A Study of the Etiology of Recurrent Epidemics of Primary Pneumonia in Children, particularly with regard to their Relation to the Weather. Adelaide, 1930-35.

by

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C O N T E N T S.

I. INTRODUCTION.

At the present time, primary pneumonia is largely an endemic disease; sporadic cases occur frequently in most well populated parts of the world. The epidemic form of the disease appears to be rare, having reached major proportions during this century only at Panama and in the mining districts of South Africa. Quoting these epidemics as exceptions, Zinsser (1) has pointed out that the epidemic form of the primary disease now only develops under such conditions as those prevailing in army camps during the cold weather, when men are crowded together in their sleeping quarters, are developing coughs and colds, and, at the same time, are exposed to unusual conditions of life, to cold and wet, to unaccustomed food and to hard work. It is thus of interest to find that the disease occurs in epidemic form among the children of Adelaide, South Australia. One of these epidemics occurred during a warm, dry Summer, and the cases were widely distributed.

Adelaide has a dry, sub-tropical climate. It is a typical city of the Southern Hemisphere; the standard of hygiene is high, the great majority of families are housed in separate dwellings, and slums are noticeable for their absence.

The curves of incidence in this study are a chronological representation of the 1250 primary pneumonias admitted to the Children's Hospital of the City of Adelaide during the years 1930-35. The cases were diagnosed by the physicians of the Hospital. As a result of extensive hospitalisation, the cases in large degree represent the morbidity from pneumonia among the children of the poorer classes. It is the custom to admit to the Wards, all cases of pneumonia, or of hyper-pyrexia of unknown origin, which are brought to the Hospital Out-Patient Department. In the case of the unemployed, many receive medical attention here for the first time, and cases of this social class, if seen by the medical practitioner, are referred by him to the Hospital. As approximately one half of all the admissions to the Hospital are from the homes of the unemployed, and the rest are from the low-wage earning classes, the figures represent most of the pneumonias of this portion of the child population. The representative nature of the epidemic curves is indicated by their sharp definition and regular unfolding.

No separate grouping has been made of the lobar and bronchopneumonias of the Hospital Records, such a grouping probably being subject to different standards of diagnosing

between the types[2]; the curves indicate the incidence of primary, pneumococcal pneumonia in children from birth to twelve years. The epidemic pneumonias seen by me, were chiefly of the type which has been described as "alveolar"[2]. The fever was high and usually mildly remittent. In the majority of cases it fell by crisis, with subsequent rapid improvement in the general condition. In the Summer Epidemic of 1935, the average duration was seven days; in only three cases out of forty, it lasted longer than ten days. In about one third of the cases, moist breath sounds were generalised, but were seldom prominent. The area of consolidation was in many cases small, a typical feature of pneumonia in childhood. The mortality was low. Death occurred in 71 of the 1250 cases, a proportion of 5.7 per cent..

II. HISTORY.

Relation of disease to the weather dates from the earliest times. Its occasional occurrence after chilling, must have forced itself upon the understanding of primitive man. We owe the first crystallisation of the thought to Hippocrates:[3] "..... To all those which have been described, the season of Spring was most inimical, and proved fatal to the greatest numbers; the Summer was the most favourable to them, and the fewest died then; in Autumn, and under the Pleiades, again there died great numbers." Two thousand years ago, the Father of Medicine thus described the influence of the seasons on the death-rate from phthisis.

(a) The Occurrence of Epidemic Pneumonia.

The relation of epidemic pneumonia to the weather, and its history up to the second half of the last century, have been extensively surveyed by Hirsch,[4] whose description I have largely followed. The first information on the epidemic prevalence of "Inflammation of the Lungs" is met with in the 16th century chronicles of pestilence in Southern Europe. In Italy, epidemics were seen at Venice

in the Autumn and Winter of 1535, at Brescia and other places in Lombardy in 1537, and over the greater part of the Peninsula in the Winter and Spring of 1563-64. The disease recurred in this country at frequent intervals throughout the next three centuries, often in severe form. Relation of epidemic pneumonia to the weather for the years 1925-28, has been made recently in Rome by Barbera, (5) who made use of the figures of morbidity among the insured population. It is, however, difficult to make out the existence of epidemics in his curves of incidence. The appearances are suggestive of endemicity. A statistical survey in the 20th century thus barely discloses the existence of epidemics in a country where its existence in epidemic form was first chronicled by clinical observers, and where this form of the disease for long occurred most frequently. It is of considerable interest to note that, towards the end of the 17th century, there was in Italy a controversy with regard to the contagiousness of the disease, opinions being almost equally divided.

During the 16th and 17th centuries, epidemics also occurred frequently in the Swiss Alps at the time of melting of the snows, and throughout France and in Germany, particularly in the Rhine basin. Severe epidemics occurred

in France with monotonous regularity during the economic stress of the pre-revolution period; these relatively widespread epidemics became epidemics of garrisons and prisons subsequent to the Revolution, and, coincident with the general improvement in the living conditions of the people, epidemic pneumonia practically disappeared from France during the second half of the 19th century.

Accounts of the disease in the United States date from the beginning of the 18th century, and "are very numerous". Scattered outbreaks occurred during the first ten years of the 19th century. These were the precursors of a pandemic of pneumonia which extended, during the years 1812-25, from Canada in the North to the Gulf of Mexico in the South. Subsequently, occasional notices of epidemic pneumonia are met with in various parts of the country. The conditions under which epidemics of pneumonia are likely to supervene in North America at the present time, have been previously quoted (p.1.).

It is not obvious from the literature whether epidemics of pneumonia ever recurred frequently in Great Britain. Huxham,(6) in his account of various diseases in Plymouth during the years 1729-36, describes "pneumonies and peri-pneumonies" as "raging very much" and "exceedingly frequent

everywhere" in the first quarter of 1731. His adequate account of what appears to have been an epidemic of influenza ("Catarrhal Fever"), during the first few months of 1733, with the "bastard pneumonies and peripneumonies" attendant upon this disease, render his observations all the more valuable. During the eight year period related by Huxham, the incidence of pneumonia was only once greatly increased in Plymouth. In his summary of the literature, Hirsch finds that accounts of epidemic pneumonia have been relatively infrequent in this country. Increased prevalence occurred in Fife in the Winter of 1736. It occurred in Plymouth again in the Winter of 1740 and in the Spring of 1746; in London in the Winter of 1805; in several villages of North Devon in the Spring of 1875; in Liverpool during the years 1876-82 (an unusual number of cases in sailors and dock labourers, treated at the Northern Hospital, and characterised by severe nervous symptoms); at Scunthorpe, Lancashire, in the Spring of 1878 (confined to certain streets); and at Dingwall, Ross-shire, in the Spring of 1883 (also limited in distribution).

While the epidemics of past centuries, from their clinical descriptions, were in the majority of cases apparently epidemics of pneumonia, this form of the disease had

largely disappeared by the time an accurate diagnosis, clinical and bacteriological, had become possible. The position was thus summarised in the 1908 edition of Allbutt and Rolleston's System of Medicine:(7) "Pneumonia has been described as occurring in epidemic form. From the Middle Ages onwards, we have accounts of acute epidemic disorders which seem more like pneumonia than any other disease; and from time to time, circumscribed epidemics in the same village or house have been reported in this country and in other parts of Europe. But there is not sufficient evidence to show that these epidemics are pneumococcal pneumonia rather than influenza or some similar infection." Since then, the disease has established itself in major epidemic form during the second decade of the present century, in the Canal Zone at Panama, and in the mining districts of South Africa.

(b) Relation to the Weather.

Hirsch,(4) summarising in 1886 the reports of many writers, concludes that pneumonia, whether sporadic or epidemic, is for its origin, dependent on weather influences proper to the seasons, and more particularly upon sudden changes in temperature and considerable fluctuations in the proportion

of moisture in the atmosphere. He records the observation of several writers that any exceptionally large number of cases of inflammation of the lungs, occurring other than during Spring or Autumn, has coincided with the prevalence of the same meteorological conditions phenomenally during that season. He points out that his conclusion is borne out by the fact that in these Northern regions (Russia, Sweden, Denmark, Germany, England, the North of France, and the Northern States of the American Union), where the most sudden and severe changes of temperature fall in the Spring, the largest number of cases occur in this season; while in the warmer and sub-tropical countries, (Italy, the islands of the Mediterranean, Spain and Portugal, Greece, Algiers, the Southern States of the American Union, Chile and Peru), which are subject to these meteorological influences for the most part in Winter, it is Winter that shows the highest occurrence of pneumonia; on the other hand, those parts of the tropics noted for the steadiness of the climate show a relatively low incidence of the disease.

Seibert,(8) (1884), studying the relation of the onset of pneumonia to the climate of New York, was of opinion that a thorough and scientific approach to the subject

could only be made by collating a large number of cases in a comparatively short time, and noting the meteorological conditions from day to day and in relation to one another. Basing his conclusions upon the returns of 768 cases made by 50 observers in New York, from March 1st, 1884, to March 1st, 1885, he pointed out that diurnal variations were followed by a rise or fall in the occurrence of pneumonia according to the meteorological states then prevailing. States, each of which promoted the occurrence of pneumonia, were "low and falling temperature, high and rising humidity, and high wind". When two of these meteorological states were present together, pneumonia was more frequent, and when all three were present, it was exceptionally frequent. Montgomery, (1935)[9], has defined chill as "the result of exposing the body to air, damp or dry, of a temperature and humidity that produces a loss of heat from the body faster than the heat mechanism is able to produce heat within the body." At a temperature of 32°F., i.e. at low temperatures, moist air feels colder than dry air, because under these conditions it takes away heat from the skin faster. It is well known that cold air in motion is more productive of chill than still air of the same temperature. Seibert's conclusions are thus in remarkable

accord with the present day physiological conception of chill, particularly when one bears in mind the cold nature of New York's Winter.

Since the advent of the bacteriological era, Rogers,(10) (1925), studying the disease in the prison population of India, came to the conclusion that the weather conditions promoting the occurrence of pneumonia were (1) a wide range of temperature during the 24 hours, (2) a low maximum temperature in the cold season, and (3) a low atmospheric humidity. He believes that chill is the most important element in producing pneumonia. Ordman,(11) (1935), studying the disease on the South African mines, also stressed the importance of wide range of temperature in the 24 hours. The disease tended to occur at the beginning of the rainy season, when the atmospheric humidity was at its lowest. Richter,(12) (1911), as a result of a comparative study of weather conditions and the onset of pulmonary disease, concluded that long periods of low atmospheric humidity (anticyclonic conditions) corresponded to periods of acute pulmonary inflammations. Barbera,(5) from his recent study of the disease in Italy, has concluded that increased relative humidity is the most important factor, cold North winds also being potential causes. The Ministry of Health (1919),

while preparing the Report on Influenza, correlated the death-rate from pneumonia to the mean temperature.[13] The closest relation was found to be to the mean temperature of the preceding week. It was shown that a drop of 1^{o} of air temperature, below the mean temperature of this week, was associated on the average with an increase of 2.5 deaths per million living above the normal average of the following week's mortality. These figures, making use of the statistics obtained in London over a period of forty years, show the ultimate relationship of the disease to cold. Young[14] (1924), correlating the number of weekly deaths from pneumonia in children with the mean temperature of each of the two preceding weeks for the same period of the year, found no evidence to believe that the mortality from pneumonia was related to the temperature of the same week, or to that of the preceding week. Young is of opinion that the difference between his conclusions and those arrived at during the Ministry of Health Investigation, is due to the influence of low temperature on the mortality from pneumonia in the aged being relatively greater. He did not find any relation of significance in his investigation of the rainfall and relative humidity.

