health (as was the case with the patients who came to our allergen-proof chambers) it must be realized that one obvious factor in the prevention of tuberculosis is to make the conditions at home similar to those in our clinic.

We feel that, for all patients who are free from symptoms of asthma in our clinic, or who, suffering from tuberculosis, are greatly relieved, the problem of their health has been reduced for the most part to a problem of technique.

REFERENCES.

¹ W. Storm van Leeuwen: *Allergic Diseases*. New York and London, Lippincott and Co. ² W. Storm van Leeuwen and W. Kremer: *Munch. med. Woch.*, 1927.

Remarks

ON

THE EPIDEMIOLOGY OF POLIOMYELITIS.*

BY

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POLIOMYELITIS is an acute specific fever which, in its complete expression, presents three distinct clinical and pathological phases. Clinically these are a short-lived initial phase of *constitutional disturbance*, followed by signs of invasion by the virus of the *subarachnoid space*, and finally by signs of involvement of the *nervous tissue* itself. Each phase has its pathological substratum. In the initial phase the virus obtains lodgement in the liver, spleen, and lymphatic structures generally, in all of which lesions are found. The virus then reaches the subarachnoid space and leptomeninges by channels which are not yet fully known, and gives rise to a reaction which is manifested by characteristic changes in the cerebrospinal fluid, and finally, invading the nervous tissue, it produces there the inflammatory lesions to which the paralysis is due.⁸ The intensity of each phase varies, as do also the time relations of the first two phases. Further, the infection may die out after the first or the second phase, thus giving rise to the so-called "abortive cases." When infection dies out after a severe meningeal reaction we speak of a "meningitic" type of the disease.

The initial constitutional phase of poliomyelitis seldom presents sufficiently characteristic symptoms for identification, and it may be so mild that the patient continues to go about and thus has ample opportunities of spreading infection, since the virus is contained in the nasopharyngeal secretions. Like other infectious diseases it has its own seasonal and age incidence, incubation period, mode of spread, and susceptibility factor. Since the publication of Wickman's observations⁹ it has been generally held that the infection is carried and spread by healthy carriers of the virus and by sufferers from the disease. From the nasopharynx of both groups of individuals a virus has been obtained capable of producing the disease in inoculated monkeys, and Flexner and Amoss⁵ concluded that the nasopharyngeal mucosa during the ten days immediately following the initial signs of infection regularly contains the virus.

The two points to which I propose particularly to address myself are the question of non-human carriage of the virus, and a view which has recently been advanced to the effect that the sole agent in the spread of poliomyelitis is the healthy human carrier of the virus. Despite the inconclusive results of extensive search for non-human agencies of spread, these have not been finally excluded. Recently, in a small American outbreak, a strong case has been made out for milk as a possible agent of spread, and the whole question has thus been reopened.

An important modification of Wickman's views as to the joint responsibility of healthy carriers and sufferers from poliomyelitis in the spread of the disease has recently been suggested by Collier,⁴ who believes that the disease

is spread exclusively by the agency of healthy carriers, and that actual sufferers from the disease in any of its phases are non-infective to contacts, owing to loss of virulence in the infecting agent immediately the sufferer becomes infected. Elaborating this hypothesis, he states that every epidemic of poliomyelitis is preceded by a "carrier epidemic," in the course of which susceptible contacts are infected and an outbreak of the disease occurs. In any given area the presence of this army of carriers leads to the practically simultaneous mass infection of all available susceptible subjects. Further, he states that epidemics of poliomyelitis are characterized by a "rapid blaze, high peak, and rapid fall," a feature due to the mode of spread he describes. In such semi-isolated communities as country villages or boarding schools this manner of spread involves the universal and simultaneous striking down of all susceptible members of the community, and precludes the further extension of the disease within the community, with the result that all those not initially infected may safely remain within the infected community. The practical bearing of this conception is that all such communities should be kept together on the appearance of poliomyelitis within them and segregated from the surrounding society.

