded in Melbourne, but these were not known to the Health Authorities until some days later. On January 20th it was ascertained that between 50 and 100 cases of a disease resembling the severe form of Influenza had occurred in Melbourne, and from there the disease spread steadily until it had invaded the five mainland states.

It is therefore claimed with some reason "that the rigid enforcement of quarantine measures throughout October, November, and December, 1918,
saved Australia from the disaster suffered by both South Africa and New Zealand, and this in spite of the fact that all the quarantine stations were kept extremely busy dealing with arriving vessels infected with a disease showing no less virulence or no less fatality than was seen in these countries". In discussing the source of the Australian epidemic, Cumpston states that it began in Victoria, where there was the least amount of active infection dealt with in the quarantine station, and the administrative details were under the charge of an experienced officer.

The epidemic spread at a slower rate from State to State and from place to place in Australia than in other countries, for, while it was well established in Melbourne on January 31st, 1919, it did not become extensive in Sydney until March. Moreover, there had been a form of influenza prevalent in Australia since July 1918, exhibiting characteristics indistinguishable from those of the disease which affected the Commonwealth in 1919, but the 1918 disease had a smaller infectivity. Dr. Cumpston concludes by saying "Nothing that is at present known of this disease is in any way inconsistent with the hypothesis of the evolution of an infection already present in Australia, to a stage manifesting greater virulence and infectivity seen in the closing quarter of 1918 in every country of the world save Australia". For this escape the work of the Commonwealth quarantine service is credited.
THE INCIDENCE OF THE EPIDEMIC.

Owing to the absence of notification during the primary wave and the disorganisation during the secondary wave, it is impossible to estimate with any accuracy the proportion of the population affected during 1918, although it is probable that during the year the majority of persons of susceptible age in the Dominion were attacked.

The returns from the military camps do not offer a reliable guide in this respect for they relate to persons at the most susceptible ages. These returns (Table VII) indicate that from 30 to 40 per cent suffered in the first wave and about 50 per cent in the second wave. The incidence on the Natives was much more severe, for among the Native soldiers at the Native Military Camp practically all were affected in the second wave.

The Chief Health Officer(11) estimates that of the whole population it is probable that about 40 per cent of persons in the Dominion were attacked during the second wave. A series of observations made in Maryland, U.S.A., showed great variation in incidence, some towns being as low as 23 per cent and others as high as 59 per cent.
MORTALITY.

The Registrar-General (N.Z.) supplies the following figures of deaths directly attributable to Influenza, which were registered in the Dominion between January 1st and December 31st, 1918. (Natives excluded).

<table>
<thead>
<tr>
<th>TABLE XI.</th>
<th>Jan</th>
<th>Feb</th>
<th>March</th>
<th>Apl,</th>
<th>May,</th>
<th>June,</th>
<th>July,</th>
<th>Aug,</th>
<th>Sept,</th>
<th>Oct,</th>
<th>Nov,</th>
<th>Dec,</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>-3</td>
<td>5</td>
<td>8</td>
<td>7</td>
<td>11</td>
<td>50</td>
<td>3294</td>
<td>2177</td>
<td></td>
</tr>
</tbody>
</table>

Total for period October - December covering second wave = 5471.

Prior to the second wave it was not customary for medical men notifying death from pneumonia and such complications to indicate that the primary disease was Influenza. It is not possible then to formulate any accurate estimate of the death rate from the primary wave, although it was accompanied by an undue proportion of fatal pneumonic complications. Taking the figures in the above table for October, November, and December as the deaths during the second wave, we have a total of 5471, excluding Natives.

MORTALITY AMONG NATIVES. Owing to the difficulty of obtaining accurate returns of native deaths it is only possible to form an estimate of the death rate among Natives during the second wave. Comparing the deaths from acute catarrhal diseases registered during October - December, 1917, with those for the corresponding period, 1918, (17) we find there were 27 as against 1150, and it is safe to assume that about 1130 of these deaths were due to Influenza.

Table XII shows the total deaths of Europeans, of Natives, and the death rate per 10,000 of the population. For purposes of comparison the death rates in South Africa, and the 96 great towns in England and Wales, are included.
TABLE XII.

<table>
<thead>
<tr>
<th></th>
<th>Deaths</th>
<th>Deaths per 10,000 mean population.</th>
</tr>
</thead>
<tbody>
<tr>
<td>N. Z. Europeans.</td>
<td>5471</td>
<td>49.60</td>
</tr>
<tr>
<td>&quot; Natives.</td>
<td>1130</td>
<td>226.00</td>
</tr>
<tr>
<td>N. Z. Europeans &amp;</td>
<td>6601</td>
<td>56.15</td>
</tr>
<tr>
<td>Natives.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Union of South</td>
<td>11,726</td>
<td>82.6</td>
</tr>
<tr>
<td>Africa Europeans.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Union of South</td>
<td>127,450</td>
<td>271.9</td>
</tr>
<tr>
<td>Africa Natives.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>England &amp; Wales (96) Great Towns.</td>
<td>44,537</td>
<td>26.9</td>
</tr>
</tbody>
</table>

Reference to Table XII shows that the death rate per 10,000 of the population for New Zealand (56.15) compares very unfavourably with that for the 96 great towns in England and Wales (26.9), whereas our rate compares very favourably with that of South Africa, 82.6 (Europeans) and 271.9 (Natives).

CASE MORTALITY. It is obviously impossible to estimate the case mortality when the notification returns are so incomplete. For instance reference to Table IX shows that from November 5th, when notification was introduced, to December 31st, 1918, 4873 cases were notified, whereas during the same period there were over five thousand deaths of Europeans from this cause. In the military camps the case mortality for the second wave was 4.5 per cent, but the population of the camps was made up of males of the most susceptible age, so that the rate for the general population would be considerably less.

AGE INCIDENCE & INFLUENCE OF SEX. The Government Statistician's return (17), Table XIII, shows the European deaths occurring during the epidemic in age groups for both sexes.

The age and sex incidence is graphically represented in Chart III. (11)
<table>
<thead>
<tr>
<th>Age-group</th>
<th>Number of Deaths</th>
<th>Death-rate per 10,000 of Male Population</th>
<th>Proportion of Population of each Age-group to Total</th>
<th>Number of Deaths</th>
<th>Death-rate per 10,000 of Female Population</th>
<th>Proportion of Population of each Age-group to Total</th>
<th>Number of Deaths</th>
<th>Death-rate per 10,000 of Population</th>
<th>Proportion of Population of each Age-group to Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 5</td>
<td>117</td>
<td>16.92</td>
<td>12.81</td>
<td>117</td>
<td>17.55</td>
<td>12.84</td>
<td>234</td>
<td>17.23</td>
<td>12.31</td>
</tr>
<tr>
<td>5 and under 10</td>
<td>24</td>
<td>3.75</td>
<td>11.86</td>
<td>25</td>
<td>4.02</td>
<td>11.05</td>
<td>49</td>
<td>3.88</td>
<td>11.46</td>
</tr>
<tr>
<td>10 &quot;</td>
<td>32</td>
<td>5.61</td>
<td>10.58</td>
<td>35</td>
<td>6.33</td>
<td>9.82</td>
<td>67</td>
<td>5.96</td>
<td>10.19</td>
</tr>
<tr>
<td>15 &quot;</td>
<td>162</td>
<td>35.35</td>
<td>8.49</td>
<td>95</td>
<td>19.89</td>
<td>8.48</td>
<td>257</td>
<td>27.46</td>
<td>8.48</td>
</tr>
<tr>
<td>20 &quot;</td>
<td>295</td>
<td>110.30</td>
<td>4.96</td>
<td>193</td>
<td>36.09</td>
<td>8.55</td>
<td>488</td>
<td>65.16</td>
<td>8.79</td>
</tr>
<tr>
<td>25 &quot;</td>
<td>546</td>
<td>152.28</td>
<td>6.64</td>
<td>341</td>
<td>68.21</td>
<td>8.87</td>
<td>887</td>
<td>105.32</td>
<td>7.78</td>
</tr>
<tr>
<td>30 &quot;</td>
<td>678</td>
<td>163.69</td>
<td>7.67</td>
<td>332</td>
<td>67.80</td>
<td>8.67</td>
<td>1,010</td>
<td>111.92</td>
<td>8.18</td>
</tr>
<tr>
<td>35 &quot;</td>
<td>602</td>
<td>144.57</td>
<td>7.72</td>
<td>220</td>
<td>48.22</td>
<td>8.10</td>
<td>822</td>
<td>94.20</td>
<td>7.92</td>
</tr>
<tr>
<td>40 &quot;</td>
<td>390</td>
<td>117.03</td>
<td>6.17</td>
<td>152</td>
<td>43.59</td>
<td>6.19</td>
<td>542</td>
<td>79.48</td>
<td>6.18</td>
</tr>
<tr>
<td>45 &quot;</td>
<td>252</td>
<td>74.36</td>
<td>6.28</td>
<td>111</td>
<td>36.66</td>
<td>5.97</td>
<td>262</td>
<td>56.11</td>
<td>5.56</td>
</tr>
<tr>
<td>50 &quot;</td>
<td>142</td>
<td>57.54</td>
<td>4.57</td>
<td>116</td>
<td>53.44</td>
<td>5.65</td>
<td>258</td>
<td>55.62</td>
<td>4.21</td>
</tr>
<tr>
<td>55 &quot;</td>
<td>72</td>
<td>37.47</td>
<td>3.56</td>
<td>62</td>
<td>36.52</td>
<td>2.86</td>
<td>134</td>
<td>37.95</td>
<td>3.20</td>
</tr>
<tr>
<td>60 &quot;</td>
<td>56</td>
<td>33.85</td>
<td>3.07</td>
<td>47</td>
<td>36.30</td>
<td>2.50</td>
<td>103</td>
<td>34.91</td>
<td>2.67</td>
</tr>
<tr>
<td>65 &quot;</td>
<td>44</td>
<td>38.23</td>
<td>2.13</td>
<td>35</td>
<td>35.44</td>
<td>1.75</td>
<td>79</td>
<td>36.94</td>
<td>1.94</td>
</tr>
<tr>
<td>70 &quot;</td>
<td>45</td>
<td>52.66</td>
<td>1.58</td>
<td>29</td>
<td>41.09</td>
<td>1.25</td>
<td>74</td>
<td>47.43</td>
<td>1.41</td>
</tr>
<tr>
<td>75 &quot;</td>
<td>30</td>
<td>48.38</td>
<td>1.15</td>
<td>30</td>
<td>64.60</td>
<td>0.82</td>
<td>60</td>
<td>55.33</td>
<td>0.98</td>
</tr>
<tr>
<td>80 &quot;</td>
<td>10</td>
<td>33.69</td>
<td>0.55</td>
<td>16</td>
<td>78.20</td>
<td>0.36</td>
<td>26</td>
<td>51.04</td>
<td>0.45</td>
</tr>
<tr>
<td>85 &quot;</td>
<td>7</td>
<td>74.47</td>
<td>0.17</td>
<td>7</td>
<td>94.85</td>
<td>0.13</td>
<td>14</td>
<td>83.43</td>
<td>0.15</td>
</tr>
<tr>
<td>90 &quot;</td>
<td>1</td>
<td>67.57</td>
<td>0.03</td>
<td>...</td>
<td>...</td>
<td>0.03</td>
<td>1</td>
<td>32.26</td>
<td>0.03</td>
</tr>
<tr>
<td>95 and over</td>
<td>1</td>
<td>333.33</td>
<td>0.01</td>
<td>2</td>
<td>500.00</td>
<td>0.01</td>
<td>3</td>
<td>428.57</td>
<td>0.01</td>
</tr>
<tr>
<td>Totals</td>
<td>3,506</td>
<td>64.96</td>
<td>100.00</td>
<td>1,965</td>
<td>34.88</td>
<td>100.00</td>
<td>5,471</td>
<td>49.60</td>
<td>100.00</td>
</tr>
</tbody>
</table>
Chart 111. Showing Death-rates per 10000 of the Population in Age-Groups.

AGE. From Table XIII and Chart III it will be seen that if the three deaths over 95 years be excluded, 2719 deaths out of a total of 5471, or almost half, occurred among persons between the ages of twenty-five and
forty, although only 24 per cent of the population are between these ages.

Under 20 years the death rate is low especially between the ages 5 to 15; it then rises; reaching its maximum in the 30-35 year age group, and then gradually declines until the 85 - 90 age group, when there is a sharp rise, but the figures among the aged are too small to yield reliable data.

The death rate according to age is not an indication of the case incidence. Making an analysis of 13,000 cases of Influenza which occurred in Maryland, U.S.A., during the epidemic, the United States Public Health report (20) says in this respect "While the incidence rate is highest among children of 5 - 14 and drops off in the older ages, the death rate is quite low among children of these ages, and is markedly high among adults of 24 - 44 and among adults of advanced ages". Table XIII shows that this applies equally to the New Zealand epidemic.

Discussing the age incidence of fatal pneumonic Influenza, Vaughan(21) offers a plausible explanation:- From a very extensive personal experience of epidemics in the American Army, he has arrived at the conclusion that young people are not more susceptible than elderly people, but are merely more frequently exposed to infection. He regards Influenza as a disease of activity.

Fischer(22) suggests that it is due to the sudden liberation of endotoxins in enormous quantities following the wholesale destruction of bacteria, caused by the powerful defences of the organism, whereas in less vigorous individuals bacteriolysis, with liberation of endotoxins, takes place more gradually.

SEX. Table XIII shows that Influenza was a more fatal disease among males than females, the death rate per 10,000 among the former being 64.96 as against 34.88 among the latter. This is clearly shown in the accompanying chart. The main incidence is still on the age groups 20 - 40, but there is a further sharp rise in the female rate between the ages of 50 - 55 dropping to the normal curve between 55 - 60. This may be a chance variant. It is rather late to be associated with changes in connection with the menopause in women.

INFLUENCE OF RACE. Both in New Zealand and South Africa the incidence and mortality was much more marked in the case of Natives than in the European population. Reference to Table XII shows that the deaths
per 10,000 in the European population were 49.60 and in the Native population 226.0. The reports of the spread of the epidemic in the Pacific also show that the Natives were exceptionally liable to attack by Influenza in the more fatal forms.

INCUBATION PERIOD & DURATION OF INFECTIVITY. It is, I believe, generally accepted that the incubation period of epidemic influenza is 48 hours or less and that cases are most infective in the earlier stages. As far as our New Zealand experience goes it is in conformity with these facts. No cases of infection amongst the attendants at the temporary convalescent hospitals are recorded but by the time these were in use most of those in attendance were probably either naturally immune or had recovered from a mild attack.

CONVEYANCE OF INFECTION FROM THE SICK TO THE HEALTHY. Until there is more definite proof whether the influenza bacillus is the primary infective agent, or whether it is, like the pneumococcus, an organism secondary to a filter passing virus, we cannot be absolutely certain that infection is always carried by the secretions of the respiratory surfaces.

Little evidence to the contrary has come to light as the result of our experience here however, except an interesting observation by Dr. MacNaughton of Samoa, who reported that a Native living in a group of islands in the Pacific developed pneumonia shortly after receiving a long letter from a native friend in hospital at the Native Military Camp in New Zealand. No other possible source of infection could be traced and this was the only case on the island.

Parsons(23) who studied the incidence of the disease in several thousand deep see fishermen and on four hundred offshore lighthouse keepers found no evidence that the disease developed except as a result of contact with the sick, or within a few days of contact with persons on shore.
PART II.

Administrative and Other Measures Adopted to Combat the Epidemic - General administration.

In using the term epidemic in the following pages I refer to the second wave of the pandemic covering the period October 26th - December 31st, 1918. Administrative measures can hardly be said to come within the scope of Epidemiology or Bacteriology except where special administrative measures, such as the prophylactic use of vaccines, are concerned.

Moreover the administrative measures which experience taught us to adopt were the result not of the experience of others who had recently passed through such an epidemic, and whose published records we might have studied, for up to the time of the outbreak the available recent literature contained little to guide us. Even the particulars of the South African outbreak, which preceded ours by some weeks, had not come to hand except in fragmentary cables. Some particulars and advice which the Government Bacteriologist at Cape Town forwarded me at the time did not come to hand until several weeks after affairs had again become normal in the Dominion. Certainly our mails were few and far between, shipping that found its way out here was urgently required for troops and for food stuffs for the Home Land, and we were more than ever cut off from our usual sources of information.

It seems strange now that the event is over that we in the South were not more impressed with the seriousness of the position in the North when the second wave broke out there. I well remember receiving a telegram from the Acting-Chief Health Officer, who had gone to Auckland when the position became serious, to the effect that the newspaper reports in no wise minimised the serious position of affairs. I remember we regarded that telegram at the time as emanating from one who was harassed by the public panic as reflected in the public press. We were soon to learn our lesson, although we had taken precautionary measures, and had had some time to consider a plan of action. I propose therefore to briefly mention the administrative measures adopted in the province of Otago with which I am more intimately connected, and to discuss at greater length certain prophylactic
measures with which a bacteriological department is more concerned, or upon which my opinion was sought by the Central Authorities.

Public Health Administration in New Zealand.
This is vested in a Minister of the Crown, with whom is associated the Chief Health Officer for the Dominion. At the time of the Epidemic there was stationed in each of the four main towns a Medical Officer of Health, known as a District Health Officer, who administered the Public Health Acts under the Chief Health Officer, in the town and province of which it was the principal centre. Each District Health Officer had several Inspectors and the necessary clerical staff.

The Public Health Act 1908 and amendments defines infectious diseases as "Dangerous Infectious Disease" and "Infectious Disease" respectively. The Governor-General by proclamation may define what shall be considered a dangerous infectious disease, and at the time Cholera, Plague and Smallpox had been so proclaimed. Other infectious diseases are made notifiable from time to time, and the list of these is much the same here as elsewhere.

The Medical Officer of Health has limited powers in dealing with the ordinary infectious diseases, but very wide and drastic powers in dealing with a dangerous infectious disease. Early in the epidemic, "Pneumonic Influenza" was proclaimed a dangerous infectious disease within the meaning of the Public Health Act 1908, and Medical Officers of Health had practically a free hand in their districts to do as they thought best for the public good.

With these powers the Medical Officers of Health administered their districts during the epidemic somewhat as follows -

Medical Officer of Health and Staff - General administration.
Central Medical Bureau - Doctors, nurses, medical students, lay helpers.

Hospitals & Auxiliary Hospitals.
Transport -
Supplies -
Educational
Prohibition of public gatherings, closing public establishments etc.
The Work of the Medical Officer of Health and his Staff. The Medical Officer of Health was in supreme control of all administrative measures and his time was very fully occupied keeping in touch with the central administration, issuing instructions, consulting with the heads of the various voluntary organisations, and with administration generally.

