THE EPIDEMIOLOGY OF MENINGOCOCCAL MENINGITIS, OR CEREBRO-SPINAL FEVER.

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THE prevalence of cerebro-spinal fever is essentially seasonal, as is well illustrated by the fact that in Ipswich, during the three years 1915-1917, 26 cases occurred in the March quarter, 33 in the June quarter, 2 in the September, and 2 in the December quarter. The maximum prevalence of the disease was in the month of March, closely followed by April. The next in order were the months of May, February, and June, but the degree of prevalence in these three months may be judged from the fact that the total number of cases for the three months was equal only to that of the month of March. After June the cases have been sporadic; the only month in which no cases have occurred has been July.

The position, therefore, is that the disease has never been entirely absent from Ipswich during the last three years, but has been most prevalent in February, March, April, May, and June, particularly in March and April. This seasonal prevalence has been characteristic of each of the three years.

Consideration of these facts would appear to establish a *prima facie* case for the association of the disease with some condition, or conditions, common to the whole year, but most in evidence in the winter and spring.

Without entering into details, we may assume that most would agree that an organism found in one part of the body, morphologically, culturally, and serologically identical with an organism inhabiting another part of the human host was one and the same organism. We are therefore justified in the assumption that a nasopharyngeal coccus and the meningococcus, which are morphologically, culturally, and serologically identical, are one and the same organism. The fact that the meningococcus in the cerebro-spinal system produces the phenomena of meningococcal meningitis, and that in the nasopharynx it produces no obvious symptoms, is quite consistent with this conclusion.

A large number of demonstrations go to prove that the nasopharynx of an individual suffering from meningococcal meningitis contains the meningococcus in the early stages of the disease. Failures to demonstrate the meningococcus in the nasopharynx of cases of meningococcal meningitis may be due to many causes, such as the extreme delicacy of the organism, faulty swabbing, possibly the exhibition of an anaesthetic before the swab is taken, the taking of swabs too late in the disease, and so on.

The practical epidemiological point is that there is sufficient evidence to justify the opinion that in every case of meningococcal meningitis the nasopharyngeal precedes the meningeal infection. If this view be accepted, then the spread of the disease is due to the transference of the meningococcus from one nasopharynx to another.

We may now inquire what evidence exists indicating that the meningococcus is spread from person to person. In this relation I quote the figures of a recent report by the Local Government Board, which epitomizes the work done in this matter by the Board's bacteriologists. They found that amongst 1,881 persons from many different sources and places who had not been in contact with cases of cerebro-spinal fever, no less than 13.5 per cent. harboured in the nasopharynx an organism which was morphologically and culturally identical with the meningococcus, and in the opinion of the Board's bacteriologists was, in fact, the meningococcus. If this statement be correct, even if the proportion given is greatly overstated, it affords definite evidence of a widespread nasopharyngeal meningococcal infection. The number of persons infected in an infected area would, on this basis, in a town of the size of Ipswich, in any one year, be very large, each being capable of transmitting the infection to others.

A normal healthy nasopharynx will rid itself of the meningococcus without treatment, or in spite of it, on the average, in something under a fortnight. An unhealthy nasopharynx, with adenoids, etc., can retain the infection for a longer period. Some individuals retain the infection for prolonged periods and become true carriers. I have known one case out of a limited number which remained

infected for over six weeks. Such persons will serve to assist in maintaining the meningococcus in a locality and will help to bridge over the summer and autumn intervals. Naturally, those in this group will be relatively few, so that the numbers infected by them will be few in relation to the total population. Nevertheless, they will assist in maintaining the reservoir.

We are now in a position to go back to the seasonal nature of meningococcal meningitis. Nasopharyngeal infections of all sorts are spread from person to person in the acts of coughing and sneezing, the immediate atmosphere being impregnated with spray or droplets, containing members of the nasopharyngeal flora. Therefore, all nasopharyngeal infections will be most readily spread during the seasons of the year when coughs and colds and other nasopharyngeal and respiratory infections are most prevalent. The spread of infection will be assisted by the closer crowding of the population during these periods of the year and by the closing up of ventilation and the complete ignorance and indifference of the public as to the mode of spread of this group of catarrhal infections.

We should, therefore, expect that meningococcal meningitis, secondary to nasopharyngeal infection, would be most prevalent in February, March, April, and May, which is precisely what does occur, as evidenced by the sudden appearance of cases of the disease in these months.

