CHAPTER IV

THE INFLUENZA PANDEMIC, 1918-19

In 1915 in the A.I.F. the disease diagnosed as "influenza" headed the list. Whether it was related in any way to the pandemic that in 1918 "put up a record" appears doubtful. . . . the notorious inexactitude in the use of the term "influenza" in peace, which was continued in the war, has resulted in a confusion far from creditable to the medical profession.¹

During the four years 1914-18 the Great War was responsible for some 27½ million casualties with some 8 million deaths among the nations engaged. In the twelve or thirteen months of the outbreak—approximately May 1918 to May 1919—it is estimated (British Encyclopaedia of Medical Practice, Vol. 7, p. 174) that some 15 million deaths were caused by the impact of a sudden infective assault by an "organism or organisms unknown". The clinical syndrome caused by them was identified, under the name "influenza", with a form of sickness which during the 19th century had been held responsible for five more or less extensive epidemics or pandemics.² What is more important, it was identified also with a strictly endemic

¹ See Vol I of the present work, 1930, p 75
² The following recorded outbreaks have been identified as caused by the "disease" which since 1580 has been known as "influenza". (Garrison, History of Medicine, p. 187). The first authentic pandemic of the disease is commonly placed in 1510. Since that date some thirteen "pandemics" have been tentatively identified, the most extensive in 1830-33 and 1847, culminating in that of 1918-19 here under review. ("Influenza, An Epidemiologic Study" by Warren T. Vaughan, American Journal of Hygiene, Monographic Series, No 1, July 1921). Sir Thomas Watson (1847) "quotes Cullen as saying that this species of catarrh proceeds from contagion"—he himself would only go so far as to agree that it was "portable" (loc cit, p. 21). One of the most characteristic features of the disease as a source of epidemics has been the fact that these major outbreaks have been interspersed with minor and local outbreaks vaguely identified as "influenzal". From the 'fifties onwards the term "influenza" was applied to almost every case of catarrh with fever. In 1889-90 a pandemic came westward. It was supposed to have originated in China and was attributed to the great floods. But it was known as "Russian influenza" just as the 1918-19 epidemic with no sounder basis was known as "Spanish". The epidemic and pandemic efflorescences of this disease and its endemic counterpart (or counterfeit) are reflected in Australian experience throughout the 19th century. Australian experience in the pandemic of 1918-19 presents certain special even unique features which are of the highest interest not only from the point of view of the history of the A.I.F but of the history of epidemics and of epidemiology. These are admirably recorded in Service Publication No 18 of the Commonwealth of Australia Quarantine Service (Influenza and Maritime Quarantine in Australia) by Dr. J. H. L. Cumpton, Federal Director-General of Health. A brief account of this experience is given in the chapter dealing with the return of the A.I.F. to Australia.
and domesticated disease, or congeries of diseases, under the title of "influenza", and was responsible for a considerable proportion of the total wastage of all armies from disease in camps, transports, and the field throughout the war. It was also—like this disease—attributed to the pleomorphic cocco-bacillus discovered in 1892 by Pfeiffer.3

Both these assumptions, and the complacency of the medical profession, received a severe jolt from the results of the clinical and other studies impelled by the pandemic, as was the world by its social results. The official summary of the debate on influenza in the Interallied Sanitary Conference on 24th March 1919 states:

The extreme gravity of the influenza epidemic of 1918, its terrible social and economic effects, cannot fail to induce our Governments to use every means at their disposal in order to pursue the study of this disease.

However, as with many another pious hope created by the shock of those tremendous events, the failure could and did occur. "The subject," as M. Pottevin said in that debate, had been "illuminated, if not by a direct ray, at least with a vague light"; but not for another fifteen years—1934—was the "direct ray" to be thrown by the isolation of the virus; and on the clinical side there is still:

no more popular dumping ground than "influenza". There is no clinical syndrome that justifies the diagnosis of influenza; and the difficulty of diagnosis will remain until the clinician and the bacteriologist by their combined efforts provide us with means for making a diagnosis in isolated cases.5

Perhaps the most extraordinary feature of this extraordinary pandemic is the furious speed with which it spread itself

8Thus in the British Official History the article "Influenza" (Vol. I, Diseases of the War, p. 174) is introduced as follows:

"Ordinary influenza was never absent from the various Army Commands during the war... In 1918 the figures were about normal until in June there was suddenly a great increase... In France the disease began by a few local outbreaks in the First and Second Armies in April and May 1918... At the end of May it reappeared with great violence in Second Army."7

*This was drawn up for the conference by Professor Calmette and approved at the fourth meeting of the plenary session. The data for this chapter are partly drawn from the official report of the discussion.

6Medical Journal of Australia, Leading Article, p. 63, July 10, 1937

The date of the discovery of the virus of influenza is commonly accepted as 1933, see Burnet, Biological Aspects of Infectious Diseases, p. 247 and Smith, Andrews, and Laidlaw British Journal of Experimental Medicine, Feb., 1934, Vol. LIX, p. 201.
throughout a community, a locality, and the globe. The term “spread” seems indeed inapplicable; more appropriate simile for that speed might be found in the action following the dropping of a crystal into a super-saturated solution. The first “wave”—to borrow the curious term used by most writers—of the pandemic would seem to have appeared almost simultaneously over a great part of the globe. In the Interallied discussion the Chinese and Japanese delegates reported their first outbreaks in March and April of 1918. In the B.E.F. also the pandemic appeared first in April 1918, and in May it was rampant in the British, French and German armies. In the Naval depots in Britain it appeared first in “the early spring”; in the infantry commands and civil community in June. From outbreaks in Spain in May it reached Portugal in June but it was prevalent in the Italian Navy in May. In India its “presence was first noted” in Bombay, Calcutta and Madras in June. From a congeries of such imperfectly recorded facts grew the idea of a sudden catastrophic visitation—a veritable Act of God.

But a feature not less evident than this seeming world-wide synchronicity was the fact of focal spread, for example from bed to bed in a ward; from some new arrival in a unit or camp; from ship to shore or shore to ship; and from over the ocean to communities isolated by sea such as Australia and South Africa.

The American Official History, Vol. IX, p. 84, presents the paradox thus, from experience in U.S.A.:

That an epidemic wave once developed is spread by contact of cases, is of course incontrovertible. But that the widespread, practically simultaneous, increase in the rates that was observed not only with this wave but also with all the preliminary and recurrent waves of the pandemic could have been accounted for by transmission from case to case of a common source seems incredible.

The two phases. Almost universally this pandemic presents itself in two very clear cut phases—the initial one being of

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6 According to Professor Jorge, representing Portugal.

7 "At the Cape of Good Hope ships were only kept free by cutting them off completely from the shore. At Sierra Leone some ships suffered very severely, the disease spreading to the shore; it was probably introduced from England by infected crews ... In North America the disease was introduced into a ship of war from the shore, and during the passage to England there were 150 cases." (Surg.-Capt. P. W. Bassett-Smith, R N—Professor of Clinical Pathology, Navy Medical School, R N, College—Proceedings of Interallied Sanitary Conference, March 1919: Report of the British Delegates)
great infective but very slight clinical virulence, mostly in the spring and early summer; it was followed after a definite though partial intermission by a second phase mostly in the autumn and winter. In this second phase, though commonly less extensive, it was relatively and absolutely a "disease" of great malignancy and in general of character widely different from that of the first phase in its clinical manifestations. The case mortality rose rapidly to a peak and continued high, while the epidemic itself petered out gradually with local recrudescences in the first few months of 1919. The date depended on the factors which determined the course of transmission. Before proceeding to an account of the course and effects of the epidemic in the A.I.F., and of its clinical features and pathogenesis, some general features call for a note.

Identity of first and second phases. The following note is taken from the introduction by Colonel S. L. Cummins, A.M.S. to the Medical Research Committee's Report No. 36, 1919.

Epidemiological features and affinities

In the B.M.J. of 28 November 1918 Captain M. Greenwood, R.A.M.C., sets out in a striking manner the characters of the curves of incidence in past influenza epidemics. In this paper it is stated that "the fundamental characteristics of a primary epidemic of influenza are a very high attack rate and an approximately symmetrical distribution in time"; . . . "the graph of the epidemic is an almost symmetrical curve; the fatality is low, rarely more than 1 per cent of the cases" . . . "a secondary epidemic affects a relatively small proportion of the population, is slower in reaching its maximum, and thereafter declines slowly and irregularly—more slowly than it increases; its distribution is asymmetrical and there is less concentration around the maximum. Further a secondary epidemic is characterized by a vastly higher fatality than a primary epidemic."