Medical & Nursing Aid. Owing to the fact that over one third of the medical profession were overseas on military service, and the remainder very fully occupied with the ordinary civilian needs, scattered as they are in a sparsely populated country such as this, the epidemic came at a most unfortunate time. It was soon realised that even in the towns the medical men available were wholly inadequate to cope with requirements. Of their already depleted ranks, many were early stricken down, thus further reducing their numbers. To economise and prevent overlapping, a central medical bureau was opened in the centre of the town and subsidiary bureaux in thickly populated suburbs. Special telephones connecting these to all parts of the town were installed and a staff of medical men, nurses, and medical students enrolled to work in rotation at the bureau. Usually one or two qualified men were constantly in attendance day and night to answer enquiries, to advise patients over the telephone who could not get medical attention, and to advise nurses and others who were assisting. Many visits were thus saved.

Many of our nurses were on service overseas and there was a pronounced shortage on this account. The hospital nursing staffs were also sadly depleted on account of sickness. To each bureau one or two nurses were attached, either those in active practice or ladies who had a nursing qualification and who offered their services. Three senior medical students were also attached in relays of 12 hours duty each, and they attended to many cases in conjunction with the nursing staff when it was impossible to send a qualified man. The town and outlying districts were divided into blocks according to population and distance to be travelled, and most of the medical men gave up their routine practices and were allotted to particular blocks, so that all requests for assistance in each block were sent on from the bureau to the doctor detailed for that block.
Lay Assistance. A sub-bureau was formed in connection with the central bureau and placed in charge of a reverend gentleman who had devoted himself heart and soul to the cause of the people. There all voluntary workers, other than medical, were enrolled, and the self sacrifice of those noble men, women, girls and boys, who gave their time, and some their lives to such a cause, is beyond all praise. Men assisted with the heavier work and even helped to dig graves when the gravediggers were unable to cope with requirements; women cooked meals, and undertook other work in the homes of the stricken, while girls and boys ran messages and carried medicine and medical comforts. In connection with the Central bureau a crèche was established, and all children whose parents were stricken, and who could not otherwise be attended to, were accommodated therein. Kitchens were also established at the Bureaux where suitable articles of diet were prepared by lady volunteers and distributed as required.

Hospitals. The resources of the general hospital were soon taxed to the uttermost and all patients, surgical and otherwise, who could possibly be moved were sent to their homes to make room for the epidemic patients. The wards quickly filled and one was struck each morning with the large proportion of new faces, many of those present the previous day having no further need for earthly assistance. As the general hospital became congested, two large Sunday School halls in the immediate vicinity were secured and fitted up by the Red Cross and Ambulance Society for the reception of patients. These soon filled, but by this time the force of the epidemic was spent, and although other buildings had been prepared in readiness, they were not required.

One important lesson learned at this time was the harm which patients suffered by being moved. It had been the practice to admit cases to one ward and to transfer them the following day to the "Dangerously ill" wards, or to the general wards according to their condition. It soon became evident that the mere moving of a patient from one ward to another often did great harm, and henceforth patients were moved as little as possible, and only sent on to the convalescent hospitals when they were out of danger.

The staffing of the hospitals, both medical and nursing, became very difficult. Several of the Honorary and Resident Staffs were ill and at least two-thirds of the Nursing Staff likewise. Moreover, several of the medical and nursing staffs were away on
military duty.

It was at this stage that the Medical School again demonstrated its importance as a National Institution. Classes were suspended, and Professors, Lecturers, and the whole of the Students, male and female, offered their services to the community through the Dean. As Sub-Dean it devolved on me to organise and distribute the students throughout the Dominion where their services were most required, and to recall and redistribute as occasion warranted it. Of the final year class of thirty-five, most were already filling posts as House-Surgeons throughout the hospitals of the Dominion as a war measure of necessity. The Fourth year class of thirty-eight, including thirteen girls, was the only source of supply for outlying districts, as we could not send third year students out unless under supervision. Of the fourth year men, 6 were sent to each of the Medical Officers of Health in the three chief towns, to be distributed throughout their districts. Several fell ill en route and had to be replaced, and it was with considerable anxiety that I followed, as well as I was able, their progress in various outlying portions of the Dominion. Fortunately all recovered and returned safely. The remainder were distributed between the local hospitals, the central bureau, and the townships throughout Otago where either there was no medical assistance or the medical practitioner was himself ill. Many frantic appeals arrived daily from Mayors and other leading citizens pleading for the services of a student. To the credit of these boys, and to the honour of their Alma Mater, one and all rendered noble service, and when affairs were again normal it was gratifying, if not amusing, to find our students returning with illuminated addresses bearing corporation seals, or with presentations of plate or gold watches. The girls were employed locally in the hospitals and at the central bureau, and whatever one's views may have been in the past as to women medical students, the gallant work of these girls, their devotion to duty to the point of exhaustion, won the admiration of all. They were often on duty for 18 hours at a stretch under most distressing circumstances, acting as medical officers, replacing nurses, or attending patients in the town.

The junior Students, numbering about 200, practically staffed the local hospitals in shifts of 8 hours each, and acted as nurses, porters, messengers, or in any capacity required.
Transport. In addition to the official ambulances, several business firms converted their motor delivery vans into ambulances, and the Motor Club under a member of the Committee, placed several dozen motor cars, with the owners, at the disposal of the central bureau. By this means doctors, nurses, and students were able to visit a large number of cases in the minimum of time, and supplies were collected and despatched with celerity.

Supplies. Fortunately the Armistice had been signed, and the Minister of Defence placed the Army Medical stores at the disposal of the Health Department. The St. John's Ambulance and the Red Cross Society likewise provided equipment, so that the task of suddenly equipping a building with 50 or 100 beds, and providing the necessary medicines and medical comforts was very much facilitated. Public kitchens were also established in connection with the Bureaux, from which soup, jellies, and other articles of light diet were despatched to afflicted houses by means of the motor service.

Educational. Circulars similar to those used in Manchester, and on the lines of the Local Government Board memorandum, February 1919, were posted in prominent places, and the columns of the press freely utilised to allay panic and to advise the public what best to do. Each day the Press, after consultation with the Medical Officer of Health and Medical Officer in charge of the Bureau, issued bulletins of a cheering and helpful nature, so that the public was fully advised as to their best course of action, how to get help, and what to do when ill. That some action of this sort was very necessary became evident, for panic seized the people and it appeared certain that a proportion of the fatal results were the result of fright.

Prohibition of Public Meetings. Exercising his powers under the Public Health Act (1908), the Medical Officer of Health prohibited public meetings of all kinds, and theatres, picture shows, and other places of public amusement, public and private schools, tea rooms, public bars and restaurants were closed.

Church services were not immediately prohibited, but the heads of the various denominations were strongly advised as a matter of precaution, and as an example to the public, to hold no public services while the epidemic lasted. Some difficulty was
experienced in this respect, and while some churches fell in with the wishes of the Department, others ignored them. The local Medical Officer of Health, falling ill himself in the middle of the epidemic, I was asked by the Chief Health Officer to assume control. Matters were at their worst, although the administration was running as smoothly as could be expected, but it was a cause for regret with me that it took the best part of a busy afternoon to get all the Churches into line. While the great majority readily acquiesced, a few held that nothing should interfere with Divine service, and others agreed to our proposals provided a definite instruction was issued so that all would be brought into line. The threat of a public order was, however, sufficient, and fortunately it was possible within a few weeks to revoke many of the restrictions placed on the public.

**Provision for a return of the Epidemic.**
As soon as matters were normal again, the Medical Officer of Health called together the various committees and others who had assisted, and in the light of the experience gained, a complete organisation was set up to provide against a return of the epidemic. Everything is in readiness so that at a few hours notice the administrative machinery can be set in operation. Fortunately nearly a year has passed and it has not been necessary to utilise this wise provision.

**The Aftermath of the Epidemic.** As already mentioned, the mail steamer "Niagara" was popularly blamed for the advent of the second wave, and it was freely stated that had the Prime Minister and Minister of Finance not been passengers, this steamer would have been quarantined by the Minister of Public Health. The latter being dragged into the controversy, which became at times very acrid, decided to set up a Royal Commission consisting of an ex-Judge of the Supreme Court and two business men, to go into the whole question. The Commission took much evidence in the four main towns and issued a report apportioning the blame and making various recommendations. Many of the recommendations were good and have been given effect to, but there was a good deal of contentious matter contained therein, and, as was to be expected, the Commissioners failed to grasp the epidemiological facts laid before them. It was unfortunate that political considerations clouded the matter and that public money was largely wasted.
Parliament has since voted £100,000 to the relief of epidemic widows and orphans and for other expenses, but it is estimated that £200,000 will be necessary before all claims are met.
SPECIAL ADMINISTRATIVE MEASURES.

PROPHYLAXIS.

The Prophylactic Use of Medicated Sprays and Inhalations. The use of medicated sprays and inhalations for the prophylactic treatment of large bodies of men was employed on a considerable scale during the late war, particularly in connection with meningococcus carriers. The two drugs which have been extensively used in this respect are Chloramine T., and Zinc Sulphate, in one to two per cent solutions, and the consensus of opinion was favourable to their use.

Similar sprays have been in use in various military camps, and especially on board troopships, to control and combat catarrhs of the respiratory tract, and according to our New Zealand experience they have been more or less successful.

Epidemic Influenza offers a less favourable sphere for prophylactic medication, but nevertheless it has been given a fairly extensive trial with, as I shall show, very doubtful results.

The prophylactic treatment generally employed in combatting influenza epidemics was to expose patients in so-called inhalation chambers, in which Levick or other spray, and Zinc Sulphate in a 1% to 2% solution was employed. The authorities to whom I have access seem divided in their opinion as to the value of this method. Most have used the Zinc Sulphate spray with a steam jet as the vaporising agent, probably because the apparatus was already available, and Zinc Sulphate and Chloramine T. had been found useful in other directions. But because these drugs have been successful in dealing with the Meningococcus, it does not follow that the Influenza virus and the Pneumococcus and Streptococcus, which usually complicated the primary influenza infection, will be similarly susceptible to their action. My own experience with both these drugs in dealing with meningococcus carriers in soldiers and civilians was on the whole favourable, but not so when diphtheria carriers were similarly dealt with. During 1917-1918 all our diphtheria convalescents who failed to give two consecutive negative swabs after they were clinically recovered, were treated in an inhalation chamber with one or other of these drugs. About 100 cases were treated thus, but I cannot attribute to it any beneficial
effect in hastening the clearing of the throat or nasopharynx of diphtheria bacilli. Several cases persisted as carriers for from four to six months and were only relieved when other methods, such as the enucleation of the tonsils, were tried. One experiment which I made in conjunction with my colleague Dr. Hughes, Medical Officer of Health, Dunedin, was in connection with a public school in which Diphtheria had broken out amongst the scholars. Three hundred contacts from the affected classes were given 10 minutes exposure in the Zinc Sulphate inhalation chamber on two occasions at 24 hours interval. Throat swabs were taken 12 hours after the last inhalation, and from six of the cultures growths of diphtheria bacilli were obtained. All the children were passed through the chamber a third time and swabs again taken, and 10 diphtheria bacillus carriers were found. So with other schools, I have found little beneficial effect follow the use of Zinc Sulphate inhalation in dealing with diphtheria carriers.

In influenza prophylaxis the use of a suitable drug might have some value, but that drug has yet to be discovered or applied. The invasion is so rapid that unless the treatment is repeated very frequently and soon after exposure to infection, the value of inhalation is reduced.

I find two opinions recorded in favour of this method of prophylaxis, both in connection with New Zealand troops. Thus the Chief Health Officer(11) reports that the opinion of the Principal Medical Officers in the Military Camps in New Zealand during the second influenza wave was that as soon as the inhalations were regularly and frequently applied, a marked reduction in pneumococcal complications resulted. On the troopships, infections of a catarrhal nature greatly lessened after the chambers were installed and used systematically. No figures are given in support of these opinions however. The Chief Health Officer adds that the value of such treatment can only be appreciated when it is available to the occupants of an institution, where it can be applied regularly and perhaps compulsorily twice a day.

Fyre & Lowe(25) examining the reports of the Principal Medical Officers of the New Zealand Military camps in the United Kingdom during the Autumn epidemic, make an important distinction between the class of cases which should be exposed to inhalation treatment. In one camp every case of mild catarrh was regarded as an infective focus and treated with
Zinc Sulphate sprays and antiseptic gargles, but the healthy non-catarrhal soldiers were not so treated and the incidence of influenza was only 3% of the strength.

In another camp, where in addition to vaccinal prophylaxis, all men, both healthy and presumably mild catarrhs were sprayed, many of the previously apparently healthy (but possibly quiescent carriers) having had the mucous membranes irritated by the spray and the surface cells devitalised, became active infections, so that the final incidence rate was 17.2%.

In a third camp where no prophylaxis at all was attempted, the incidence was 36%.

Eyre & Lowe emphasise that no attempt should be made to lower the vitality and resisting power of Nature's first line of defence - the surface cells of the mucous membrane of the upper respiratory tract - but they apparently approve of the spray as a prophylactic measure when mild catarrh exists. In this opinion they differ from Leonard Hill, whose work is reviewed later. The experience of Cumpston(28) is unfavourable, and he considers that inhalation with Zinc Sulphate is of no value for the control of an epidemic in full swing.

The case for the inhalation chamber receives universal condemnation when applied to the general public. In New Zealand, public chambers were set up on the wharves, at railway stations, and in almost every town and township throughout the country. They were usually the cause of crowding, and in our experience the difficulty of preventing chills when people passed from the warm atmosphere to the cool outside air, counteracted any beneficial effect that might have been derived. In Christchurch the Zinc Sulphate spray was generated by compressed air jets and this objection overcome.

I am indebted to Dr. Chesson, Medical Officer of Health, Canterbury district, for the following details of the compressed air sprays as arranged in his district during the epidemic.

"The plan we adopted here was to have the "compressor working outside the inhalation chamber. A "long pipe ran from this along one side of the chamber "at a convenient height, with a number of short branch "pipes, each fitted with a tap. These branch pipes
were each fitted to spraying nozzles dipping into a
"Winchester containing the Zinc Sulphate solution.
The apparatus could be arranged for any number of
"spray nozzles, any or all of which could be working
"as required. The chambers were well ventilated and
"there was no unpleasant stuffy feeling in them.

"Persons to be treated entered at one end of
"the chamber, each opposite a nozzle and about seven
"feet distant from it. The distance was maintained by
"means of a barrier in front of which each person
"stood. The subjects passed through the chamber con-
tinuously, each one remained for about 5 minutes in
"the fine spray, and breathed deeply through the nose
"and open mouth. On the wharf, two railway carriages
"were fitted up in a similar manner, and driven from
"the compressor of a locomotive engine.

"In the suburbs, tram cars were used as
"chambers, the compressed air being obtained from the
"brake compressors.

"In country districts, compressors were
"driven by motors of various kinds."

Dr. Chesson claims the following advantages
for this system -

1. The compressed air is obtained from
outside the compartment and therefore clean fresh air
is continually being forced in.

2. There is no heat generated and the
atmosphere is always sweet.

3. The subject does not get overheated and
therefore is not subject to chill on leaving the
chamber.

4. The spray operating continuously, the
chamber is able to be efficiently ventilated, and
crowding of waiting people can be to a large extent
avoided.

Dr. Chesson concludes his letter by saying
that he is rather sceptical as to the value of whole-
sale spraying owing to the bringing together of
people who would not otherwise come in close contact.
He favours individual spraying however.

Well ventilated waiting rooms should be
provided and people admitted to the chamber only in
small numbers at a time, and the steam jet should be
replaced by the compressed air jet if public inhal-
ation chambers are to be recommended at all.
The Influenza bulletin(27) issued by the Editorial Committee of the American Public Health Association emphasises the futility of sprays and gargles, which instead of protecting the nose and throat from infection are held to tend to remove the protective mucus, to spread infection, and to increase the chance of infecting organisms gaining an entrance.

To the harassed Medical Officer of Health in times of public panic such as existed in New Zealand at the height of the epidemic, the inhalation chamber came as a veritable boon. The moral effect was remarkable, until the possible risks became known, for there was positive evidence that something was being done to protect the public, and the fitting up of any sort of inhalation chamber in the outlying townships and villages did much to allay the anxiety of the residents.

Although opinion is generally against the value of the inhalation chambers, as used, in checking the spread of Influenza, the recent work of Hill(28) seems to show that provided a suitable reagent is employed, and conditions of excessive heat and humidity are avoided, there is a pouring out of fluid from the respiratory tract, whose function is to dilute and wash away a chemical irritant and so wash and cleanse the part. Hill points out the important influence which cool air has in bringing more arterial blood to the respiratory membranes, and increasing evaporation from, and therefore flow of tissue lymph through it, whereas warm moist atmospheres are against this natural washing and immunising defence.

Hill mentions the fact (pp. 149) that only three per cent of the employees of the London Electric Railway Company, who worked in rooms ventilated by ozonised air, were absent due to Influenza in the 1918 epidemic, and 10 per cent of those who worked in rooms not so ventilated. Ozone, as other irritant gas poisons, excites exudation, and Hill says it is possible that the traces of ozone added to the air may, by stimulating transudation, keep off infection. The recent papers of Shufflebotham(29) and Gregor(30) show that the incidence of the recent influenza epidemic on workers in various poison gas factories, in gas works, and in a tin mine where SO2 fumes were discharged, was very much less than on those living under similar conditions but not exposed to fumes. Exceptions were noted in the case of those engaged in the production of phosgene gas, and tri-nitro-toluene.
Hill claims(31) that all these agencies protect by reason of their irritating action on the respiratory mucous membrane, which is washed and cleansed by the drawing out of lymph. Apparently he considers the result is the effect of the mechanical rather than bacteriicidal action.

To combat the influenza infection, he recommends the breathing of cool air, and as an adjunct any spray, gargle, or snuff which enhances the outflow of secretion from the respiratory membrane of the nose and throat, and suggests(28) that Sodium bisulphite might be given a trial as a smelling salt. It is said to give off SO₂ and a few minutes inhalation night and morning might suffice to prevent infection.

I am interested in these reports by Hill, Shufflebotham, and Gregor because some weeks after the New Zealand epidemic I received a letter from the manager of a fellmongery in the North Otago district to the effect that the workers who were exposed to the fumes of the sulphuric acid in one portion of the works, had not suffered from the prevailing epidemic, whereas others in the same works, but not exposed to these fumes, had suffered in common with the rest of the community. At the time I was not inclined to attach much importance to the statement, as the number of workers exposed was too small upon which to base any opinion, but when I subsequently read the experience of others based on larger groups of workers my opinions were modified.

