FURTHER OBSERVATIONS ON THE BEARING OF ATMOSPHERIC HUMIDITY ON OUTBREAKS OF CEREBROSPINAL MENINGITIS (ALEXANDRIA, 1930-1932).

By ARTHUR COMPTON, M.D., D.Sc.

Director, Municipal Public Health Laboratories, Alexandria.
Late Captain (T.C.) Royal Army Medical Corps.

In the August number of the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS, 1932 [1], I gave an account of the steps that led me to enunciate the humidity theory of cerebrospinal meningitis, which connects outbreaks of the disease with high atmospheric humidity, particularly indoors, when carrier-rates are high.

The present communication relates to Alexandria, where during the past two years the disease has shown a marked recrudescence, as elsewhere in Egypt. Authoritative figures for the decade 1920-1929 give 327 cases with 209 deaths for the whole of Egypt, or an average of 33 cases a year [2]. The year 1930 is marked by an increase to 98 cases with 58 deaths, and the year 1931 by a startling increase to 871 cases with 510 deaths. The corresponding figures for Alexandria are: 1930-1931 (July-June), 77 cases with 45 deaths; 1931-1932, 55 cases with 32 deaths.

Since the climate of Alexandria is characterized by high humidity, which is greatest in summer (June-August) and least in winter (November-February), and as this is the reverse of what happens in Cairo and the interior of Egypt [3], it may seem surprising that Alexandria suffered no more than the rest of Egypt during the outbreaks of 1931 and 1932. Cerebrospinal fever, as met with in Egypt, differs in no essential way from its appearance elsewhere. The seasonal incidence is winter and spring, the same as in the more temperate climate of Europe; and sporadic cases likewise keep turning up throughout the year, indicating that the disease never totally disappears between one epidemic period and the next.

This Alexandria study reviews, in terms of the humidity theory, data relating to the outbreaks of 1930-1931 and 1931-1932, and concerns mainly case-distribution and carrier-rates.

Kenawy [4] has made an interesting study of the distribution of cases over the various districts of the city for the epidemic period—November, 1931, to April, 1932. He has analysed 105 cases notified to the Sanitary Service, and finds that of the nine districts into which the city of Alexandria is divided, it is the Moharrem Bey district, sheltering a prosperous, comparatively easy type of inhabitant, which heads the case-list over the period, with 23 cases per 100,000 of its population. Gomrock, a working-class district, comes second on the list with 22 cases per 100,000, and the fashionable suburb of Ramleh third, with 19 cases per 100,000;
192 Cerebrospinal Meningitis and Atmospheric Humidity

while Minet-el-Bassal and Karmous, inhabited by the very poor, figure actually at the bottom of the list, each with only 8 cases per 100,000. Can this peculiar case-distribution be accounted for on our humidity hypothesis? Unless it can, the hypothesis would stand greatly weakened.

The better-class district of Moharrem Bey is notably well wooded, and trees are known to constitute a cause of atmospheric humidity; besides it is in parts rather low-lying, and is widely traversed by the Mahmoudieh and Farkha Canals (fig. 2). It also houses the Alexandria Water Company's plant with its large "settling basins" and constant evaporation therefrom. It is further rich in many large gardens, which necessitate frequent watering. All these features tend to maintain a high outdoor humidity, with its repercussion on indoor humidity.

![Fig. 1.](image-url) (1) Left: Giving an arrangement of the nine districts of Alexandria in terms of density of population per Km². (2) Right: Giving the case-distribution of cerebrospinal meningitis in the same nine districts over the two-year period 1930-1932.

Again, the Gomrock district, second on the case-list, is a more or less water-logged area, with damp subsoil, such that, as I am informed by our technical service, subsoil water can be tapped in parts at 60 cm. below the surface; besides it is bounded on three aspects by the sea. Further its density of population is the greatest of all the districts: 61.8 inhabitants per 1,000 square metres (fig. 1 (1)). It is not surprising then, that excessive outdoor and indoor humidities must characterize this area.

