POLIOMYELITIS IN MALTA, 1950 - 1951

An epidemiological and clinical study of the disease as it affected the Armed Services

by

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PREFACE

This work was done while I was Physician at the David Bruce Military Hospital, Malta G.C. during 1950 and 1951. I then had the opportunity of studying an outbreak of poliomyelitis among servicemen, their families, and some nurses. Some of the features of the outbreak were of interest from the epidemiological, and to a lesser extent, from the clinical point of view. I have made observations on the following:

(a) The incubation period of poliomyelitis.

(b) The mode of spread of poliomyelitis virus, and the relationship of concurrent minor illnesses to the outbreak.

(c) The relationship of activity in the early stages of poliomyelitis to paralysis.

(d) Clinical features of the outbreak.

(e) Pregnancy and poliomyelitis.

(f) Treatment.

I have summarised the work under these headings, and drawn conclusions, at the end of each section; there is, therefore, no general summary and list of conclusions at the end of the thesis.

I was helped in the clinical work by A.J. Craig, Esq., M.D., F.R.C.S., Professor of Surgery at the Royal University of Malta and by Captain J.P. Caley, R.A.M.C. Colonel H.C. Benson, who was then commanding the hospital, gave me much encouragement in pursuing the work.

I. INTRODUCTION

(a) Historical.

There was a small epidemic of poliomyelitis in Malta in 1902 but it was not until the winter of 1942 - 43 that the first large epidemic occurred. Indeed, the total recorded incidence during the period 1921 to 1941 was only 61 cases, and the largest number of cases in any one year was 9, in 1921. It was the opinion of Seddon et al. (1945) that more than 61 cases occurred during these 20 years because one of the authors noted the after effects of poliomyelitis while examining recruits for the Armed Services during the 1939 - 45 War. This opinion has also been expressed to me by Professor A.J. Craig (Professor of Surgery in the Royal University of Malta) in a personal communication.

Between November, 1942, and March, 1943, 483 cases were notified of which 57 were in servicemen. (Seddon et al. 1945). A second outbreak occurred during the winter of 1945 - 46 when there were 79 civilian cases and 6 service cases. In the latter half of 1947 there were 57 civilian and 18 service cases.

(b) The outbreak.

In the epidemic of 1950 - 51 the first case was a Maltese child who became ill on 27th May, 1950. Cases continued to occur during the hot summer months, through the cool winter ones, until February, 1951, when 230 cases had been notified. My

observations /

observations are drawn from a study of 51 cases among servicemen, their families and some nursing sisters; for convenience all my cases are classified as service cases since they were all admitted to the David Bruce Military Hospital. Their distribution is shown in table I.

(c) Age distribution.

The age group distribution of the civilian cases showed the infantile pattern of the 1942 - 43 and the 1945 - 46 epidemics, thus: 81.9% aged 0 - 4 years, 5.6% aged 5 - 9 years, 5.6% aged 10 - 19 years, and 6.7% over 20 years. Although there was a tendency in the civilian cases towards a rising age incidence as compared with earlier epidemics, the infantile pattern is clear. This tendency to a rising age incidence conforms with a general epidemiological trend noted by Burnet (1940) and Sabin (1949a). The infantile pattern is the rule in most tropical and semi-tropical countries where standards of hygiene tend to be low. McAlpine (1945) stated that poliomyelitis was not uncommon among native Indian children while comparatively rare in adults, and according to Paul et al. (1944) there was a fairly high endemic rate in Egyptian children under the age of 5 years with a low rate in Egyptian adults. In a later study in Egypt Paul et al. (1952) were able to show that the age of onset of poliomyelitis in Egypt is purely infantile; i.e. two thirds

of the cases occurred before the age of 2 years. They compared this with Miami, Florida, which is roughly on the same latitude as Cairo, where two thirds of the cases did not occur until children were 12 years old. They also found that serum neutralization tests in Egyptians ranging from one month to 40 years of age showed that by 2 years of age about 80 per cent. had acquired Lancing type antibodies, and throughout subsequent life the level was maintained at about 100 per cent. Similar studies were made with the Brunhilde and Leon types of poliomyelitis virus and they deduced from their results that most children in Egypt acquired antibodies to all the three known types of poliomyelitis virus between the ages of 1 and 4 years. In Japan (Paul 1945) the disease is endemic and affects mainly children, while in West Africa the disease is well known to local inhabitants in young native children but uncommon in native adults (Findlay, Anderson and Haggie 1946). The service cases in Malta in 1950 - 51 were mostly adults (table II), of whom two were Maltese (civilian nursing sisters working in the Military Hospital); one was a Canadian, one a New Zealander, and the remainder were of British nationality. The two Maltese nursing sisters were binovular twins who were away from Malta during the 1942 - 43 epidemic of poliomyelitis. As in the previous recorded outbreaks no Maltese troops were affected.