Analysing the figures of the B.E.F. Colonel Cummins reached the conclusion that

There can be no doubt that the two epidemics were identical in nature. The difference observed between the cases admitted in the summer and those seen in the autumn were differences in degree and depended on the enormously increased virulence of the infective agent in the secondary wave.9

Identity with past epidemics. At the discussion on influenza at the annual meeting of the B.M.A. in 1919 Captain Green-

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8 In the Northern Hemisphere.
9 It is certain that he should have added "or agents".
wood raised two issues "the solution of which" he said, "is necessary before we can fully understand the epidemiology of influenza".

The first . . . is whether the form of the first influenza wave in our recent experience . . . differed materially from that of the primary manifestation during the last great pandemic—the winter influenza in 1889-90.

His analysis of selected figures led to the provisional conclusion

that there is no clear-cut formal difference between the outbreak of 1889-90 and that of 1918, nor between its evolution in a mixed population and in one homogeneous with respect to age and sex.

Biological factors. The second issue—left open—was

whether the common features of the epidemic curves, the rapid rise, and the less rapid decline are sufficient to ground some hypothesis of the biological factors responsible.

Theories of epidemic origin. Various hypotheses were put out to explain the genesis of the epidemic:

(a) That it broke out in many centres throughout the world as the result of a pandemic "constitution" produced by the war—or an epidemic time factor. (b) That it spread (i) from east (China) to west, (ii) from west (Spain or America) to east. (c) That it was an epidemic exacerbation of the endemic disease. (d) That it was a development from the local epidemics of "purulent bronchitis" in Europe and epidemic empyema in the camps in U.S.A.¹⁰

Effects of the pandemic. Even the meagre facts recorded of the pandemic are of great interest. A few relevant features are given in the following summary:

Great Britain. The incidence of the disease in the general population is stated (Report No. 4, British Ministry of Health, 1920) as follows: "In 1918 there were 3,129 deaths from influenza per million of population in England and Wales. The corresponding figure for 1919 was 1,170."

¹⁰ The report of the Interallied Sanitary Commission's meeting says: "The reports presented to the Commission do not suffice to establish the source of origin of the epidemic which from March 1918 spread with extreme rapidity" (throughout the world). At this conference a French delegate, Dr. Louis Martin said: "It appeared to have started in March 1918 in China." Professor Jorge: "Other epidemics have spread from the east (Asia) to the west but the present epidemic seemed to start in Spain." The British Official History, (Diseases, Vol I) "Influenza", p. 175, says: "The disease was world-wide, and its course seemed to be from west to east. It prevailed in America in 1917. In 1918 the first European epidemic on a large scale took place in Spain in May." Reading this account together with those of outbreaks of "purulent bronchitis" (p. 213) one might gather that endemic or "ordinary" influenza was, in fact, epidemic throughout the war, that in the spring of 1918 it suddenly assumed a new form, but reverted on a vast scale in the autumn.
The disease occurred in epidemic form in ships of the British Navy from the Shetlands to the Scilly Isles. It was prevalent throughout the Mediterranean and the Adriatic, the East Indies and the Persian Gulf. At the Cape of Good Hope ships were "kept free by cutting them off completely from the shore". At Sierra Leone one ship with a complement of 718 had 662 cases and 38 deaths. For the year 1918 the total cases of influenza on ships was 80,144. The total case mortality was 2·8 per cent. Mortality was much higher in the autumn than the spring; at one depot there were 928 with no deaths in the first epidemic, but 468 with 14 deaths in the second.

The incidence of influenza throughout the war is shown in the following table, which is a summary of information given in British Official Medical History of the War—Statistics, page 86:

<table>
<thead>
<tr>
<th>Campaign</th>
<th>Period</th>
<th>Admissions</th>
<th>Rate per 1,000 of ration strength.</th>
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<tbody>
<tr>
<td></td>
<td>1915</td>
<td>44,392</td>
<td></td>
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<tr>
<td></td>
<td>1918</td>
<td>313,938</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(24 weeks)</td>
<td></td>
<td></td>
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<tr>
<td>Macedonia</td>
<td>Oct.-Dec., 1915</td>
<td>795</td>
<td>13·06</td>
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<tr>
<td></td>
<td>1916</td>
<td>282</td>
<td>2·04</td>
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<tr>
<td></td>
<td>1917</td>
<td>98</td>
<td>5·39</td>
</tr>
<tr>
<td></td>
<td>1918</td>
<td>19,862</td>
<td>154·27</td>
</tr>
<tr>
<td>Dardanelles</td>
<td>1915</td>
<td>3,126</td>
<td>26·70</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>1914</td>
<td>6,047</td>
<td>4·72</td>
</tr>
<tr>
<td></td>
<td>1915</td>
<td>31,300</td>
<td>21·21</td>
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<tr>
<td></td>
<td>1916</td>
<td>36,072</td>
<td>22·57</td>
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<tr>
<td></td>
<td>1917</td>
<td>28,980</td>
<td>16·98</td>
</tr>
<tr>
<td></td>
<td>1918</td>
<td>139,683</td>
<td>86·55</td>
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</tbody>
</table>

**British India.** India suffered more severely than any other country. Figures given at the Interallied Conference showed nearly 5 million deaths out of a total population (census 1911) for British India of 238,527,625 with a death rate per thousand ranging from 66·7 to 4·7, and averaging 20·7. The report of the conference states: "Influenza is estimated to have killed not less than 6 per cent. of the population of the central India states, and was responsible for the deaths of upwards of 5 millions in British India and not less than 1 million in the native states; this estimate is conservative. The disease was excessively prevalent and fatal in Afghanistan, Baluchistan, Persia, Turkestan, etc. . . . Females suffered much more than males: the incidence of mortality was highest between the ages of 10 and 40, and among the troops the incidence was higher among British than among Indians, but the case mortality for British troops was 4·3 and for Indian 13·7 per cent" . . . The mortality in the first epidemic wave was almost nil: the second commenced in September and by the end of November the mortality rates approximated the normal throughout the country.

**French Army.** ("La Grippe"). The disease appeared in the Armies between 10th and 20th April 1918 in two small villages north of Compiègne. At the end of April it was diffused over the whole front.
It developed in two periods, May to August and September to December.11

In the first period the number attacked was 43,620. In the second 188,825—a total of nearly a quarter of a million.

Incidence per 1,000 in French Army

<table>
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<tr>
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</thead>
<tbody>
<tr>
<td>1918-19</td>
<td>9.47</td>
<td>4.54</td>
<td>1.11</td>
<td>1.12</td>
<td>9.15</td>
<td>28.54</td>
<td>12.35</td>
<td>7.01</td>
</tr>
</tbody>
</table>

The case mortality ran from less than 0.2 per cent. in May, June, July to 8.1 in August and thereafter maintaining approximately this level.

American Army. Statistics for "inflammatory diseases of the Respiratory Tract" are given below. The American Medical History says that in the U.S. Army

"There were in all, during the World War, 3,515,464 admissions to sick report for disease. Of these, 32 per cent. were primarily for respiratory disease . . . 18.33 per cent. or 1 man to every 5.17 contracted influenza in the service, 6.27 per cent. bronchitis, 0.86 per cent. broncho-pneumonia, and 0.17 per cent. lobar pneumonia. The 1,159,177 cases of respiratory diseases represent 26.63 per cent. of the total number of men in the Army, or 1 to every 3.5 men. Venereal disease was responsible for the next largest number of admissions, followed by mumps.12

The figures given are

United States Army in France, Ratio per thousand per annum.

Influenza. Bronchitis Broncho-pneumonia Pneumonia. Total.

137.38 46.14 9.33 8.93 201.78

German Army. The figures for "Grippe" in the German Army are taken from Volume III of the official Sanitätsbericht über das Deutsche Heer, page 121. The following table gives the actual numbers for the field army and army of occupation, treated regimentally or in the hospitals of the army and lines of communication. The sudden apparent rise of the endemic disease "influenza" to epidemic proportion is particularly striking. (The table is as printed in the German work.)

<table>
<thead>
<tr>
<th>Year</th>
<th>Regimental</th>
<th>o/oo of strength</th>
<th>In Hospitals</th>
<th>o/oo of strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>1914-15</td>
<td>131,189</td>
<td>29.5</td>
<td>30,943</td>
<td>7.0</td>
</tr>
<tr>
<td>1915-16</td>
<td>252,357</td>
<td>37.8</td>
<td>68,880</td>
<td>10.3</td>
</tr>
<tr>
<td>1916-17</td>
<td>283,800</td>
<td>39.2</td>
<td>74,509</td>
<td>10.3</td>
</tr>
<tr>
<td>1917-18</td>
<td>896,266</td>
<td>126.0</td>
<td>176,671</td>
<td>24.8</td>
</tr>
<tr>
<td>1914-18</td>
<td>1,543,612</td>
<td>242.2</td>
<td>351,003</td>
<td>55.1</td>
</tr>
</tbody>
</table>

The two phase character of the epidemic was almost universal with this apparent exception that in an unvisited community—on a ship, for example—the epidemic took the form in which it occurred in the person who transmitted it.