The various investigations have in the main proved

the importance of chill as a factor in producing increased incidence of pneumonia, and such variations as may be present in their conclusions are probably largely due to the different proportions in which the chill producing elements of the weather are present in different climates.

(c) The Influence of Deficient Hygiene.

The frequent occurrence of the epidemic form of the disease in prisons and garrisons indicates that adequate hygiene is of importance in preventing this manifestation. Inefficiency may occur in several ways, of which overcrowding is of particular note on account of its importance in the propagation of carrier disease. Two classical epidemics are of interest in this connection. One was described by Akershus in Christiana (1866-67).(15) In a certain prison of this city, pneumonia occurred in 63 out of 360 prisoners. There was not a single case in the common prison of the city, and "the real reason had to be sought for in the excessive crowding and bad ventilation of the sleeping quarters".(4) The second instance occurred on three ships of the English Mediterranean Fleet, on which pneumonia was epidemic in 1860-61. Investigation disclosed that the sleeping quarters of the crew on the lower deck

had been crowded to excess, and that they were damp and ill-ventilated. The crews were transferred to other ships "with the best results".

Woods, (16) (1927), investigated the relation of social conditions to the occurrence of pneumonia in the age group 0-5 years. She used as her instances of social conditions, (a) overcrowding, as measured by the percentage more than two in a room, and (b), poverty, indicated by the numbers of pawnbrokers and moneylenders per 1000 occupied males (census 1921). She found that the mortality from pneumonia in the age group 0-5 years is closely related to social conditions, being much higher in the children of the poor and overcrowded. This factor appeared to be still important with regard to the mortality from pneumonia in middle life, but less so in later life.

III. THE EPIDEMIC CURVE, ADELAIDE, 1930-35.

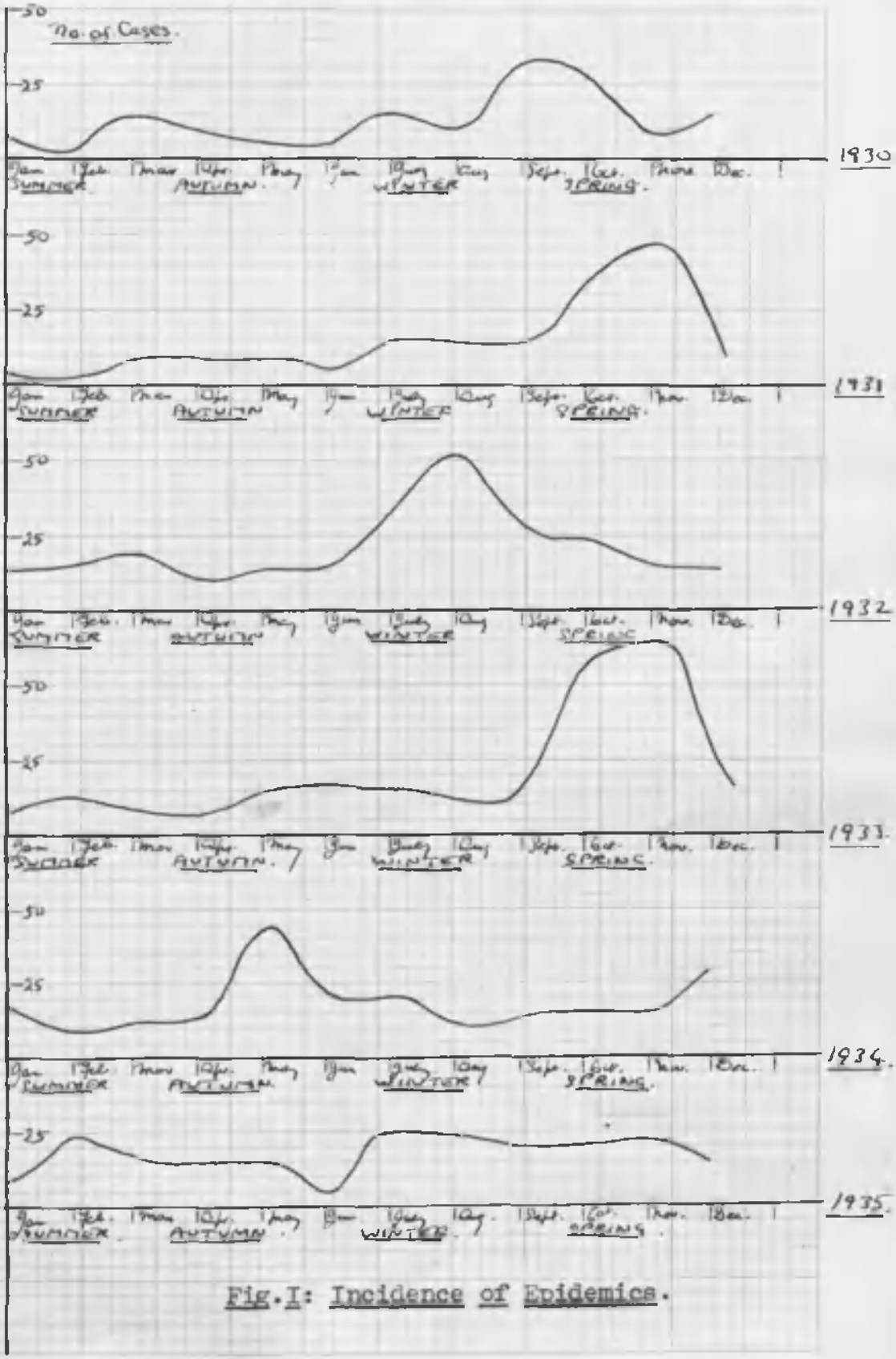
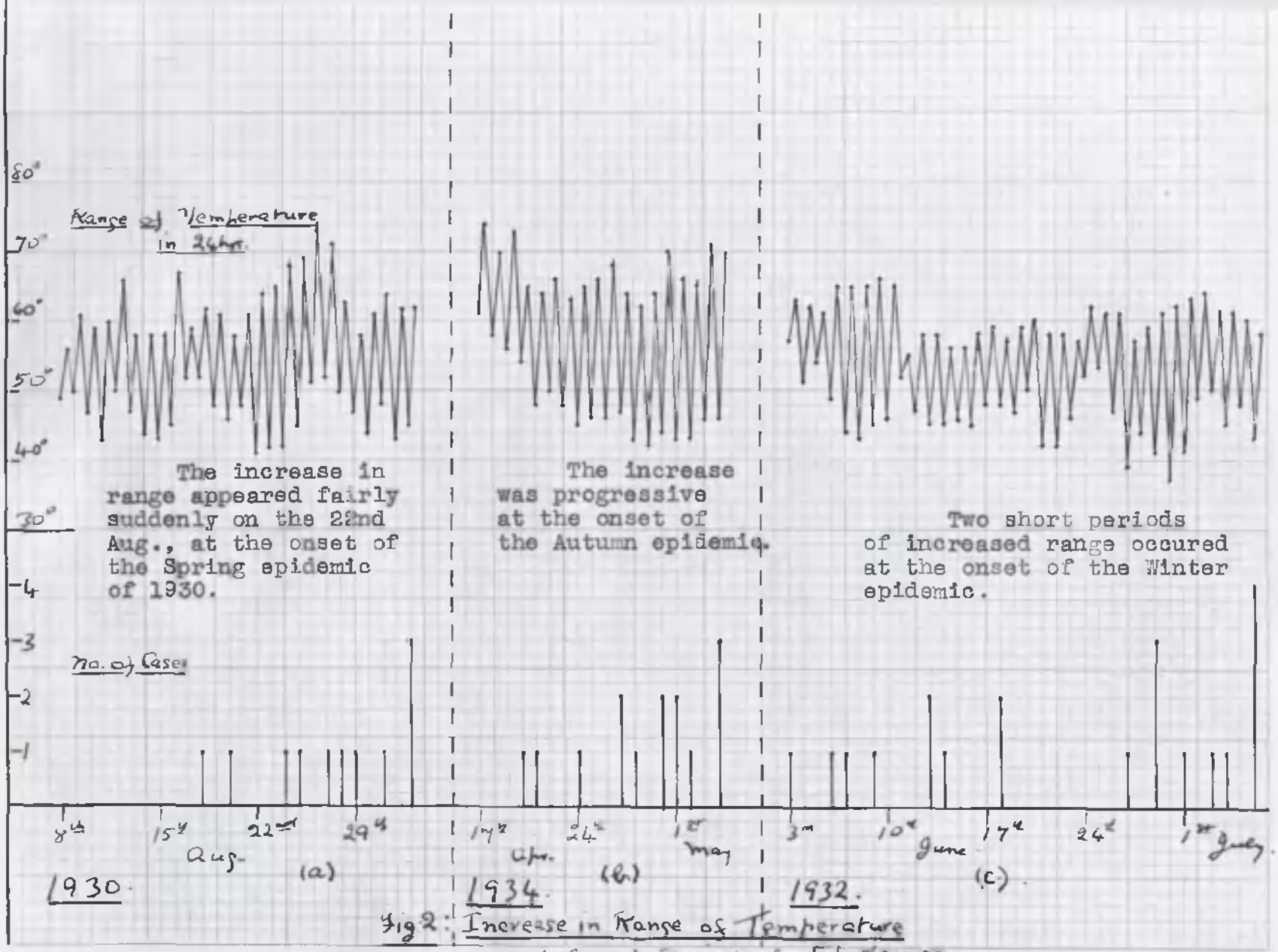


Fig. I: Incidence of Epidemics.