(d) /

(d) Geographical distribution of service cases.

Malta and its neighbouring island of Gozo are together rather smaller than the Isle of Wight. The area of Malta is 95 square miles and that of Gozo is 27, (see map). The approximate civil population in 1950 was 310,000. The islands are thus densely populated.

The David Bruce Military Hospital is situated at Imtarfa (see map), where also a small number of service families were quartered in 1950 and 1951. Seventeen cases, including 12 of the hospital staff, lived at Imtarfa. Thus one third of the cases came from this area. If the 12 hospital staff are excluded, there would still be 5 out of 51 cases, (i.e. 9.8 per cent.) who lived near the hospital. These 5 people had ample opportunity of coming into contact with most of the staff of the hospital in shops, barracks, or day to day encounters in the open.

I think that all this supports the view that contact is an important factor in the spread of poliomyelitis. (See Chapter III).

II. THE INCUBATION PERIOD OF POLIOMYELITIS

The Incubation Period of poliomyelitis has not been accurately established; the period usually given is 7 - 14 days but estimates of from 3 - 35 days have been given.

Wickman in 1907 tried to calculate the Incubation Period from the occurrence of multiple cases in families and others followed his lead. Thus Aycock and Eaton (1925) studied the interval between multiple cases in 253 families, and found that a large proportion of the so called secondary cases arose from a common source with their respective primary cases; a much smaller proportion of secondary paralysed cases in the same family occurred 10 - 18 days after the primary cases and were regarded as true secondary cases. They concluded that the Incubation Period of poliomyelitis varied from 10 - 18 days.

Other methods of calculating the Incubation Period have been used since, thus:

(a) Limited exposure to a known case.

Aycock and Kessel (1943) studied 49 cases which followed a limited exposure to a previous case. They found that in the majority of cases the Incubation Period varied from 6 to 14 days; exceptionally it was as long as 20 days. In the Mauritius epidemic of 1945 (McFarlan, Dick and Seddon 1946) the Incubation Period estimated from 7 cases with short

and /

and only possible exposures and from 6 cases with longer but limited exposures was found to range from 8 to 14 days. Silverthorne et al. (1949) observed 7 instances in which the deduction seemed reasonable that a certain patient had been infected by another during a restricted period of contact. From these instances they thought that the Incubation Period might lie between 7 and 21 days.

(b) Contact with a known case outwith the district.

Casey (1942) collected a series of instances in which the initial victim in a district either visited or was visited by a patient with acute poliomyelitis from some other district. His estimation of the Incubation Period by this method was 5 to 35 days.

(c) From the study of milk-borne infections.

This method of spread of poliomyelitis is thought to be rare but several examples of it have been reported, e.g. Wickman (1907) mentioned an outbreak, probably due to milk, in 1905, where there was a simultaneous onset of cases in fairly widely separated houses all supplied with milk from the same farm, in which a case of poliomyelitis had occurred, 14 days previously. He assumed that the Incubation Period was upward of 14 days. Also in an apparently milk-borne infection in Spring Valley, New York, reported by Dingman (1916) /

(1916), the interval between primary and secondary cases was between 14 and 18 days. In another milk-borne outbreak of 8 cases in Cortland, New York, Knapp et al. (1926) estimated the Incubation Period at 7 to 18 days. Finally, Aycock (1927) reported an outbreak of poliomyelitis in Broadstairs in Kent, in which the majority of cases occurred between 7 and 14 days following the period during which they were all using the same milk supply.

(d) From the occurrence of poliomyelitis following tonsillectomy.

Aycock and Luther (1929) presented evidence which indicated a causal relationship between tonsillectomy and the occurrence of poliomyelitis in 16 cases all of whom developed the disease between 7 and 18 days following operation.