Since this hypothesis raises vitally important questions of public health and departs from the generally accepted body of teaching, it is essential that the data on which it is based should be carefully studied. Briefly, as formulated by Collier, these are: (1) the literature of the disease records no authentic example of case-to-case infection. Multiple cases within households are rare, and spread from a patient in a hospital ward is unknown. (2) The "rapid blaze, high peak, and rapid fall" of epidemics of poliomyelitis can be explained only on the basis of a preceding "carrier epidemic," and does not accord with case-to-case spread. (3) Finally, there is the categorical statement, for which no evidence is submitted, that at the moment of infection the virus ceases to be infective, thus rendering case-to-case spread impossible.

Before proceeding to discuss the first two postulates, let us summarily dismiss the third. It has been established by Flexner and Amoss that the nasopharyngeal secretions within the first ten days of onset of the disease contain a virus not less infective for the monkey than is that of a healthy carrier. For this susceptible animal there is, then, no loss of virulence. In the circumstances it is incredible that there should be a selective loss of virulence for the other susceptible animal, man. Throughout the whole range of infective agents pathogenic to man there is no parallel for the state of affairs thus alleged for the virus of poliomyelitis, and in the absence of evidence we may dismiss it as a speculation incompatible with the facts.

It is perhaps more difficult to establish unequivocally the occurrence of case-to-case infection; despite the strongest indications, it is always easy to maintain that somewhere in the background is the healthy carrier who is in fact responsible for spread; and while it may be impossible to demonstrate his existence at the relevant time and place, it may be equally impossible to exclude it. Nevertheless, we may reasonably object to the invocation of this factor in respect of every epidemic unless there is more substantial evidence of its operation than our inability to prove a negative.

In referring to the literature for examples of case-to-case infection it is essential to go direct to original sources in which all the facts of observation are recorded irrespective of their possible bearing upon the question at issue. For this purpose we cannot do better than turn to Wickman's original monograph on the Swedish epidemic of 1905. Wickman concluded, upon evidence which left him no alternative, that poliomyelitis tends to spread along the lines of human traffic, borne thereon by healthy carriers of the virus and by sufferers from the disease, more particularly during its initial phase and in its abortive form. Impressed as he was by the importance of the healthy carrier, he drew attention to the equally important role of the infected patient. Amongst the numerous foci of the disease analysed by him those in the parish of Trästena and on the islands of Sirkö and Borgö provide clear examples of a state of affairs incapable of explanation on

* These remarks were made in opening a discussion on the notifiable infectious diseases of the nervous system at the Annual Congress of the Royal Sanitary Institute, July, 1927.

the basis of a preceding "carrier epidemic," and establishing beyond reasonable doubt the preponderant role of case-to-case infection. The accompanying figure is a diagrammatic representation of the Trästäna outbreak, modified from Wickman's diagram.

The rectangles represent houses, and the figure included within each is that of the number of cases of poliomyelitis in the house. Beside each house is given the dates of development of each case. Except in respect of the first and last cases the month is July. S = the school and attached schoolhouse. The arrows represent direction of spread, and those with interrupted lines indicate households infected by a healthy carrier. In the other households the initial case was a child actually attending school. The asterisk indicates the date (July 15th) on which the school was finally closed. (Modified from Wickman's Figure 2.)

In 1905 Trästäna was a remote woodland village of some 500 inhabitants living in 119 houses; 49 cases of poliomyelitis occurred in nineteen houses, the first case developing on June 28th, and the last thirty-seven days later, on August 4th. The maximum number of cases developing on any single day was 4. Multiple cases in households were the rule, occurring in no fewer than thirteen of the nineteen infected houses. The maximum number of cases in a single house was 5. In the first eight households infected the initial case was a child attending school. The school was infected by the first case in the village, and the schoolhouse was the second household involved, four of the schoolmaster's children developing the disease. From this point household after household was infected, in six instances a school child who remained healthy carrying infection home from school. In nearly every instance an incubation period of three days or over intervened between the appearance of successive cases within a given household. The school was closed on the 15th of the month, and after that the epidemic rapidly declined.

Here we have a characteristic example of gradual spread of the infection within a semi-isolated community, the school being the centre of infection. The day-to-day case incidence both in the village as a whole and within individual households indicates a case-to-case spread, and is inexplicable on the basis of a preceding "carrier epidemic," and simultaneous infection of all available susceptible subjects.