In my opinion the rise in meningococcal meningitis coincides with an increase in the density of the nasopharyngeal infection, whilst the summer and autumn fall coincides with a corresponding drop. We thus get a vicious circle produced by (1) a rise in the usual winter coughs and colds, a proportion of the sufferers harbouring the meningococcus; (2) a number of people are infected by these, the number varying with the opportunities of infection; (3) these in turn cough on the infection, and thus with an increase of catarrhal infections a constantly increasing number of persons become infected with the nasopharyngeal infection; (4) when the finer weather comes on, coughs and colds diminish rapidly, and so does the meningococcal nasopharyngeal infection.

The question may now be asked, granting all this, Why are there not more cases of meningococcal meningitis? My answer to this is that the invasion of the meninges by the meningococcus is an accident dependent on conditions as yet undetermined. If this view is correct, it is merely a mathematical proposition that the greater the number of individuals involved, granting the possibility of the accident, the greater will be the number of accidents. I believe that the existing evidence tends to show that the height of the meningeal epidemic corresponds to the greatest density of the nasopharyngeal epidemic and that the sporadic cases occurring during the other parts of the year merely indicate the existence of a reservoir of nasopharyngeal meningococcal infection.

If these views are correct, then the present mode of dealing with a few positive contacts of a few cases of meningococcal meningitis is futile.

In support of this view of the epidemiology of the disease is the quite marked independence of individual cases. True, on occasion, more than one member of the same family may be attacked. Thus I have seen three cases in one family and two in another. Nevertheless, the general fact is that the cases are single and independent and occur amongst individuals who have no connexion of any kind with one another. This, in my opinion, is strong evidence of a widespread generalized infection.

A FATAL CASE OF PARATYPHOID B SIMULATING TYPHUS FEVER.

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THE following fatal case of paratyphoid fever due to the *Bacillus paratyphosus* B seems to be worth noting, not only from the extreme severity of the lesions found *post mortem*, but also from the clinical course of the disease.

On admission to this unit the patient presented a clinical appearance almost indistinguishable from an advanced stage of typhus fever. He was in a state of

low muttering delirium with carphology, coma-vigil, and retention of urine; there was a faint purple petechial and macular eruption on the chest and abdomen. Had we not had the advantage of skilled bacteriologists to examine the patient both before admission to this unit, and also while here, the condition would almost certainly have been diagnosed as an advanced stage of typhus fever. Owing to our situation at the front, it has been impossible for us to search the literature and ascertain whether many other such cases have been already recorded. As far as we have been able to learn the number of severely toxic cases of paratyphoid is very small.

The kidney lesions in this case were remarkable, and Hurst in his book, *Medical Diseases of the War*, seems to describe a somewhat analogous case suffering from paratyphoid A, in which death occurred from suppurative nephritis in the fifth week. Death occurred in this case in the third week from what might be called "a pyaemic condition," and in all the abscesses the *Bacillus paratyphosus* B was found.

Captain Lindsay of the 13th Mobile Laboratory discovered the bacillus before the case was admitted to this hospital, and we are grateful to Captain J. Cruickshank, Officer Commanding 18th Mobile Laboratory, for further bacteriological investigations and the help he gave at the necropsy. We are indebted also to Captain A. M. Davie, R.A.M.C.T., for notes on the patient at the onset of his disease in the line, and to Captain Gray, R.A.M.C., for some of the earlier clinical notes.

Condition of Patient in the Line.—The patient had had two doses of antityphoid vaccine in December, 1915, and two doses of the triple vaccine (T.A.B.) in June, 1917. He had served nine months in France, and had reported sick at odd times before with very minor ailments. On September 17th, 1917, when the battalion was in the line, he complained of pains in the stomach and diarrhoea of sudden onset. Next day he complained of pains in the head and legs, with shivering; temperature 101.2°; diarrhoea better. He was sent by the regimental M.O. to the field ambulance as P.U.O., and was detained there for the night. On September 19th he was evacuated to a clearing station, complaining of pains in the head and across the abdomen, with pain also in the muscles of the thigh and calf. On September 20th there was pain and tenderness in the epigastric region, with cramps in the hands and legs, vomiting, rigors, and cough. On September 24th abdominal pain was complained of, the lower abdomen being tender, but not rigid. The patient complained of retention of urine, but passed a small quantity after suprapubic fomentation. From September 25th to 29th the condition was much the same; the urine passed in twenty-four hours varied between 4 and 12 oz. On September 29th *B. paratyphosus* B was found in the blood on culture. The spleen was palpable and the urine smoky. He was transferred to the infectious casualty clearing station.

Condition on Admission.—The patient was semi-comatose, with dusky and livid face. There was retention of urine and the bowels were confined. Epistaxis occurred. After a while there was low muttering delirium and carphology. The abdomen was slightly distended and tender, the spleen readily palpable; there were no typical rose spots, but on the chest a fine petechial eruption with subcuticular mottling in the axillae. Next day the patient was much worse, wildly delirious, and trying to get out of bed. Signs of consolidation were found in the left lung and at the base of the right lung. A simple enema was given with satisfactory result. Ten ounces of bloody urine were drawn off by catheter. The patient died at 5.45 a.m. on September 31st.