But what about the Karmous area, whose comparative indemnity from attack, on Kenawy's analysis, is so striking; and this in spite of its huge population (fig. 2), and the fact that it represents the poorest district of the city, where overcrowding must at times be very rife? It owes its comparative immunity, I think, largely to its geographical situation, being situated for the most part well above sea-level, with a dry subsoil. This
means drier outdoor air, and consequently better circulation between outdoor dry air and such indoor moist air, as results from overcrowding, i.e., freer ventilation in this district.

But six months represents a very short period, and the resulting conclusions may not be wholly exact. Accordingly I have re-investigated the question, collecting from the archives data relating to a two-years' period: July 1, 1930 to June 30, 1932. Some 232 cases are represented, and their distribution over the various districts in terms of 100,000 of the population has been worked out (figs. 1 and 2). The total case-incidence over the two years is seen (fig. 1 (2)) to follow very closely that worked out by Dr. Kenawy for the shorter period. Moharrem Bey again heads the list,

but Gomrock and Attarine now tie for second place, while Labbane ties with Ramleh for third place. Then come in descending order Karmous, Manchia and Minet-el-Bassal. Further, fig. 1 shows that there is no obvious connexion between density of population and case-incidence.

Attarine now steps into prominence. Why? No doubt because it adjoins the comparatively heavily infected Moharrem Bey area, with some overflow of contagion, which is better brought out by these studies over the longer period. That the district may have harboured a large proportion of "carriers" from the previous epidemic period seems to be indicated by fig. 1 (2). This figure indicates the cases of the respective districts distributed in terms of the four half-yearly periods during which they
occurred. The months January to June are considered epidemic months and July to December non-epidemic (or endemic) months. Attarine shows a fairly pronounced endemic period separating its epidemic periods.

It was primarily with the object of attempting to ascertain whether there was any district-immunity carried over from one epidemic season to the next that the distribution recorded in fig. 1 was worked out. The figure establishes no such immunity; in fact the contrary is the rule; the districts most attacked in the epidemic months of 1931 were most attacked again in 1932. This is the outstanding lesson of the chart, and it is not encouraging for 1933, unless the non-epidemic months of 1932 should show a great falling off in sporadic cases. So far as the figure goes it seems to show no justification for the assumption that bacteriophage-immunity, in the sense conceived of by d’Herelle for the “cholera carrier,” plays any part in the story of “carriers” of meningococci. In this connexion I may add that I have worked hard to find a phage for the meningococcus, but hitherto without success.

The case of Manchia is of interest. Next to Minet-el-Bassal it shows the lowest total case-incidence. It escaped the epidemic and endemic periods of 1931 entirely, but it figures fairly prominently in the epidemic of 1932. Its geographical situation is bad, being low-lying and sandwiched between Gomrock and Attarine, both of which, over the two years’ period, were attacked twice as severely. Its density of population is practically the same as that of Karmous, without possessing the favourable geographical situation of the latter. To what, then, does it owe its comparative immunity? To some extent possibly to the fact that Manchia represents on the whole a wealthier type of inhabitant than Gomrock, also with less overcrowding in the proportion of 46 : 62, or approximately 3 : 4 (fig. 1), with possibly a correspondingly lower carrier-rate. Its escape, however, seems to me to be accidental. It will be of some interest to see what happens in this district during the next epidemic season.

These observations, then, of Kenawy, and their extension, are of considerable interest. They afford on the whole a handsome confirmation of the humidity view, and one that we had every right to expect ex hypothesi.