In another outbreak cited by Collier himself we find evidence of the same order as that provided by the Swedish epidemic. I refer to the outbreak of poliomyelitis in the Devon village of Stokes Rivers in 1911.⁷ At the relevant time this village contained 119 inhabitants living in eighteen houses. There were reported 36 cases of the disease, occurring in fourteen houses. Four houses, containing 8 children, escaped entirely. The main facts of the outbreak are as follows: The thirty-sixth and last case developed as late as forty days after the first; not more than 2 cases developed on any one day; multiple cases occurred in

eleven of the fourteen infected households. In one house 9 cases occurred during a period of twenty-two days, with a definite incubation period between the onset of each. Collier's summary of this outbreak—namely, that it was "a swift scourge, which, lighting upon the little village of Stokes Rivers, could lay low in a few days 45 persons out of a total population of 119," is so out of harmony with the facts as to lend no support to his theory.

Finally, Burrows and Park⁸ have analysed a very interesting small outbreak in a block of flats in an American city which provides further convincing evidence of the important role of case-to-case spread in epidemic poliomyelitis. In this instance 10 children out of a total of 18 were successively infected after exposure to preceding victims in the series.

These outbreaks are characterized by a complete lack of the features held to establish the exclusive role of the healthy carrier and the occurrence of a preceding "carrier epidemic" in the epidemic incidence of poliomyelitis. They show neither a "rapid blaze, high peak, and rapid fall" nor any subsequent immunity of susceptible subjects not infected at the outset. Multiple cases appearing successively within a single household predominated over single cases, while susceptible subjects proved liable to infection, with its attendant risks to life and limb, throughout the whole period of their stay within the infected community and until the epidemic had finally smouldered out.

If we consider the special case of a semi-isolated community of young children, containing in all probability a high proportion of susceptible subjects, there seems no doubt that their retention within an infected community is fraught with considerable risk of infection throughout the entire period of their inclusion.

The theory of the "carrier epidemic" and the exclusive infectivity of the healthy carrier, therefore, falls to the ground, and the practical bearing thereof is that the keeping together of a community in which poliomyelitis has appeared is dangerous to every member of the community in question, and cannot be regarded as a scientific measure of preventive medicine.

We must continue to hold, therefore, that Wickman's theory of case-to-case and carrier infection in poliomyelitis is correct, and must be the basis of procedure when epidemics occur. That there are no published records of the spread of the disease within hospital wards, either by carriers or by sufferers, cannot be interpreted as excluding case-to-case infection without also ruling out carrier infection. In any case the argument lacks force, since there are obvious factors contributing to this lack of hospital examples. For over twenty years poliomyelitis has been generally recognized as a contagious disease, and children admitted to hospital suffering from it in its acute phases are treated with the precautions usual in cases of infective disease, and their opportunities of spreading infection are restricted in consequence.

The question has also been approached from another point of view by Aycock and Eaton,² who, making a comparative study of the incubation periods of measles, scarlet fever, and poliomyelitis, find that in each disease incubation periods fall into two distinct groups, a short and a long. The former indicates simultaneous infection from a common source; the latter indicates true secondary case-to-case infection. On this basis, although true secondary cases of poliomyelitis are less common than those of the other two diseases, their occurrence seems undoubted.

There remains the question of non-human carriage and spread of the disease. From his analysis of a large series of cases Aycock¹ has come to the conclusion that transmission by human contact appears inadequate to account for some of the features of the epidemic occurrence of poliomyelitis, and that spread may occur in the apparent absence of this factor. He suggests the existence of non-human modes of spread, and indicates milk as one such possible agency for the carriage of the virus. He has subsequently reported, with Knapp and Godfrey,⁶ a small outbreak in which there was evidence that milk may act as a vehicle of infection. Of this outbreak of ten cases, these authors remark:

"As to the Cortland outbreak, we would point out that the only cases to occur in a city of 15,000 people subsequent to the

appearance of three cases more than two months previously were, with one possible exception, in persons who drank milk from one dealer who sold less than 3 per cent. of the city's total supply; that the cases were distributed widely enough to make it improbable for the milk supply to be a mere coincidence of geographic distribution. When to these facts we add that an actual paralytic case of the disease existed on the dairy farm from which most of the milk came; that this patient was milking the cows and handling the milk for a period of four days while in the acute stage of the disease, and as the time which elapsed between this period and the dates of onset of the subsequent cases were consistent with the generally accepted minimum and maximum incubation periods of poliomyelitis, the chain of circumstances is as complete as is found in the majority of milk-borne outbreaks of other diseases. While this outbreak points to transmission by milk, we are of opinion that this is not the usual mode of spread of poliomyelitis."

In such outbreaks as are characterized by indications of common source or mass infection, therefore, such a non-human mode of spread must be considered. The sudden outbreak of poliomyelitis in a Kent sea-coast town last year comes to mind in this connexion, and suggests the possibility of such a mode of spread. It would indeed be unfortunate if an uncritical acceptance of the hypothesis of a preceding "carrier epidemic" were to lead to any omission to investigate searchingly every possible means of transmission, non-human as well as human, of this dread disease. One of the most striking dangers of speculations of the kind is that they are apt to make us think that we possess an adequate explanation of phenomena which are, in fact, extremely obscure in their causation, and thus to lead to a slackening of that rigorous and exhaustive search by which alone knowledge is to be gained.

REFERENCES.

- ¹ Aycock: *Journ. Amer. Assoc.*, 1926, 87, 75.
- ² Aycock and Eaton: *Amer. Journ. of Hygiene*, 1925, 5, 724 and 733.
- ³ Burrows and Park: *Arch. Intern. Med.*, 1917, 20, 56.
- ⁴ Collier: *BRITISH MEDICAL JOURNAL*, 1927, i, 751.
- ⁵ Flexner and Amoss: *Journ. Exper. Med.*, 1919, 29, 379.
- ⁶ Knapp, Godfrey, and Aycock: *Journ. Amer. Med. Assoc.*, 1926, 87, 635.
- ⁷ Reece: *Proc. Roy. Soc. Med.*, Epidem. Sect., 1912, 5, 59.
- ⁸ Walshe: *Lancet*, 1927, i, 326.
- ⁹ Wickman: *Beiträge zur Kenntnis der Heine-Medinscher Krankheit*. S. Karger, Berlin, 1907.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

TWO CASES OF INJURY TO THE CERVICAL VERTEBRAE.

THE two following cases of injury to the cervical vertebrae—one of them ending in death—seem worthy of record. There seems little doubt that in the first the nerve symptoms, though absolutely symmetrical in the upper limbs, did not result from cord injuries as the result of fracture of the sixth cervical vertebra, but from bilateral identical injury to the brachial plexus. All cords were involved, but mainly the inner, the injury to none being sufficient to abolish sensibility. The nature of the injury, the method of its production, and the complication of a fracture of the cervical vertebra make the case interesting and unusual.

CASE I.

A man, aged 58, whilst fishing fell from a bridge, a distance of about 12 feet, landing upon his head. He did not lose consciousness.

On admission to Bristol General Hospital on March 21st, 1927, he had a scalp wound extending for about 7 inches over the frontoparietal region, with no skull injury. Neck movements he performed with care, but complained of no pain in that region. Both wrists were dropped, with complete loss of power. The thumbs were slightly abducted, and the fingers held flexed at the interphalangeal joints and extended at the metacarpophalangeal joints. His grip was extremely feeble, equally so on both sides. Shoulder and elbow movements were weak and sluggish. Anaesthesia was absent in both hands and arms, the patient being able to appreciate a touch with wool, but he complained of a tingling sensation passing from his wrists to his fingers.

Abdominal reflexes were present; the knee-jerks were normal and equal on both sides; the plantar reflexes were flexor. There was no loss of function or power in the lower limbs and there were no sensory changes.