11 These facts are from Le Service de Santé pendant la guerre 1914-18 (Médecin Inspecteur Général A. Mignon) Tome IV. This work carries the history only till the end of 1918.

12 History of The Medical Department of the United States Army in the World War, Vol IX, p. 67.
THE INFLUENZA PANDEMIC

In the troops in France. The experience of the several formations of the A.I.F. reflects, naturally, that of other troops in France and, as elsewhere, presents two fairly clear-cut phases.

The epidemic in the A.I.F. A description given by the Chief Consultant Physician B.E.F. may be epitomised as follows:

It is impossible to fix the exact date at which an epidemic begins. But there seems no doubt that the spring epidemic of influenza began by small local outbreaks on the northern part of the front during the month of April. These did not spread immediately, but about the middle of May the cases began to increase rapidly. In the First Army Major McNee had reported that "since about June 9th the disease has spread rapidly through that Army". Cases were observed in the Third Army on May 21st. "At the various bases it was noticed at the end of April in Rouen, Havre and Marseilles; early in May at Boulogne and Calais."

The numbers affected were "very great", but could not be estimated by reason of the mildness of the epidemic. From June 25th "the rate fell rapidly". Daily admissions to C.C.S. in Second Army had fallen from a maximum of 623 on June 25th to about 50 in the middle of August.

In the A.I.F. the disease appeared first in the 1st Division in Second Army, but by the middle of May was general throughout the formations in Fourth Army also. The following from Captain R. E. Nowland (one of the Australian medical officers lent to the British Army in 1918) gives a good picture of the first epidemic:

(early in May) P.U.O. broke out in the brigade which, in my opinion, was the influenza that has been rampant ever since. The C.O. got it first, then it spread "like wild-fire" right through the brigade . . . At that time I had seen no literature on the subject, but recognized it was very infectious and that fatigue, bad ventilation, etc. predisposed to it. I isolated all cases and any man feeling "seedy". I had as many as 60 men in bed in one day, but of the whole number—between 400 and 500—only 2 were evacuated. The course most of the cases ran was that they were very sick for about 2 days, with intermittent temperatures up to 102°, severe frontal headache, constipation and general malaise. About the third day the temperature would subside, they would start to eat, and by the fifth day were able to get about, but were still a bit shaky; many had a cough with a good deal of muco-purulent expectoration, and some were very hoarse for a week. In from 7 to 10 days the epidemic had subsided and by the end of June we were practically clear of it . . . Circumstances favoured us, we were out of the line, the weather was fine, and we had good billets.

Sir Wilmot Herringham opening the discussion on "Influenza" at the annual meeting of the B.M.A. 1919 See B.M.J. of 1 Apr 1919

He was attached to an artillery brigade.
The following summary of the experience of Captain A. H. Barrett with the 36th (Aust.) Heavy Artillery Brigade, illustrates the change in type but the essential identity of the two phases.

"On May 8th we had our first cases of the influenza epidemic. On that date I was called to the 1st Aust. Siege Battery billets to see 18 men down with vomiting, some headache, pains across the lower part of the chest, legs, and down the back. 7 had a temp. of over 104°, and all were quite helpless; 1 was delirious, was labelled 'flue and was evacuated.' In another battery, caught while on the move, 9 men were left in an ambulance dressing station and on the next day we had 20 men in bed (in billets). They were strictly isolated, but in 2 days we had 52 and the two medical orderlies were working night and day. They were all put in one barn, isolated, and none were evacuated. All got quite well though most of them for days had no appetite for meat, but took well to beef extract and custard made with milk and sugar. For a week the battery was practically out of action, but, after these cases cleared up, we had little trouble with influenza in this battery."

Other batteries however, suffered severely in the second phase.

Later in the year the character of the disease changed definitely. The onset was similar in most cases: sudden and with similar pains and the helpless feeling; but some men, when first seen, were already becoming dyspnoeic, with engorged appearance of the face. In these, moist sounds could generally be heard but no definite dullness, and the prognosis was bad. Various orders came from Army saying "all cases of influenza, however slight, should be evacuated." In this way we should have lost nearly our whole strength, while I am certain the spread of the disease would not have been stopped. And I so often had complaints from men evacuated, of their experiences when being moved about so much in transit, that—if I felt sure a man was going to recover—he was kept in the battery and made comfortable. Later in the year an increasing proportion had to be evacuated. In January 1919 13 men of one battery were suddenly ill within 12 hours, all in hospital for a long time, one died, and it was touch and go with 5.

At the Field Ambulances. In the B.E.F. from the middle of May though the nature of the outbreak was in doubt, an effort was made to tackle it by isolation. In the 1st Australian Division the A.D.M.S. (Colonel R. B. Huxtable) noted at the end of May:

Sick return normal except toward the end of the month when an epidemic of febrile nature broke out and spread rapidly. Of mild type, leaves very little disability, and clears up in about 5 days.

In Second Army No. 17 C.C.S. was set apart for "influenza". In all the field units we find much the same picture—a
febrile disease lumped under “P.U.O., N.Y.D. pyrexia, and trench fever”, with the heading “influenza” appearing fairly frequently in June. In the 1st Field Ambulance only 30 cases were reported as treated for “influenza” in May. But in June “P.U.O. and influenza” have 613 out of a total of 875. By July,

the influenza epidemic has died out, and although one or two men have been seen with what appeared to be a recurrence of the true influenza (sic) there has been no large outbreak similar to the one we had last month.

At the Casualty Clearing Stations. Here the same picture presents itself. In No. 3 A.C.C.S. in April, “influenza” does not appear; “P.U.O., trench fever, and N.Y.D. pyrexia” are grouped and furnish 171 of a total of 897 medical cases (bronchitis furnishes 28, pneumonia 10). In May P.U.O. and its associates claim 336 out of 1,313; another 55 are labelled “influenza”. It is added that influenza “became epidemic and all these cases were sent elsewhere” to special stations. In June the trio furnished 290 out of 1,431 and influenza 313. Summarised, the diary of No. 3 A.C.C.S. says:

“As the influenza epidemic continues, its virulence seems to be increasing, complications more common, and convalescence, before rapid and complete, now passes through a phase of prostration. Broncho-pneumonia is the most severe complication in 18 cases of which there were 5 deaths.” This unit had “received the worst cases from the wards of several field ambulances”. “To combat the epidemic and to reduce wastage” D.M.S. Second Army set aside all clearing stations not specially occupied and these “became stationary hospitals for the reception, treatment and discharge to duty of these cases”. As these filled “all the tentage we could spare were given up to these cases—the acute medical ward took broncho-pneumonias, 3 tents equipped with stretchers took purely pyrexial cases”, and a large store tent was allotted for convalescents.

In July the diagnosis P.U.O. was cut out in this unit.15

15 By a new and keen O.C Maj H H Woollard. He wrote: “Efforts have been made to stimulate M.O’s to diagnose their cases and avoid subterfuges such as P.U.O., N.Y.D.—not so much in the direction of accuracy of diagnosis as of increasing the observation of individual cases.” At this time an officer of the American Medical Service (Maj. W. Fischel) was in charge of the medical ward. His opinion is recorded, that, “in a large per cent. of cases differential diagnosis between trench fever and other pyrexias . . . cannot be made with certainty at a C.C.S.” without longer observation.
Influenza gives 255 of a total of 808. "The influenza epidemic has subsided and the amount of serious sickness diminished." In August "the number of cases diagnosed influenza steadily goes down but the duration of disability seems to increase". In September there is "very little of importance to report in connection with the medical work of the C.C.S. during the past month". "Debility had risen from a trifling matter to the second highest 'disease'."

**October: the second phase.** Here begins a vastly different tale. In the 2nd Field Ambulance "the epidemic form of influenza with respiratory complications accounted for a large percentage of the evacuations during this month". In No. 3 A.C.C.S. of 1,426 admissions for sickness 498 were for influenza.

These (says the unit's war diary) at first were admitted generally as P.U.O., being cases of pyrexia with prolonged convalescence often followed by debility. Quite a number were admitted for diarrhoea. The type of disease is much more serious, toxæmia marked, patients peculiarly "dopey" with frequent pneumonia of a catarrhal type and tendency to marked cyanosis, delirium and low temperature.

From among the men of the unit itself there were only 14 admissions from influenza "not of a severe type", and general health "remained good"—as, it may be noted, it did in that unit throughout the epidemic.