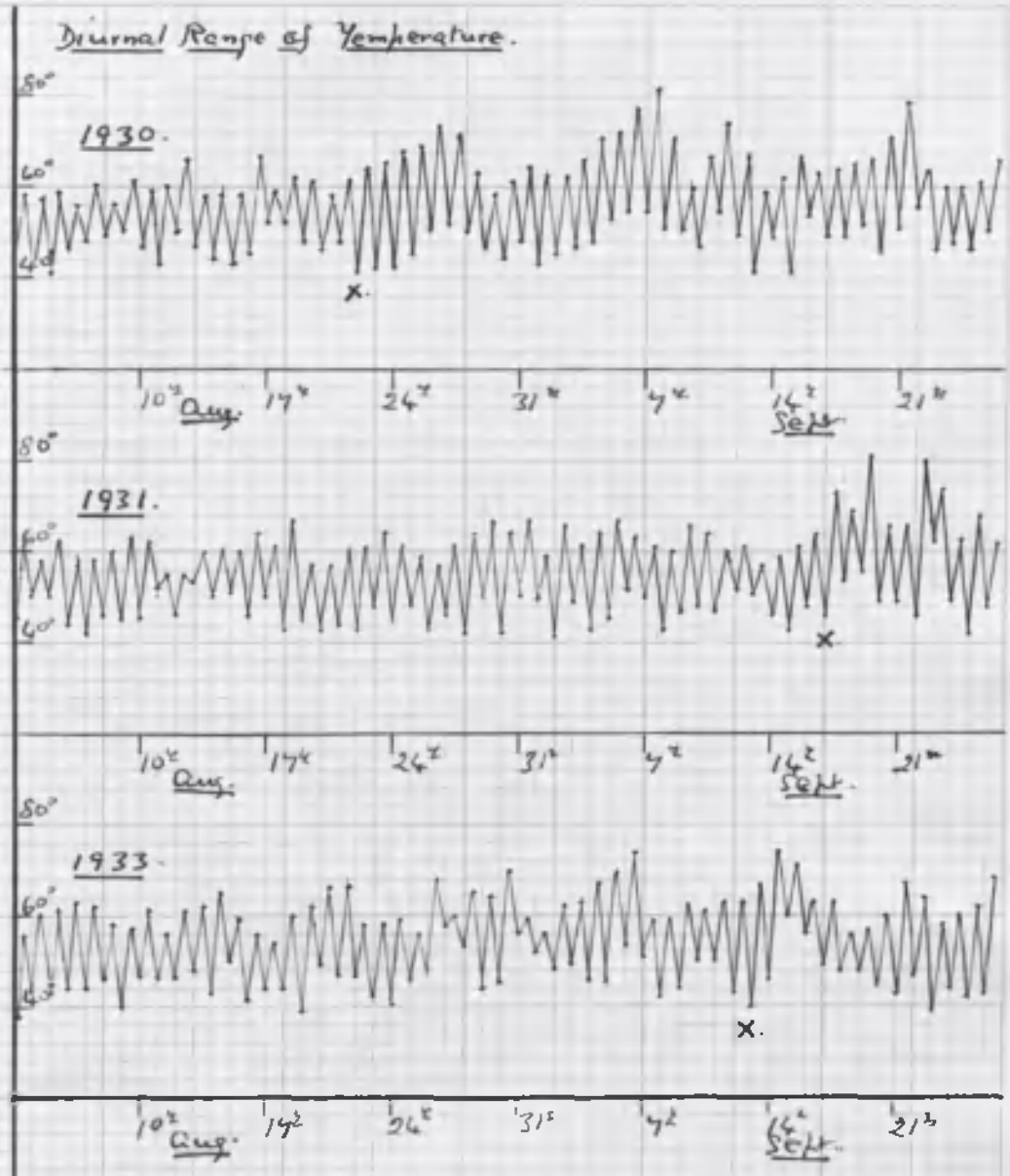


The increase in range appeared fairly suddenly on the 22nd Aug., at the onset of the Spring epidemic of 1930.

The increase was progressive at the onset of the Autumn epidemic.

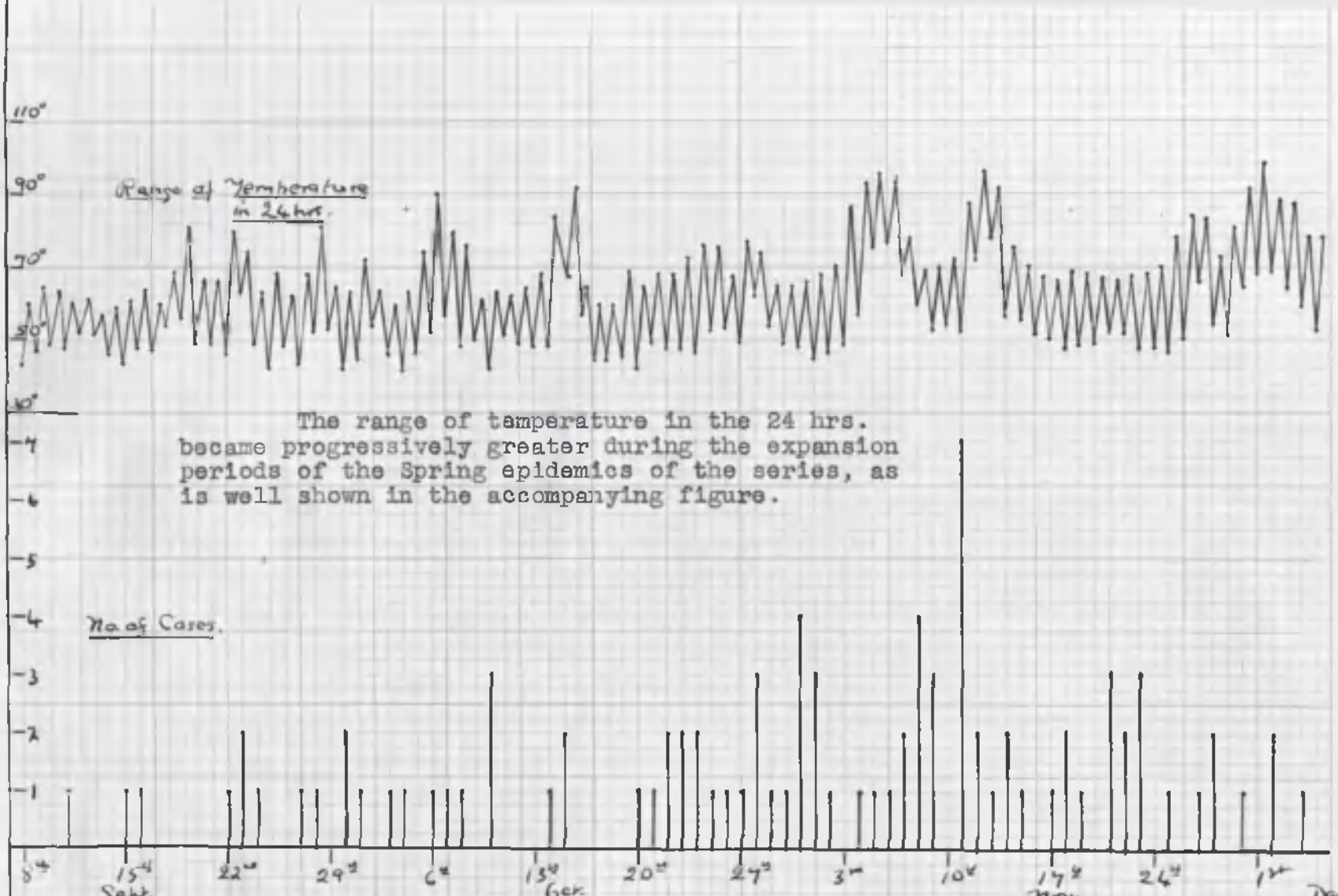
Two short periods of increased range occured at the onset of the Winter epidemic.

Fig 2: Increase in Range of Temperature at Onset Period of Epidemics.



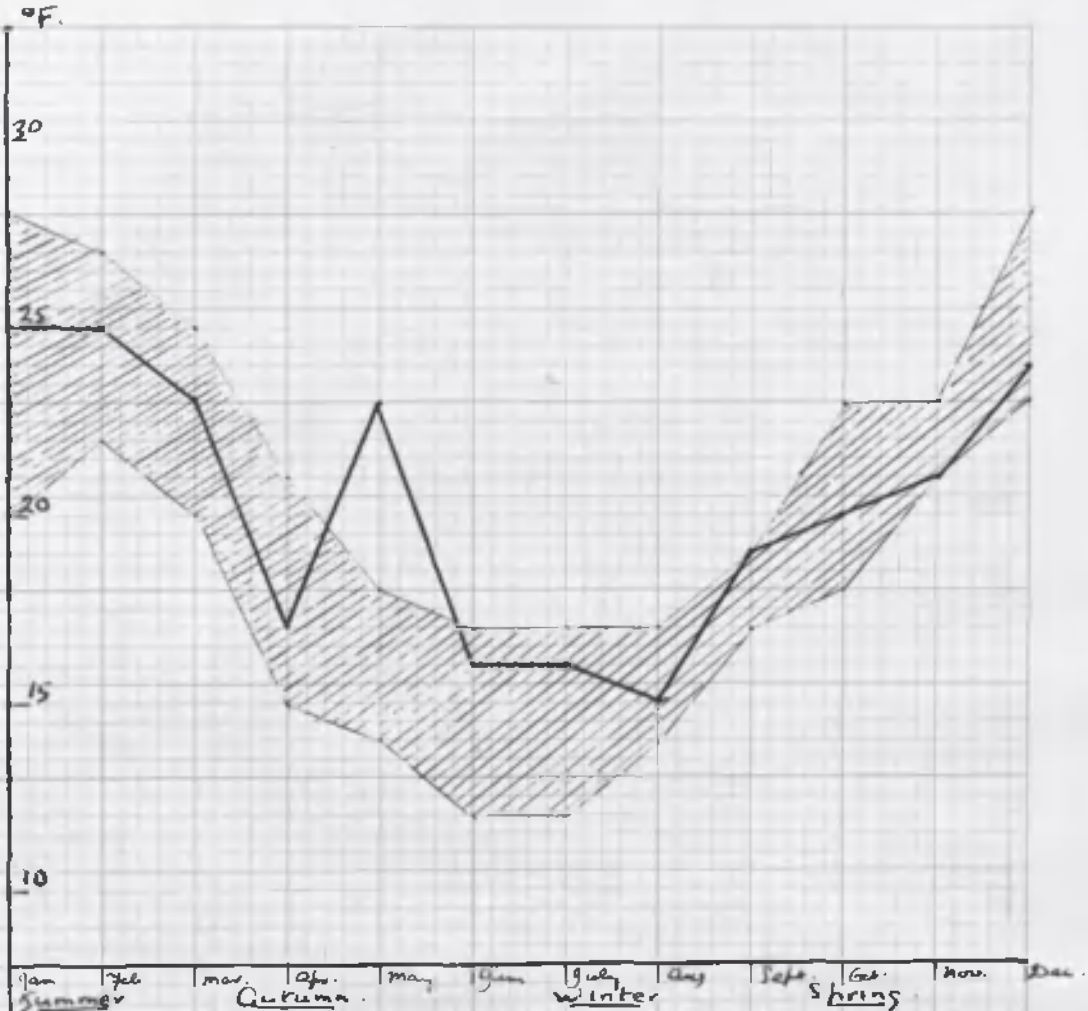
X marks the onset of increased diurnal range of temperature in the Springs of I930, I93I and I933. This appeared earlier in I930 than in I93I and I933. It is evident from Fig.I that the Spring epidemic of pneumonia of I930 appeared correspondingly earlier than that of I93I and that of I933.

Fig. 3.



The range of temperature in the 24 hrs. became progressively greater during the expansion periods of the Spring epidemics of the series, as is well shown in the accompanying figure.

Fig. 4 : A Typical Spring Epidemic of Pneumonia.