Francis et al. (1942) described the occurrence of poliomyelitis in a family of 5 children, the 5 eldest of whom developed bulbar poliomyelitis 9 to 14 days after tonsillectomy carried out on the same day. This study also emphasised the danger of tonsillectomy during the months in which poliomyelitis is prevalent even though cases of the disease have not been recognised in the community.

Some of the variation in the above is due to lack of uniformity of interpretation among clinicians as to what constitutes /

constitutes the onset of disease; and the onset itself is extremely variable. There are several ways in which the disease may commonly begin (Horstmann 1949). Thus there may be a first phase (minor illness), which is entirely non-specific, followed by a few days of well-being before a second phase with all the first phase symptoms, plus those of involvement of the central nervous system. There may be no interval between first and second phases; the first phase may be entirely absent or so mild as to be entirely missed; and occasionally paralysis may occur after 10 days or more of vague non-febrile prodromata. Some clinicians take the onset of the prodromal or first phase, while others the secondary (meningeal) phase, as the end point of the Incubation Period. As has been shown, these are by no means always clear cut, and in my cases the very first day of illness is taken as the end of the Incubation Period.

In addition to the epidemiological studies of the Incubation Period much experimental work in animals has been done. In experimental poliomyelitis variation in the Incubation Period is common, depending on the animal species, dose, route of inoculation and strain of virus.

Thus Faber and Silverberg (1941), inoculating rhesus monkeys with 40 recently isolated human strains of poliomyelitis virus by various portals, found that the Incubation Period varied from 4 to 64 days.

Finally /

Finally an attempt at finding the Incubation Period has been made by isolating the virus, either in experimental poliomyelitis in animals, or in human poliomyelitis. In animal experiments Melnick (1946) found that virus introduced into the skin of monkeys and chimpanzees could be isolated from their stools several days before the clinical signs of the disease appeared. In human poliomyelitis, Francis et al. (1942) reported presumptive evidence of the presence of the virus 30 days before the onset of the disease; Brown et al. (1945) found virus in the stool 19 days before the clinical onset, Gear and Mundel (1946) isolated virus 12 days before the onset and Zintek (1947) could not recover virus from the throat washings or stools of any member of an infected family 4 days before the onset of poliomyelitis, although 8 days later an additional member of the family developed paralytic poliomyelitis and at this time the virus was recovered from every other member of the family.

By these methods, then, the estimation of the Incubation Period has given widely varying results; this is especially so in experimental poliomyelitis and in the work concerned with virus isolation. In the Malta outbreak of 1950 - 51 twelve of the 51 service cases were members of the nursing staff of the David Bruce Military Hospital. By studying the contact history of 7 of these it was possible to make an estimation of the Incubation Period.

The circumstances were these:

The first of the service patients in the outbreak of poliomyelitis in Malta in 1950 was Mrs. W., the wife of a Naval Officer; she was admitted to hospital on 15th June. The second patient, the wife of Lieut. D., Royal Navy, was admitted on 24th July in the paralytic stage with severe flaccid paralysis of both legs and a lesser degree of paralysis of the right arm. She had been ill at home for 8 days and was four months' pregnant. Lieut. D. was admitted four days later on 28th July complaining of severe headache, vomiting, fever and generalised body-pains of 2 days' duration. On admission he had no paralysis and his condition was typical of the preparalytic stage of poliomyelitis. Paralysis of all limbs and of the main muscles of respiration developed during the next twelve hours. In spite of treatment in an artificial respirator he died 36 hours after admission to hospital.

Until he was put in the respirator Lieut. D. was nursed in a single bedded room measuring 15 x 10 x 14 feet the floors and walls of which were of Tarrazzo. The weather at the time was warm and humid; the maximum and minimum temperatures during the last week in July were 94°F. and 72°F., respectively, while the highest relative humidity was 97%. The rainfall was nil. The windows of the room were kept open and flies were excluded by fly proof screens. The ventilation was adequate and was assisted by an electrical table fan. There was little dust.