**At the General Hospitals.** At the end of October the Australian Corps moved out of the line to the Abbeville area, and its experience in this phase is reflected in the records of No. 3 A.G.H. which like all the other Australian General Hospitals, bore a full share of the tremendous labours and strain imposed on the medical and nursing professions throughout the world by this gargantuan parasitic debauch. A report from No. 3 A.G.H. (written by Major F. B. Lawton) says:17

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16 The experiences of No. 3 A.C.C.S. which accompanied Fifth Army to Cologne may briefly be noted. In December admissions for influenza numbered 162 out of 619; in January 285 out of 772, with 11 deaths. In February "influenza with or without complications accounted for the bulk of admissions... At Cologne a very virulent type of the epidemic raged. Of 13 deaths 12 were from influenza, admitted under a great variety of diagnoses—P.U.O., influenza, bronchitis, tonsillitis, diarrhoea, pleurisy, appendicitis, gastritis, rheumatism and pneumonia." In March and April influenza still provided the bulk of medical admissions, "with a recrudescence of pneumonia of the catarrhal type so fatal earlier in the year". In May the unit entrained for Le Havre and England.

17 One of the best accounts of the epidemic in a single article is that by an Australian medical officer, Maj S. W. Patterson (late Director Walter and Eliza Hall Institute of Research, Melbourne) who at the time was working in a British
Cases of influenza usually mild were seen at times from the opening of the hospital in France. At the end of February 1918 there was an outbreak of influenza among the orderlies of the hospital. This was not severe though the men were very sick for a few days. It was not till May 1918 that we had many cases, though there were a few in April and there were some broncho-pneumonias of which delirium was a feature . . . Each patient had a card attached at the field ambulance stating that he was not to be evacuated (because of the apparent mildness of the disease). Most of these cases were not very severe. Some of them developed broncho-pneumonia.

After this wave, which subsided about the middle of July, there was a period in which we had only occasional cases. This lasted till September 9th, when we received a batch of patients from the Australian Corps School at Rue on the coast near St. Valery. All were very sick on admission, and some of them had broncho-pneumonia and the others all developed it, and six of the first thirteen died. They were all seen by Sir John Rose Bradford. In the next few days others came from the same place with the same condition, and soon cases came from other places. Ward after ward was taken for these patients, till in October we had in H Block one ward of 30 beds and 4 wards of 44 beds each full of broncho-pneumonia patients . . . On September 29th there were 1,037 patients in the hospital. In October two more large wards—Adrian huts of 48 beds each—were taken and filled with cases of broncho. During December the numbers decreased a little, but it was not till January 1919 that the numbers became small, and, though there have been a few recurrences, it has not been necessary to isolate more than two wards at a time.

When the epidemic was at its height the whole staff had to work hard, the Sisters hardest of all . . .

The following are the figures (for this hospital) for the months of September to December, when influenza and broncho-pneumonia were most severe.

<table>
<thead>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sick admissions</td>
<td>1,161</td>
<td>3,186</td>
<td>3,107</td>
<td>2,257</td>
<td>9,711</td>
</tr>
<tr>
<td>Admissions—influenza</td>
<td>183</td>
<td>1,252</td>
<td>1,334</td>
<td>540</td>
<td>3,309</td>
</tr>
<tr>
<td>Deaths—influenza</td>
<td>4</td>
<td>80</td>
<td>134</td>
<td>20</td>
<td>238</td>
</tr>
</tbody>
</table>

In the A.I.F. Depots in Great Britain the epidemic followed much the same course as in the civil community in Britain, but it seems to have begun earlier. The following account is an epitome from the full and accurate records kept by Colonel McWhae as A.D.M.S.:

unit in Rouen. Writing in M.J.A., 6 March 1920, he says of the first outbreak: "We had read of the spread of the so-called Spanish Influenza in the newspapers, but our first contact with it in Rouen was the arrival, in April, of a hospital train from Italy . . . Most of the R.A.M.C. personnel and patients had suffered during the journey from a three to five-day fever of great contagiousness. Several were admitted for investigation . . . on Thursday to No. 25 Stationary . . . On the following Sunday afternoon I found that 26 orderlies, several nurses and five medical officers including the whole of the laboratory staff had been taken ill on the previous days. Lt.-Col C J Martin, in his indefatigable way, was carrying on the investigation although suffering from a severe attack"
The only other period (than the winter of 1916-17) in which there was serious menace to the health of the troops was during the influenza epidemic of 1918-19. Owing probably (sic) to the fact that the personnel in A.I.F. depots was always rapidly changing, influenza attacked the troops in three distinct waves, with a well marked interval of freedom between each wave. The following schedule describes the epidemic:

<table>
<thead>
<tr>
<th>Outbreak</th>
<th>No. of Cases</th>
<th>Duration</th>
<th>Average Strength</th>
<th>%/oo Troops Quarterly</th>
<th>Respiratory Complications</th>
<th>Deaths</th>
<th>% Case Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3,324</td>
<td>9 May-8 Aug. 1918</td>
<td>34,200</td>
<td>97</td>
<td>6 per cent.</td>
<td>10</td>
<td>0.3</td>
</tr>
<tr>
<td>2</td>
<td>4,424</td>
<td>16 Sept.-3 Dec. 1918</td>
<td>38,000</td>
<td>116</td>
<td>11 „</td>
<td>113</td>
<td>2.55</td>
</tr>
<tr>
<td>3</td>
<td>1,766</td>
<td>16 Jan-20 Mar. 1919</td>
<td>38,100</td>
<td>75</td>
<td>12 „</td>
<td>80</td>
<td>4.5</td>
</tr>
</tbody>
</table>

The first (summer) outbreak was due to a mild form of influenza, the second and third to an extremely virulent form in which the rate of incidence was kept down only by the enthusiastic co-operation of all ranks both combatant and medical. The type became progressively more virulent; but during the last wave of the epidemic a thousand soldiers were arriving from France every second day, and the journey to England in very cold weather, sometimes in crowded and comfortless cattle trucks may have lowered the resistance.

**Prophylaxis.** In view of the large numbers exposed to risk in the constantly changing population of the depots the results achieved by the vigorous steps taken were remarkably successful. The use of prophylactic vaccine is examined elsewhere, but the following is an epitome of McWhae’s account of the other measures:

In the first epidemic to decrease the risk of infection in huts 15,000 soldiers were placed under canvas, not more than 6 men in each tent, and all were well aired once daily. In this way the number of men in the huts was much reduced. In the later outbreak it was not possible to carry this out, but the ventilation in huts, canteens, and so forth was rigidly enforced and crowding prevented. During the height of the epidemic, public places of amusement were put out of bounds. In the huts the men slept with feet and head alternating and gargled pot. permang. (1 in 5,000 of normal saline) which was also sniffed through the nose. Blankets of cases were sterilised and contacts were kept from public resorts for three days.

“General instructions” warned all soldiers against “allowing themselves to become chilled when cooling after exercise... The matter is one of commonsense. Soldiers undergoing severe exercise should not be over-clothed but they should put on any additional clothing when the exercise is finished.” Definite arrangements for hot meals were made, “the supper meal to be procurable at a fixed hour”. An order specifying the
early symptoms of influenza, and instructing officers, N.C.O's, and men to report for medical examination immediately they noticed any of these symptoms, was read on Company parade daily and a copy posted in each hut of the Command. Medical officers were required to admit to hospital every soldier whose temperature was above normal, and combatant officers were made responsible for reporting to the R.M.O. any soldier too ill to attend sick parade; he was to be seen at once by the R.M.O.

Transports to Australia. A most important and difficult problem was that of preventing outbreaks of influenza in the troop transports to Australia carrying invalids and, after the Armistice, in those repatriating the rest of the A.I.F. The whole subject of transportation, however, is dealt with in a later chapter. The dramatic circumstances of the influenza epidemic as experienced by the Light Horse in Syria have been described in Volume I.

The specificity of a "disease" may be determined by a constant clinical syndrome and pathology; a homogeneous pathogenic agent; or an identifiable epidemic distribution. The specificity of influenza still rests almost wholly on the last. The difference between a case of the early "three-day fever" and a fulminating case in October—in which men were felled with an overwhelming toxaemia and became livid and died within 36 hours or, after recovery from widespread pneumonia and the brink of death, might stay crippled for many months or for life—could not be adjusted by clinical discrimination either at the time or now. In the lack of a specific pathology or pathogenesis, identity could not be fixed by laboratory analysis. There remain the epidemiological features of the disease and the fact of a

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18 The appalling possibilities presented by such an occurrence is illustrated by American experience. "On September 16th the Nestor shipped 2,807 troops. On the second day out influenza began to appear and the following day she put into Nova Scotia and landed 660 men including all sick and contacts. Over 1,000 more were taken ill between here and Liverpool, of whom 240 had broncho-pneumonia, 17 died at sea, and others were landed in a dying condition." (Herringham, loc. cit.) The experience of the Olympic early in October was even more tragic, 141 out of the 5,600 troops carried having died during the voyage or on landing, or within two days of their landing in England—a case mortality of 6 per cent.