During the later stages of the New Zealand epidemic, when the value of the Zinc Sulphate inhalation chambers was being called in question by members of the medical profession, I conducted a simple experiment to determine the germicidal action of solutions of Zinc Sulphate on the epidemic pneumococcus, which was at that time being found in the lung tissues and oedema fluid in almost pure culture. The experiment was arranged after the Rideal-Walker method for testing disinfectants, and emulsions of cultures of the pneumococcus on blood agar, once removed, were tested in varying strengths of Zinc Sulphate solution from ½% to 3%. It was found that the solutions of Zinc Sulphate which could be tolerated, i.e. 1% to 2%, by patients, had little if any germicidal action on the pneumococcus up to 20 minutes contact. I considered that the same organism when protected by mucus and the nasopharyngeal secretions, would not be affected to any extent by the 10 minutes exposure which patients underwent in the inhalation chambers.
The case for medicated sprays seems then to require careful reconsideration. The practice of crowding those to be treated in small rooms and exposing them to fumes or chemical sprays in a steaming, humid atmosphere is wrong, and any good derived from chemical action, whether mechanical or bactericidal, is more than counterbalanced by the crowding together and the humid atmosphere, leading to cross infection and diminished vitality of the mucus membranes. We must in the future endeavour to avoid these errors if we are to use public inhalation chambers at all, by providing room to "space out" the subjects, and after selecting a suitable chemical (or irritant, if we agree that the irritant action is the beneficial one) vaporise it by the use of a cool jet of compressed air. We should thus secure the moral effect of an inhalation chamber with few of its defects.

Since writing this study, the work of Dr. Gregor, British Medical Journal, November 1st, 1919, has just come to my notice - the journals for that date having just arrived in New Zealand. Dr. Gregor finds that air charged with these gases is distinctly bactericidal and such gas impregnated air, when inhaled, has the effect of rendering the secretion of the upper air passages acid. Such an acid medium is unfavourable to the growth of the majority of pathogenic microorganisms, and Dr. Gregor is continuing his promising investigation under the auspices of the Medical Research Committee.
The utility of Face Masks in preventing infection. It is generally accepted that respiratory catarrhs are for the most part conveyed from the sick, or from the healthy carrier, to the healthy, by means of nasal or oral droplet infection. Fomites may under certain circumstances be involved, but the great mass of infection is as indicated. Shackelton records an outbreak of "colds" amongst his men in the Antarctic after opening parcels of clothing which had arrived from Home. The men had previously been singularly free from colds, but while there is the possibility of the infective organisms having survived in particles of mucus, there is the probability of infection from carriers on the relief ship.

Attention has been drawn to the value of the face mask during the influenza epidemic as a means of protecting the healthy from infection, and numerous investigations have been made to ascertain the value of masks under experimental conditions.

While there is no doubt that a properly constructed mask intercepts a large proportion of the microorganisms which would otherwise enter the mouth or nose of the wearer, the fact must not be lost sight of, that more than one authority has directed attention to the possibility of infection with the influenza virus through the conjunctiva, while some go so far as to blame the hands as the principal means of conveying infection. Thus Vincent & Lochon\(^{32}\) state that since a person may become infected by receiving the organisms on the conjunctiva or the mucus membrane of the nose or throat, a mask which covers only the nose or mouth is not efficient. They recommend a mask which surrounds the head and prevents the gauze from touching the face.

Vaughan\(^{33}\) speaking at the Annual meeting of the American Public Health Association in Chicago in December, 1918, expressed the opinion that the most frequent way in which respiratory diseases are conveyed from one to the other is from hand to mouth, and he attaches more importance to this than to droplet infection.

A mask to be efficacious must be of sufficient size to cover the mouth and nose at least, and as air currents conveying droplet infection may pass under the mask it must be sufficiently pliable to mould itself to the contours of the face. The masks generally recommend-
ed consist of from 4 to 8 layers of fine mesh gauze, sewn together at the edges and measuring about 5 x 4 inches. To each corner tapes are attached for tying behind the head, or looping round the ears. Weaver(34) recommends three layers of gauze with a mesh of 40 threads or more. Vincent and Lochon, already quoted, found that at least five layers were necessary to prevent penetration of microorganisms through the mask. Leete(35) found butter muslin more satisfactory than surgical gauze, and recommends at least four layers.

Llewellyn(36) conducted a series of ingenious experiments in which air laden with vaporised pneumococcus vaccine containing 1000 million per c.m. was pumped with a bellows through an apparatus, under conditions in certain respects approximating to those of natural respiration. When the bacteria laden vapour was aspirated through four layers of gauze (butter muslin was used) there was a stoppage of 46% of organisms; six layers effected a stoppage of 60%, and eight layers of 68% of organisms. Llewellyn explains the comparatively slight increase in the stoppage effected by eight as compared with six layers of gauze, as partly due to the extremely small size of the droplets which would reach the seventh or eighth layers. He assumes that if everyone were to wear a mask of four layers, the dangerous droplets would have to pass through eight layers of gauze, and a wide air space, in order to reach the recipient. At the time he published his experiment, the Australian Government had made the wearing of masks in public compulsory, or at least they attempted to do so.

Sahli, discussing experiments carried out at his suggestion by Lauterbure(37) in which a nebulised watery spray of B. Prodigiosus was found to pass freely through the mask used in the experiments, agrees that the mask may serve some purpose by holding back the coarser particles of excretion, but he holds with the Flugge school that these visible pellets play a role subordinate to that of the infected droplets which may remain suspended for hours, and be carried direct into the lung alveoli.

One can agree with Sahli from one's personal experience and feelings when wearing masks in the presence of patients, that the appearance of a masked attendant must arouse a feeling akin to fear in the mind of the patient.
Simon(38) is a strong advocate of the gauze mask in preventing infection from patients suffering from pneumonia, diphtheria and influenza, and he considers all those in attendance on such patients should be compelled to wear masks, not only for their own protection but in order to prevent the possibility of their becoming carriers. Chauffard, Netter, Vincent and Bezangon(39) recommended that masks should be worn by the medical and nursing staffs during influenza epidemics, and by the patients themselves when they begin to get up.

Newsholme(8) while doubting whether persons affected with catarrh will in the future submit to wear masks, suggests that nurses of influenza patients should adopt this precaution. Vaughan(33), as already pointed out, is of opinion that we have over-estimated the value of the mask. When, however, he visited one of the American soldier camps during the influenza epidemic where the mask was not in use, he addressed the medical officers as follows "Shall we use the mask or not? It is not compulsory. But every doctor who attended cases of pneumonic plague (place not mentioned) and did not wear a mask, died from it, and every man who cared for pneumonic plague cases and did not wear a mask contracted the disease. You can do as you please."

Certain American States and the Australian Commonwealth Government attempted to make the wearing of masks compulsory during the influenza epidemic, with moderate success only. In Australia a vigorous press campaign was waged for and against the measure, and one of the "antis", a well known medical practitioner, was fined for appearing in public without a mask. The attempt was eventually given up, and the sequel was an action by the practitioner mentioned in which he secured £150 damages for wrongful arrest.

The objections brought forward against the wearing of masks apply especially to their use by the public. Since we are not yet certain whether the infective agent is a filter passing virus or not, it is not possible to ascertain whether four or more layers of gauze are efficacious. It has been suggested that the saturation of the inner layers of the mask for some hours by the moisture of the breath may tend to catch the majority of the microorganisms in the inner layers thereof, and that if the mask be reapplied before their death the wearer may get a sudden mass infection. That this is by no means an unreal danger must be apparent to anyone who has experienced the difficulties and discomforts attendant on the wearing of masks during hospital work.
Hill(31) voices the feelings of such when he says that the wearing of masks by raising the temperature and humidity of the air breathed, is against the natural defensive mechanism.

Our experience with masks in the New Zealand epidemic was limited. Where they were made available in hospitals and medical bureaux they were not generally adopted. They were uncomfortable, especially if worn for any length of time, and the frequent putting on and off of the same mask without facilities for sterilising it was, as I have pointed out, undesirable. Attempts to sterilise one's mask by placing it when not in use in pocket containers with cottonwool impregnated with volatile disinfectants, such as formalin, rendered the mask too irritating to wear near the conjunctiva. Finally I adopted the plan of providing a large number of masks for members of the staff whose duties brought them into contact with patients, and a fresh mask was used each time - the used mask being placed in a container with formalin and subsequently sterilised in the hot air oven ready for future use.

It is certain that no measure making the wearing of masks in the public streets compulsory would be successful, even if tolerated, although it might be possible to enforce their use in trains, trams, and other public conveyances. The moral effect on the wearer must not be lost sight of and lay helpers would probably derive some comfort from their use when brought into contact with patients.

The masks then should be of at least 4 layers of fine gauze and of sufficient size to cover the nose and mouth and reach to just below the eyes. Their use by doctors, nurses, and attendants in hospitals, ambulances, or when attending the sick in their homes, should be encouraged, and means should be provided for issuing a standard pattern mask, free of charge, in large numbers. Printed instructions should be issued with the masks, or posters displayed in prominent places, advising that each mask should only be worn once without disinfection, and containers or receptacles should be provided in convenient places in which used masks should be placed ready for removal to a central disinfecting depot.
The prophylactic use of bacterial vaccines in Epidemic Influenza.

It may be said that our knowledge of the application of vaccines in the prophylaxis of epidemic influenza was based on previous experience of similarly composed vaccines in the treatment and prophylaxis of catarrhal diseases of the respiratory tract.

In those countries where the epidemic appeared with little warning and spread with great rapidity, there was little time or opportunity to effect a general prophylactic inoculation of the public, even if such a course had been considered desirable or feasible, but in many institutions, camps, and other localised collections of people, vaccinal prophylaxis was attempted with, I think, a limited measure of success.

The literature for the latter half of 1918 and the first half of 1919 already abounds with reports on the value of this measure, but a great deal of it must be discounted on the score of the small numbers of persons treated and the inadequate controls set up. Moreover, it is difficult to compare results when the composition and dosage of the various vaccines used varied to so great an extent. Out of this mass of evidence it is possible, with a very limited personal experience of the use of vaccines in the New Zealand epidemic, to form the opinion that prophylactic vaccination does not protect against the primary attack of Influenza, but it does afford some protection, limited in amount and in duration, against the secondary pneumococcal and streptococcal complications which are frequently responsible for the fatal issue. Thus, in numerous instances reported in the literature, previous vaccination has not resulted so much in a lessened incidence of the disease, as in a diminished case mortality - those contracting the disease after vaccination suffering from milder attacks.

If we are to consider that Influenza is caused by a filterable virus or some organism not yet discovered, we can understand the failure of the vaccines in general use to prevent an attack, whereas prevention of the secondary complications is explicable if we consider that the Influenza bacillus, the Pneumococcus and Streptococcus are the principal agents responsible for these secondary manifestations.
Composition of the Vaccines used. These would be comparable only if we could assess the antigenic value of vaccines of varying polyvalency, grown on media of different composition, sterilised by heat or chemicals, or prepared in special ways such as the sensitised, lipoidal, or detoxicated vaccines.

Generally the vaccines used have included several strains of each of the microorganisms found in the sputum or in the lungs post mortem in any particular locality. They have been prepared from cultures as little removed from the primary culture as possible, and with a few exceptions they have been sterilised by heat or by chemicals. In these respects the different vaccines are to a certain extent comparable, but the factor of dosage still further complicates any effort at comparison. Doses differing greatly in size have been recommended by those who have had considerable experience in the use of vaccines, as the following table shows - (Table XIV, see next page).

A glance at the table will show that the doses recommended and used, vary from the extremely moderate doses recommended by Eyre & Lowe and the War Office Committee of which Eyre was a member, the moderate doses used by Wynn, and by Anderson in Cape Town, the large doses recommended by Allen, to the massive doses used by the Americans, Minaker and Irvine. No doubt the method of preparation of the vaccine, the virulence of the organisms, and the personal experience of the author have influenced the result in each case, but it is obvious that if the War Office Committee's doses are those to be accepted for use during an epidemic, then the large doses of Allen, and Minaker and Irvine, must either be out of all proportion to what is required for satisfactory immunisation, or they must result in a dangerous negative phase during a time when virulent organisms are broadcast. If the larger doses are harmless and necessary, then the smaller doses should have little value, or effect at most an evanescent protection. While the numbers of persons treated by the smaller doses is considerable and affords an opportunity for estimating their value, the records of persons treated by the larger doses are insufficient to afford a criterion for comparison.

The Protection afforded by Mixed Catarrhal (Influenza) Vaccine. The value of any vaccine will depend to a certain extent on whether it includes the organism or organisms responsible for the infection, on its polyvalency if it is not autogenous, on details of its preparation which will determine its
## TABLE XIV.

**Influenza - Vaccines in Prophylaxis.**

<table>
<thead>
<tr>
<th>Authority recommending Vaccine &amp; reference</th>
<th>1st. 2nd.</th>
<th>1st. 2nd.</th>
<th>1st. 2nd.</th>
<th>1st. 2nd.</th>
<th>1st. 2nd.</th>
<th>Interval between doses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Committee of War Office, October 1918. (40)</td>
<td>30 60</td>
<td>100 200</td>
<td>40 80</td>
<td></td>
<td></td>
<td>10 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>A third dose recommended for Colonial &amp; Native contingents.</td>
</tr>
<tr>
<td>Eyre &amp; Lowe (41) New Zealand troops.</td>
<td>10 30</td>
<td>50 100</td>
<td>10 50</td>
<td>200 500</td>
<td>25 75</td>
<td>10-14 days</td>
</tr>
<tr>
<td>Minaker &amp; Irvine, U.S.A. (42)</td>
<td>2.500</td>
<td>500 2000</td>
<td>4000 100</td>
<td></td>
<td></td>
<td>3 doses at 3 day intervals</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No particulars</td>
</tr>
<tr>
<td>Penfold, Commonwealth of Australia. (44)</td>
<td>25 125</td>
<td>10 50</td>
<td>10 50</td>
<td>25 125</td>
<td></td>
<td>7 days</td>
</tr>
<tr>
<td>Wynn, (45)</td>
<td>100 200</td>
<td>100 200</td>
<td>100 200</td>
<td>500 1000</td>
<td>500 1000</td>
<td>3 doses recommended, prefers 14 days.</td>
</tr>
<tr>
<td>Allen, R.W. (46)</td>
<td>1000 2000</td>
<td>500 1000</td>
<td>500 1000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vaccine prepared &amp; used by Author.</td>
<td>50 100</td>
<td>100 200</td>
<td>50 100</td>
<td>250 500</td>
<td></td>
<td>10 days.</td>
</tr>
</tbody>
</table>
antigenic value, and the size of dose and time of administration. With regard to Epidemic Influenza there was no experience of the use of a mixed influenza vaccine, for vaccine therapy was either unknown or in its infancy when previous pandemics swept over the world. A considerable amount of work had been published, however, before 1918 on the use of mixed catarrhal vaccines in the prophylaxis of catarrhal conditions of the respiratory tract, including the use of pneumococcal vaccines in the prophylaxis of pneumonia. Notable among these are the contributions of Wright and his co-workers, and Lister, in South Africa, whose work in combating pneumococcal infections amongst the native mine workers is well known. Several American workers, including Cole and his fellow-workers at the Rockefeller Hospital, have used pneumococcal vaccines in a sufficient number of cases to indicate the value of their work, and in Great Britain Eyre has studied the effect of prophylactic inoculation against respiratory catarrhs amongst the New Zealand troops.

Lister (47) using massive doses of a polyvalent pneumococcal vaccine representing the local types of pneumococci, claims considerable prophylactic value for his vaccine.

Cecil and Austen (48) inoculated 12,519 men against Types I, II, and III pneumococci. Three or four doses were given at intervals of 5 to 7 days, the total dosage being 6000 - 9000 millions of Types I and II and 4,500 - 6000 millions of Type III. During the ten weeks subsequent to the inoculation, and while the men were still under observation, no cases of pneumonia due to these three types occurred among men who had received two or more doses of vaccine, while in a control of approximately 20,000 men there were twenty-six cases of pneumonia due to these types during the same period. The pneumonias that did prevail were mostly due to type IV pneumococci.

Eyre and Lowe (49) state that "Amongst those who are in the habit of employing vaccines therapeutically in civilian practice the impression has steadily gained ground that sufferers from catarrhal conditions of the respiratory tract are benefited by inoculations prophylactically administered". They proceed in their paper to detail their experience of the use of such vaccines on a nominal roll of 1000 New Zealanders in camp in Great Britain in 1918, an outbreak of measles complicated by purulent bronchitis having occasioned the Authorities to consult Dr. Eyre on the matter. The organisms present in the sputum
in these cases were *B. Influenzae*, *Micrococcus Catarrhalis*, and *Streptococcus Pyogenes Longus*. The vaccine was prepared in much the same doses as subsequently recommended by these Authors for the prophylaxis of Influenza (see Table XIV) and was administered to 1000 men in two doses at an interval of 10 days.

The authors state that the few serological tests they were able to carry out indicated that an immunological response had been made by some of the inoculated individuals to the prophylactic vaccination, but in June Nature stepped in and provided clinical proof of the protection conferred, for in this month a widespread pandemic of "Influenza" commenced.

They publish a table showing the records of admissions to hospital with respiratory complaints from a nominal roll of 1000 inoculated men and from 1000 uninoculated New Zealand men in the United Kingdom. The results are not strictly comparable, for the two groups of men were not living under exactly the same conditions, but from the table it is seen that among the inoculated men the incidence of Influenza was 2 per 1000, as against 28.4 per 1000 of the uninoculated men. They consider the result amply justifies the prophylactic use of a mixed catarrhal vaccine.

Wright and his pupils have published extensively on this aspect of vaccine therapy, so much so that Allen has published a book on "Bacterial Diseases of Respiration", and Carmalt Jones devotes a chapter to the subject in his "Introduction to Therapeutic Inoculation".

I have indicated sufficiently that up to the outbreak of Influenza in 1918 there was a steadily growing opinion in favour of the value of vaccines in the prophylaxis of infections of the respiratory tract due to the *B. Influenzae*, *Pneumococcus* and other organisms. Upon this knowledge was based the application of vaccines in the prophylaxis of epidemic influenza, with the results of which I now propose to briefly deal.

**Prophylactic Vaccination during the 1918 Epidemic.**

Many opinions have been expressed in a general way supporting the contention that Influenza vaccine affords definite protection against the disease, but while these opinions are favourable they are based on the results in small groups of people and can only be
accepted as such. A few opinions unfavourable to the use of the vaccine have been expressed, especially to its use during an epidemic. The results would have lent themselves to comparison more readily had the same vaccine and the same procedure been universally applied, but the very nature of the epidemic, its sudden appearance, and decline, and the abnormal demand which it made on the medical resources of the countries visited, left little opportunity for a thorough trial of a method which is almost universally acclaimed in preventing such infections as Typhoid Fever.

The following is a summary of the opinions of those who have had some experience of the use of this vaccine during the 1918 epidemic.

In Britain - Eyre and Lowe (25) summarise the result of prophylactic vaccination in their report on "The Autumn Influenza epidemic (1918) as it affected the New Zealand Expeditionary Force in the United Kingdom". The total number of men is not stated but their opinion is based on the results in several thousand men. They found the infection incidence in the inoculated (see Table XIV for composition of vaccine) as against the non-inoculated was 1 to 3. The risk of death amongst all cases infected was for the inoculated 1 to 9 for the non-inoculated.