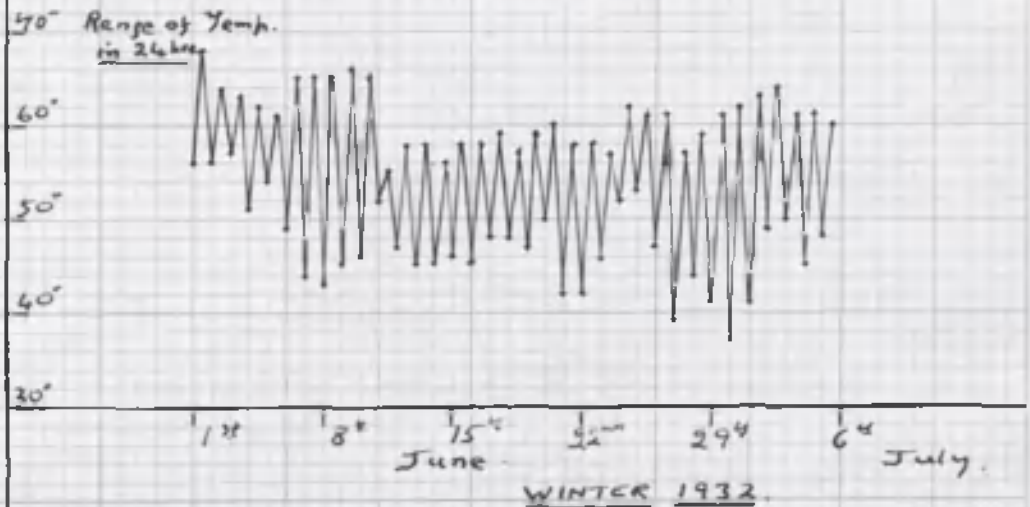


Average Monthly Range of Temperature in 24 hrs. during the Years 1930,'31,'32,'33,'35. (Limits of).

Average Monthly Range of Temperature in 24 hrs. during 1934. The abnormal range in the Autumn of this year determined the only epidemic of the series which occurred during this Season.

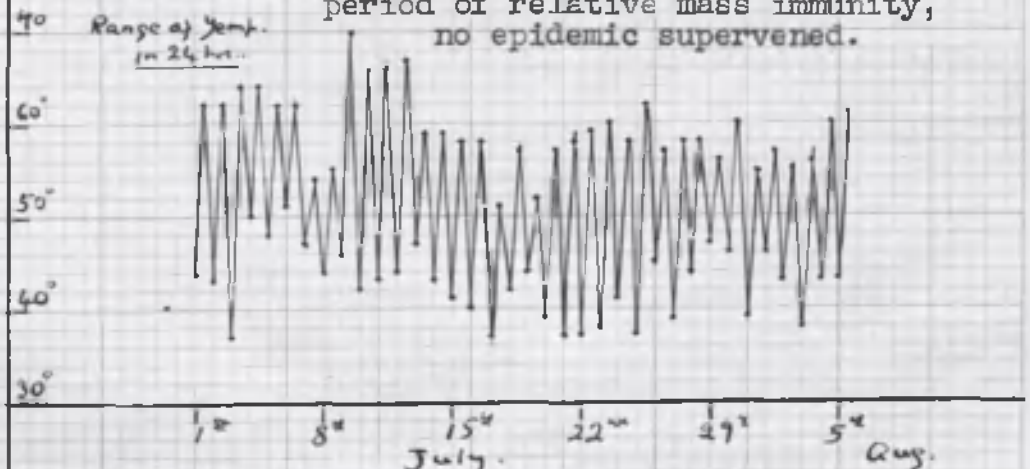
Fig. 5.

The Diurnal Range at the Onset of the Winter Epidemic, 1932. (Vide Fig.2c.). Two short, four-day periods of increased range occurred.



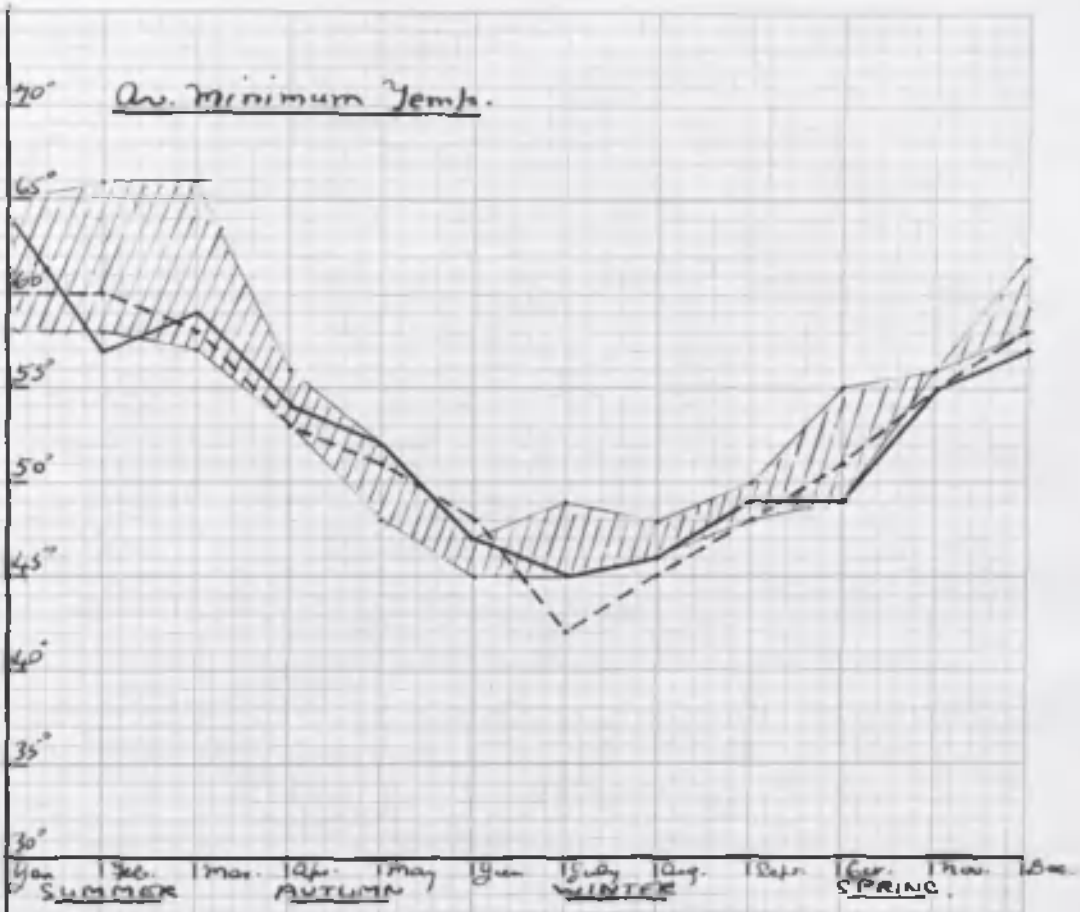
WINTER 1932.

The Diurnal Range was also increased, and during a somewhat longer period, in the month of July, in the Winter of the following year, 1933. In spite of a longer preceding period of relative mass immunity, no epidemic supervened.



WINTER 1933.

Fig. 6.



—— Qu. Minimum Temp. 1932.

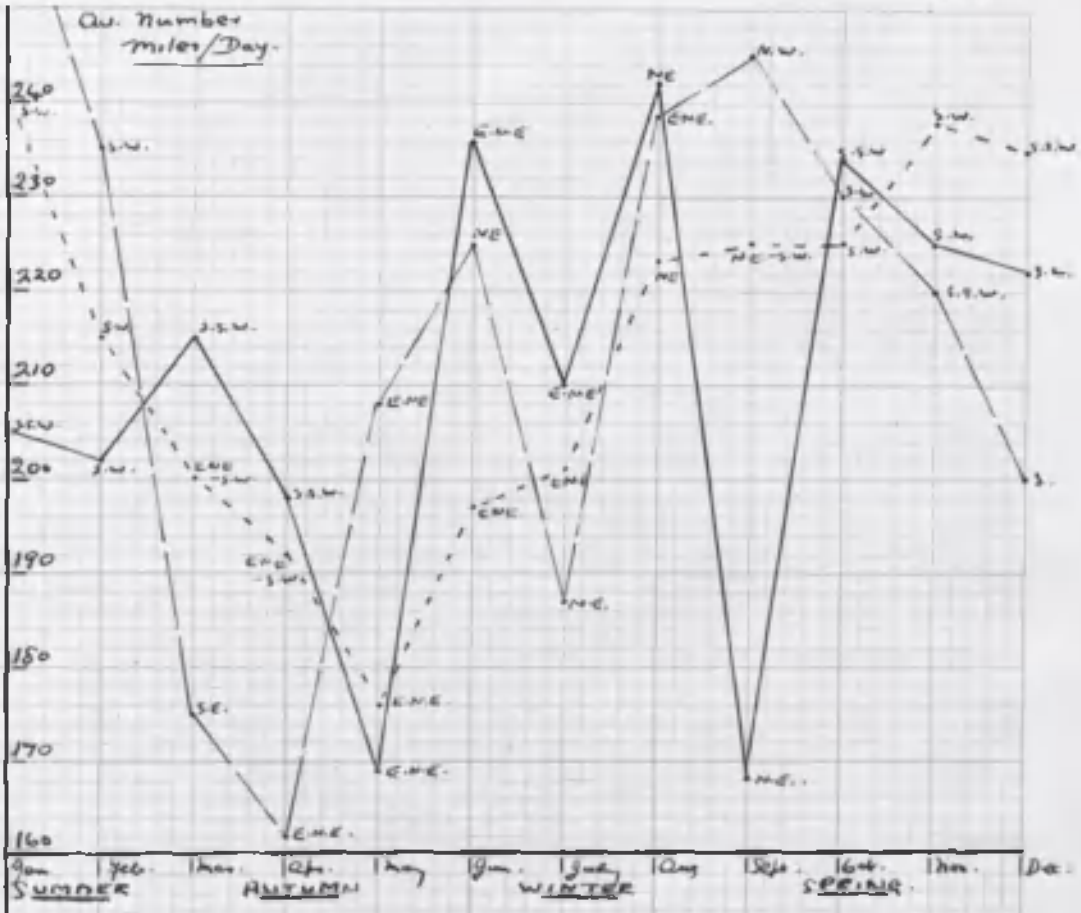
---- Qu. Minimum Temp. 1933.

Limits of Qu. Minimum Temp. during the other four years of the Series.

The Winter of 1933 was the coldest of the series, but no epidemic occurred, although the previous epidemic was 12 mths. removed.

After a similar period of relative mass immunity, the only Winter epidemic of the series occurred in 1932, the Winter being one of only average coldness.

Fig. 7.