19 p. 735 and elsewhere

20 Amid much other evidence may be noted the fact that only two deaths from influenza are recorded of the Light Horse. The War Office issued a general letter on the identification of influenza as a cause of death in pneumonia. Herringham (see B.M.J., 1 Apr 1919) sums up thus—"Looking back at the 5 years of war it seems to me that the cases we have been calling influenza this winter are of the same character as those we have seen in most previous winters . . . and when I recollect the epidemic of 1889 . . . I am very much tempted to ask whether the present disease is influenza at all . . ."
gradual clinical merging of the first type with the second. The symptoms of the disease in its first phase and its merging with the second have already been described. No attempt can be made to present a comprehensive clinical picture of the complex of morbid states encountered in this second phase. The following descriptions of the “malignant” type are from Australian records:

Clinically the most noteworthy features were the tendency to haemorrhage, including epistaxis, haematemesis, blood-stained sputum, which was usually profuse and watery, profound toxaemia and little evidence of consolidation of the lungs (Major S. W. Patterson, M.J.A., 6 March 1929).

The report of the Medical Officer, No. 3 A.C.C.S. says. Two types of case were recognised:

1. Those with frank pneumonia, nearly always of lower lobe and generally bilateral, with little or no cyanosis, with high temperature, ending by crisis or lysis with general improvement on the fall of temperature. These are very severe while they last, and some are probably ordinary croupous pneumonia. The sputum is usually rusty and viscid. These cases do well, and compared to the next group cause little anxiety.

2. Men with bad colour, either distinctly blue cyanosis, or dusky, often without any definite physical signs of consolidation, dull to flat on percussion, especially behind, some râles or fine cracking crepitations and diminished air entry with weak or absent breath sounds. Pyrexia moderate or for a time high and falling without improvement in the general condition or physical signs.

Some of these have no sputum, others develop a profuse purulent sputum—I think often when improving—others a haemorrhagic sputum, while in others in whom consolidation appears, a rusty pneumonia sputum is seen. These cases may continue cyanosed, without developing consolidation and with chiefly negative signs, and die. Others hang fire, as it were, for a time and gradually recover, others develop deep patches of consolidation and increase in râles, the consolidation gradually increasing, often involving both lower lobes, usually unequally, and part of the upper lobes, with râles present all over front and back. The temperature may fall and the progress of the disease continue, until the patient finally succumbs from exhaustion or toxaemia. A particularly bad case is the man with pallor and lividity of the lips and ears and pinched appearance; these closely resemble the picture of cases poisoned with phosgene gas. Many of these cases appear to be deprived of oxygen and the lower part of the chest may be noticeably indrawn with the respiratory movements. I have several times noticed a cyanosed face clear temporarily and become pink after the propped-up patient has coughed and taken a few deep inspirations.

One is impressed with the feeling that the course of the disease

Curiously enough, thus has been accepted as typical of the “influenza” of 1918-19 though as will be seen the clinical syndrome and the underlying pathological changes depended chiefly on the nature of the various symbiosing agents that were the cause of the respiratory complications found in influenza, in common with measles, and probably other virus diseases, met with in training camps in the war.
and the result is dependent largely upon the toxaemia. This may be intense, as in the fulminating cases which die before the development of definite physical signs, or it may so sap the patient's strength as to leave him unable to recover when the disease has apparently burnt itself out, and the physical signs have apparently begun to improve. The pulse often keeps comparatively good until nearly the end, and I have seldom found definite evidence of marked dilation of the heart. The pulse is often noticeably slow for the temperature. In a few cases the morning temperature is higher than in the evening.

**Cyanosis.** Of this extraordinary and dreaded sign Herring-ham writes thus:

In many cases the patient was admitted in a state of cyanosis which was not accounted for by the physical examination of the chest ... I have never in my life seen anything like the picture presented. Entering a ward you might see 6 or 8 of these cases, some heliotrope, as it has been well called, others really purple yet not appearing as much distressed in their breathing as from their colour you might expect ... I do not know the explanation. It did not seem to me that there was in these cases such failure of the circulation as would account for it. ... In some cases the same may be truly said of the state of the lungs. The condition of congestion with oedema seen in others recalled phos-gene poisoning.22

Investigation of the morbid anatomy of a disease, that is to say the departure from structural normality induced in the subject by the *materies morbi* has two purposes: to explain the physical signs and symptoms; and to ascertain which particular cell-complex, system, organ or tissue is the physical or chemotactic *point d'appui* of the "object"—i.e. the parasite or its toxin—or of the tissue reaction of the "subject".

In the influenza of 1918-19 the task of elucidation that faced the pathologist was a truly appalling one. In the first place, save that it was known to be parasitic and alive the cause was hypothetical; as to whether the pathogenic agent generally accepted—Pfeiffer's *B. Influenzae*—was in truth responsible, scientific circles of the profession were seriously sceptical. Second, and consequentially there was no criterion, clinical or pathological, to discriminate any specific disease at all. That out of this chaos, and in the dust and heat of the supreme crisis of the war, the medical profession of the world should have reached an understanding which subsequent research has

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22 Professor J. S. Haldane, F.R.S., suggested (*Brit. Medical Journal, 15 June 1929*) that the coloration might be due to meth-haemoglobinemia
confirmed, is an evidence of rational poise that gives hope for humanity.

In a considerable proportion of the cases coming to post-mortem the evidence of damage entirely or mainly obscured that of any reaction and, when present, the latter covered the whole gamut of tissue reaction found in acute disease of the respiratory tract. The dominant feature was haemorrhagic and serous effusion. The following description is compiled from the notes of a number of medical officers of the A.I.F.

Frothy, sanious fluid was often exuding from the mouth and nostrils. When the thoracic cavity was opened it was seen that the front of the turgid lungs was pushed upwards, usually full of air and crackling. Rupture of the air vesicles had taken place in many cases leading to patches of acute emphysema beneath the pleura. Frequently there were small areas of sub-pleural haemorrhage. Some cases showed recent soft fibrinous adhesions sometimes very dense, and in tearing through them blood-stained fluid exuded from the mouth and nostrils, as it was expressed from the lung and bronchioles.

The most striking feature was a general engorgement and water-logged condition of the lungs.

Microscopically the capillaries of the pleura, alveoli, and bronchi were engorged and often ruptured. The alveoli were full of a homogeneous, coagulated, albuminous exudate, often containing blood, and in the more affected parts leucocytes and endothelial cells.

To this primary inflammatory slimy oedema and congestion were added the following types of broncho-pneumonic involvement: (i) Peribronchial type; (ii) The usual broncho-pneumonic type; (iii) Purulent bronchitis; (iv) Acute emphysema. Protean combinations of the pathological conditions thus outlined were sometimes found throughout the lungs.

The following independent account is epitomised from Medical Research Committee Report No. 36.

62 cases. Pharynx congestion was common, sometimes purple coloration was found here, as also in the larynx. Tracheal inflammation was always present, sometimes intense. Contents were usually frothy fluid, on the pleural surface superficial haemorrhages were common. Thin fibrinous pleurisy was often seen—it was dense over areas of pneumonic consolidation. In the lungs "very striking changes were found". These are summarised as (a) "oedema", (b) "gelatinisation", (c) haemorrhage, (d) broncho-pneumonia, and (e) abscess (3 early cases).

It was found possible to relate some of these to physical signs, but not usually. Of the first type "the affected area is firm, heavy, crepitant;
on section no consolidation, cut surface pours out frothy brownish fluid; no pus". The gelatinised lung was thinly coated with fibrin; on section the lung was red, homogeneous and glassy, with excess of fluid and nearly airless. Broncho-pneumonia and capillary bronchitis were common in all grades in late cases. Miliary broncho-pneumonia occurred in four cases.

Captain Connor’s histological investigation included the areas of haemorrhage, of broncho-pneumonia, of oedematous pneumonia, of grey hepatisation and of areas of solid but "dripping" lung. In general the picture presented was that of an "acute haemorrhagic inflammation with toxic spoiling of the capillaries" passing through all grades of reaction to a typical broncho-pneumonia. In general there was little leucocytic invasion of the alveoli, in which endothelial cells were more common. Haemorrhagic and oedematous areas, grey hepatisation, and broncho-pneumonia might all be found in the one lung.

For the elucidation of the aetiological conundrum presented by this clinical and pathological anarchy three lines of enquiry were available: pathological research, epidemiological reasoning, and clinical analogy. The first has given us the "open sesame" to a final, if still far off, solution. But the other two made material contribution.