W. D'Este Emery (50), discussing the use of vaccines as recommended by the two previous workers and by the War Office Committee, considers that these vaccines have a considerable prophylactic value.

F. L. Armitage (51) Bacteriologist to the N. Z. Stationary Hospital, E. F. F. France, considered the dosage of the mixed catarrhal vaccine of Eyre and of the War Office Committee ineffective and prepared a vaccine using half the doses recommended by Allen (see Table XIV). The N. Z. Stationary Hospital to which Armitage was attached acted as the Infectious Disease Hospital for the Second Army, E. F. F. Force, France, during the period of the epidemic.

Of 133 cases of all ranks inoculated with this vaccine, there were no cases of either "influenza", "cold", or pneumonia during the epidemic period. While admitting his numbers are small, Armitage states that "it seems wonderful that the staff should have escaped; since during the epidemic the hospital received hundreds of cases of Influenza and Pneumonia".
In America. Minaker and Irvine (42), already referred to (see Table XIV for composition and doses of vaccine) state that of 1080 of the civil population inoculated, the morbidity was 1.4 per cent and mortality nil. In the non-inoculated population with whom these persons mixed, the morbidity was 5.3% and the mortality 9.2%. Of 1950 marines inoculated and 8232 not inoculated the morbidity and mortality ratios were 1.8 : 2.8 per cent as compared with 15.7 : 5.0 per cent.

Wallace (52), reporting on his experience in the use of vaccine in the Wrentham State School, Mass. states that the vaccine was used during the epidemic itself. Of 129 employees at the School, 71 were inoculated and of these 5 contracted the disease. Of 58 not inoculated, 38 contracted the disease. In a building in which lived 150 inmates, 28 were vaccinated and only one contracted Influenza, while of the 128 unvaccinated, 64 contracted Influenza.

In South Africa. Anderson (43) formed the opinion that vaccines were useful in prevention and in mitigating the severity of the disease if it were contracted.

Australia was afforded an excellent opportunity to test the value of prophylactic vaccination, not only in institutions, but of the public generally, just as the opportunity for demonstrating the value of maritime quarantine presented itself. Australia, as I have already pointed out, did not suffer like South Africa and New Zealand, and the epidemic wave was delayed there until well on in 1919. There was therefore not the element of surprise in her case, and there was ample time not only to prepare adequate supplies of vaccine, but to vaccinate the public before the advent of the epidemic.

Public Vaccine stations were established and promised to be largely used had not a regrettable press controversy arisen, in which leading members of the medical profession joined, and so clouded the issue in the minds of the public that comparatively few availed themselves of the opportunity. Thus Chapman, who had recently been appointed to the Chair of Pharmacology, publicly deprecated the use of vaccines without a knowledge of the infection they are presumed to combat, stating that from his experimental work inoculation appeared to lessen the resistance of the patient to an attack of Influenza, if the organisms used in the vaccine are not those infecting the patient. Chapman's
views were strenuously combatted by Welsh, the Professor of Pathology, who was acting for the Health Authorities.

Statistics dealing with a large number of men have been published by Penfold (44) of the Commonwealth of Australia Serum Laboratories, under whose direction large quantities of vaccine were prepared (Table XIV). In order to ascertain the prophylactic value of the vaccine, particulars of its use by the Victorian Railway authorities are supplied. 11,402 men are employed in the metropolitan and suburban districts, and of these 745 were inoculated once, 4,410 were inoculated twice, and 6,247 were not inoculated at all. The period of observation began on January 24th, 1919, and extended for 6 weeks. The incidence of the disease in each group was as follows -

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
<th>Number infected.</th>
<th>Proportion infected.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uninoculated</td>
<td>6247</td>
<td>252</td>
<td>4 per cent</td>
</tr>
<tr>
<td>Once inoculated</td>
<td>745</td>
<td>61</td>
<td>8.2 per cent</td>
</tr>
<tr>
<td>Twice &quot;</td>
<td>4410</td>
<td>95</td>
<td>2.1 per cent</td>
</tr>
</tbody>
</table>

It appears from the above table that while the value of a double inoculation is established, a single inoculation offers no protection, but apparently increases susceptibility. The numbers receiving one inoculation were few however, and Penfold attempts to explain the unfavourable results - an attempt for which he is taken to task in the same issue of the Medical Journal of Australia.

It is probable that the doses he used erred, like those of Eyre and the War Office Committee on the side of safety, so that a single dose, which is only half the size of the second dose, would be too small to arouse any but the slightest immunity, and that, probably, after an initial period of increased susceptibility.

In New Zealand little opportunity was afforded us of either preparing or using a vaccine as a
prophylactic, for the second wave of the epidemic overwhelmed us so suddenly that there was little time in which to act and no recent literature to guide us. Moreover, we were faced with the problem of giving a vaccine in the midst of an epidemic with little data as to a possible period of increased susceptibility following its use. While, however, the wave was spreading south, I made what preparations I could for this eventuality, but owing to the disorganisation in the North it was impossible to obtain any but the most meagre information as to the bacteriology, and quite impossible to obtain any cultures for the purpose of preparing a vaccine. With the advent of local cases their bacteriology had to be first determined and vaccines were then prepared for limited use. Our staff was depleted and could not have prepared vaccine in large quantities, particularly as the newer cultural methods whereby large quantities of influenza bacillus emulsion could be quickly prepared only came to hand towards the end of the epidemic.

Five out of six available members of my staff received two prophylactic injections (for doses see Table XIV) and of these none contracted the disease, though constantly exposed to infection. The non-inoculated member of staff contracted the disease, but in a mild form only.

The experience in New Zealand is practically confined to the troops[11] and here the experience of the medical officers was encouraging, not as a prevention of infection so much as a means of reducing the more serious complications by raising the resistance of the body to pneumococcal and streptococcal complications.

Before considering the points raised in this discussion, there remains one important question to answer, namely:- Is the use of a mixed Influenzal vaccine, such as was generally used, followed by a negative phase or period of increased susceptibility on the part of the inoculated one, and if so does this prohibit the use of such vaccines while an epidemic is in progress?

According to the work of Wright, a negative phase or period of diminished resistance follows the injection of bacterial protein in the case of those already infected with the microorganisms represented in the vaccine. This negative phase may be very slight, if present at all, if the dose of vaccine is carefully regulated, but it may be marked and followed by unfort-
unate results to the patient if the dose is too large or if it is administered at an unsuitable time. Every patient being a law unto himself in this respect, general directions for guidance only can be issued, and the vaccine therapist usually "feels his way" with the first one or two doses of a vaccine.

We know from experience, however, what the prophylactic dose of such a well known vaccine as the Typhoid-Paratyphoid vaccine should be, but we were in an altogether different position with the Influenza vaccine. Here we had little previous experience to guide us, and we were dealing with a disease of much greater infectivity, and one in which the course of the disease was so rapid that it might run its whole course within the period of increased susceptibility should such follow the injection of vaccine. It is held by many that the negative phase has been made a bogey, and that no period of increased susceptibility follows the exhibition of a vaccine provided the patient is not at the time suffering from an infection by organisms included in the vaccine. If this is so there is no risk in giving Typhoid vaccine to contacts during a typhoid outbreak, and there should be no risk in giving Influenza vaccine during an Influenzal epidemic.

Eyre and Lowe(49), already quoted, are quite emphatic on this point. They say "The experimental work upon which prophylactic and still more therapeutic vaccination was originally based, showed clearly that quite apart from the immediate local and general reactions there supervened a period of lowered resistance, or increased susceptibility towards infection by microorganisms of the same species as those constituting the antigen, and that this so-called negative phase varied in extent from a few hours to two or three weeks in accordance with the size of the dose of the antigen, the general physical condition of the inoculated subject, and other factors. We were, of course, fully alive to this aspect of our experiment and anticipated that during the seven days following the first inoculation, and the ten days following the second, there would be an increase in the number of catarrhal affections reported from the troops comprising our nominal roll, and in order that none might be missed arrangements were made for all these cases to be admitted to hospital for observation. The accuracy of our surmise was proved by subsequent events, as will be seen from the accompanying curve showing the relevant admissions from day to day for a period of three weeks from the date of the initial inoculation.
It will be seen that 15 admissions were reported between the administration of the first and second dose of vaccine, and 82 cases during the 10 days following the injection of the second and larger dose of vaccine.

These figures, according to the authors, emphasise the reality of the negative phase and prompt the suggestion that the initial dose might with advantage have been somewhat larger, or preferably that the interval between the two inoculations should have been longer.
In a later paper(41) the same authors consider that vaccination should be continued in the face of an epidemic, but while no protection from attack is given during the period of increased susceptibility, up to 10 days following the injection, apparently the risk of death is very much diminished.

Several other writers in the papers already quoted see no objection to the use of the vaccine during an epidemic, and most of those who have had personal experience of its use, and have observed the results in a sufficient number of cases, are agreed that a suitable vaccine administered with care and in suitable doses and at correct time intervals does give a large measure of protection to the inoculated individual, not so much from attack but from the severe secondary complications which so often determine a fatal issue. Opinion also favours the use of the vaccine during an epidemic if, as would be desirable, there has not been time or opportunity to exhibit it prior to the epidemic. When one considers the history of influenza epidemics, it is readily seen that it would seldom be possible to protect a community in readiness for an epidemic, partly because of the suddenness of its appearance and the rapidity of its spread, partly because of the difficulty of preparing sufficient quantities of suitable vaccine at short notice, even if the antigen were already available, and partly because of the short duration of the protection afforded, which would necessitate inoculation at intervals of a few months - a wholly impracticable procedure in the case of the public.

Summarising these results it is seen that a mixed catarrhal vaccine including the microorganisms usually found in catarrhal infections of the respiratory tract, i.e. the B. Influenza, Pneumococcus, Streptococcus, Micrococcus Catarrhalis, and others less frequently encountered, does offer some measure of protection against attack, but more particularly does it modify the severity of the disease by eliminating the secondary complications. The inoculation is probably followed by a short period of increased susceptibility, but this is not a sufficient reason for withholding the vaccine from the public in face of an epidemic, as even then the risk of death in the inoculated is less than in the non-inoculated.

Its use is especially indicated just prior to an epidemic if this is possible, but at any time in institutions, camps, or in isolated communities, where the advent or dissemination of the disease can be better controlled.
If it be granted that prophylactic vaccines in Influenza have all the merits claimed for them, and provided that ample supplies are available, one may ask whether we have in this procedure a measure which is at all likely to play any great part in the prevention of the spread of epidemic influenza in the community generally. I am afraid the answer must be in the negative, for the outbreaks are as brief as they are severe, and before any considerable numbers in a community could be protected, the epidemic would have exhausted itself. Moreover, the British public, and their cousins overseas, do not take readily to any measure of compulsion, and short of this the natural dread of injections of all kinds, especially when coupled with objectionable word "Vaccine", would be sufficient to deter the majority from availing themselves of this useful measure.
PART III.

NOTE ON THE PATHOLOGY OF 25 FATAL CASES.

The accompanying table (Table XVI) gives a summary of the pathological and bacteriological findings, along with the main clinical features. The clinical notes available are unavoidably very scanty owing to the congestion of the wards and the shortage of staff during the height of the epidemic. With regard to duration of disease, while the duration in hospital is known, the date of onset is doubtful in many of the cases. Any attempt to correlate the pathological condition with the clinical duration fails from lack of clinical data.

Certain features are common to all these cases, i.e. Tracheitis, bronchitis, (a few showed marked laryngitis), haemorrhages, oedema, and a variable amount of catarrhal pneumonia, acute lymph glandular changes, and toxic changes in liver, kidney, spleen and heart, but the cases can be roughly grouped into two main types -

(1) Those showing intense toxaemia with haemorrhage and oedema of the lungs as chief features, catarrhal pneumonia being present in parts but only demonstrable microscopically, and

(2) Which includes the cases where there was definite pneumonia - all grades of pneumonia being found from small patches of broncho-pneumonia to definite lobar consolidation.

Even in type II the haemorrhagic oedema was present. There are 7 cases which can be put into the first group (673, 674, 675, 677, 678, 680, 684), and sixteen cases which show varying degrees of definite pneumonia, into the 2nd group. Two cases must be mentioned by themselves, i.e. 679, which shows an old standing bronchiectasis, with pneumonia and abscess formation around, and 691, in which the lungs were riddled with Staphylococcal abscesses. (Microscope slides and photomicrographs, illustrating these cases, accompany this study.)

In the earlier cases, which formed mostly group I, a gram positive capsulated diplococcus, which was later definitely identified as a pneumococcus, was present in enormous numbers in the lung tissue and haemorrhagic oedema fluid. The B. Influenzae was demonstrated in the bronchial secretion of a few, but
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Date Adm.</th>
<th>Date Death.</th>
<th>Duration in Days</th>
<th>Sputum</th>
<th>Pneum</th>
<th>Cyanosis</th>
<th>B. Inf.</th>
<th>Pneumococ.</th>
<th>Staph. aureus</th>
<th>Laryngitis</th>
<th>Bronch.- By.</th>
<th>Lobar Lobar</th>
<th>Membranous</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-673</td>
<td>42</td>
<td>18.11.18</td>
<td>17.11.18</td>
<td>2</td>
<td>++</td>
<td></td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Old heart lesions.</td>
</tr>
<tr>
<td>2-674</td>
<td>21</td>
<td>18.11.18</td>
<td>16.11.18</td>
<td>?</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>3-675</td>
<td>19</td>
<td>16.11.18</td>
<td>8.11.18</td>
<td>?</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>4-676</td>
<td>54</td>
<td>15.11.18</td>
<td>19.11.18</td>
<td>4</td>
<td>rusty</td>
<td></td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Old T.B.</td>
</tr>
<tr>
<td>5-677</td>
<td></td>
<td>16.11.18</td>
<td>17.11.18</td>
<td>10</td>
<td>scanty</td>
<td></td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>6-678</td>
<td>38</td>
<td>17.11.18</td>
<td>19.11.18</td>
<td>10</td>
<td>-</td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>7-680</td>
<td>42</td>
<td>12.11.18</td>
<td>8.11.18</td>
<td>9</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>8-682</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>9-683</td>
<td>45</td>
<td>3 weeks</td>
<td>19.11.18</td>
<td>2+ weeks Bl.etn.</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>10-686</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Chronic interstitial myocardiitis.</td>
</tr>
<tr>
<td>11-688</td>
<td>31</td>
<td>22.11.18</td>
<td>23.11.18</td>
<td>?</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>12-689</td>
<td>42</td>
<td>7 days</td>
<td>25.11.18</td>
<td>9</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Pneumococcal meningitis.</td>
</tr>
<tr>
<td>13-688</td>
<td>26</td>
<td>14.11.18</td>
<td>25.11.18</td>
<td>11</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Conglomerate Streptococcus.</td>
</tr>
<tr>
<td>14-688</td>
<td>57</td>
<td>5 weeks</td>
<td>26.11.18</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Empyema &amp; abscesses in lung.</td>
</tr>
<tr>
<td>15-689</td>
<td>29</td>
<td>No notes available</td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Old T.B. Lung.</td>
</tr>
<tr>
<td>No.</td>
<td>Name</td>
<td>Age</td>
<td>Date of onset</td>
<td>Date of admission</td>
<td>Duration in days</td>
<td>Sputum</td>
<td>Pneumonitis</td>
<td>Cyanosis</td>
<td>B. Inf.</td>
<td>Pneumoc.</td>
<td>Staph. aureus</td>
<td>Laryngitis</td>
<td>Bronchitis</td>
<td>Phlegmon &amp; abscesses in lung</td>
<td>Jaundice</td>
</tr>
<tr>
<td>-----</td>
<td>------</td>
<td>-----</td>
<td>---------------</td>
<td>------------------</td>
<td>-----------------</td>
<td>---------</td>
<td>-------------</td>
<td>----------</td>
<td>---------</td>
<td>----------</td>
<td>---------------</td>
<td>------------</td>
<td>------------</td>
<td>---------------------------------</td>
<td>---------</td>
</tr>
<tr>
<td>16</td>
<td>Miss J. 16</td>
<td>16-11-18</td>
<td>23-11-18</td>
<td>27-11-18</td>
<td>11</td>
<td>?</td>
<td>+</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>17</td>
<td>Miss L. 16</td>
<td>16-11-18</td>
<td>23-11-18</td>
<td>27-11-18</td>
<td>4</td>
<td>?</td>
<td>+</td>
<td>?</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>18</td>
<td>Miss B. 20</td>
<td>22-11-18</td>
<td>27-11-18</td>
<td>5</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>19</td>
<td>Mrs. F. 33</td>
<td>17-11-18</td>
<td>24-11-18</td>
<td>29-11-18</td>
<td>12</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>20</td>
<td>Mrs. McK. 36</td>
<td>26-11-18</td>
<td>2-12-18</td>
<td>8</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>21</td>
<td>Mrs. G. 38</td>
<td>27-11-18</td>
<td>2-12-18</td>
<td>28</td>
<td>?</td>
<td>+</td>
<td>Jaundice</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>22</td>
<td>Mrs. D. 53</td>
<td>30-11-18</td>
<td>3-12-18</td>
<td>5</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>23</td>
<td>Mrs. S. 28</td>
<td>2-12-18</td>
<td>5-12-18</td>
<td>8</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>24</td>
<td>Mrs. J. 27</td>
<td>2-12-18</td>
<td>5-12-18</td>
<td>9-12-18</td>
<td>8</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>25</td>
<td>Mrs. S. 7</td>
<td>4 days</td>
<td>7-12-18</td>
<td>10-12-18</td>
<td>8</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
was not nearly so prominent or so readily demonstrable, either in films or cultures, as in the group II cases.

In group I the following microorganisms were recovered in smears and cultures from the lungs and bronchi:

- **B. Influenza** in 4 (674, 678, 680, 684).
- **Pneumococcus** in 6 (673, 674, 675, 678, 680, 684).
- **Streptococci** in 0.
- **Staphylococcus Aureus** in 2 (673, 675).

The organisms most abundantly present in this group were - Pneumococci in 6; B. Influenzae in 4; Staphylococcus Aureus in 2. No Streptococci were found in this group. No. 677 is interesting in that no microorganisms were found in any of the fluids or tissues examined, except a few Staphylococcus Albus in the heart blood and lung smears.

In group II, including the two aberrant cases specially mentioned, the B. Influenzae was more prominent and the pneumococcus less in evidence. The Staphylococcus aureus was frequently found and appeared in considerable numbers in some of the cases, while in the secondary complications Streptococci were found in addition.

Unfortunately the members of the staff who had undertaken the examination during the life of the patient, had not a view of the prevailing epidemics and these were not suspected. A summary of the main features found in these four principal organisms mentioned as being most frequently found. B. Aerogenes Capsulatus was found in the heart blood and spleen in 3 cases; B. Friedlander in heart blood in one case; Mic. Catarrhalis in lungs and bronchi in 3 cases. (These findings will be discussed in the section on Bacteriology).