Post-mortem Examination.

On the abdomen and front of chest there were a few pale petechial spots.

Left Lung: Adherent all over (old adhesions). Large haemorrhagic infarction in lower lobe; area of infarction with pneumonia in upper lobe. *Right Lung:* Free from adhesions; lower lobe completely consolidated; early grey hepatization, with red hepatization of contiguous portions of other lobes. There was inflammatory exudate between the lobes.

Ileum congested along whole length, with acute inflammation of mesentery in this area; glands enlarged, congested, and haemorrhagic; Peyer's patches along last three feet of ileum swollen and congested; no ulceration. Large intestine contained fluid faeces; diffuse inflammatory changes along caecum and ascending colon.

Surface of left kidney studded with yellowish-white nodules and in places definite abscesses with fluid pus; on section kidney riddled with abscesses. Similar septic condition of right kidney.

The liver showed cloudy swelling; gall bladder markedly distended. Spleen enlarged and congested; haemorrhagic spots on section. The bladder showed well-marked purulent cystitis. Brain and medulla: Some excess of fluid, but no meningitis.

B. paratyphosus B was grown from urine, kidney abscesses, bladder, and spleen.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

PREVENTION OF NERVE BULBS IN STUMPS.

PAINFUL bulbs on the ends of nerves following amputation are so frequent and troublesome as to cause a very protracted convalescence in a large number of cases. In an article in the *BRITISH MEDICAL JOURNAL* of August 25th, 1917, I described a method of dealing with nerves which experience has further shown to be valuable in preventing these neuromas.

Recently I had the advantage of seeing a median nerve which had been treated in this way four months previously together with an ulnar nerve which at the amputation had been severed in the ordinary way. The median end was the shape of a pointed pencil, and the ulnar nerve had a large and tender bulb.

It would appear that when a nerve is severed in amputation the axis cylinders grow out in search, as it were, of those of the other severed end. These nerve elements from the central end multiply, enlarge, and get involved in and surrounded by fibrous tissue, which on contracting squeezes and renders tender and painful the sensitive nervous tissue involved in the mass. A bulb may exist and not be painful or tender until this contraction takes place.

My experience tends to show that if the sheath of a nerve is cut and stripped back along its trunk with a piece of gauze between the finger and thumb for about half an inch in the form of a cuff, and then brought forward and tied after the denuded trunk has been severed, the sheath acts as a limiting resisting membrane, and this protrusion of axis cylinders is prevented.

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COINCIDENT INFECTIONS OF GONORRHOEA AND SYPHILIS.

FROM 5 to 10 per cent. of all cases of gonorrhoea subsequently develop syphilis. The infections are acquired simultaneously or within a short period. In an experience during the last twelve months of over 200 cases of double infections I have never seen complications or retardation of cure caused by either mercurial or arsenical preparations, as suggested by Captain Lumb (*BRITISH MEDICAL JOURNAL*, March 9th, p. 285). The cases were treated with injections of novarsenobillon, either intravenously or subcutaneously, and 1 grain of mercurial cream weekly. Smears for gonococci were taken on admission, and afterwards weekly smears, the first thing in the morning. If the smear was negative and the patient dry for seven days, a urine test was done after prostatic massage. If this test was negative the case was considered cured.

Patients in the syphilis wards gave negative smears sooner than those in the gonorrhoea wards, due, as I concluded, to their lying to rest more, owing to their injections and perhaps a greater incentive to get rid of a loathsome combination by inducing them to irrigate thoroughly. Venereal hospitals are a refuge for many slackers and shirkers. Among these 200 cases two of epididymitis occurred during treatment, and another about a month after the completion of antisyphilitic treatment following the passage of a bougie. No cases of prostatitis occurred. The treatment for gonorrhoea was irrigation of the bladder with potassium permanganate and prostatic massage occasionally. No drugs were given, and vaccines only in cases of rheumatism. The patients, except in rare instances, were cured of gonorrhoea before the course of antisyphilitic treatment was completed.

The urethral discharge is sometimes increased following an injection of neosalvarsan, especially in early secondary syphilis with wide generalization of the spirochaete—the class of case in which a rise of temperature and malaise follow a first injection. Any depressing cause will increase the urethral discharge temporarily. In early primary syphilis reactions seldom occur.

A small proportion of cases are either lamed by intramuscular injections of neosalvarsan, or have so much pain as to make the method unsuitable in private practice. The deep subcutaneous method causes less pain and