The day following admission to hospital his temperature rose to 102° F., and his pulse from 64 to 98. He became dull, and towards evening he was slightly delirious. The paresis remained unchanged. He had not passed urine. The third day his mental apathy continued, and there was oedema of forehead and eyelids, extending down both sides of the neck and affecting most of the posterior triangles. He lay with his arms across his abdomen. He passed urine normally, and a normal motion of his bowel had taken place. X-ray examination showed an oblique fracture of

the body of the sixth cervical vertebra with a slight degree of compression.

From this day he commenced to improve. The weakness and stiffness in his arms gradually disappeared; he became able to move the wrists, but his grip was still feeble. There were no abnormal urinary or bowel symptoms, nor at any time evidence of any affection of the lower limbs. After six weeks in hospital he was discharged as a walking patient, wearing a neck plaster; the only remaining symptom was a rather weak hand-grip.

CASE II.

A youth, aged 21, fell from a roof on to the back of his neck; he was very dazed but not unconscious. On examination in the out-patient department he was found to have a small cut on the back of the head. He was quite conscious, and complained of pain in the neck and down the right arm. Movements were free, and there were no sensory changes. He refused to remain in hospital.

The following day he walked to the out-patient department, but, his symptoms having become worse, he was admitted to hospital. The pain in his neck and arm was still present, but he now held his head stiffly, and turned towards the left. Paresis of the right arm was marked, but no sensory changes could be elicited. That same evening he suddenly collapsed and died. *Post-mortem* examination revealed a fracture of the posterior arch of the atlas, with some oedema and minute haemorrhages in the cord.

I am indebted to Professor E. W. Hey Groves and to Mr. Duncan Wood for permission to publish these cases.

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CARCINOMA A COMPLICATION OF PILES.

It is not unusual to see piles the result of carcinoma, but the following case illustrates the reverse condition:

A lady, aged 62, had been operated on for piles fifteen years previously, but the symptoms recurred about nine months ago. The clinical features were characteristic of external and internal piles, which became inflamed and prolapsed at intervals. A slight amount of blood and a little mucoid discharge were noted. In addition to the prolapsed internal haemorrhoids several external piles were present, but one was larger and firmer than the others. It seemed to be an organized thrombosed pile with considerable inflammatory thickening and slight ulceration of the surface. The inguinal glands were not enlarged.

At operation the possibility of this pile being malignant was considered and its excision with a fairly wide margin of skin and mucosa was carried out in conjunction with the operation for the prolapse. The base was not adherent to the external sphincter.

On section the usual structure of an organized thrombosed pile was seen, but in addition the surface skin showed definite malignant changes—squamous-celled carcinoma with cell nests, downgrowths of epithelium, and round-celled infiltration. Subsequently bilateral inguinal adenectomy was performed, but the glands showed no sign of invasion.

I examined the patient six months after the first operation and found no sign of recurrence.

This case illustrates the importance of submitting to microscopical examination all tissues removed at operation. Without it this condition could not have been diagnosed. More recently I met with a case in which there was advanced leucoplakia of the skin overlying an external pile—presumably a pre-cancerous condition.

I am indebted to Mr. K. W. Monsarrat, surgeon to the Northern Hospital, Liverpool, for permission to publish these notes.

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LONG UMBILICAL CORD COILED ROUND NECK.

THE following case of an unusually long cord coiled six times round the neck appears worthy of record:

A multipara, aged 29, was delivered of a full-term female child, weighing 5½ lb., in University College Maternity Hospital on July 7th, 1927. Vaginal examination, ten minutes previous to the delivery, showed the head in a deflexed attitude, the anterior fontanelle being well down. The position was right occipito-anterior. After the head was born one loop of cord was pulled over it, before the rest of the child was rather precipitously delivered. There were then five more coils round the neck. The length of the cord was 58 inches (147 cm.). The insertion of the cord was central, and the placenta and membranes were complete. No signs of foetal distress were apparent before delivery, and the child commenced breathing quite normally when born.

The average length of cord is given as about 50 cm., and the limits are from a few millimetres to 198 cm. The source of this much quoted figure is Hyrtl's book *Die Blutegefäße der menschlichen Nachgeburt* (1870). De Lee (1913) states that cords over 100 cm. in length are very