Where the Staphylococcus aureus or Streptococci occur, the lesions seem more pronounced. Where a Staphylococcus aureus infection was present in the lungs, the leukocyte reaction was abundant round these organisms. In fact in case 673 one can predict where Staphylococci are likely to be found with the low power from the clumps of leukocytes seen. In case 691, the Staphylococcus aureus was present in great masses to the exclusion of all other organisms, and there is intense leukocyte reaction and definite abscess formation.
Complications. In Type I (haemorrhagic oedema type) though the lungs were so involved there was no pleurisy, but in many of Type II cases Pleurisy or even Empyema occurred, the causal organisms being either Pneumococci, Streptococci, or Staphylococcus aureus. B. Influenza was never found in these lesions. It was not possible to examine the brain in every case, but of those examined only one showed a definite menigitis (693) - pneumococcal in origin. The other brains examined from different types of cases showed only some engorgement of the vessels, oedema of brain, and clear cerebro-spinal fluid, with increased globulin content. In no case was pericarditis or peritonitis present, and no case showed gastric or intestinal lesions other than a few petechial haemorrhages with erosion of the gastric mucosa.

Haemorrhage occurred in various sites besides the lungs, and lymph glands. For example, there were three cases with definite haemorrhages in the rectus abdominis muscles; two cases with haematosalpinx (the clinicians frequently met with severe uterine haemorrhage).

The suprarenals occasionally showed minute haemorrhages.

Jaundice occurred in several cases without demonstrable liver or gall bladder lesions other than acute toxic changes.

Unfortunately the member of the staff who had undertaken the blood examination during life, early fell a victim to the prevailing epidemic and these were perforce discontinued.

The above gives a summary of the main features found in the 25 cases under discussion, and while it would be needless repetition to give full details of all the cases, the following have been selected as illustrating the main pathological changes. I am indebted to my clinical colleagues and my friend Professor Drennan of the Pathological Department for access to their records.
TYPE I. Case 680. J.C. Haemorrhagic oedemato lungs.

Patient's Name. J.C. Age. 42
Under care of Dr. Fitchett. Ward. Plunket.
Date of Death 20.11.18 Address Richardson St., St. Kilda.
Date of P.M. 20.11.18 Date of Admission to Hospital. 18.11.18
Occupation. Labourer.
Disease. Influenzal Pneumonia.

CLINICAL NOTES.
Patient has been ill about seven days. Took ill suddenly. Headache and backache. Soreness behind sternum. Cough, sputum not blood stained.

On admission: Temperature 104.2; Pulse 98; Resp. 45; No epistaxis.

Chest examination: Broncho-vesicular breathing over whole of left chest. Inspiration loud; expiration faint.

Right lung: Sonorous rhonchi and crepitations over whole lower lobe.

Died, 20.11.18.

POST MORTEM REPORT.
Body is that of a young, well developed man. R.M. present.

Trachea and large bronchi much congested and contain blood stained frothy fluid.

LUNGS: Haemorrhages in pleura. On section - deep red throughout from intense congestion and oedema. No definite pneumonic consolidation. The deep red portions sink in water. The smaller bronchi are moderately congested.

Glands along trachea, bronchi, and at root of lung are swollen, soft and red, and some show small haemorrhages.
HEART: All chambers engorged, but especially right side, which is moderately dilated; muscle pale, firm, and cloudy.

LIVER, SELLER, & KIDNEYS show only acute toxic changes. A few calcified glands in lower part of mesentery, otherwise no old lesions.

STOMACH & INTESTINES - showed slight engorgement.

BRAIN - Not examined.

SUMMARY: -
Influenza.
Acute congestion and oedema of lungs.
Bronchitis.
Engorged heart.
Acute toxic changes.

BACTERIOLOGY:
Cultures and films from - Heart blood.
Lung.
Bronchus.
spleen.

MICROSCOPICAL:
LUNGS: General engorgement of all blood vessels, especially of alveoli. Haemorrhages into the alveoli are numerous and in places they appear distributed along small bronchioles. A few alveoli are empty. The majority contain blood, either as a dense haemorrhage, or as scattered corpuscles mixed with inflammatory cells, with or without fibrin. Some have only a delicate fibrin network with few cells, and some only oedema. The cells of the alveolar exudate are not numerous and are about equal proportions of polymorphs and mononuclears, the latter actively phagocytic towards red and white cells. The small bronchi show acute catarrh.

Organisms - Scanty Gram positive diplococci are scattered amongst the alveolar exudate and in the small bronchi, many being in phagocytes. No definite influenza bacilli could be demonstrated in sections.

TRACHEA: Only moderate engorgement of the capillaries. The epithelium is intact except in a few places where the cells are rather ragged. The mucous glands are active but the cells are not disintegrated. A few Gram positive diplococci lie on and between the epithelial cells.
LARGE BRONCHI: Show acute bronchitis with marked engorgement of all blood vessels and haemorrhages into the lumen. One slide shows the actual point of rupture. The mucous glands are distended with mucus. There are Gram positive diplococci present at the point where the epithelium is ruptured and mixed with blood, there masses of them occur free and in phagocytes. In the polychrome blue sections some minute organisms occur faintly stained and too indefinite to name influenza organisms.

LYMPH GLANDS: All blood vessels engorged, and scattered haemorrhages. Cells of lymph follicles separated by oedema. Simases distended and filled with cells, mostly large mononuclears, many phagocytic. A very occasional diplococcus occurs, and then in a phagocyte.

TONSIL: Moderate engorgement of blood vessels, epithelium intact. A few lymph follicles show hyperplastic "germ centres". A few crypts contain necrotic epithelial cells.

SPLLEN: Markedly oedematous pulp containing many large mononuclears of which a number show phagocytosis of red corpuscles, pigment and cell debris.

LIVER: Cloudy swelling, and engorgement of blood vessels. Little fatty change. There is a typical cavernous haemangioma in one part.

KIDNEY: Cloudy swelling, engorged glomeruli, interlobular and straight vessels.

HEART: Great engorgement of capillary network. Striation of many fibres is blurred. No fatty degeneration seen.

THYROID: Appears in normal colloid state.

SUPRARENAL: General engorgement of all blood vessels.

STOMACH: Capillaries of mucosa engorged and a few small haemorrhages. Oedema of mucosa and sub-mucosa.
TYPE II. Case 686. J.F. A haemorrhagic broncho-pneumonia.

Case 693. A.F. A lobular pneumonia with pleurisy, empyema, and meningitis.

------------

CASE 686.

Patient's Name. J. F. Age. 26
Under care of Dr. Colquhoun Ward. Plunket
Date of Death. 25.11.18 Address. Riversdale
Date of Adm. to Hospital. 20.11.18
Occupation. Farm Labourer.
Disease. Pneumonic Influenza; relapse from influenza.

CLINICAL NOTES.

On admission to hospital - Temp. 104; Pulse 92; Respir. 40.

Has been ill about six days. Took ill suddenly while walking about the street. Sore throat, especially when he coughs. No epistaxis. Sputum not blood-stained. No other previous illness.

Chest examination: Moist crepitations over left lower lobe. Rest of chest - nothing abnormal found. Occasional crepitations over right lower lobe.

21.11.18 - Only occasional crepitations over left lower lobe in front. Posteriorly over left lobe; abundant crepitations and bronchial breathing.

A few crepitations over right base posteriorly. Over rest of the chest - loud inspiration, and very quiet expiration.

Sputum abundant - being treated quinine and 9 grs. ammon. carb. q.q.

6 p.m. Patient has a definite attack of dyspnoea - respirations 42 - followed by a fit of coughing. Patient wanders a little.

22.11.18 - Patient is rather more cyanosed at intervals - breathing rapid - 45 p.m.

Chest examination: Anteriorly bronchial breathing over both lungs - louder on right side.

Posteriorly - Bronchial breathing and crepit-
ations over both bases. Crepitations fine.

24.11.10. Loud tubular breathing everywhere without crepitations. Cyanosis more marked. Laryngitis present. Respiration at present rapid and deep - 70 p.m.

Chest examination:

Right Lung
- Anterior - Tubular Breathing.
- Posterior - " "
- Apex - " "
- Axilla - " "

No crepitations to be made out anywhere - loud tubular breathing all over the chest.

Left Lung:
- Anterior - " "
- Posterior - " "
- Apex - " "
- Axilla - " "

Extent of involvement not easy to determine by auscultation alone. Both lungs apparently involved - but extent indeterminate.

Died, 25.11.18

POST MORTEM REPORT.

Body is that of a young well-nourished man. R.M. general. P.M. lividity posteriorly and especially of head and neck. Larynx and upper part of trachea slightly congested. Lower trachea and large bronchi much congested and contain fœthy blood stained fluid mixed with mucus.

LUNG: No pleurisy or effusion. Both lower lobes firm, feel solid and are deep purple in colour with pleural haemorrhages. O.S. deeper red with grey patches around small bronchi, which are much congested. Upper and middle lobes feel less solid, but O.S. show similar broncho-pneumonic patches with haemorrhages especially along bronchi, and oedematous congested lung between.

Glands at root and along bronchi - moderately enlarged, soft, oedematous and congested; along trachea they are congested and some show small haemorrhages.

HEART: Right side dilated and engorged with blood clot, left side engorged, muscle pale brown and opaque, firm. Valve cusps normal. Aorta - only a few fatty streaks in intima.
LIVER: Mottled with acute congestion and yellow, fatty, lobules.

Spleen: Enlarged, firm. O.S. Uniform deep purple throughout.

Kidneys: Show only cloudy swelling of cortex, and engorged vessels.

Bladder: Distended with urine, also ureters and renal pelvises filled with urine.

Summary:
Influenza.
Tracheitis and Bronchitis.
Acute congestion and oedema of lungs, with haemorrhage and broncho-pneumonia.
Acute toxic changes in organs.

Microscopic.

Lungs: General engorgement of all vessels. Haemorrhage is prominent, either in groups of alveoli along distribution of a bronchus, around abscess areas, or more scattered. It also occurs in pleura and some small bronchi.

The upper portions of lung show many alveoli with only a few cells or oedema, though even here are broncho-pneumonic patches and haemorrhage.

The lower portions have more extensive pneumonic consolidation - confluent broncho-in type - and haemorrhage. Here also are small abscesses amid the pneumonic patches. In these abscess areas there are masses of Gram positive cocci and necrotic cell debris, with intense leucocyte infiltration around.

Elsewhere organisms are few, an occasional Gram positive diplococcus being found, also a number of poorly staining cocci.

Large Bronchi: Intense engorgement of blood vessels, epithelium ragged and shed in parts, the fragments being mixed with leucocytes, wall oedematous, mucous gland active.

At the breaks in the epithelium are many Gram positive cocci, mostly in pairs and short chains (pneumococci?), also large Gram positive cocci (Staphylococci), and a few rather indefinite minute Gram negative bacilli (? Influenza).

Trachea: Shows similar appearances, with haemorrhages through epithelium.
LYMPH GLANDS: Follicles oedematous. Sinuses greatly distended and filled with large mononuclears, many phagocytic for red blood corpuscles, cell remnants and diplococci.

Spleen: Acute general engorgement of pulp.

Liver, Kidney, & Heart: Engorged capillaries and cloudy swelling.

Suprarenal: Engorged capillaries and cloudy swelling.

CASE 693.

Patient's Name: A.F. Age: 33
Under care of Dr.: Fitchett Ward: Plunket
Date of Death: 29.11.18 Address: Roslyn
Date of E.M.: 29.11.18 Date of admission to Hospital: 24.11.18

CLINICAL NOTES.

On Admission: Temperature 103; Pulse 100; Resp. 25; Pulse full and strong.
Took ill seven days ago - in the evening - just after tea - felt well at tea, but vomited soon after and then felt ill. Headache pretty severe for two days.
Backache - but not severe.
Vomiting - for several days; unable to retain food.
Epistaxis - slight in amount.
Cough - about three days after commencement of illness. Pain behind lower part of sternum down to epigastrium. Occasionally blood in sputum.
General appearance - Moderate cyanosis present; lips blue.

Chest examination:
Right Lung: Anterior - Negative. Posterior
Apex
 Axilla
Breathing very shallow and quiet.

Left Lung: Anterior - A few fine crepitations inferiorly
Left Lung (Contd.).

Posterior - Fine crepitations over the base and tubular breathing.

Apex - Negative.

Axilla - Fine crepitations.

Trouble at present seems limited to left lung and chiefly to lower lobe.

25.11.18: Right Lung - Still negative.

Left Lung - Crepitations in axilla and base anteriorly show involvement of two lobes.

Died, 19.11.18.

POST MORTEM REPORT:

Body is that of a young, slightly built man.

R.M. absent.

HEAD: Skull cap normal. Excess of turbid cerebro-spinal fluid.

BRAIN: Oedema of pia-arachnoid and thin yellow purulent exudate over vertex, along sylvian fissures, and around base and cerebellum. All vessels engorged. O.S. - brain oedematous, ventricles dilated and filled with slightly turbid cerebro-spinal fluid. Middle ears and bony air cells normal.

LARYNX: Not congested. TRACHEA: In its lower third, and large bronchi congested and covered with yellow muco-pus. Glands, along bronchi and at bifurcation enlarged, soft, and congested.

LUNGS: Right - No pleurisy, firm and red.

O.S. - Intense general engorgement, but only moderate oedema.

Left - About six ounces turbid yellow fluid in pleural cavity. Pleura covered with shaggy yellow fibrinous exudate all over. O.S. - Anterior margin of upper lobe cripitant but congested and oedematous. Rest of upper lobe and whole of lower lobe consolidated, grey granular surface which exudes slimy yellow pus. Lower margin is collapsed, fleshy, dark slaty red. In the consolidated parts are sharply defined areas of lobules and adjacent are haemorrhagic and congested areas.

The smaller bronchi are congested and contain muco-pus.
**HEART:** No excess of pericardial fluid; over apex is a flake of fibrin. All chambers filled with p.m. clot, but right side only moderately distended. Muscle congested and cloudy. Otherwise nil.

**LIVER:** Slightly swollen; mottled red and yellow (congestion and fatty).

**Spleen:** Not enlarged, very soft, pulp congested.

**Kidneys:** Show only cloudy swelling, and moderate engorgement of vessels.

**Suprarenals:** Nil.

**Stomach & Intestines:** Nothing of note.

**Summary:** Influenza.
- Purulent lobular pneumonia with pleurisy,
- Acute congestion and oedema of lungs.
- Bronchitis.
- Meningitis.
- Acute toxic change in organs.

**Microscopical.**

**Lungs:** Right - Areas of distended alveoli mingled with areas of alveoli filled with haemorrhage, others with inflammatory cells. All blood vessels are engorged, especially marked in the haemorrhagic areas. No pleurisy. The small bronchi are filled with inflammatory cells and their walls congested and oedematous, and some have haemorrhages in the walls.

**Left -** Consolidated areas - recent fibrinous exudate with many acute inflammatory cells on pleura. Alveoli contain acute inflammatory cells - mostly polymorphs, also large mononuclears, and many necrosed cells. In some cases these cells fill the alveoli completely, in other cases only a few are present in lumen. Fibrin is scanty.

Adjacent to consolidated areas are alveoli overdistended, also some with oedematous content, and some with a few inflammatory cells.

**Lower border -** Alveoli are collapsed, capillaries greatly engorged. Scanty haemorrhage is present in some alveoli. Small bronchi are filled with inflammatory exudate.

On surface there is a ragged fibrinous exudate and great numbers of acute inflammatory cells - mainly polymorphs.
LARGE BRONCHI: Epithelium shed in places and mixed with inflammatory cells and some red corpuscles. Intense engorgement of all vessels in wall; oedema of wall and infiltration with inflammatory cells; mucous glands filled with mucus. In short, an intense acute bronchitis.

TRACHEA: Moderate congestion. Epithelium shed in parts and ragged. Changes much milder than in bronchi.

Organisms: In consolidated parts there are many Gram positive diplococci, some intracellular. Similar organisms occur to a less extent in haemorrhagic areas, and to a greater extent in the pleuritic exudate.

In large bronchi there are diplococci as above, also some in short chains, and large cocci (? Staphylococci). In the polychrome blue section there also appear some poorly staining minute diplococci; these are not definitely recognisable in the Gram stained section and are too vague to warrant the suggestion that they may be influenza bacilli.

LYMPH GLANDS: The changes are similar in all and vary only in degree in individual glands and in different parts of the same gland.

There is much engorgement of all blood vessels, and oedema of gland tissue. Lymph follicles are not hyperplastic. Haemorrhages are frequent, some amongst the follicles, some in the sinuses, and often both areas are involved and almost solid with red corpuscles. The sinuses are dilated, markedly so in many cases, and filled with inflammatory cells - large mononuclears, often phagocytic, and many polymorphs - and in places with red corpuscles. In one small tracheal gland there is practically an abscess at the periphery where a sinus is enormously distended and filled with polymorphs and large mononuclears. In some sinuses there are threads of fibrin, usually associated with groups of organisms.

Organisms - There are Gram positive diplococci scattered amongst the haemorrhages, and in the sinuses; and in places, in some sinuses, are tangled masses of streptococci - usually associated with fibrin as noted above.

SPLEEN: There is general engorgement of the pulp. Lymph follicles are compressed and not hyperplastic. In the pulp spaces are many large mononuclears, some filled with red corpuscles, or blood pigment, or cell debris.
LIVER: Moderate engorgement of capillaries. Considerable fatty change throughout lobules.

KIDNEYS: Malpighian bodies and straight vessels moderately engorged. Convoluted tubules dilated with granular debris in lumina, cells ragged and disintegrating and nuclear staining poor in many.


HEART: All capillaries engorged. Many fibres show blurring and faint striation. No fatty change.

BRAIN: Scanty fibrinous exudate and great numbers of inflammatory cells in meshes of pia. Blood vessels engorged. Brain oedematous. The inflammatory cells are mostly polymorphs, with some large mono-nuclears, often phagocytic. There are great numbers of diplococci amongst the exudate.

POST-MORTEM EXAMINATION.

Body is that of a young girl, emaciated; deeply jaundiced. N.M. absent.

GENERAL: Half pint bile stained turbid fluid in right pleura. Left pleura - two oz. clear fluid; in pericarditis.

Right pleura covered by shaggy exudate, especially over lower and posterior part quite posteriorly adhesion keep the two sides adjacent.

Left lung: posterior part has scanty exudate. Mesoenteric glands prominent - several being moderately congested.

Intestines: Nothing abnormal.

SPECIAL: Bronchial glands very much enlarged.

Right Lung - Section - large number small abscesses; abscesses larger at base, have yellow membrane; around is intense congestion and haemorrhage. Bronchus in case of lower lobe connect with abscess and mucosa is covered with purulent exudation.
ABERRANT TYPE. Case 691. M.A. *Staphylococcus aureus* lung abscesses.