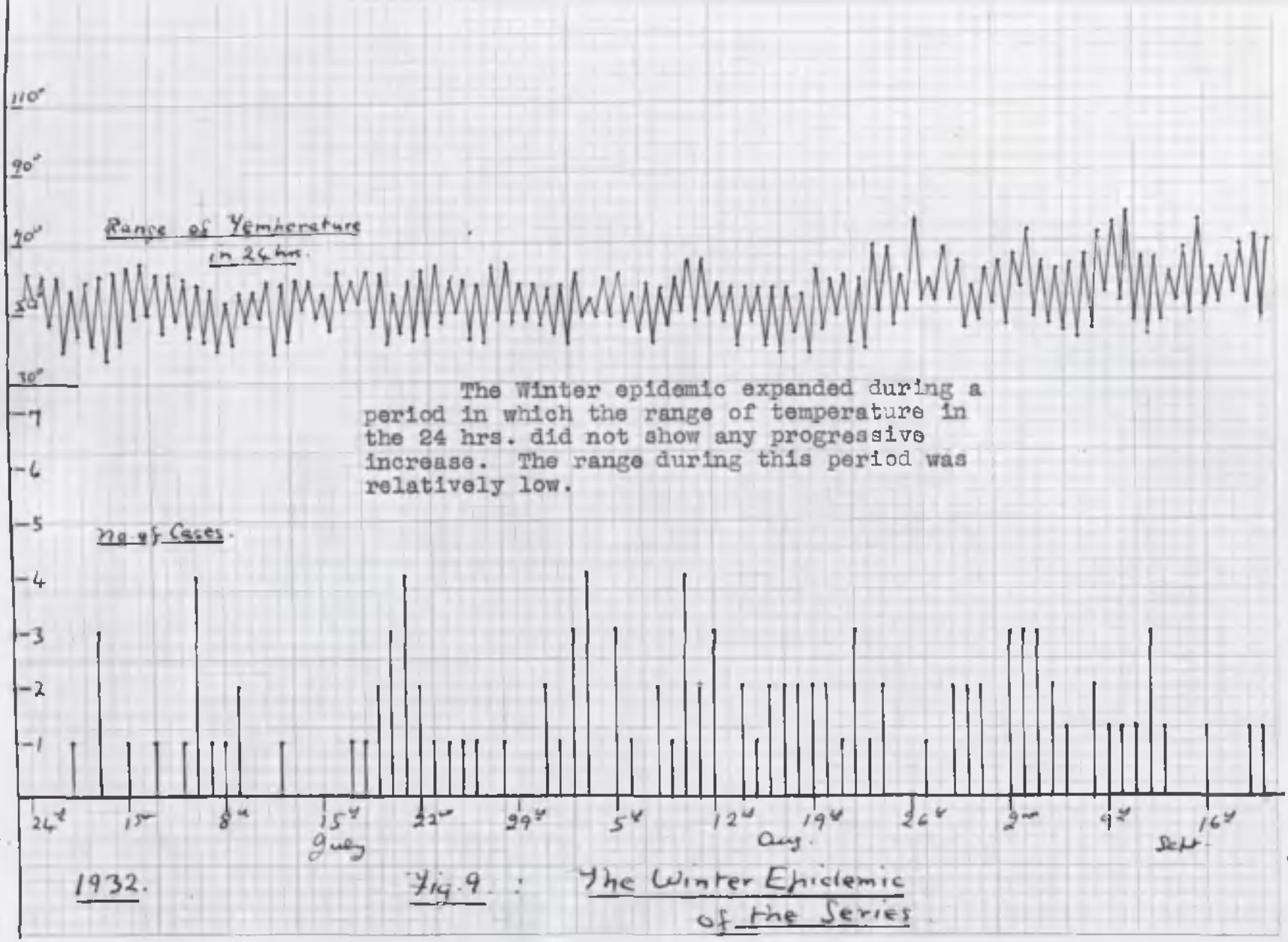
——— Av. Wind Speed 1932.

——— Av. Wind Speed 1931

- - - - Six Years Av. 1930-35.

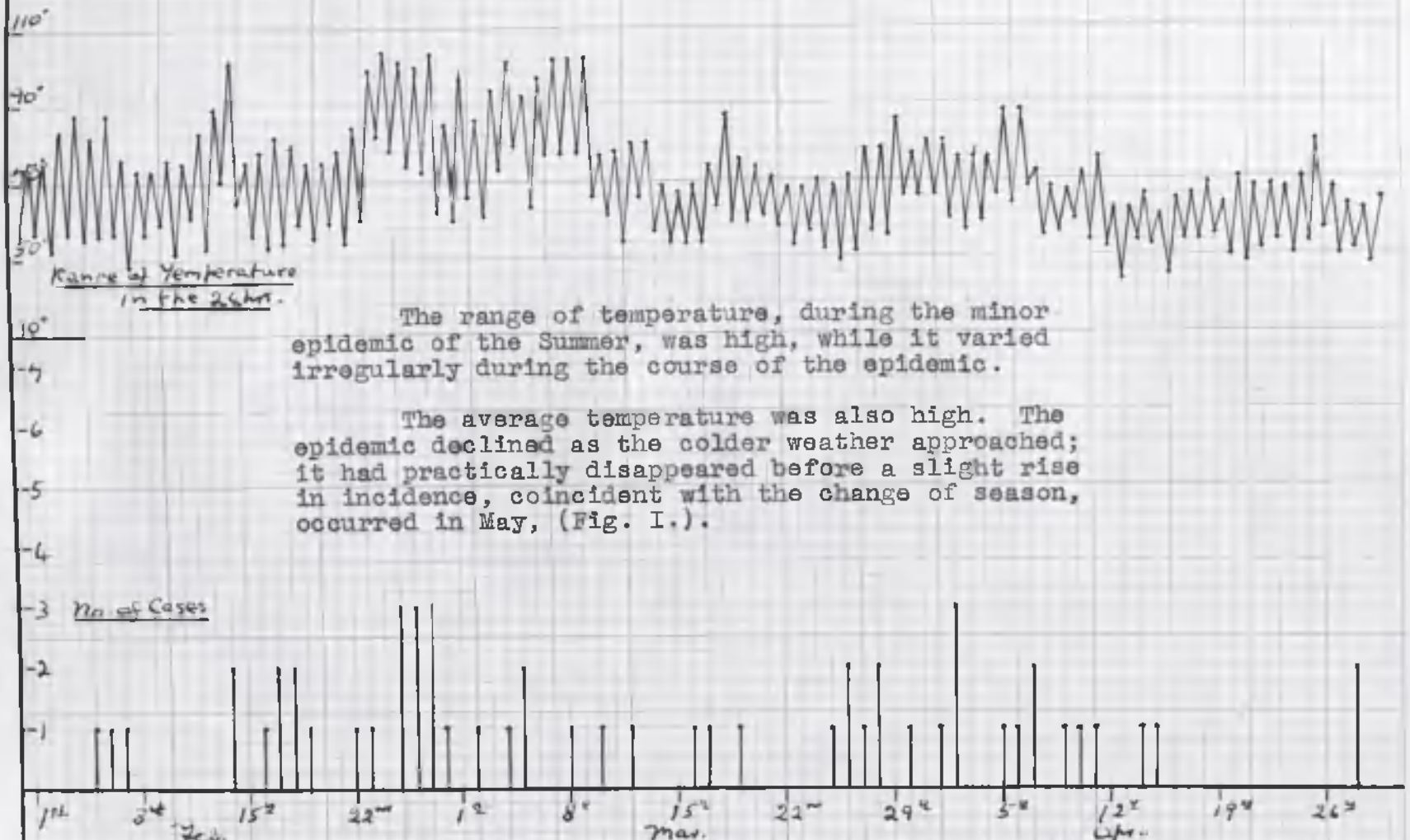
The Average Wind Speed was abnormally high in the Winter of 1932. While similar, if slightly lower Wind Speeds, were registered in the Winter of 1931, without a Winter Epidemic occurring, the raised Wind Speed in the Winter of 1932 may have been of significance in the promotion of the Epidemic. There is, however, no close relation to the unfolding of this Winter Epidemic. While increased incidence first appeared in June, and progressive increase in the number of cases occurred in July (viz. Figs. 2c & 9) the average Wind Speed was less in July than in June.

Fig. 8.



The Winter epidemic expanded during a period in which the range of temperature in the 24 hrs. did not show any progressive increase. The range during this period was relatively low.

Fig. 9 : The Winter Epidemic of the Series.



The range of temperature, during the minor epidemic of the Summer, was high, while it varied irregularly during the course of the epidemic.

The average temperature was also high. The epidemic declined as the colder weather approached; it had practically disappeared before a slight rise in incidence, coincident with the change of season, occurred in May, (Fig. I.).

Fig. 10 : The Minor Epidemic of Late Summer, 1935.

THE EPIDEMIC CURVE.

From the curve of incidence depicted in Fig.1, it is evident that a well defined major epidemic of primary pneumonia occurred among the children of Adelaide during each of the five years 1930, 1931, 1932, 1933 and 1934. Each epidemic was of approximately three months' duration, steady increase in the number of cases taking place over a period of one and a half to two months, at the end of which time the maximum number of cases was occurring. The subsequent regular decline occupied a somewhat shorter period (Fig.4). Out of 1041 cases recorded during these five major epidemic years, 516 occurred during epidemics. The proportion of the yearly morbidity taking this form varied from 42 per cent. in 1934 to 57 per cent. in 1933. An index of the epidemic nature of the disease is given by comparing the incidence in November, 1933, to that of the same month in 1934. The seasons are reversed in the Southern Hemisphere, so that this is the last month of the Australian Spring. During these two months, the weather was similar, but in the former 65 pneumonias were admitted to the Hospital, in the latter only 14. The morbidity, during a month of changeable weather coincident with a major epidemic, was

therefore almost five times as great as during the corresponding month of the following year, the weather of which month was equally changeable, but without accompanying epidemic pneumonia.

In 1935, the last year of the series, a minor epidemic occurred in the late Summer and early Autumn (Fig. 10); it resembled the major epidemics in appearance and duration, but there were fewer cases. After the disappearance of this epidemic, the next marked rise in incidence occurred in mid-Winter during the same year (Fig. 1); this high level of incidence persisted during the remaining five or six months of relatively cold weather. No epidemic peak occurred during this time. During these cold weather months of 1935, the incidence resembled that of endemic pneumonia.

(a) The Relation of the Epidemics to Season.

In 1930, 1931 and 1933, the major epidemics occurred in the Spring, in 1934 in the Autumn, and in 1932 in the Winter. Four out of the five major epidemics of the series thus occurred during the change of season. The preponderance of epidemics fell in the Spring (Fig. 1).

(b) Relation to Temperature.

Degree of Temperature: Epidemic occurrence bore no close relation to the average degree of coldness. The Winter of 1933 was abnormally cold, (Fig.7), and, as is obvious from the curve of incidence (Fig. 1), a year of relative immunity had elapsed since the previous epidemic. In spite of these two circumstances, the epidemic of 1933 did not occur till the Spring. The average degree of coldness was greatest during the Winter months, yet only one of the epidemics occurred during the Winter, and it was a Winter of only average coldness (Fig. 7). It is of interest to note that the lowest temperature recorded during this epidemic of the Adelaide Winter, was one of 37°, or 5° above freezing point. The temperature was not depressed below this level during any of the epidemic periods. The lowest temperature recorded during the minor epidemic of the Summer of 1935 was one of 48°; the epidemic unfolded during the month of February, when the average temperature was 72°. Such an average temperature is very much higher than that of a fine July in England. (July is the corresponding month of an English Summer). These epidemics occurred, therefore, in a climate of which the temperature was relatively high; one of the epidemics occurred when

the average temperature was very high. While these epidemics occurred in a relatively warm climate, and bore no close relation to the degree of cold, it must be borne in mind that the major epidemics of this series occurred only during the colder months of the year.

Range of Temperature during the 24 hours: During the Winters, the minimum temperature and the range of temperature in the 24 hours were at their lowest average level. When the average temperature rose in the Spring, the minimum temperature, recorded in the early hours of the morning, stayed relatively low. The rising average temperature was consequently accompanied by a progressive increase in the diurnal range, (Fig. 4). The weather of Adelaide is unusually stable; sudden changes of weather are infrequent. The diurnal range in temperature thus increased in a regular fashion in the Spring, the increase continuing over a large number of days, as is well shown in the same Figure. Each of the Spring epidemics was preceded for a few days by this increased range of temperature, associated with low minimum temperatures; as the epidemic progressed, the range of temperature became greater. The close relationship between epidemic onset and increase in the diurnal range of temperature, is exemplified in the

following instance: In 1930, the increase in diurnal variation arrived in August, while in 1931 and in 1933, it arrived in September (Fig. 3). As is evident from the curve of incidence in Fig. 1. the epidemic of the Spring of 1930 correspondingly preceded those of the Spring months of 1931 and 1933.