Some general remarks on the climate of Alexandria in relation to outbreaks of the disease and our hypothesis seem called for. The city is characterized by a high humidity all the year round, but more so in the hot months of summer, which follows from its geographical situation, being surrounded by water on three aspects: on the north and west by the Mediterranean, and on the south by Lake Mariout (fig. 2). Yet the seasonal incidence of the disease is winter and spring, just as in Europe. The cause of the seasonal incidence cannot therefore be humidity, since the disease is for the most part represented by only sporadic cases during summer months. I think the seasonal variation is explained by the fact that many of the very poor sleep on the roofs of their dwellings during
summer, thereby bringing into play the protective factors of isolation and good ventilation, and are only driven to sleep indoors with the approach of winter when nights are cold and the rains arrive, conditions corresponding to the situation in Europe in winter and spring. Then overcrowding with high indoor humidity must exist. It follows that it is this driving indoors in winter of the carrier into overcrowded, badly ventilated dwellings which brings about, by contact, propagation of the carrier-state, and, by excessive indoor humidity, explosions of the disease.

Another protective factor in the situation as concerns Alexandria is that during the non-epidemic months of summer, and even during epidemic months, relatively few carriers are about. Throat-swabs from 857 contacts were sent to the Municipal Laboratories for examination during the epidemic period of 1931, and only six carriers were found, representing a carrier-rate of 0.7 per cent. During the non-epidemic months of 1931, 250 non-contacts, inhabitants of the Hadra Prison, were examined, and only one carrier was found (0.4 per cent). These carrier-rates may not be wholly exact, because practical difficulties intervened to prevent the determinations being ideal, notably as concerns the swabbing not being done personally, and the specimens being collected not on West's swabs but on ordinary slightly curved diphtheria swabs. While admittedly not perfect they nevertheless give, I think, a good approximation to the reality, especially when considered alongside an ideal determination carried out towards the close of the epidemic season of 1918 [6]. On that occasion 310 military non-contacts (from Mex and Mustapha Camps, and Nos. 19 and 21 General Hospitals) were swabbed and examined by Staff-Serjeant J. W. J. Leighton, B.Sc.(Lond.), R.A.M.C. (T.), and myself, working with West's nasopharyngeal swabs, pea-flour serum-agar medium from the then Central C. S. F. Laboratory, London, and a minimum of personal errors arising from the fact that both of us had had a long experience of such work in England. Only three carriers were found, representing a carrier-rate of one per cent, compared with a much higher rate among troops at the time in England.

Carrier-rates, then, on the whole would appear to be comparatively low in Alexandria, which is no doubt due to the sunshine and the out-of-door life of the place. It is well known that out-of-door life in the sunshine quickly clears up the throats of carriers of the meningococcus. I have frequently noted this fact in England [7]. Hence the probable explanation of why a city with such a humid climate as that of Alexandria escapes more serious infection.

To sum up, the foregoing facts concerning the disease at Alexandria harmonize perfectly with the view that outbreaks of cerebrospinal meningitis and their intensity are simply a function of carrier-rates and of atmospheric humidity, the latter acting most probably through an oedematous, spongy, diminished resistance state of the nasopharyngeal mucosa permitting penetration by the microbe [8]. Since the occurrence
Cerebrospinal Meningitis and Atmospheric Humidity

of cases varies definitely with high carrier-rates and with raised atmospheric humidity, it is evident that when no carriers are about it matters not what the humidity conditions are, there can be no cases; and conversely, when there is no elevation of humidity it matters not how many carriers may be about there should be no cases. Thus we have "that intelligible whole which alone we call a picture or a view." And this, in a nut-shell, is the theory regarding the etiology of cerebrospinal meningitis which I had the honour first to sponsor some seventeen years ago, and which I think still explains most of the facts of the disease.

Prophylaxis, then, should obviously aim at keeping indoor humidity as low as possible, by avoiding low-lying regions, particularly in the neighbourhood of trees; as the latter tend towards excessive outdoor humidity with repercussion on indoor humidity, when selecting sites for military camps, schools, and the housing of large numbers of individuals. Also, efficient drainage should be provided to eliminate subsoil moisture. In times of an epidemic, overcrowding, the drying of wet clothes in living quarters, and any other cause of excessive indoor humidity, should be scrupulously avoided. At all times good ventilation of sleeping apartments should be insisted upon, as this is the simplest means of combating excessive indoor humidity, which in conditions of overcrowding facilitates by contact the production of the carrier-state and by increased humidity the production therefrom of cases.

REFERENCES.