**Patient's Name:** M.A. **Age:** 6 years.

**Under care of Dr.** Fitchett **Ward:** Houghton

**Date of Death:** 27.11.18 **Address:** Dunedin

**Date of P.M.:** 28.11.18 **Date of admission to Hospital:** 23.11.18

**Country where born:** New Zealand. **Disease:** Pneumonic influenza. (Jaundice lasted at least four days).

**CLINICAL NOTES.**

The records show practically nothing, the case being admitted during the height of the epidemic when the staff was particularly depleted through illness, but clinically the case corresponded to the epidemic type. When in hospital the child developed an empyema from which pure cultures of *Staphylococcus aureus* were obtained. A day or two before death jaundice appeared.

**POST MORTEM REPORT.**

Body is that of a young girl, emaciated, deeply jaundiced. R.M. absent.

**GENERAL:** Half pint bile stained turbid fluid in right pleura. Left pleura - two ozs. clear fluid; no pericarditis.

Right pleura covered by shaggy exudate, especially over lower and posterior part quite posteriorly adhesions keep the two sides adjacent.

Left lung: posterior part has scanty exudate. Mesenteric glands prominent - several being moderately congested.

Intestines: Nothing abnormal.

**SPECIAL:** Bronchial glands very much enlarged.

Right Lung - Section - large number small abscesses; abscesses larger at base; have yellow membranes; around is intense congestion and haemorrhage. Bronchus in case of lower lobe connects with abscess and mucosa is covered with purulent secretion.
Left Lung - Same appearance to lesser degree. Area at base and posterior margin shows multiple abscess etc. Bronchi intensely congested and haemorrhagic. Contain blood-stained fluid. Glands at root and bifurcation enormously swollen. On section - oedematous, pale and mottled.

**TRACHEA:** Larynx, and upper part trachea congested. Shows superficial necrosis of mucosa. Large bronchi similar. Tonsils not enlarged. Thyroid appears normal. Paratracheal glands congested.

**HEART:** Right side engorged. Muscle firm, pink and cloudy. Valve cusps healthy.

**GALL BLADDER:** Distended with clear bile-stained mucus. Ducts patent.

**LIVER:** Swollen. On section - orange yellow mottled with congestion; no abscesses.

**KIDNEYS:** Jaundiced, cloudy swelling; no abscesses.

**SUPRARENALS:** Marked congestion. Small haemorrhages in both.

**Spleen:** Slightly swollen. On section - moderate congestion.

**STOMACH & INTESTINES:** Nothing to note.

**SUMMARY.**


**MICROSCOPICAL.**

**LUNG:** Extensive abscess formation in lungs with congestion and haemorrhage around. Masses of staphylococci in abscesses. In places the abscesses are definitely related to small bronchi, and the walls of these are lined by masses of cocci. There is acute pleurisy on the surface.

**LIVER:** Fatty changes; no abscesses; no catarrhal changes found in small ducts.
A SUMMARY OF THE BACTERIOLOGICAL ASPECTS OF THE EPIDEMIC IN NEW ZEALAND.

When it became evident that New Zealand was involved in the pandemic of Influenza, it became necessary to study the local Bacteriology as far as it was possible at a time when the great cry was for medical aid, and when laboratory work of any kind was regarded as non-essential. The incessant call for medical assistance, and the depletion of the staffs upon which we had to rely, made it a matter of some difficulty to do our duty, and at the same time conduct a sufficient number of bacteriological examinations to give some permanent record of the Bacteriology of the local epidemic.

The scheme which I outlined with my colleague the Professor of Pathology would no doubt have yielded more satisfactory and more complete results had the members of our team remained well. One after another fell ill, however; and although at first we attempted to replace them by students from our classes it soon became impossible to do other than routine bacteriological examinations. Fortunately the pathological material we were able to obtain was reserved for future examination and report.

I have accordingly divided the bacteriological work into two groups - (1) The antemortem findings, (2) the post-mortem findings, - a summary of which follows.

(1). Ante-mortem Bacteriology. The bacteriological examinations which I was able to make during life were comparatively few, and it was in this phase of the investigation that I was hampered by the frequent changes through illness in the staff that I had arranged to assist in the carrying out of the different portions of the work.

Of those examinations which it was possible to complete in some detail, there were 18 sputum examinations, 18 blood cultures, and six specimens from secondary complications.

Sputum examination. A student was detailed to collect with aseptic precautions sputum from every severe case as soon after admission to hospital as possible, with the object of ascertaining the bacterial flora before secondary organisms invaded the bronchial
secretions and confused the picture. Special sterilised covered enamelled mugs were provided and flasks of boiled water with which to rinse the patients' mouths prior to taking the specimen. Attempts were made to obtain specimens with as little mouth contamination as possible. As soon as the morning specimens were collected they were brought across the road to the laboratory and washed in two or three changes of sterile, recently prepared saline, and Gram films and cultures on freshly prepared rabbit blood agar plates prepared for examination. The cultures were incubated at 37°C. and examined daily for five days. It soon became apparent that under our peculiar circumstances little reliable information would be obtained by carrying on this procedure. In many cases there was either no sputum at all, or at best a little tenacious, frothy or mucoid material which teemed with the usual mouth organisms in spite of all attempts to avoid that contamination. Attempts to cleanse the patients' mouths, which were often dry and cracked, caused much distress and after having instructed three students in turn as to the procedure to be followed, and after having examined only 18 suitable specimens, I abandoned sputum examinations altogether. For similar reasons attempts to obtain a series of nasopharyngeal swabs was abandoned.

Other workers have noted the same difficulty. Thus Abrahams, Hallows and French(53) state that with one exception they have been struck by the relative paucity and even the entire absence of sputum, and that the cases which occur in any particular district tend to conform to one type in regard to the amount of sputum.

Keegan(54) in America, and C. J. Martin(55) in France, have noted a similar difficulty to that which I experienced in obtaining reliable information from sputum and nasopharyngeal mucus in the earlier stages of the epidemic.

In tabular form I have included my findings and compared them with those of several others who were more fortunate in securing better or more numerous specimens upon which to base their reports.
<table>
<thead>
<tr>
<th>Investigator</th>
<th>No. of Cases</th>
<th>Influenza Bac.</th>
<th>Pneumococci</th>
<th>Streptococci</th>
<th>M. Catarrhalis</th>
<th>Staphylococcus aureus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author</td>
<td>18</td>
<td>3 cases in cultures. 9 in films.</td>
<td>18</td>
<td>11</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Martin. (55)</td>
<td>20</td>
<td>14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Influenza Committee Advisory Board D.G.M.S. France. (56)</td>
<td>222</td>
<td>91</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McIntosh. (57)</td>
<td>25</td>
<td>21</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fildes, Baker &amp; Thompson. (58)</td>
<td>106</td>
<td>45</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irving &amp; Co-workers at Camp Upton U.S.A. (59)</td>
<td>145</td>
<td>5</td>
<td>117</td>
<td>18</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>Lamb &amp; Brannin. (60)</td>
<td>80</td>
<td>37</td>
<td>72</td>
<td>32</td>
<td>67</td>
<td>16</td>
</tr>
<tr>
<td>Patrick. (61)</td>
<td>50</td>
<td>1</td>
<td>45</td>
<td>21</td>
<td>22</td>
<td>16</td>
</tr>
<tr>
<td>Eichhorst. (62)</td>
<td>?</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whittingham &amp; Sims. (63)</td>
<td>23</td>
<td>54%</td>
<td>65%</td>
<td>82%</td>
<td>65%</td>
<td>44%</td>
</tr>
</tbody>
</table>
The above necessarily somewhat incomplete table represents the findings of different investigators in Britain, Europe, America and New Zealand. The results are not strictly comparable, for the methods of investigation are as a rule not stated in detail, and the object in view in most cases has been to demonstrate the presence or absence of the Influenza bacillus. The findings must necessarily vary with the experience of the investigator, the technique employed, the stage of the epidemic at which the specimens were taken, and the facilities for collecting and working up the specimens.

The general experience seems to have been that the Influenza bacillus was found in a varying percentage of cases, though by no means in all. For instance the earlier reports of German workers, including Pfeiffer himself, show that the Influenza bacillus was relatively seldom found in their earlier cases, while several American workers found it in a considerable percentage of their cases.

Very scanty reference is made in the literature at my disposal as to the presence of the Influenza bacillus in the nasopharynx of healthy persons. Fildes, Baker & Thompson in the paper quoted in the above table found 15 positive cultures in 71 apparently healthy persons — these results being obtained, however, during the epidemic period.

Pritchett and Stillman, reporting the results of an investigation to determine the incidence of B. Influenzae, found that in 177 persons who gave no history of having had influenza, 42 per cent had influenza bacilli in cultures from the throat or saliva.

Where the expectoration was scanty and collected with difficulty — as was the case in the earlier epidemic cases in New Zealand — the percentage findings were low, but as the wave declined the percentage findings rose. That this was our experience here will be seen later from the results of findings in post-mortem material.

Pneumococci and Streptococci were found in a much larger percentage of cases. In many of our cases capsules pneumococci seemed to be the predominant organism; but owing to the fact that these organisms are frequently present in normal mouths, percentage findings in Influenza cases are not of great value unless the specimens of sputa are collected without mouth contamination. In our cases, as has been pointed out, this was an extremely difficult matter. Thus
Simmons and Taylor (65), in an investigation into the bacterial carriers in the upper respiratory tract, found that in 3174 persons examined by cultural methods over half (56%) were carriers of the Streptococcus haemolyticus, and 25% were carriers of various types of pneumococci.

**Blood Cultures.** In our series of cases a student, who had had considerable experience in the technique of blood culture, was detailed to take cultures from each case on admission which the clinicians considered a typical case of pneumonic influenza. 18 cultures were thus obtained before the student fell ill, and it was impossible to replace him until the epidemic was on the wane.

**Technique:** 10 c.c. of blood was drawn during life and within a few hours of admission to hospital from an arm vein by means of a sterile syringe, previously washed out in boiling saline. Cultures were only collected when the temperature was over 101°F, and in most cases it was between 103°F and 105°F when the blood was withdrawn.

The blood was immediately ejected into a flask containing 100 c.c. of citrate peptone broth, and after thorough mixing of the blood and broth the flask was incubated at 37°C, for seven days. Subcultures on to fresh rabbit blood agar plates ready warmed in the incubator were made daily, and films stained by Gram. (N.B. The particulars regarding the medium of Matthews and the "K" medium, which gave much better growth of B. Influenza, had not at this time come to hand).

In no case was the Influenza bacillus found, and the pneumococcus in one case only. This case subsequently came to post mortem and proved to be one of lobar pneumonia, although both pneumococci and influenza bacilli were recovered from the lungs and bronchi.

These blood cultures did not suggest that the disease was of a septicemic nature at the time the examinations were made. That our results agreed with some and disagreed with others will be seen from the following table.
<table>
<thead>
<tr>
<th>Investigator</th>
<th>No. of cases</th>
<th>Influenza found in</th>
<th>Pneumococcus found</th>
<th>Other organisms found</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Influenza Committee, D.G.M.S., France (55)</td>
<td>18</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>510</td>
</tr>
<tr>
<td>Martin, C.J. (57)</td>
<td>68</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>40</td>
</tr>
<tr>
<td>McIntosh (57)</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Whittingham &amp; Sims (63)</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Orticoni &amp; Barbie (66)</td>
<td>50</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Lamb &amp; Brannin (60)</td>
<td>62</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Howard (67)</td>
<td>248</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>510</strong></td>
<td><strong>40</strong></td>
<td><strong>5</strong></td>
<td><strong>5</strong></td>
<td><strong>510</strong></td>
</tr>
</tbody>
</table>

**TABLE XVIII.**

**BLOOD CULTURES.**

- Influenza Bac. found
- Staphy. albus 1
- Streptococcus 5
- Streptococcus 1
- Repeated in several cases.

- Pneumococcus found in:
- 1
- +
Though the percentage findings of B. Influenzae differ considerably in sputum examinations, most workers have found it at one time or another during the epidemic - the varying percentages found lending themselves to one of several possible explanations.

In the blood culture group there is a general agreement in the negative findings both of Influenza bacilli and pneumococci, though the latter appears more frequently than the former, if for the present the results of Orticoni and Barbié are excluded. Thus the Influenza Committee to the D.G.M.S., France, reports a single positive Influenza blood culture by each of two workers only out of 68 cultures reported. No details are available as to methods employed or as to controls set up. Whittingham and Sims (Table XVIII), out of 50 cultures, did not find the Influenza bacillus once, and Lamb and Brannin in America (Table XVIII) out of the large total of 246 cultures similarly failed to find the Influenza bacillus. These authors express surprise at the small percentage of positive blood cultures in their cases during the epidemic, for in two previous series of pneumonia cases in 1918, the one of 92 cases and the other of 180 cases, they obtained pneumococci in blood cultures in 26% and 18% respectively. The technique was the same in each series.

In the light of these reports it is difficult to explain the results obtained by Orticoni and Barbié (Table XVIII). Out of 62 blood cultures the Influenza bacillus was found in 38, either in pure culture or along with pneumococci or streptococci. It seems impossible to conclude that their technique was so far in advance of that of the other workers mentioned that the latter missed the Influenza bacillus if present in the blood. Even considering that their cultures were all taken shortly before death and the latter some days before death (which was not the case), the difference could hardly be explained. If we are to accept their results as accurate and reliable (and there seems no reason to doubt them unduly if the Medical Research Committee has considered their work worthy of review in the Medical Supplement to the Review of the Foreign Press), one can only suggest that the local conditions, or the susceptibility of their patients or the virulence of the Influenza bacillus in their locality, may have had some influence in determining a septicemic condition which would appear not to have been the case in the other localities mentioned.
Note on the Peptone Broth used for Blood Cultures.

My failure to obtain any growth of Influenza bacilli or pneumococci in blood cultures except in one case, which coming to Post mortem proved to be a case of lobar pneumonia with grey hepatisation, led me to wonder whether the peptone which was used in making up the beef broth was responsible. A communication from the Research Laboratories of the Royal Institute of Public Health has shown(68) that since Witte's peptone has been unprocurable several of the brands of peptone on the market are unsuitable for bacteriological use, in that they retard bacterial growth, and results obtained with one sample of peptone in testing disinfectants by the Rideal-Walker method varied considerably from those obtained with other brands of peptone. While this might not matter with strongly growing organisms, it might make all the difference with the pneumococcus and influenza bacillus when, in addition, they would have to contend with any bactericidal properties of the added blood.

Several batches of broth prepared with three different brands of peptone gave easily recognisable growths of pneumococci from the heart blood of rabbits used for testing the virulence of the strains however, and a similar result was obtained from the heart blood of several human cases which was obtained post mortem and shown to contain pneumococci microscopically. I concluded therefore that the negative findings did not depend on the culture medium used.

The Bacteriology of Secondary Complications during Life.

These are of less interest from the epidemic standpoint except in so far as they show the absence of the Influenza bacillus in the exudates of empyema, meningitis, and other complications, and the frequency with which the other organisms which are generally regarded as secondary invaders were found. The Influenza bacillus tended to confine itself to the respiratory tract, and the pneumococci and streptococci spread from there to involve lung, pleura and meninges as the case might be.

Of the complications (other than pneumonias) which I had an opportunity of examining during life, there were four purulent pleural fluids, one middle ear abscess, and one cerebro-spinal fluid. These seem to
represent the chief complications found by Abrahams, Hallows and French in England (53), and by Lamb and Brannin (60) in America. The organisms found were as follows -

<table>
<thead>
<tr>
<th>No.</th>
<th>Lesion</th>
<th>Microorganism found</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Empyema fluid.</td>
<td>Pneumococcus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>M. 455</td>
</tr>
<tr>
<td>2.</td>
<td>P. 112</td>
<td>Streptococcus haemolyticus</td>
</tr>
<tr>
<td>3.</td>
<td></td>
<td>IV</td>
</tr>
<tr>
<td>4.</td>
<td>Otitis media, pus</td>
<td>Streptococcus haemolyticus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Staphylococcus aureus</td>
</tr>
<tr>
<td>5.</td>
<td>Cerebro-spiral fluid, pus</td>
<td>Pneumococcus.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not grouped</td>
</tr>
</tbody>
</table>

TABLE XIX.
Although careful search was made both in direct films and in cultures in the six cases tabulated above, the Influenza bacillus was not found. Two of the pneumococcal strains were grouped subsequently, as a fresh supply of the Rockefeller type sera was not received until some weeks after the epidemic. The pneumococcus mucosus (type III) was tested for virulence by injecting 0.5 c.c. of a thin emulsion of the empyema pus and saline intraperitoneally into a young (half grown) white rabbit. Death followed in 20 hours, and from the Heart-blood, Spleen and peritoneal exudate a capsulated diplococcus was recovered, which gave the characteristic slimy colonies of this type on blood agar.

In the paper of Orticoni and Barbis previously commented on for their remarkable number of positive blood cultures, the Influenza bacillus was found in the pleural fluid in 12 cases and in the cerebro-spinal fluid in two cases.

Silbermann, quoted in the same review, found the Influenza bacillus in almost pure culture in the empyema fluid of one case, and as the predominant organism in association with Streptococci or Pneumococci in another.

Hicks and Gray report finding the B. Influenzae in the pleural fluids in two cases and diplococci resembling Streptococcus mucosus in most though they do not mention the number of cases they investigated, nor do they differentiate between Pneumococcus mucosus and Streptococcus mucosus.
(2) Post-mortem findings - 25 cases.

In the chart accompanying the Post-mortem Summary there are set out in tabular form the principal organisms recovered from the lungs or bronchi of the 25 fatal cases of pneumatic influenza which I investigated with my colleague Professor Drennan.

In the following pages I have summarised the complete bacteriological findings in these cases in so far as I was able to work them out at the time, and the technique which was employed. Thereafter I have discussed at greater length certain aspects of these findings which I was able to work out in greater detail when the epidemic was over.

**Technique.** In every case the autopsy was made as soon after death as possible, generally within 4 hours of same. In two cases the examination was delayed for 12 hours. In every case smears and cultures were made from the heart blood, spleen, lungs and bronchi, and subcultures made where necessary.

**Heart blood.** At the autopsy the pericardium was opened aseptically, and the surface of the heart seared by the Pathologist. With a sterile syringe and needle the left ventricle was punctured and from 5 to 10 c.c. of blood withdrawn and distributed into two flasks each containing 100 c.c. citrate beef broth. A few drops were then spread over the surface of a plain agar plate and smears made for microscopic examination.

The culture flasks were incubated for 5 days and examined microscopically and by subcultures on rabbit blood agar plates daily when necessary. Any organisms found were then worked out in detail, the staining methods and culture media used depending on the microorganism or microorganisms under investigation.

The smears made in the post-mortem room were examined by Gram's stain, two smears from each case being prepared by this method; and where diplococci resembling the pneumococcus were found, smears were stained by Muir's modification of the Pitfield Capsule stain.