Association of the Autumnal change of weather with the onset of an epidemic, occurred only in 1934. During the Autumns of 1930, 1931, 1932, and 1933, the high diurnal temperature variations of the Summer had become relatively small before the onset of the colder weather. Towards the end of April, 1934, however, an abnormal period of anti-cyclonic weather occurred; this abnormal weather persisted throughout the month of May. Instead of pursuing its customary downward trend of the Autumn, the diurnal range became greater (F.g 2b), thus following a similar course to that ordinarily occurring in the Spring. The first few days of this abnormal weather coincided with an increase in the number of pneumonias, which progressed regularly to major epidemic dimensions. This was the only period of weather of this type which occurred in the Autumn during the years studied (Fig. 5). It coincided with the only epidemic that occurred during this season of the year.

The four major epidemics which have been described, had thus developed with the arrival of weather during which the diurnal range of temperature steadily increased over a period of several weeks, while the minimum temperature was relatively low. The onset of the fifth, in the Winter of 1932, did not coincide with the onset of a period of maintained increase in the range of temperature during the 24 hours. At the beginning of the epidemic, two short, four-day periods of increased range occurred (Fig. 2c). In the following Winter, however, after a similar period of relative mass immunity, similar variations of temperature occurred, but failed to promote an epidemic, (Fig. 6). While the two short four-day periods of increased range at the beginning of the Winter epidemic cannot be disregarded in view of the great importance of this factor in the etiology of the pneumonia of this series, increase of the diurnal range is obviously of diminished importance in the causation of this epidemic. Increased range was quite absent during the month of July, when the incidence steadily increased, (Fig. 9). The unfolding of the Winter epidemic was thus unrelated to the extent of change of temperature during the 24 hours, a phenomenon contrasting markedly with the unfolding of the other major epidemics.

Like the Winter epidemic, the minor epidemic of the late Summer of 1935 failed to show any close relation to the diurnal range of temperature. The latter varied considerably during the epidemic period, but showed no constant relation to the course of the epidemic, (Fig. 10). All the range of temperature manifestations of this Summer were reproduced in the other Summers of the series, without the contemporary occurrence of epidemics. The epidemic appeared in February and rapidly attained its maximum number of cases during this month; the average range of temperature of the month, was one of 24^{o}, exactly that of the average in February for the six years of the study.

Summarising, one may conclude that degree of temperature was not intimately connected with the causation and course of these epidemics, although the major epidemics only occurred when the minimum temperature was low. The meteorological factor of chief importance was that of increased range of temperature in the 24 hours, a period of maintained increase usually coinciding with the onset and unfolding of the epidemics. This period, however, was practically absent in the case of one of the five major epidemics; it was of no apparent significance during the minor epidemic.

(c) Other Weather Factors.

Wind: No consistent relation was demonstrated between wind speed and epidemic onset. The climate of Adelaide is not a windy one. The highest speed per day averaged for any one month of the six years of this study, was that of the Summer month of January, 1934, a value of 265 miles in the 24 hours. The winds were highest in the Summer, falling to a low level in the Autumn, and rising again in the Winter and Spring. During the cold weather, the winds were usually Easterly, while during the warm weather they were Westerly.

It may be of significance, that, during the six years, the highest average wind speed recorded in the Winter month of June, occurred in 1932, (Fig. 8); the weather, particularly during the middle fortnight of this month, was cold and squally. While the wind speed of the same month in the following year was similarly raised, if not quite to the same extent, (Fig. 8), and without relation to epidemic occurrence, the month of June, 1932, saw the beginning of the Winter epidemic of the series. As has been shown, the importance of increased range of temperature was diminished with regard to this epidemic. It seems not un-

likely that, at the onset period of the epidemic, these abnormal winds acted in conjunction with the short periods of increased range of temperature which were shown to occur contemporaneously, thus creating a chill period of considerable intensity.

While abnormal winds may have promoted the onset of the Winter epidemic, they would not appear to have borne any close relation to its expansion; the epidemic appeared in June and expanded in July, but the average wind speed of the former month was greater than that of the latter, (Fig. 8). It is thus of considerable interest to note that both the chill factor represented by increased diurnal range of temperature, and that represented by abnormally high winds, diminished during the month of July, 1932, the month of expansion of the Winter epidemic.

Similarly, the highest monthly average for the Winter month of July, during the six years of the study, was registered in 1935, a value of 221 miles/day, as opposed to a six-yearly average for the same month, of 201 miles/day. This month also saw a rise in the incidence of pneumonia, (Fig. 1), which was not heralded by increased diurnal range of temperature; during the period of onset of this increase in incidence, the range was almost exactly that of the

average for the six years of the study.

In 1931, however, the rise in average wind speed in the Spring, preceded that of 1930. The positions with regard to epidemic onset were reversed, the onset of the Spring epidemic of 1930 taking place a month earlier than that of the Spring epidemic of 1931. Also, the lowest wind speed averaged during any one month of the whole six year period, occurred in May, 1934; this was the period of expansion of the Autumnal epidemic.

One must therefore conclude for this series, that periods of abnormally high wind, occurring during the colder weather, may be factors in the production of increased incidence of primary pneumonia, and may occasionally assist in the promotion of the epidemic form of the disease. Notwithstanding, the intimate relation which has been shown to exist between epidemic pneumonia and increased diurnal range of temperature, has no counterpart in its relation to wind speed.

Relative Humidity: During the periods of onset of the various epidemics, the average relative humidity corresponded largely to that of the average for the same periods in other years. The lowest average recorded during a month of epidemic onset, was that of February, 1935,

when the Summer epidemic appeared; this was 33 per cent. as compared with a six yearly average for the same period of 39 per cent.. The highest figure, recorded during the Winter epidemic was 72 per cent., as compared with a six yearly average of 71 per cent. for the same period. Of the four major epidemics occurring during the change of season, the average relative humidity in two, at the period of onset, was a little higher than that of the six yearly averages for the respective periods; in the two others it was less.

Relative humidity does not appear to be a factor of importance in the causation of the epidemic pneumonia of Adelaide children.

Rainfall: The average rainfall of the six year period showed that more inches of rain fell during the latter half of the year. The rain fell less consistently, but in heavier falls, during the first six months of the year. The heavy falls of rain were unrelated to the epidemics. The rainfall of Adelaide is relatively small. It bears no relation to the epidemic pneumonia.

Hours of Sunshine: Adelaide, like many other cities of the Southern Hemisphere, has a proportion of sunshine which has only to be experienced to be appreciated. While

the influence of sunlight on the incidence of pneumonia is not intimate, it is of interest to note that an epidemic of pneumonia, if a minor one, established itself during the month of February, 1935, when the sunshine averaged nine hours per day. During the three months of the Autumnal major epidemic, the sunshine averaged between four and five hours per day, an amount of sunshine at least equal to that of some English Summers. This latter epidemic occurred soon after a Summer during which the amount of sunshine could only be described as maximal. The average amount of sunshine per day for the mid-Winter month of July, during the six years of this study, was slightly over four hours. It may therefore be noted that these epidemics occurred in a climate which enjoys an unduly large proportion of sunshine, and that the disease may establish itself when the daily sunshine is maximal.

(d) The Epidemic Rhythm.

A period of approximately one year separated each of the first four epidemics, (Fig. 1). This rhythmic occurrence of epidemics was completely altered by the Autumnal epidemic of 1934, which, as has been shown, was determined by a period of abnormal weather. An interval

of only three months separated this epidemic from the preceding one. Subsequent to the close apposition of these two epidemics, a period of approximately four months elapsed before the next rise in incidence in December, 1934. These four Spring months had previously been the period of greatest atmospheric stimulus from the point of view of epidemic promotion; they had not been survived before without the superimposition of an epidemic period. It therefore seems likely that a short period of high relative mass immunity occurs after each epidemic of pneumonia. In this series, the inter-epidemic period varied from three months to one year; the weather ordinarily determined the duration of the interval, but a short period of about three months probably followed each epidemic during which time cold, changeable weather had no power to promote an epidemic.

The raised incidence in December, 1934, was not productive of an epidemic, probably because this month introduced the Summer. The incidence in the following month of January, 1935, was not above the average for the time of the year, but the disease proceeded to minor epidemic proportions in midsummer in February. The epidemic ran its course throughout February, March and April. While the number of cases was relatively small, the epidemic

closely resembled the major epidemics in appearance and duration. The maximum number of admissions per day occurred during a heat-wave; this unusual happening provoked the curiosity which resulted in this study. As the Summer epidemic occurred about six months after the last few cases of the previous epidemic, it happened during a period when the relative mass immunity had probably declined appreciably; the major epidemics previous to the fore-runner of the Summer epidemic had all occurred in the latter half of the corresponding years, while that previous to the Summer epidemic had occurred in the first half of the year. As this Summer occurred later in the immune period than the previous Summers, it is possible that the Adelaide Summer, with its high diurnal range of temperature, is potentially dangerous with regard to respiratory infection.

Subsequent to this Summer epidemic, increased incidence re-appeared in the mid-Winter month of July, 1935. This increase in incidence was maintained during the next five months, but no true epidemic tendency was shown; the appearance was more that of endemicity, (Fig. 1). It would appear that this recurrent form of epidemic pneumonia is not stable, as might have been expected in view of

its relative rarity.

(e) Notes on the Summer Epidemic.

The average temperature during the occurrence of this Summer epidemic was high; the weather was extremely dry, and the proportion of sunshine during the three months of the epidemic averaged almost seven hours per day; the relative humidity was consistently low. The diurnal range of temperature was, on the average, no greater than in the other Summers of the series. It seems likely that the part played by the weather in the promotion of this epidemic was relatively small.

It was therefore investigated with regard to the distribution of cases. With two exceptions, it was found that the forty cases of this minor epidemic came from different streets of the city, and from widely scattered areas. Two cases, however, came from the same street and were admitted within three days of each other, a fact of some interest when it is remembered that the epidemic period was of three months' duration. About the middle of the epidemic period, a one year old in-patient of the hospital developed a typical primary pneumonia.