Epidemiology. Any exact epidemiological study of the disease was vitiated, first by the fact that no exactly definable "disease" existed, and second, that of the syndromes in which the outbreak showed itself only the pneumonic complications were notifiable in any civil community.28 Army figures however gave a clue to the incidence of the general morbidity. Numerous observations confirmed the generalisation that the incubation period of the mass disease was from 24 hours to a few days. Many careful observations in most armies determined that men who had suffered from influenza in the first phase obtained a degree of immunity to the second, but that after a brief time limit, it was at best relative. On these facts and the features of the epidemic curves Greenwood endeavoured to "ground some hypo-
thesis of the biological factors responsible” for epidemic influenza. The premises he set out as follows:26

We may lay it down that the realization of an epidemic demands three essentials—namely, an infectable subject, an infective object, a favourable environment. (a) The object is in general organized; under constant environmental conditions it will tend to pass through a cycle of changes expressing themselves by means of different effects upon the subject, and these changes may be hastened or retarded by variations of the common environment. (b) The subject being necessarily a living creature, is, mutatis mutandis, susceptible of changes congruent with those just predicated of the infective object.

(c) The environment must be understood to comprise not merely the direct reactions of say, climatic or economic conditions upon the subject and object, but also the effects of the morbid evolution upon both, together with the results of interference ab extra, for instance, in response to the activities of sanitary administrators.

On these premises and certain figures, military and civil, a tentative deduction was reached:

While the actual forms of the waves are plausibly explained, either by intra-epidermic changes of infectivity due to the parasite or by varying susceptibility, the fact of our having such waves at all in this year, and not having them in previous years, is attributable to the existence of a double periodicity to be referred to the coexistence of two distinct strains of infecting organisms or, if the reader prefers, two armies of infecting organisms, which may or may not possess a bacteriological specificity.

The proposal, prima facie “suggestive” that the pandemic was caused by the “augmented infectivity of the organism induced by the herding together of young susceptibles” was negatived by the fact that “in point of time” (as it appeared) “influenza became epidemic in neutral countries before those at war were affected”. The possibility that “a considerable number of epidemic manifestations during the past decade were really epidemiologically regarded influenza” was tempting but might “lead us into greater difficulties than it escapes”.

The situation in 1919 was summed up by the statement that, from the epidemiologist’s point of view, “much further investigation” was needed “before we shall have a clear view of the problem”.

Clinical analogy: endemic influenza, measles and some

26 From paper read by Capt. M Greenwood, at Joint Meeting; sections of Medicine and Preventive Medicine and Pathology (British Medical Association, 1919).
Apart from “lobar” (pneumo-coccal) pneumonia—a distinct and clear-cut disease entity with a history of its own—the whole problem of infective disease of the respiratory tract in the war hinges on the question what did “influenza” mean. In the records prior to the pandemic, influenza presents itself (1) as a fairly constant cause of local wastage from a non-descriptive congeries of pyrexia, corresponding more or less to the pre-war idea of “influenza”; (2) at camps of training in outbreaks, approaching an epidemic incidence, of grave and fatal disease of the lungs, in which measles, rotheln, and influenza were associated with a great variety of morbid states of lungs and pleura. These comprised the following conditions:

(a) Pleuro-pneumonia. In Australian experience such an outbreak occurred in 1914-15 in the Mena camp and has been described in Volume I. It was associated chiefly with measles. The most characteristic feature was a fibrinous pleurisy, so dense that it gained a local name “the Mena Shawl”. In these cases besides the pneumococcus the bacillus of Friedländer was prominent. “Influenza” was not regarded as a factor of any significance. Streptococcal infection was infrequent.

(b) In British Command camps at Aldershot and also in France there were identified local epidemics of a very fatal and apparently specific condition “purulent bronchitis”. The British Official History accepts this disease as identical with some elements in the disease mass of “pneumonic influenza”. Whether they were indeed “influenzal”, and, if so, why they remained isolated, remains for the future to show.

(c) The history of respiratory infections in the American camps of training is of extraordinary interest. Very extensive and fatal outbreaks occurred. The outstanding feature of these was at first (end of 1917) the occurrence of epidemics of pleuro-pneumonia and empyema associated with measles. The clinical picture is strikingly reminiscent of Mena. For the most part a broncho-pneumonia, purulent bronchitis or empyema

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27 The chief sources of information under this heading are the records of the A I F. that deal with the outbreak of respiratory disease at Mena (1914-15); the Review of War Surgery and Medicine, A E F., and Vol. IX, Official Medical History, U.S.A., which deals with the camp outbreaks in America; and the British Official Medical History, Pathology dealing with “influenza” and “purulent bronchitis”.

28 See Lancet, 8 Sept., 1917 “Purulent Bronchitis; its Influenzal and Pneumococcal Bacteriology” by Aldophe Abrahams, N F. Hallows, J W H. Eyre, and Herbert French.
is imposed on the specific disease. But here, in particular, the streptococcus group emerges and comes to dominate the situation. So definite was this invasion that special wards were set apart for measles cases in which haemolytic streptococci were found in the throat. The outbreaks in different camps had apparently no connection with one another.

Investigations of the maladies prevalent in camps in the past winter (1917-18) revealed the streptococcus as a widespread invader and the cause of many conditions in which we have hitherto believed it played only a minor and secondary role. Moreover it seems to incite conditions having a fairly precise clinical prognostic and anatomical picture so as to be quite readily distinguishable from similar conditions due to other micro-organisms.²⁹

At the end of 1917 in America, against this background, influenza emerges, and, in the second half of 1918, the haemorrhagic type of lung infection. It is difficult to escape the conviction that, in America also, the pandemic owed its specific character to the unknown factor rather than to the symbiotic microbes, whether Pfeiffer, streptococcus, or other.

Respiratory infections in camps of training. The camp outbreaks of respiratory disease in various armies present such striking analogies as to compel the conviction of some common aetiologial factor beyond the occasional coincidence of infective agents. Integrating A.I.F. experience with that of other countries, the picture emerges of intimate and complex but exactly apprehendable mass-interaction between the infective object and the infectable subject. Environmental factors, largely controllable, seem to have determined the issue. On the side of the agent, the extent of the attack depended on diversity of method, ability to seize the day, to exploit success with augmented virulence, and so forth. The subject defended himself with biological, instinctive, and rational reactions, varying with race, experience, and culture. Medical science has so far advanced in knowledge that the clash of these forces in the environment of military camps can now be followed with much exactness. In the crowding, cold, overwork, dissipation, change of diet, dirt, promiscuity, and so forth we can note, pari passu with the coincidence of some virus infection, the emerging of one or more "facultative parasites", the identity of which will depend largely

on the coincidence of its saprophytic presence in those subjects first or most severely exposed. Apparently it assumes pre-eminence in virtue of the impetus gained by precedence, and also by the increase of virulence with successful passage.

Pathological research. The clinical syndrome characteristic of bacterial diseases is not grossly pleomorphic. It was inevitable therefore that in the study of influenza scientific physicians should have been profoundly dissatisfied with the obscurantist effect of a family name and an accredited organism as a cause.30

The dissatisfaction was expressed in the early years of the war by the creation of "P.U.O." as a substitute for "influenza" as an interim diagnosis; in the last year of the war it was evident in the manner in which medical scientists leapt at the opportunities for study offered by a pandemic of the real disease. Within an hour after the arrival of the first batch of Spanish influenza at Rouen an intensive research was begun into its bacteriology by the Adviser in Pathology A.I.F., Lieut.-Colonel C. J. Martin.31

The same thing was happening throughout the B.E.F. and in every national army. Experimental research quickly followed, and was continued throughout the epidemic. If the circumstances and competing interests are considered, the bulk of this research was prodigious, and the results must command admiration. The Australian Medical Service was in the centre of this research and a note upon it is therefore not out of place.

The case for Pfeiffer's bacillus. Until the advent of the three-day fever, "influenza" had received during the war no more scientific or critical attention than in the pre-war years. It was accepted as a diagnosis which did not involve scientific discrimination—at best a clinical nondescript. Purulent bronchitis had been studied and an epidemic tendency observed. The first reaction to three-day fever was to search for Pfeiffer's bacillus and with much success. Thus at Rouen it was found in nearly every case, and a definite relation was established with the course of the disease and evidence of phagocytosis. In the then prevalent worship of laboratory research, the discovery of this

30 The position of the clinical influenza in 1918 was not unlike that occupied by rheumatic fever to-day. The B. Influenzae of Pfeiffer had a standing akin to, if perhaps more secure than, that of the coco-bacillus of Poynton and Payne.

31 At this time Col. Martin was stationed at No. 25 British Stationary Hospital and engaged on his important work on dysentery.
organism was made the final diagnostic criterion of influenza. When it appeared to be absent, this was attributed, often correctly, to the technical difficulties of its isolation. But in general the verdict was that pronounced by Colonel Cummins in the *M.R.C. Report No. 36:* "not only is *B. Influenzae* present in nearly every case but it tends to invoke immune responses early in the disease".