**Spleen.** The capsule having been seared and punctured with a sterile knife, smears from different portions were made and several loops full of pulp inoculated into two tubes of peptone broth. A rabbit blood agar plate was also inseminated direct
from the pulp. After preliminary incubation a further rabbit blood agar plate was inoculated from the broth spleen pulp emulsion. Cultures were examined daily for five days and subcultures made where necessary. The smears were stained by Gram, Leishman, or by the Muir's-Pitfield capsule stain, similarly to the Heart blood and other smears.

Lungs. In many of the earlier cases the lungs when removed from the body were dripping with haemorrhagic oedema fluid, and it was difficult to obtain material for examination free from bronchial contamination. Sections were made with a sterile knife through the seared pleura and material taken for films and cultures as far away from any of the bronchi as possible. The procedure subsequently carried out was similar to that already indicated for the Spleen.

Bronchi. The trachea and larger bronchi having been laid open so as to avoid contamination, broth cultures and blood agar plates were inseminated with the aspirated mucus or mucopus if present in sufficient amount, or from a large platinum loopful obtained by rubbing the loop over the surface of the mucosa when the secretion was scanty. Films were prepared from the trachea and from the larger and smaller bronchi, and in all cases stained by Gram, Leishman, and the capsule stain. When the bacterial flora as revealed in direct films was profuse, care was taken in spreading blood agar plates from the broth tubes inseminated in the post-mortem room, to have the resulting colonies sufficiently isolated to facilitate recognition. It was unfortunate that the excellent K medium described by Fildes, Baker and Thompson in the paper already quoted was not known to me until the epidemic was on the wane, or the work in investigating the Influenza bacillus would have been much simplified, and my percentage findings probably increased. A note of this medium is appended.

Criteria adopted in differentiating the microorganisms found. It soon became evident that in the majority of cases either the Influenza bacillus, a gram positive capsulated diplococcus resembling the pneumococcus, or the Staphylococcus aureus, was the microorganism most frequently and most profusely present in the lungs and bronchi of the fatal cases. Streptococci were relatively few in numbers even in those cases in which they were demonstrated, so that in the limited time available I did not differentiate them other than by noting their appearance on blood agar plates (i.e. appearance and size of colony,
haemolysis or other change in the blood present, and growth in peptone broth and litmus milk) and the microscopical appearances of the growths on these media.

In determining the presence of the Influenza bacillus, the morphology in tissues and cultures, staining reaction with Gram stain, the appearance of colonies and rate of growth on blood agar, and absence of growth on non-haemoglobin containing media, were considered sufficient under the circumstances. Symbiotic growth with the Staphylococcus aureus, which was frequently present in cultures from the lungs and bronchi, assisted in the recognition of the bacillus in several cases, but the pneumococcus did not seem to influence its growth one way or the other.

The importance of keeping the culture plates in the incubator and examining them daily for five or six days was brought home to me during the routine examination of the culture plates from the lungs and bronchi, particularly the latter. I noticed in two of the earlier cases which I had, after the third day's examination, noted as showing no growth of Influenza bacilli, that as late as the fifth day several Influenza-like colonies were showing when the plates were scanned with the hand-lens, whereas they had not been visible by this method previously. Subsequently no plate was recorded negative as far as the Influenza bacillus was concerned until after the fifth or sixth day in the incubator. Generally the colonies were easily recognised after 48 hours incubation, as the plates I used were rather deeper than usual and thus the drying effect of the incubator temperature was minimised as far as possible.

Note on "K" medium for growth of the Influenza bacillus. In their paper on the Pathology of the Influenza Epidemic already quoted, Fieldes, Baker and Thompson state that they were using a digested blood medium for growth of the Influenza bacillus as recommended by Matthews of the St. Mary's Hospital Laboratory. They state that they gave this up for the medium of Levinthal, which is simpler to prepare. They based the preparation of their medium on the reference to Levinthal's work in the Daily Review of the Foreign Press (October 1st, 1918) compiled by the Medical Research Committee.

The principle of this medium is the addition of fresh blood to a flask of melted agar in the proportion of 1 in 20. The medium is brought to the boil and the coagulum aseptically filtered off through
"gauze or paper."

Fildes and his co-workers state that there are factors in the preparation of this medium which are still obscure and that they have several times made up batches which are entirely useless. They are satisfied that the factor in these cases is the filtration through gauze or paper. Neither of these substances should be used but glass wool only, presumably because the paper absorbs the essential vitamins (Jordan Lloyd).

I can corroborate these remarks, for batches not prepared strictly according to directions are liable to give very disappointing results. Thus filtration through paper in the ordinary hot water funnel gave a medium which was inferior to fresh blood agar. On two other occasions, being unable to obtain fresh human blood, I used the blood we had obtained from the Abattoirs for sheep cells for the Wassermann test. Knowing this blood would not be sterile, I steamed the tubed medium after filtration with disastrous results as far as growth effects were concerned.

The technique of preparation given by the authors in their paper must therefore be carefully followed, and I prefer to incubate my culture tubes as well as plates overnight, as it is not easy, owing to the method of preparation, to avoid contamination of a few in each batch.

The effect of this medium on the growth of the influenza bacillus is remarkable. The effect of adding the soluble products of a small quantity of boiled blood to ordinary agar is to produce either a copious confluent growth in 24 hours or discrete colonies of from 2 to 4 mm in diameter. The growth is as copious as, or even more so, than that of the meningococcus on serum agar, and is quite distinct from that of pneumococci or streptococci, neither of which seem to be particularly influenced in their growth by this medium.

For the preparation of influenza vaccine this medium is excellent, for as much emulsion is obtained from a single slope as would be obtained from a dozen or more slopes of ordinary blood agar.

In differentiating the pneumococcus some difficulty was at first experienced, for the primary colonies on rabbit blood agar were unusually large, measuring from 1 to 1.5 mm in diameter after 48 hours incubation and in the water of condensation in blood agar slopes to which the colonies were transferred for further investigation, unusually long chains of lanceolate diplococci occurred. All cultures of this organism were passed through Hiss's inulin-serum-water and tested for bile solubility. Virulence was
tested in young rabbits in several cases where bile solubility was delayed or incomplete. Unfortunately the Rockefeller type sera were not available at the time, as I had experienced considerable difficulty and much delay in renewing my stocks. A tentative classification only could be made at the time, depending on morphology, gram staining, appearance of cultures on blood agar, on agar, and in broth, fermentation of inulin, and bile solubility and virulence for the young white rabbit. White mice were not procurable at the time, owing to an accident whereby a laboratory boy liberated the breeding stocks during the previous holidays.

Summary of Bacteria found in 25 fatal cases with the Technique detailed above.

TABLE XX.

<table>
<thead>
<tr>
<th>Microorganisms</th>
<th>Heart Blood</th>
<th>Spleen</th>
<th>Lungs &amp; Bronchi (grouped)</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. Influenzae</td>
<td>0 cases</td>
<td>0 cases</td>
<td>20 cases</td>
</tr>
<tr>
<td>Pneumococci</td>
<td>6</td>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td>Staphylococcus Aureus</td>
<td>3</td>
<td>4</td>
<td>17</td>
</tr>
<tr>
<td>Streptococci</td>
<td>2</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Friedlander’s Bacillus</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>B. Aerogenes Capsulatus</td>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Mic. Catarrhalis</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Diphtheroid bacilli</td>
<td>1</td>
<td>0</td>
<td>- not noted</td>
</tr>
</tbody>
</table>

At the time this work was done there were several references, in the European literature available, to a gram positive diplococcus which differed from the pneumococcus, but which was frequently associated with the Influenza bacillus in the lungs and bronchi of fatal cases.
The pneumococci which I isolated and which were present in profusion in the lung juice of the earlier haemorrhagic oedema type of case, also differed from the pneumococcus with which I was familiar in routine work.

At the time I was unable to do more than note the appearances of this diplococcus in the cultures used for the investigation, but cultures were prepared from typical cases and put aside for further study. A stock culture served as a control. In this way 15 cultures were available for study obtained from the following sources.

**TABLE XXI.**

| Blood culture | Case B.115. (This case subsequently came to post mortem. (P.M. 4-676).) | 1. |
| Empyema fluid | Cases M.433 and P.112 | 2. |
| Lung puncture | Two cases not included in the post mortem series in which the lungs were punctured just after death. L.P.1, L.P. 4. | 2. |
| Post mortem cases | 1-673, 3-675, 4-676, 15-689, 19-693, 20-694. | 6. |
| Cultures obtained from heart blood of rabbits after virulence tests. P.M. 3-675, P.M. 4-676, P.M. 19-693. | 3. |
| Stock culture | pneumococcus isolated from Empyema case several months previously. | 15. |

These cultures were put up on rabbit blood agar slopes previously tested for sterility, capped, and left at incubator temperature (37°C) and transferred to fresh slopes every 5 or 6 days. While waiting for supplies of Rockefeller type pneumococcus sera and a further supply of white mice, they were plated out to determine their purity.
Unfortunately, it was several weeks before these supplies came to hand, and meantime the cultures were kept going and experiments made with peptone broths of varying reactions and different composition to obtain a broth giving the most copious growth of pneumococci for bile solubility and agglutination tests. While one undoubtedly obtains clearer results when pneumococcal emulsions are prepared for agglutination and bile solubility, from surface slopes, there is the added burden of washing each emulsion, for Avery, Chickering, Cole and Dochez(70) state that the presence of serum in pneumococcus cultures inhibits or completely arrests the lytic action of bile. I was therefore anxious to obtain a sufficiently copious growth in broth to answer both purposes and to avoid the use of solid culture growths if possible. Satisfactory and uniform results not being obtainable with all strains with the ordinary peptone broth in use in the laboratory, I made up a batch of the carbonate beef infusion dextrose broth recommended by Hiss(71) and several strains were tested for rapidity and volume of growth in this. At first sight a copious growth resulted but on examination it was found that the turbidity which developed on incubation depended on the presence of the carbonate, even when coarse particles only were added to the culture medium. The growth of pneumococci was not more marked than in ordinary broth and the turbidity rendered the growth quite unsuitable for my purpose. I was unable to overcome this turbidity, although I followed the directions given by Hiss very carefully. For this reason I abandoned this broth and experimented with a beef infusion broth made up without carbohydrate, and titrated against phenolphthalein both when hot and when cold.

I finally found that, using Witte peptone, of which I was able to obtain a small supply, a beef broth of a reaction +10 to Eyre's scale, and sterilised in the steamer on three successive days, gave sufficiently satisfactory growths of the cultures in 18 hours for my purpose. If growth was continued for 24 hours, there was a gradual clearing of the fluid due to autolysis of the cocci. It is probable that I obtained a more copious growth than I should have done with freshly isolated cultures, for by this time the cultures had been transferred several times, and were accustomed to growth outside the body.

Having obtained a suitable culture medium, the subsequent technique employed in investigating these pneumococcus cultures was as follows -

(a) Growth in Inulin serum water. (b) Bile solubility. (c) Agglutination with Rockefeller sera
Types I, II, and III.

(a) Inulin serum water was prepared according to the method of Hiss (see Rockefeller Monograph 7, pp. 20) with 1 per cent inulin. Duval and Lewis(72) have noted inconsistent results with different makes of inulin, some of which were shown to contain dextrose; and they noted considerable difference in the ability of their pneumococcus strains to ferment this carbohydrate. They recommend replacing the inulin serum water with inulin serum broth advised by Theobald Smith. I found no difficulty with this medium, however, prepared as recommended by Avery and his co-workers.

(b) Bile solubility tests. In carrying out this test a 10% solution of sodium taurocholate in normal saline was added to an actively growing 18 hour broth culture of the pneumococcus in the proportion of 1 c.c. to 10 c.c. culture. A control of an equal portion of the same culture was set up in an exactly similar tube for purposes of comparison. Solution was usually rapid, and, where delayed more than a few minutes, the tubes were placed in the incubator.

My experience with the bile solubility test has not been altogether satisfactory. Three of the cultures under investigation failed to clear completely, although different brands of bile salt were used, and finally emulsions of washed cocii from the surface of blood agar slopes were tried. The culture medium and the bile salt used seemed to make no difference, but the failure to completely dissolve seemed an inherent part of the particular strain tested. Others have noted the same difficulty, so that this test has only a relative value and must be considered along with other methods. Neufeld, quoted by Avery and his co-workers(70) reports the insolubility of certain avirulent strains of pneumococci, but Avery found that all of several hundred strains isolated from cases of pneumonia were dissolved, and he places considerable reliance on the bile reaction.

(c) Agglutination tests. Workers at the Rockefeller Institute, New York, (70) have shown that pneumococci fall into two great classes, the larger of which comprising about 80% of all strains encountered in disease consists of three subgroups, designated I, II, and III. Stillman(73) reports that there are at least twelve subgroups of atypical Type II pneumococci. Type III consists of the Pneumococcus Mucosus, an organism distinguished from the other types by morphological, cultural, as well as immunological differences. The smaller of the two main classes
(Type IV) represents about 20% of the strains isolated from cases of lobar pneumonia, and consists for the most part of strains which are not interrelated. They are said to be less virulent than the other fixed types and to be frequently encountered in the mouth secretions of normal individuals.

The technique employed in the agglutination tests with my strains was that recommended by Avery, Chickering, Cole and Dochez (70), pp. 25. 18 hour broth cultures were used and when the majority of the strains appeared to belong to group IV, i.e. failure to agglutinate with type 1, 2, and 3 sera, I repeated the whole series, using the washed growth from blood agar slopes in normal saline in place of the broth cultures. The results were, however, the same.

The pneumococci isolated from cases of pneumonia following influenza have fallen largely in this group, and for this information we have to look largely to the American literature. Thus Opie, Freeman, Blake, Small, and Rivers, in a report to the Surgeon-General on Pneumonia following Influenza at Camp Pike, U.S.A. (74) found pneumococci type IV in 53.6 per cent, type III in 7.7%, type II in 4.9%, atypical type II in 21.7%, and type I in 13.1%. Similar results are quoted by Medalia (75), who determined the type of pneumococci from 440 cases of influenzal pneumonia, and found the larger percentages to be represented by Type IV 85.8% and type II (atypical) 3.4%.

Of the types found among the British armies in France, Sladden (56) found those he studied in detail to conform to type IV of the Rockefeller classification.

Of our New Zealand strains, I was only able to type 15, including one control, and of these, as will be seen from Table XXII, 10 conformed to type IV, 2 to type II (atypical), 1 to type III, and the control to type I. One culture (L.P.1) was contaminated before it came the length of the agglutination tests for a streptococcus survived, and this culture was lost for purposes of comparison.

Of my two atypical type II strains, which were both from the same case 19-693, one from the lungs and the other after passage through a rabbit, I was at a loss to group them seeing that at first sight they appeared to belong to group IV, not being agglutinated by type sera I, II, or III after one hour incubation at 370 as recommended by Avery (70), (pp. 25). After standing in the ice-chest overnight, typical
agglutination of the emulsion had taken place with type II serum, quite different to the slight sedimentation which had occurred in the other tubes. I referred these results in detail to Cole of the Rockefeller Institute, who has just replied to the effect that he considers them Atypical type II strains.

It was unfortunate that I was unable to group my stains of pneumococci at the time of the epidemic, for more than one authority has suggested that the types are not stable and are liable to vary under cultural conditions. Thus Rosenow(76) noted that the pneumococcus-streptococcus group of organisms underwent definite changes in morphology, infecting power, and immunological reactions when subject to the influence of an altered environment.

See Table XXII, on following page.
### TABLE XXII.

**PNEUMOCoccus CULTURES.**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Gram stain.</th>
<th>Capsules.</th>
<th>Growth on blood agar.</th>
<th>Inulin fermentation</th>
<th>Bile Solubility</th>
<th>Type I</th>
<th>II.</th>
<th>II.(atyp.)</th>
<th>III.</th>
<th>IV.</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.433</td>
<td>+</td>
<td>+ marked</td>
<td>Typical flat col.</td>
<td>+ 60 hrs.</td>
<td>+ partial</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>L.P. 1</td>
<td>+</td>
<td></td>
<td>Proved later to be a Streptococcus - discarded</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.P. 4</td>
<td>+</td>
<td>+</td>
<td>Typical flat col.</td>
<td>+ 48 hrs.</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 1</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 3</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 3 rabbit</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 4</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 4 rabbit</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>B.115</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 15</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>+ 60 hrs.</td>
<td>+ partial</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 19</td>
<td>+</td>
<td>+</td>
<td>Smaller than above</td>
<td>+ 48 hrs.</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 19 rabbit</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.M. 20</td>
<td>+</td>
<td></td>
<td>&quot; &quot;</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>P.112</td>
<td>+</td>
<td>+</td>
<td>Typical flat col.</td>
<td>&quot; + &quot;</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Stock culture</td>
<td>+</td>
<td></td>
<td>Smaller than above</td>
<td>+ 60 hrs.</td>
<td>+ +</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

---

*(Note: Certain immunological observations made during the recent epidemic also lent support to the view that the E. Influenzae was the cause of the Epidemic (this, however, cannot be said to support the...)*
DISCUSSION OF RESULTS.

The bacteriological findings summarised above do not add any further information as to the cause of epidemic influenza, or as to the role of the Influenza bacillus in etiology. They merely indicate what groups of bacteria were most frequently encountered in the epidemic as it presented itself in New Zealand, and show that these correspond more or less with those found by numerous workers in other lands. They also indicate that the secondary organisms depended to a certain extent on a working up in virulence of local strains, and that the same group of organisms did not assume like virulence in all countries. As regards the role of the Influenza bacillus in etiology, present opinion is divided along two lines - one holding that it is the primary infecting agent and that the pneumococcus or streptococcus and other microorganisms (such as the Staphylococcus Aureus locally) gained sufficient virulence to account for the invasion of the tissues and many of the fatal pneumonias and other complications; - the other holding that the primary infecting agent is an unknown virus possibly belonging to the class of filter-passing viruses, and that the influenza bacillus is to be grouped with the pneumococcus-streptococcus secondary organisms.

Numerous authorities might be quoted in support of either view, but the opinions of a few will suffice. Thus the report of the Influenza Committee of the Advisory Board to the D.G.M.S., France, (56) concludes with a consideration of the evidence pointing to the B. Influenzae as the cause of the Epidemic Influenza, and amongst other evidence states -

(1) That the Influenza bacillus is frequently found in the respiratory tract of persons suffering from the disease.

(2) The failure to recover the bacillus from early cases, or cases early in the disease, may be attributed to the bacilli multiplying in the mucous membrane of the trachea and bronchi without at first producing an excess of reactive secretion. (Others blame inexperience of the worker or faulty technique, or unsuitable culture media).

(3) Certain immunological observations made during the recent epidemic also lend support to the view that the B. Influenzae was the cause of the Epidemic (this, however, cannot be said to support the
primary role of the influenza bacillus, but merely helps to establish its etiological relationship as an infecting agent).