IV. DISCUSSION.

The wide field of what was probably the epidemic pneumonia of the pre-bacterial era, has contracted greatly. The disappearance has been so radical that the epidemics of past centuries have been described (7) as "epidemics of inflammation of the lungs, more like pneumonia than any other disease." The epidemics of Panama and of the mining districts of South Africa, occurring after primary pneumonia had become a clinical entity, proved that typical primary pneumonia could establish itself on major epidemic scale. This study was made under circumstances in which the diagnosis, where necessary, was supplemented by modern methods, and where the weather was of such a stable nature as to render the etiological value of its separate elements fairly accurately assessable. It would indicate that the epidemic form of true pneumococcal pneumonia bears a relation to the weather resembling that of the "epidemic inflammation of the lungs" of pre-bacteriological days. It also shows that epidemic pneumonia can occur in a city where the standard of hygiene compares favourably with that of most large cities. The epidemic form of the disease has been unmasked by the hospitalising of a large proportion of the

cases occurring among a certain class. It is doubtful whether the individual general practitioner to the lower-wage-earning classes, with the small number of cases at his disposal, could have noted that increased incidence occurred over well marked periods of three months at varying times of the year. It is quite certain that he could not have had a sufficient number of cases of pneumonia with which to detect the epidemic form. It seems not unlikely that epidemic pneumonia may recur undetected in some cities where hospitalisation of pneumonias is minimal. Circumstances, however, probably render Adelaide unusually suitable for the epidemic propagation of pneumonia. Melbourne, the nearest large city, is 400 miles away, and intermingling of the populations is desultory, particularly with regard to the lower classes. The surrounding territory is agricultural and sparsely settled; it contains only small towns and scattered villages. The community of Adelaide is thus, to all intents, a closed one. The climate exhibits well sustained, recurrent chill periods. It has been known from the earliest times that chill is often the precursor of pneumonia. Sudden changes of temperature during the 24 hours were recognised to be of importance as early as the first half of the 19th century.(4) In hot, dry

climates, such as those of India or Adelaide, the extensive range during the 24 hours would appear to take the place of the sudden changes of the more temperate zones. In Adelaide, this factor increases in a regular way at the beginning of the Spring, so that, during the first few weeks of this season, a constant period of meteorological stimulus occurs yearly. One might have expected a progressive increase in the number of cases as the range of temperature increased, with a subsequent regular decline in the number of cases as the susceptibles of the population were weeded out. Such an hypothesis might have explained the Spring epidemics, and the Autumn epidemic of the series. It was probably the conception of the pre-bacteriological era. Hirsch (4) wrote in 1885 that pneumonia was ordinarily a disease of the Spring and Autumn, and that the occurrence of an unduly large number of cases other than during these seasons, was usually due to the weather being changeable out of season, particularly with regard to temperature and humidity. In this series, the epidemic pneumonia occurred most frequently in the Spring; the Autumn was not usually a period of marked temperature fluctuations. The diurnal range of temperature was, however, greatly increased throughout one of the six Autumns studied, and this determined the

only Autumnal epidemic. As the Autumn was not usually a period of excessive temperature fluctuations, this epidemic might have been said to have occurred as a result of the weather having been "changeable out of season". With regard to the Winter epidemic, it has been shown that the onset coincided with the appearance of two short periods of increased diurnal range of temperature, allied to abnormally high winds and cold weather.

As has been pointed out, however, while relating the disease to temperature and to wind, neither of these factors appeared to bear any close relation to the period of expansion of the Winter epidemic; the chill factor diminished during this period. Similarly, the minor epidemic of the series occurred during the Summer, when the temperature was relatively high and the weather could only have been classified as fine. In neither of these epidemics was there a meteorological manifestation of such a nature as to cause the uniform development of an epidemic. One must conclude for these epidemics that factors other than the weather played a large part; they were probably still of importance where the chill factor became progressively greater as the epidemics expanded.

It is well known that the distribution of the pneumo-

coccus is worldwide. It exists as a harmless saprophyte in the upper respiratory passages of a varying proportion of normal people. The disease only supervenes when the micro-organism gains the upper hand. The three great factors in the struggle, particularly with regard to epidemic occurrence, are (1) the virulence of the organism, (2) its mode and extent of transmission, and (3) the resisting powers of the host.

(a) The Virulence of the Organism.

The pneumococcus becomes less virulent as a result of frequent subculture on artificial media. This loss in virulence may be counteracted by frequent passage of the organism through laboratory animals. It has been pointed out by Griffith (17) that this loss of virulence is to some extent due to the dissociation of the organism into R (rough) forms, which are relatively avirulent, and into S (smooth) forms, which retain their virulence; the injection of the partially attenuated colonies into animals, results in the elimination of the R forms, only those colonies surviving which are best adapted to survival in the animal body. Felton and Dougherty, (1924) (18), have shown that this may not be the only reason for the increase

in virulence. By frequent transfers in milk at four-hourly intervals, they succeeded in greatly increasing the virulence of a strain of pneumococcus arising from a single cell.

Netter (19) observed a chronic carrier of the pneumococcus during a period of three years. Inoculating mice with the saliva of the carrier, he found that the weeks during which infection did not supervene, corresponded to a diminution in the number of deaths from pneumonia in Paris. From such a finding, one must conclude either that the virulence of the pneumococcus must alter with the seasons, or that the susceptibility of the mouse pursues a course parallel to that of man.

Dissociation. The importance of dissociation in the life history of the organism, was made clear by Griffith in 1923. (20) Growing the organism on Type specific antisera, he correlated the loss of virulence with the change from the S to the R type of organism. (21) Shibley has shown that the R forms are recoverable from the lungs and exudates of patients recovering from pneumonia. The change has been shown to take place 'in vivo'; Wadsworth and Sickles (22) have recovered the R form from horses undergoing immunisation with virulent strains.

The evaluation of dissociation has been extended in other diseases and for other organisms. Felix and Pitt[23] have shown for B.Typhosus that there is a marked parallelism between the virulence for mice and the resistance to agglutination. The occurrence of this biological form of dissociation has been confirmed by Kauffmann.[24]

Vogelsang[25] found only S forms of B.paratyphoid in 51 individuals sick with the disease or convalescent from it. On the other hand, among carriers who had been cured for some time, he found sometimes S, sometimes R forms, but often the two in association.

Describing epidemic cholera, Hornus[26] relates the finding of non-agglutinable vibrions in the water at the beginning of an epidemic, while actual cases are occurring. Then, little by little, proportionate to the extension of the epidemic, agglutinable vibrions appear. Later, when the epidemic diminishes in extent, but before it quite disappears, the vibrions resume their state of inagglutinability.

The importance of dissociation in the life history of the pneumococcus, indicates that it may also be a factor in the epidemiology of pneumonia.

Pneumococcal Types. The strains of the organism are

now generally classified into three main serological Types, I, II, and III, on the one hand, and into a Group IV on the other hand, representing these strains which are not agglutinable by any of the three chief Type sera. Working in America, Cooper (27) has been able to demonstrate 29 additional Types among the strains of Group IV. Griffith (17) gives the following figures as representative of the approximate proportions for the various types occurring in lobar pneumonia in Great Britain and America: Type I - 30 to 40 per cent., Type II - 20 to 30 per cent., Type III - 0 to 16 per cent., and Group IV about 30 per cent.. He contrasts these figures with the occurrence of pneumococci in the normal mouth secretions of 297 persons examined at the Rockefeller Institute; there was no history of contact with a recent case of lobar pneumonia given by any of these. Strains of pneumococci were recovered from 116. Of these, one example of Type I was found and none of Type II; Type III occurred 34 times, a percentage incidence of 28. The remainder belonged to Group IV. The chief Types tend to disappear from the nasopharynx during convalescence, a result which may either be due to the strains dying out, or as Griffith has shown to be possible, perhaps the result of a transformation to Group IV by the action of the immune

substances developing during recovery. Griffith (28) was the first to show that pneumococcal Types are convertible. He inoculated into the subcutaneous tissues of mice, an attenuated R strain derived from one Type, together with a large dose of virulent culture of another Type, killed by heating to 60°. This resulted in the formation of a virulent S pneumococcus of the same Type as the heated culture. He suggests that the pneumococcus, apparently residing as a harmless saprophyte in the nasopharynx, may in this way acquire virulent powers. "So long as it retains certain potentialities, indicated by the possession of S antigen, the most attenuated pneumococcus may develop the full equipment of virulence. The first essential is a situation in which it can multiply, unchecked by the inhibitory action of a healthy mucous membrane. In the nidus thus formed, the pneumococcus gradually builds up from the material furnished by its disintegrating companions, an antigenic structure with invasive properties sufficient to cope with the resistance of the host."

Such an hypothesis indicates the possibility of increased incidence occurring without the intervention of the transmission factor.

The variation in the virulence of the pneumococcus

has been adequately proved. It is manifested in its powers of dissociation and probably in its multiplicity of Types.

It has been shown by Topley (29) that variations in virulence may be of importance in the realms of experimental epidemiology. Infecting mice with B. Aertrycke, he found that an initially avirulent strain may become virulent while spreading within a closed community, such an event, however, by no means always happening.

It seems likely that some part is played by variations in virulence of the pneumococcus in the epidemic pneumonia in man, though to what extent, it is not known.

(b) The Factor of Transmission.

It is usually not possible to relate the average case of primary pneumonia to other cases of the disease occurring in the same district or city. That the relatively avirulent pneumococcus ordinarily an inhabitant of the nasopharynx, has in certain circumstances been converted experimentally to a virulent Type, indicates that pneumonia may occur spontaneously. Cases sometimes occur, however, in which a contact is smitten with the disease. Hospital practice provides evidence that persons suffering from

pneumococcal pneumonia, or its suppurative complications, may be sources of contagion and infect other patients.(7) The results of a study by Baermann(30) in 1914, are of interest with regard to the incidence of pneumonia where it is prevalent. The cases, occurring on a Javan plantation, he divided into three groups, (1) small localised epidemics, limited to groups of labourers in one hut or otherwise closely associated, and not dependent on weather conditions, as these were similar all over the plantation. He was of opinion that these depended on the passage of strains of special virulence from man to man. (2) A generally increased incidence of pneumonia all over the plantation at certain seasons and dependent on weather conditions. (3) A small stream of hospital infections of severe course, which he assumed to be due to a strain of pneumococcus more prone than others to persist in the wards. The first and last groups of Baermann's series would appear to be to a considerable extent dependent on the transmission element, the middle group more on weather conditions. Zinsser(1) investigated an epidemic which occurred in a regiment of soldiers who had been marching for long distances in wet weather and had been camping on wet ground; 26 pneumonias occurred within 16 days. Analysis showed that

they were caused by all four pneumococcal types, without particular relationship between contacts and types. He was of opinion that exposure, rather than transmission, was the important factor. Zinsser contrasts this with the apparent importance of the transmission factor in the South African epidemics, in which control of this factor coincided with marked diminution in the prevalence of the disease. He is of opinion that the greater susceptibility of the coloured racess increased the importance of this factor in this instance. Increased susceptibility seems undoubtedly to have been present,(11) particularly among the group of natives classified by Lister as "tropical".(10) Rogers has drawn attention to the inadequate clothing of the native races as a factor of importance in this connection.

It has been adequately shown that active cases of the disease result in an increased number of carriers of the pneumococcus.(31) Rosenau, Felton and Atwater, investigating the epidemiology of the disease in America, found among 180 normal persons who had not been exposed to pneumonia, carriers of the pneumococcus in the proportion of 2.2 per cent., 4.4 per cent., and 9.9 per cent., in Types I, II and III respectively. Among 270 persons who had been in contact with 28 cases, 14 became carriers of these Types;

carriers of the three chief Types were present in the proportions 8.1 per cent., 4.8 per cent., and 17.8 per cent. respectively. The raised proportions in Types I and III ware of particular significance in that only these Types were present among the cases to which the contacts were exposed. While this relatively high percentage of carriers resulted from contact with cases, carriers themselves were not found to be very productive of carriers; of 78 persons exposed to 14 carriers, only one became a carrier. Taken in conjunction with this last finding, it is of considerable interest to note that their study of 14 healthy carriers of the three chief Types, among whose families were no cases of pneumonia, failed to disclose the source of the organism. For the material of their investigation, they concluded that "the channels of spread of the pneumococcus are past finding out from the history of contacts usually available." These observers quote the result of an investigation relating the percentage occurrence of the chief Types in the nasopharynxes of normal individuals in a district where pneumonia was epidemic, to the occurrence in a similar group where pneumonia was not epidemic, (Table 1.). It indicates that a raised carrier rate may exist during epidemic prevalence of pneumonia.