But as the pandemic developed the pathological picture became more and more confused. Many skilled observers failed to find Pfeiffer's bacillus; negative findings were recorded in September 1918 by Kolle and even Pfeiffer himself. Other organisms were found to predominate. In particular from America came amazing reports of extensive epidemics of broncho-pneumonia and empyema in which the streptococcus group played a dominant rôle, both independently and as a symbiotic especially in measles and "influenza". It became obvious that, if any single organism was the determinant of the disease, the clinical features of the pneumonic type involved several organisms beside Pfeiffer's bacillus. This suggested the presence of a master agent which caused the type disease and opened the way for symbiotic invaders. Many still believed that this was the rôle of Pfeiffer's bacillus.

*Search for a "filter-passer".* The existence of disease due to ultra-microscopic or filter-passing agents had been known since 1898 (Foot and Mouth Disease). The long search for such a cause of the "common cold" had begun—founded on the technique created by Noguchi in his research on infantile paralysis. Three teams tackled the problem of influenza on these lines: Nicolle and Lebailly in the Pasteur Institute; Rose Bradford, Bashford and Wilson at Etaples; and a team, selected by Colonel Cummins in the laboratory of No. 3 A.G.H. The officers here chosen were Major H. G. Gibson, R.A.M.C., Major F. B. Bowman, C.A.M.C., and Captain J. I. Connor, A.A.M.C. Each of these teams obtained positive results by the inoculation of monkeys and other animals with filtrates and direct suspensions of mucus from influenza patients. Their finding that the disease might be communicated to animals was accepted provisionally by British and French bacteriologists, but with strong reservation in respect to specificity. The English observers identified certain minute but microscopic organisms;
these they sub-cultured and with them produced haemorrhagic pneumonia in monkeys and mice. The Interallied Conference in 1919 in its “provisional conclusions” set out:

We have as yet no precise knowledge as to the causal agent of influenza. The coco bacillus of Pfeiffer is almost always present in the excretions and around the lesions of influenzal broncho-pneumonia; but it is also found in the bronchial excretions of many subjects attacked by various affections besides influenza. Doubtless it plays an important part in the primary complications, but it is not the specific agent of the disease. It is probable that the specific agent of influenza is an ultra microscopic virus, capable of passing through filters of porous porcelain.

Repetition of these investigations in America and Japan were negative; and, so far as concerns the virus cause of influenza, here the matter remained until the historic and far-reaching discoveries initiated by Laidlaw and his co-workers.

The *British Official History (Pathology)*, written in 1923, sums up the case in favour of Pfeiffer.

*The new outlook 1919*

Our conclusions are: (1) that there is one common and original microbic cause of influenza; (2) that, if this be not the *B. Influenzae* of Pfeiffer, it is in the earliest cases constantly associated therewith (p. 454).

It was held that the lesions which the American observers and others had found associated with streptococci and other organisms “are not the lesions seen in uncomplicated influenzal pneumonia”; and that a succession of lesions of the respiratory tract is recognisable in these influenza cases that is in itself absolutely specific. The individual lesions may not be specific but the succession and association are such as are peculiar to this one disease (p. 463).

Nevertheless many will prefer the statement of the case as set out by Colonel Cummins in the Medical Research Committee’s report:

An analysis of the relation of the type of organism isolated to the type of lesion present does not reveal any features striking enough to definitely ascribe any type of lesion to infection with any one particular organism. An important factor underlying the production of secondary lung complications would appear to be the degree of injury to pulmonary tissues by the primary aetiological agent of the disease. The resulting areas of haemorrhagic oedema afford a ready portal of entry and ideal conditions of growth for the organisms already existing upon the surface of the respiratory tract.

The whole clinical and pathological picture becomes intelligible if we can postulate a primary aetiological agent acting locally upon the respiratory surfaces and generally through its toxic products in such
a manner as to prepare the way for invasion by the prevailing respiratory flora. The work of Gibson, Bowman and Connor at Abbeville and of Wilson at Etaples holds out a promise that this explanatory link may now be supplied.

The amazing chemotactic affinity of this virus for human protoplasm seems almost to rule out any rational means of prophylaxis. The following summary has been made of prophylactic measures taken or attempted in the war:

(1) *Administrative procedures.* While A.I.F. experience affords no precise evidence regarding the value of local *notification* and *isolation*, the view held by the A.D.M.S. A.I.F. Depots (Col. McWhae) that the infection was spread from man to man and that this fact should be rationally exploited seems thoroughly justified by recorded observations. Colonel Barber (D D.M.S Australian Corps) also believed that after the Armistice “the epidemic was to a great extent held in check” by the wide dissemination of the troops in the billeting area.

(2) *Direct measures.* Gargles and sprays were used in the Command Depots but no good word has been found for the practice. The following from the *American Official History* is pertinent to the whole problem of respiratory prophylaxis: “The actual mechanics of the mode of transmission of the virus of influenza is a point over which argument has taken place. There is to-day substantial agreement that the disease is transmitted from individual to individual, rather than by aetian convection...” *(Volume IX, p. 111).* The suggestion is made that—

“The obvious fact that infective material is constantly sprayed into the air by the coughing patient, from which it is equally readily inhaled by those near by, has tended to render us oblivious of other possibilities perhaps as important. The rôle of the hand in the spread of these diseases has been emphasized, particularly by Lynch and Cumming... It is entirely probable that both methods play their part in the process.”

(It may be noted that the War Office made regulations on these lines in connection with the prevention of C.S.F. and diphtheria.)

(3) *Specific immunity.* No aspect of this extraordinary pathogenic conundrum better illustrates the chaos that remained to be resolved than the results reported of the use in the war of vaccines containing *B. Influenzae.*

In the middle of 1918 the War Office commended the use as prophylactic of a mixed vaccine containing *B. Influenzae* 60 millions, pneumococcus 200 millions, streptococcus 80 millions per c.c. The results reported by various competent observers defy scientific explanation on the present hypothesis concerning the pathogenesis of the “disease”.

1. Leishman’s summary of figures for troops in Britain.32

32 The question of the identity of the *B. Influenzae* as isolated in 1918-19 with *Haemophilus influenzae* of to-day would seem to be still a matter of some interest. Cultural methods varied much in the war as well as results.

33 Col Sir W B Leishman The A I F experience is summarised on p 218 The tables summarise figures given by Professor Alexander Fleming, in *Recent Advances in Vaccine and Serum Therapy*, p. 331. (London: J. and A Churchill, 1934.)
Incidence Ratios per 1,000 Pulmonary complications.

<table>
<thead>
<tr>
<th>Inoculated</th>
<th>Uninoculated</th>
</tr>
</thead>
<tbody>
<tr>
<td>14.1</td>
<td>1.6</td>
</tr>
<tr>
<td>47.3</td>
<td>13.3</td>
</tr>
</tbody>
</table>

2. Eyre and Lowe's figures for N.Z. troops in U.K.

<table>
<thead>
<tr>
<th>Inoculated</th>
<th>Uninoculated</th>
</tr>
</thead>
<tbody>
<tr>
<td>13.0</td>
<td>1 - 6</td>
</tr>
<tr>
<td>41.0</td>
<td>2.25</td>
</tr>
</tbody>
</table>

3. Duval and Harris's figures (for patients inoculated with B. Influenzae 1,000, 500, and 1,000 millions).

<table>
<thead>
<tr>
<th>Inoculated</th>
<th>Uninoculated</th>
</tr>
</thead>
<tbody>
<tr>
<td>33.0</td>
<td>433.0</td>
</tr>
</tbody>
</table>

The conclusion of the Interallied Sanitary Conference as to the value of inoculation may be commended as a model of scientific restraint:

Trials in preventive vaccination, bacteriotherapy and serotherapy made by various experimentalists have as yet given only imperfect results. It cannot be otherwise, since (the specific virus of influenza being unknown to us) these trials could be carried out only with various non-specific organisms, such as the coco bacillus of Pfeiffer or pneumococci or streptococci, etc. Vaccines or sera prepared from those organisms act only on the secondary bacterial conditions associated with influenza.

As to the specific treatment of influenza one observation made in the A.I.F. is strongly confirmed by wide contemporary experience, namely the value of rest. Within obvious limits imposed by circumstance, the less any case of influenza was moved the better. This factor, it would seem, might often determine whether the course of the attack was to be benign or malignant.

Serum therapy. Favourable results in the treatment of the primary disease by serum from horses recovered from an influenza-like epidemic known as "la Gourbe", and of pneumonic cases by anti-pneumococcus and streptococcus sera, were reported to the Interallied Conference. This elicited from Dr. Martin, Director of l'Hôpital Pasteur, the question, "May not these results depend on some non-specific action of the horse serum itself?"

Vaccine therapy. The influence of vaccine on the recovery rate has been noted above under prophylaxis.