Spilsbury(77), in discussing the morbid anatomy of the disease, said he regarded the condition as a primary infection of the air passages by Pfeiffer’s bacillus, the failure to find the organism in 40% of his post mortem cases being due either to the search not being sufficiently thorough, or to the organism having disappeared before death. Following this infection an invasion of the air passages and linings by pneumococci or streptococci occurred, one of these organisms being responsible for the pneumonias. The changes found elsewhere in the body were due to acute toxaemia, and in no case had he found a pyaemic or septicaemic condition.

Opie and his co-workers(74), in the report already quoted, suggest a similar sequence of events, i.e. the Influenza bacillus descends into the bronchi, pneumococci, usually type IV (in their experience) invade the infected bronchi, then the lung, and produce pneumonia. Haemolytic streptococci may descend and infect the pneumonic lung, or they may infect apart from pneumococci.

We have then in these views a reasonable explanation of the sequence of events - a primary injury to the mucosa of the respiratory tract by the influenza bacillus with the production of toxins, which on absorption produce a general malaise. In a certain percentage of cases, probably depending both on the virulence of the influenza bacillus itself and of the secondary organisms, an infection of the damaged tissues takes place, resulting in one or other form of pneumonia.

Conclusive evidence against these views and in support of the contention that the Influenza bacillus is of secondary import is still lacking, although there is a strong body of opinion in its favour. We may for purposes of illustration liken the former views to those once held with regard to the etiological significance of the B. Suipestifer in Hog Cholera, which we now know not to be the primary cause. As regards Influenza it is probable that we shall have to wait for another epidemic to solve the problem, for our materia morbi quickly appears, and as quickly disappears, at least in epidemic form.
Two authoritative opinions in support of the contention that the Influenza bacillus is secondary to an unknown virus will suffice.

The Medical Research Committee (78) in an exhaustive summary of the literature dealing with the influenza epidemic in Europe up to September 1918, survey the facts and opinions marshalled in the various laboratories of Europe and arrive at the conclusion that the influenza bacillus appears to have been recovered much more frequently from soldiers in the battle zones than from civilians. Nevertheless, they hold that the "cold logic" of the post mortem room leaves no doubt that the bacillus of Pfeiffer does not play any more important part than does the ubiquitous diplo-streptococcus commonly reported about that time. The real virus, said to be invisible or a filter passer, on account of its unknown nature, remains to be discovered. W. H. Park, Director of Laboratories, New York City Health Department (79), summarising several representative American investigations, states that the results appear to throw the influenza bacilli in the cases studied into the class of secondary invaders, and that other microorganisms, such as certain streptococci and pneumococci which are under suspicion in different localities, will be found not to possess the necessary identity of characteristics to allow them to remain under serious consideration as the primary agent in the epidemic, but rather, like the influenza bacillus, to be reckoned among the most important of the secondary invaders. His final conclusion is that the microorganism causing this epidemic has not yet been identified.

What direct evidence can be brought forward in support or otherwise of the latter views? Or, in other words, has an invisible virus been demonstrated to the satisfaction of all?

Some years ago, apart from the question of epidemic influenza, it was contended with some experimental proof that common colds were caused by a filter passing virus. The first to bring forward evidence of a filter passing virus as the primary cause of epidemic influenza was, I believe, Nicolle, (80) but his experimental proof was meagre. Still it drew the attention of others to this possibility, and several papers have been published on the subject. All lack sufficient experimental proof, a few infected monkeys usually being the only experimental animals available, whereas it would be difficult to avoid the criticism, if human material were used for inoculation experiments,
that in the presence of an epidemic they were not naturally infected.

An interesting summary of an experimental research in Japan is reported by Yamanouchi, Sakakami and Iwashima.\(^\text{(81)}\) An emulsion of the sputa from 43 influenza patients was made in Ringer's solution and this emulsion injected into the nose and throat of 12 healthy persons. A filtrate of the same emulsion was injected into the nose and throat of 12 other healthy persons. Of these 24 persons, 6 had influenza and the others apparently had not. The six were not affected, while all the other 18 subjects were attacked by the disease after an incubation period of two to three days. Similar results were obtained with a filtrate of the blood of influenza patients, but no infection followed the application of pure cultures of influenza bacilli, pneumococci, and streptococci recovered from the sputa of influenza patients. The authors conclude that the virus of influenza is a filterable virus.

Recently considerable attention has been directed to a preliminary report by H. Graeme Gibson\(^\text{(82)}\) and to a later report by Bradford, Bashford, and Wilson\(^\text{(83)}\) on the presence of a filter passing virus in certain diseases, including Influenza.

The apparent ease with which these workers recovered what were held to be filter passing viruses in a number of diseases of obscure etiology caused one to wonder why they had not been discovered before, although the experimental proof, other than by cultures, was meagre.

It remained, however, for Arkwright\(^\text{(84)}\) to discredit the findings of these workers, and to show that they were mislead by impurity and natural degenerative changes in their cultures. It seems amazing that the fallacies noted by Arkwright had not been detected before this work was made public, and emphasises the feeling which was engendered here with regard to most of the work on filter passing viruses in connection with Influenza, that the different workers were in too great a hurry to be the first in the field to announce what would be, if true, a notable discovery.

There remain but two further observations in connection with this bacteriological summary. It is noteworthy that whereas in many of the published reports in Great Britain the Microoccus Catarrhalis occupied a prominent place, it was only met with three
times in the lungs or bronchi of my 25 fatal cases. It is to be expected that returns including sputum examinations and nasopharyngeal swabblings would show a higher percentage finding of this microorganism than those excluding these sources of contamination. On the other hand the Staphylococcus aureus was prominent in my series, being present in the lungs and bronchi in 17 out of 25 cases examined, and in one case (17-691), except for a few pneumococci, it was the only organism found in these situations and that in great numbers.

Patrick (61), whose paper has already been referred to, found it prominent in his cases in Malta, it being present post mortem in the heart blood or lungs of 10 out of 11 fatal cases in which broncho-pneumonia was a complication of Influenza.

Chickering and J. H. Park Junior (85) have endeavoured to establish a grave etiological role of Staphylococcus aureus in the pneumonia following Influenza at Camp Jackson, U.S.A. They deal with 153 cases of what they term Staphylococcus pneumonia. In 61 cases, however, other organisms such as Influenza bacilli or pneumococci were found associated with the Staphylococci. In 14 cases cultures of Staphylococcus aureus were obtained from the lungs after death and when death took place late in the cases with Staphylococci, numerous small abscesses were found in the lungs. This was the appearance found in one case (17-691). These cases serve as a further illustration of the fact that in different localities different microorganisms assumed importance as secondary invaders, whereas in other localities, although the general effects of the epidemic were as severely felt, a somewhat different secondary flora was responsible.

We remain ignorant then of the true cause or causes of epidemic Influenza, although the work of the last year has indicated useful and promising lines along which research might be profitably directed in the future.
1. New Zealand suffered from the 1918 Pandemic of Influenza in common with the rest of the world. The local manifestation consisted of a primary wave in August and September, and a secondary wave of great severity, which, commencing in Auckland about October 26th spread south, covering the whole Dominion in about two weeks.

2. Meteorological conditions predisposing to an undue prevalence of respiratory catarrhs during the year were an excessive rainfall and prevailing southerly or south-westerly winds.

3. The low crude death rate, the low infantile mortality rate, the absence of overcrowding and the healthy conditions under which most of the people live indicate that epidemic diseases should not obtain as favourable a soil for dissemination as is the case in countries less favourably situated.

4. The New Zealander is less resistant to Respiratory and Catarrhal diseases than the Britisher, and this fact probably accounted for the undue severity of the secondary wave of the pandemic.

5. The primary wave was characterised by a morbidity and case mortality in excess of the average for the years immediately preceding, but it left no impression on the minds of the people.

6. The secondary wave was characterised by excessive morbidity and mortality and affected New Zealand more severely than most other countries, with the exception of South Africa.

7. The incidence and case mortality was excessively heavy on the Native race as compared with Europeans.

8. The influence of age and sex is seen in higher death rates in males than in females and in both sexes between the ages of 20 - 45 years. Similar results have been recorded in all other countries.

9. There is no definite proof that any particular vessel brought the secondary wave to New Zealand, although popular opinion blames the R.M.S. "Niagara". Influenza was prevalent in a severe form in the Dominion before the arrival of this steamer, and it is probable that the arrival of
several overseas ships about this time with returning troops, each added its quota towards the massing infection.

10. The position in Australia is interesting in that a rigid maritime quarantine, Interstate quarantine, and compulsory wearing of masks, and free public vaccination seem to have delayed the advent of the second wave and to have modified the severity of its incidence.

11. The Administrative measures adopted to combat the Epidemic were similar in essential details to those adopted elsewhere. The division of the larger cities into blocks to which one or more medical practitioners were attached prevented overlapping in medical attention. The bureaux established in the cities assisted the Medical Officer of Health in the medical and lay organisation, and were of great value.

12. Provision should be made for a return of the epidemic and the organisation set up, with improvements which experience suggested should be kept in touch with, but there is no indication that New Zealand will be visited by such a severe epidemic until the next world pandemic.

13. The prophylactic use of medicated sprays is a measure of doubtful administrative value. A drug which has a selective action on the influenza virus has yet to be found. The public spray probably allays panic, and satisfies the public that something is being done. If used, open well ventilated rooms should be provided, and the spray generated by compressed air. Steam jets should be avoided as they raise the temperature and humidity of the chamber and predispose to chill.

Provision should also be made to "space out" the subjects, both those waiting and those actually in the inhalation chamber, to avoid cross infection as far as possible.

N.B. Since writing the above, Gregor's work on the effect of gas impregnated atmospheres has come to hand, but it is too early yet to say whether that line of research will evolve any satisfactory means of personal or public prophylaxis.
14. Face masks are a useful means of prophylaxis for medical attendants, nurses, and those coming into intimate contact with patients. They should be made of at least four and preferably six layers of fine mesh gauze, such as butter muslin, and of a sufficient size to cover the mouth and nose and mould into the contours of the face. An ample supply should be available, so that each mask is only worn once and then placed in a convenient receptacle ready for sterilisation. The wearing of masks by the public is a matter for personal consideration, and may give confidence to the excessively nervous. It is not a measure which is likely to have any influence in staying the spread of an epidemic.

15. Prophylactic vaccination with mixed catarrhal vaccines is not a measure which can be offered to the public with any prospect of success, even if sufficient supplies could be procured in time. Vaccination should be attempted in the case of troops, and in institutions or more or less isolated communities, and in these cases is likely to be of value. There seems no reason to withhold vaccination in face of an epidemic, although care should be taken to avoid excessive dosage in view of a possible negative phase for several days after the larger second dose.

16. Pathologically the fatal cases could be roughly grouped under two heads, (1) Those in which there was intense toxaemia with marked haemorrhagic oedema of the lungs as chief features, catarrhal pneumonia being present in parts but only demonstrable microscopically, and (2) Those cases in which there was definite pneumonia, either broncho-pneumonic or lobar in type. The haemorrhagic oedema was present in several type II cases, but was a much less marked feature than in type I.

17. A pneumococcus showing certain unusual cultural features and failing to agglutinate with the type sera of the Rockefeller Institute (type IV), was found in considerable numbers in the haemorrhagic oedema fluid, more especially of the earlier cases.

18. Microscopically type I cases were characterised by a general engorgement of the vessels, haemorrhage and oedema into the alveoli. Definite pneumonic consolidation was absent in most parts
though prolonged search revealed small areas of catarrhal pneumonia. There was intense bronchitis and frequently tracheitis. The lymph glands showed evidence of intense toxic absorption.

In the pneumonic type (type II), all grades of pneumonic consolidation were met with, from small patches of definite broncho-pneumonia to large areas of the lobar type. The cellular reaction in these varied considerably, in some it was intense, in others scanty, the majority of the cells being polymorphonuclear leucocytes. The variety of appearances may depend on the different microorganisms, as in those cases where Staphylococci were found microscopic abscesses were not infrequent. In these pneumonic cases the haemorrhagic oedema appearance was present in parts other than those actually pneumonic.

19. Sputum examinations were not found satisfactory on account of the difficulty of collecting specimens during the earlier part of the epidemic wave. The flora during this stage was largely pneumococcal, but towards the close of the epidemic this microorganism was much less prominent and the Influenza bacillus became increasingly evident.

20. Blood cultures with one exception were sterile and did not indicate that the disease was in the nature of a septicaemia. This seems to have been the general experience, but in isolated instances influenza bacilli were recovered in blood cultures.

21. No particular microorganism seems to have been responsible for the secondary complications (excluding pneumonias). Influenza bacilli were not found except in the respiratory passages and lungs.

22. In 25 fatal cases, influenza bacilli were found in the lungs and bronchi in 20 cases, pneumococci, mostly type IV, in 19 cases, and the Staphylococcus aureus in 17 cases. Streptococci did not play any great part in these cases. The pneumococci were found in profusion in the earlier cases, but less frequently in the later cases. The Influenza bacillus was more prominent and more readily found in the later cases.
23. In a further study of 15 strains of pneumococci, 2 from empyema fluids, 2 from lung punctures in fatal cases not included in the 25 post mortem cases, 10 from post mortem cases and 1 a stock culture, it was found that 10 belonged to group IV Rockefeller classification, 1 to group III, 2 to group II (atypical), 1 to group I. One culture proved to be a Streptococcus.

24. The Bile Solubility test is useful in differentiating pneumococci from streptococci, but certain strains are only partially soluble. This may be the fault of the culture medium used or of the bile salt, but there was no indication that this was so.

25. Inulin was fermented in Hiss's serum water by all the strains tested. The rate of coagulation of the medium varied with different strains, some being very active and others delayed. It was complete in three days with all the strains tested.

26. Opinion is divided as to whether the Influenza bacillus or an unknown virus is the cause of Epidemic Influenza, though the latter view is the more generally accepted. Definite proof that a filterable virus is the cause is still required.
REFERENCES.


10. Compiled from respective returns of Registrar-General, England & Wales, (quoted report Director-General Medical Services, N.Z., 1919) and the Registrar-General, New Zealand.


12. Special Report to District Health Officer, Auckland, 1919.


15. Personal Communication.

31. Hill. - ibid. pp. 239.
46. Allen, R.W. - "Practical Vaccine Treatment". pp. 95.


DESCRIPTION OF PLATES AND PHOTOMICROGRAPHS.


Lung. Presents a uniform dull red appearance more prominently red in the lower lobe. This is the appearance presented by the haemorrhagic oedema type. The upper portion is more grey owing to the oedema being more pronounced there. There is a haemorrhage in the pleura along the lower margin. There is no pleurisy.

PLATE II. (Coloured) Trachea & bronchi. Case 680.

Trachea & bronchi. Show congestion, increasing in intensity from above downwards. Along the trachea and the bronchi appear swollen lymphatic glands dotted with haemorrhages.

PLATE III. (Coloured). Case 686.

Lung. This illustrates one variety of the pneumonic type. (Type II of text). In upper lobe are red patches of haemorrhage, following the distribution of the smaller bronchi. Mingled with these there occur in places yellow points of purulent bronchitis and early abscess formation. The rest of the lobe is pinkish grey from haemorrhagic oedema. The lower lobe shows more extensive haemorrhagic broncho-pneumonia and intense bronchitis in the large descending bronchus. The ground work is the same as the upper lobe, i.e. haemorrhagic oedema. There are haemorrhages in the pleura but no pleurisy.

PLATE IV. (Coloured). Case 693. Lung. Another variety of the pneumonic type.

The larger portion is from left lung - the smaller from base of right lung.

Left Lung. (Larger portion). There is a typical shaggy exudate of pleurisy over the surface. The cut surface (which represents the lower lobe of this lung) shows an extensive area mottled grey and pink of confluent broncho-
pneumonia. In parts the individual broncho-pneumonic patches can be distinguished. The base shows mainly haemorrhagic oedema.

Right Lung. (Smaller portion). Presents mostly the red appearance of haemorrhagic oedema, with several grey patches of actual pneumonia. There is no pleurisy in this portion.

PLATE V. Photomicrographs. (Case 673). Haemorrhagic oedema.

Fig. 1. Lung. The alveoli are filled with red corpuscles.

Fig. 2. Same case. Trachea shows tracheitis (a) ragged shed epithelium, (b) engorged vessels. There is general oedema of the tissue.

Fig. 3. Case 680. Portion of large bronchus, (a) haemorrhage. Over this the epithelium is ruptured and disintegrated.

Fig. 4. Same case. Lung, showing alveoli containing many red corpuscles, more abundant at (a).

PLATE VI.

Fig. 5. Case 686. (a) indicates lumen of a small bronchus which shows an intense bronchitis. The dark area around consists of engorged vessels and haemorrhage into the alveoli. The looser tissue is lung tissue with inflammatory cells in the alveoli.

Fig. 6. Same case. Shows a similar appearance round a smaller bronchus. (a) indicates bronchus disintegrated and filled with pus. (b) is a haemorrhagic patch, and (c) the pneumonic lung adjacent.

Fig. 7. Same case. Lymph gland from along trachea. The dark patch (a) is a haemorrhage, the looser tissue (b) is a greatly engorged sinus containing inflammatory cells.
PLATE VI. (Contd.)

Fig. 8. Case 676. Portion of lung from a definite pneumonia showing the usual consolidated alveoli and (a) a small bronchus filled with inflammatory exudate and shed epithelium.

PLATE VII.

Fig. 9. Case 693. Portion of consolidated lung showing the alveoli filled with inflammatory cells.

Fig. 10. Same case. Lymph gland from along bronchus. The gland structure is obscured from extensive haemorrhage and inflammatory cells filling the sinus system.

Fig. 11. Same case. Shows typical bronchitis with marked engorgement of the vessels (seen as black areas) and consolidation of the adjacent alveoli.

Fig. 12. Case 691. Termination of small bronchus, whose lumen is indicated (a). The distal portion is dilated from destruction of the walls, lining which are masses of Staphylococi (b). Similar masses are seen in the lumen and in the lung tissue around. The lung tissue adjacent to the bronchus is largely necrotic.
Plate 2 Case 680 Trachea & Bronchi.
Plate 3  Case 686  Lung.
Plate 4  Case  693  Lung
PLATE V.

DB. 763 Lung—haemorrhagic oedema
Fig 1.

DB. 763 Tracheitis
Fig 2.

DB. 680 Haemorrhage in large bronchus
Fig 3.

DB. 680 Lung—haemorrhagic oedema
Fig 4.
DB/686 Bronchitis and haemorrhagic broncho-pneumonia

Fig 5.

DB/686 Bronchitis and haemorrhage.

Fig 6.

DB/686 Lymph Gland.

Fig 7.

DB/676 Acute Bronchitis in Lobar pneumonia area.

Fig 8.
DB/693 Lobar type of Pneumonia
Fig 9.

DB/693 Lymph Gland - Haemorrhagic
Fig 10.

DB/693 Bronchitis and Pneumonia
Fig 11.

DB/693 Staphylococcal Bronchitis
E commencing abscess.
Fig 12.