Table I.

Incidence of carriers of pneumococcus in district where pneumonia was epidemic as opposed to incidence in nearby district where pneumonia was not epidemic.

	Baltimore, Sparrows Point, where pneumonia epidemic existed.	Baltimore, John Hopkins Hospital Dispensary.
Total persons examined:	100	50
Type I	6 per cent.	0 per cent.
Type II.....	10 " "	4 " "
Type III	6 " "	2 " "
Group IV	35 " "	32 " "
No pneumococci	43 " "	62 " "

Lenz, (32) (1917) has suggested that epidemic pneumonia is the result of a rise in the percentage of pneumococcal carriers in the community, subsequent to the widespread prevalence of catarrhs. He advanced this theory after observing an epidemic of pneumonia in a prisoners' camp in Bavaria in warm Spring weather during 1915. In subsequent years, much worse weather periods occurred without any great increase in the number of cases of pneumonia. A widespread epidemic of febrile catarrhs occurred simultaneously to this epidemic of pneumonia, and he was of opinion that the two were associated. Gordon, (33) (1921), using as his material a group of Chicago students, found pneumococci present in the nasopharynx in as many as 47 per cent. of cases of

bronchitis, in 36 per cent. of cases of cold, and in only 21 per cent. of healthy subjects. He described a localised epidemic of sore throats with unusual clinical features, among school children; investigation disclosed that a Group IV pneumococcus was present, showing similar immunological reactions in all cases, and distinct from pneumococci of the same Group isolated from healthy children in the school. It seems not unlikely, therefore, that febrile catarrhs of the upper respiratory passages may occur, of which the pneumococcus is the causal organism. Epidemics of these would lead to a carrier state of the community. The pneumococcus thus appears to possess properties common to the organismal causes of carrier diseases such as cerebrospinal fever.

Schroder and Cooper (34) have recently described an epidemic of pneumonia which occurred in a children's institution; the disease in this instance was highly infectious, the incidence falling where the opportunity for transmission was most marked. The morbidity was caused by a Group IV organism which proved to be Type V of Cooper's series. Cases occurred in which the pyrexia and malaise were of short duration; these received the clinical designation of "Colds". Of the cases which were seriously ill,

radiological evidence was necessary in some cases to confirm the presence of involvement of the lung; in the majority of the severe cases, however, the involvement of the lung was observable clinically.

The notes on the Summer epidemic of this series indicate that the epidemic pneumonia of Adelaide is not highly infectious. Cases, however, occasionally occur spontaneously in the wards of the Hospital. It is of interest to note that the father of one of the pneumonias admitted to the Children's Hospital by me, was in the General Hospital of the city suffering from lobar pneumonia.

During the six year period studied, many children were admitted to the Hospital suffering from what was described in the Hospital Records as "Nasopharyngitis". The clinical appearances were limited to nasopharyngeal injection, with accompanying marked toxaemia. The temperature was high over a period of one or two days, at the end of which time recovery set in with extraordinary rapidity. It is significant that the largest number of cases of "Nasopharyngitis" admitted during any one period, was during the Autumnal epidemic of pneumonia.

The steady rise in the number of cases over a period

of five to eight weeks, with an equally regular decline in incidence, show an epidemic form unlike that of highly contagious diseases such as influenza, where the largest number of cases often occurs early in the epidemic.

Summarising, it may be said that in the epidemic pneumonia of the children of Adelaide, case to case infection is infrequent; the epidemic pursues a regular course which is not necessarily dependent on weather changes; epidemic catarrhs of the nasopharynx may occur simultaneously. The appearances are those of carrier disease.

As has been shown by Schroder and Cooper, pneumonia may exist in highly contagious form; what is apparently a carrier form of the disease has been occasionally described; the disease exists ordinarily in endemic form, the cases occurring sporadically. It seems not unlikely that the manifestations of the transmission of the pneumococcus and of its power of infectivity, may be as kaleidoscopic as the morphology and typology of the organism, and, perhaps, as the appearances of the disease.

(c) Susceptibility of the Host.

Only a relatively small percentage of the child population of Adelaide became clinical cases of pneumonia dur-

ing each epidemic period. It is thus evident that a correspondingly high degree of resistance to the disease exists. A study of this factor entails a knowledge of the route of infection. It is significant that pulmonary lesions have rarely, if ever, been produced experimentally by subcutaneous, intraperitoneal, or intravenous injections. Probably the most successful experiments were those of Blake and Cecil,(35) who succeeded in obtaining a high percentage of positives by the injection of Type I culture into the tracheas of macacus monkeys; out of 31 monkeys thus inoculated, 26 developed pneumonia. Infection of the nasal passages and intravenous inoculation failed to produce pneumonia. McLeod,(36) in an extensive survey of the literature, suggests that the development of the pneumonic lesion in the human being is most probably explained by the gaining of access of virulent pneumococci to the trachea or bronchi, "some of which penetrate some part of the mucous membrane more deeply to invade the peribronchial tissues and, by continuous extension, all the adjacent lung tissues, until their progress is interrupted by an adjacent interlobar septum, or by the concentration of the body's anti-bacterial agencies."

It therefore seems likely that pneumococcal infection

in man is air-borne, and that the first line of defence is the respiratory mucous membrane. Resistance to the pneumococcus may therefore be advantageously discussed under two headings: (1) a local resistance, residing in the respiratory mucous membrane, and (2) the general resistance of the body to infection, depending on many factors.

(1) Local Resistance to Infection. It is well known that dust particles and noxious gases have an injurious effect on the respiratory mucosa, but the air of Adelaide is singularly free from these.

Chilling, on the other hand, has been shown to be of great influence in the promotion of these epidemics. It seems likely that this factor acts by causing a diminution in the power of resistance of the respiratory mucosa. Muecke and Hill (37) have noted the changes which take place in the respiratory mucosa when the subject is chilled. In a warm room, particularly if the feet are in a draught, the nasal mucosa is congested and swollen, and may be covered with thick mucous. On passing out into a cold atmosphere, the mucosa becomes pale as a result of vaso-constriction, but for some little time, it remains swollen and boggy. These observers suggest that "the ciliated cells, white corpuscles and lymph may be chilled, and the

velocity of the vital reaction reduced when the blood vessels constrict." They further point out that the air passing over the vasoconstricted mucosa is not properly heated in its downward passage. Deficient secretion of mucin may also occur. McDonald and Leisure[38], studying the ciliated epithelium lining of the maxillary sinus, have shown that, while warming increased the ciliary action and raised the local resistance, cooling had the converse effect. Kerr and Lagen[39] have shown that the transmission of the common cold is difficult in the absence of chilling. Isolating groups of individuals in an air-lock, the temperature and humidity of which could be maintained at any desired level, they showed that the common cold does not supervene, even under ideal circumstances for transmission, provided that the temperature is maintained at a constant level.

Observers are unanimous in assigning to chill an important part in the etiology of pneumonia. As has been previously suggested, the chill factors are probably present in different proportion in the different climates. It is of interest to note that the more recent commentaries, such as those of Rogers and Ordman, stress the importance of fall in temperature at night. As in Adelaide, those

studies were carried out in hot, dry climates. It seems likely that this factor is of particular importance in such climates, and that other factors, such as humidity and wind, and the sudden changes in temperature associated with weather changes, assume greater importance in colder and more equable regions, where they are more difficult to assess in consequence of their erratic nature.

(2) General Resistance. It was shown by Malling Hansen[40] in 1886, that the growth of children varies with the seasons. It is greatly diminished in the Winter and Spring, and is maximal in the late Summer and early Autumn. Palmer,[41] in America, has recently verified this conclusion, and Fitt,[42] working in Melbourne, has shown that a similar physiological cycle occurs in the Southern Hemisphere, where, as a result of the inversion of the seasons, the increase in weight takes place more rapidly during the months of December, January and February. The constituents of the blood alter during the seasons, variations having been observed in the size and number of the erythrocytes,[43] and in the percentage proportions of the white cells.[44] Variations may also occur in the weight of the thyroid gland,[45] and in its iodine content,[46] the latter being highest in the late Summer.

These variations are not only confined to physiological manifestations. Perkins[47] has shown that the blood content of diphtheria antitoxin is highest in the Autumn; Tobiasz[48] found that the highest proportion of Schick positive reactions were given during the Winter, the lowest during the Autumn. It was shown[49] in the classical experiments of Sudmersen and Glenny, (1909), that the guinea-pig is more susceptible to diphtheria toxin in the Spring.

The epidemic pneumonia of this series established itself once during the Summer and once during the Winter. As has been previously shown, the Summer epidemic occurred when the temperature was high, and it was probable that the chill factor operated only in minor degree. While the onset of the Winter epidemic was marked by a period of chilly weather, during the major part of its course the chill factor did not operate to any great degree, and the minimum temperature did not at any time reach freezing point. These two epidemics stood out from the other epidemics of the series, in all of which a steady increase in the chill factor apparently accounted for their onset and to some extent explained the course. The Summer epidemic occurred in minor form, while the Winter epidemic reached proportions equalling those of the major epidemics of the

change of season. While remembering the difference in average temperature of the two seasons, it is not unlikely that seasonal variations in susceptibility may influence the course of this epidemic form of pneumonia.

V. CONCLUSIONS.

(1) It has been shown that pneumonia may take epidemic form in a white population residing in a warm, dry climate.

(2) This form of pneumonia has been shown to recur regularly among the children of a large city.

(3) The weather factor of chief importance in the promotion of the majority of these epidemics, was a period of maintained increase in the diurnal range of temperature, when the minimum temperature was relatively low. This factor would appear to be of outstanding importance in the etiology of pneumonia in climates which are hot and dry, or are subtropical. In these climates, it is an index of the chilling due to the fall in temperature occurring at night during certain seasons of the year, or during periods of abnormal weather.

(4) Periods of abnormally high wind, occurring during the Winter when the average temperature is low, may occasionally cause increased incidence of pneumonia in Adelaide, and may even assist in the promotion of the epidemic form of the disease.

(5) In this climate, the chill element of the weather is thus chiefly defined by the cold weather occurrence of

periods of increased diurnal range of temperature, and, occasionally, of abnormally high wind. It is significant that chill periods occurred at the time of onset of all the major epidemics.

(6) With one exception, such periods persisted while the major epidemics expanded. Ordinarily, as in the case of the Spring epidemics, the diurnal range of temperature became progressively greater while the incidence of the pneumonia was increasing. The chilly weather which was present at the beginning of the Winter epidemic, however, practically disappeared soon after the appearance of the epidemic. The epidemic subsequently expanded normally in the absence of weather during which the chill factor was marked. As in the case of the minor epidemic of the Summer, there was no meteorological cause of such a nature as to promote the uniform development of an epidemic.

(7) A short period of about three months probably followed each epidemic, during which time cold, changeable weather had no power to promote an epidemic.

(8) The appearances are those of carrier disease.

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