A brief note linking past with present will perhaps illum-
ate the future. The post-war history of “influenza” is concerned with a series of epidemics of which Professor Alexander Fleming remarks it is true that epidemics labelled influenza occur, but there is no certainty that they are the same disease as existed during the pandemic which ended in 1919.

In 1933 Laidlaw, Andrewes and Wilson transmitted from humans to ferrets a disease from which a virus was isolated, and strong evidence was forthcoming that it might be the primary infective agent in human influenza. At least there had been gained “a new starting point and new hope for the ultimate solution of the problem”, and it had been brought within the range of experiment. The confirmation of the research in America, the discovery of a close immunological relationship with the virus of Shope’s “hog influenza”, and also the discovery of important symbiotic relations between that virus and a bacillus resembling Pfeiffer’s *Bacillus Influenzae*, are now history. In 1935 Dr. F. M. Burnet working at the Walter and Eliza Hall Institute, Melbourne isolated from a local epidemic a virus which serological tests identified with the British strain. He says:

The present report adds nothing new to the knowledge of the virus, but, taken along with the American findings, adds a substantial corroboration to the view expressed by Laidlaw and Francis, that typical epidemic influenza is a distinct disease entity due to one specific virus, the serological characters of which do not vary significantly from epidemic to epidemic or from country to country.

The history of the 1918-19 pandemic illumines the wide possibilities opened up by these discoveries; but it also suggests that this is the beginning, not the end, of definitive research. The autumn epidemic of 1918 has still to be explained.

Nothing is more striking (says the report of the Medical Research Committee whose team worked at No. 3 A.G.H.) than the unanimity with which all observers mentioned haemorrhage as a prominent feature of the disease in the autumn epidemic.

Whatever the materies morbi transmitted to the experimental animals at No. 3 A.G.H. may have been, the “disease” transmitted was clinically identical with “pneumonic influenza”.

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The production of haemorrhagic oedema is a constant occurrence... (in the inoculated animals) the lung haemorrhages form the essential pathological lesion.

No link up of the ferret or swine disease with "pneumonic influenza" can be complete which does not explain also this haemorrhagic diathesis as a constant symptom, the world over, of the pandemic disease, and seen in slight or moderate cases as well as in the most severe.

*Duplicate of the diagnosis "influenza" tested by A.I.F. figures.* The present writer found that a preliminary study of the Australian war-records relating to the clinical syndrome known as "influenza" in its endemic and epidemic forms, and especially a study of the views expressed at the Interallied Sanitary Conference of March 1919, tends to confirm his strong personal conviction based on war experience, that the endemic and epidemic elements in the sickness recorded in the admission and discharge books as "influenza" represented at least two distinct syndromes, probably reflecting a similar multiplicity in the specific extrinsic causative agents.

On this presumption the Australian statistical records were at first analysed to distinguish as epidemic (or "pneumonic") influenza the cases recorded in the minor and major outbreaks of 1918-19, relegating to a distinct class the "influenza" recorded of the years 1914-15-16-17. Though crude, this method of cutting the statistical Gordian knot would have much clinical justification. It had to be abandoned, however, in view of the uncertainty that surrounded the question and consequently both in the present chapter and in the general tables at the end of this volume (Chapter XVII) all "influenza" is lumped together as a "disease". The scientific impropriety of this is obvious, and the figures are given with this warning. (The relation between "influenza" and other respiratory infections is also shown.) The American figures given earlier in the present chapter form an interesting comparison. The German figures illustrate the very regular incidence of what we have termed the endemic influenza and the sudden onset of the pandemic disease.\(^\text{36}\)

The following table sets out the incidence of "influenza" in the A.I.F., and relates this to the various stages of the war and to other respiratory diseases.

**Statistics**

\(^{36}\) American and German figures on p 196
Comparative table of A.I.F. admissions in France for Influenza, and for other Fauetal and Respiratory Tract Infections shown by yearly periods from March to March with their comparative incidence.

<table>
<thead>
<tr>
<th>Admissions to</th>
<th>1916-17</th>
<th>1917-18</th>
<th>1918-19</th>
<th>Total</th>
<th>1916-17</th>
<th>1917-18</th>
<th>1918-19</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Field Ambulances ...</td>
<td>6,304</td>
<td>3,922</td>
<td>11,721</td>
<td>21,947</td>
<td>9,186</td>
<td>6,434</td>
<td>3,733</td>
<td>19,353</td>
</tr>
<tr>
<td>Percentage in year ...</td>
<td>28·9</td>
<td>17·9</td>
<td>53·2</td>
<td>—</td>
<td>47·4</td>
<td>33·3</td>
<td>19·3</td>
<td>—</td>
</tr>
<tr>
<td>Cas. Clr. Stations</td>
<td>3,980</td>
<td>2,368</td>
<td>5,941</td>
<td>12,280</td>
<td>7,704</td>
<td>4,805</td>
<td>2,588</td>
<td>15,097</td>
</tr>
<tr>
<td>Percentage in year ...</td>
<td>32·3</td>
<td>19·3</td>
<td>48·5</td>
<td>—</td>
<td>51·0</td>
<td>31·8</td>
<td>17·2</td>
<td>—</td>
</tr>
<tr>
<td>Exp. Base Hospitals ...</td>
<td>4,214</td>
<td>1,517</td>
<td>5,351</td>
<td>11,082</td>
<td>9,140</td>
<td>3,851</td>
<td>3,785</td>
<td>16,776</td>
</tr>
<tr>
<td>Percentage in year ...</td>
<td>38·0</td>
<td>13·7</td>
<td>48·3</td>
<td>—</td>
<td>54·5</td>
<td>23·0</td>
<td>22·5</td>
<td>—</td>
</tr>
<tr>
<td>Evac. to U.K. ...</td>
<td>905</td>
<td>573</td>
<td>3,956</td>
<td>5,434</td>
<td>3,376</td>
<td>2,544</td>
<td>2,752</td>
<td>8,672</td>
</tr>
<tr>
<td>Percentage in year ...</td>
<td>16·8</td>
<td>10·5</td>
<td>72·7</td>
<td>—</td>
<td>38·9</td>
<td>29·3</td>
<td>31·8</td>
<td>—</td>
</tr>
</tbody>
</table>

The great drop in “other respiratory” in 1918-19—figure 19·3 etc., in penultimate column—would indicate:

(a) that influenza was in effect epidemic throughout the war; or
(b) that during the epidemic many diseases were called “influenza” that were not influenza; or
(c) that there is a great mass of respiratory disease whose breeding, being unknown or promiscuous, is labelled by the symptom-complex that best fits its clinical manifestations; but that when a specific disease like influenza is discovered—or invented—there is a natural tending in the medical profession to identify it more exactly than is justified.

Moral, why not “P.U.O.” in civil practice?

The history of the epidemic in Australia is necessarily dealt with later in connection with the return of the A.I.F. to Australia and with the quarantine precautions taken there, with considerable success, to prevent or delay the entrance of the pandemic invader. 87

87 This chapter is based, for the most part, on contemporary records of the 1914-18 war. It has not been brought precisely into line with present-day views. However, the facts and even the opinions recorded have a practical, as well as historical, interest in view of the possibility that the Second World-War pandemic may again be a respiratory infection. For the experiences of the Royal Australian Navy see Chapter VII.
SYNOPSIS OF CHAPTER V

THE SCIENTIFIC BACKGROUND OF ARMY MEDICINE IN THE WAR

THE SCIENTIFIC OUTLOOK IN 1914.
(a) General.
(b) In Australia.

GENESIS OF SCIENTIFIC MEDICINE IN THE ARMY.
(a) In the British Army.
(b) In the Australian Military Forces.

Structure of scientific medicine in the Army.
Clinical research
Administration of research.
The R.A.M.C. laboratories.
The Medical Research Committee
Agencies for dissemination
Individual.
Collective dissemination.
Publications.
Interallied Sanitary Commission.
The Annual "Conferences".

A HIGHLIGHT OF COMBINED RESEARCH—THE "DISCOVERY" OF TRENCH FEVER.

Scope and Limitations.
The Medical and Surgical Consultants.
The Adviser in Pathology.

Medical research in the Eastern Theatre.
Pathology—The Anzac Field Laboratory.
Pathology—Research at No. 14 A.G.H.
Clinical Medicine—The discovery of rectal Bilharziasis.
Collection of Museum specimens.

Medical research in the Western Theatre.
Pathology: The A.I.F. Central Laboratory.
The episode of Cerebro-spinal Fever.
A.I.F. Laboratories in France.
Bio-physical research: Problems of Flying.
Clinical Science: The Physicians in the A.I.F.

Surgery in the A.I.F.
Surgery in the Gallipoli Campaign.
Surgery in the Palestine Campaign.
Surgery in the Western Theatre.
Collection of Museum Specimens.