The /

The patient was constipated for 3 days before admission and he had no bowel action while in hospital. The naso-pharynx was not inflamed and the patient had no excessive throat or mouth secretions except for a brief period after he was put in the respirator when it was necessary to aspirate some accumulated saliva on one occasion only. This was done by me using a rubber catheter and mouth suction taking care that no aspirated secretions reached my mouth. This action was probably foolhardy but the situation for the patient was desperate and no other means of suction was immediately available; I did not develop poliomyelitis.

Table III shows the dates of onset of poliomyelitis in seven members of the hospital staff. That Lieut. D. was the source of infection in some of these cases there is little doubt. He was in hospital for only 36 hours (28th - 30th July). The work of Paul, Havens and Van Rooyen (1944), in virus isolation experiments in the Middle East, suggested that the amount of virus present in the intestinal tract was greater in the more severe, than in the milder cases of poliomyelitis. If this was so in Lieut. D's. case, then he was probably a highly infectious case.

Of his attendants, numbering three doctors, six nursing sisters and three orderlies, seven later developed poliomyelitis; these are listed in table III. Case I, a Naval Nursing Sister, nursed /

nursed two other cases of poliomyelitis, both beyond the acute stage, while working for four days only at the David Bruce Military Hospital but she returned to duty at the Royal Naval Hospital, some miles away, where there were no cases of poliomyelitis, after Lieut. D. died. Her contact with known cases of poliomyelitis was therefore brief and did not take place after Lieut. D's death. Cases 2, 3 and 4 came into contact with no other known case of poliomyelitis.

The known contact of case 5 was very short, only long enough to take a sample of blood for a differential white cell count, a few hours after Lieut. D's. admission to hospital and before he developed signs of paralysis. In this connection, the work of Horstmann (1952) on viraemia in experimentally induced poliomyelitis is interesting. She was able to demonstrate viraemia in seven out of ten cynomolgus monkeys and three out of four chimpanzees with experimentally induced poliomyelitis. The viraemia persisted on some animals over the 4th, 5th and 6th days after virus ingestion. The virus was not isolated from blood specimens collected during the first three days after feeding, nor on the 7th day in the two instances in which it was tested, and the interval between viraemia and the appearance of paralysis varied between three and seven days in the cynomolgus monkeys.

Bodian (1952) also described the isolation of virus from the /

the blood of four chimpanzees fed Lansing type virus. In these cases also the virus was isolated before the onset of paralysis in three of the chimpanzees which became paralysed and before a rise of antibodies was detected.

I think that the chance of the laboratory technician becoming infected through Lieut. D's. blood is remote. His contact was nevertheless brief and during part of it, i.e. while blood was being sucked into the pipette, no protective mask was worn.

The contact of cases 6 and 7 with other cases was diffuse and less reliance can be placed on the time of onset of the Incubation Period. These two cases were the Maltese twin sisters already mentioned. They were of good family and are likely to have been less exposed to infection with poliomyelitis virus, than the average Maltese citizen, even although they had not been away from the island during the 1942- 43 epidemic.

Scrutiny of table III shows that in cases 2, 3 and 4 the Incubation Period was 20, 22 and 22 days respectively; it seems reasonable to assume that cases 1 and 5 became infected with virus at the same time, while in cases 6 and 7 this seems unlikely but nevertheless possible. The instance of the Naval Nursing Sister (case I) is most striking, since she left the Military Hospital, where all the service cases of poliomyelitis were nursed, after a short exposure to infection from a seriously /

seriously ill case. The Incubation Period in this instance was not less than 18 days.

As pointed out by Aycock and Eaton (1925), in their study of multiple cases in families, true secondary cases occur 10 to 18 days after the primary ones. This seems likely to have been the case with Lieut. D. who became ill 10 days after his wife sickened.

Lieut. and Mrs. D's. only child, a girl aged six, did not develop clinical poliomyelitis although contact with both her mother and father was both intimate and prolonged. Presumably this child had an inapparent infection or an immunity to the virus.

SUMMARY:

Since the realisation of the infectious nature of poliomyelitis many efforts have been made to work out the Incubation Period. Estimates have varied, even in clear cut cases of known contact, from 5 to 35 days. In cases presumed due to milk-borne infection, the Incubation Period has been given as 7 to 18 days and in cases following tonsillectomy the same figures have been quoted. Experiments in animals, and virus isolation work in humans, has given even wider variations.

The contact history of seven cases, attendants of a patient with poliomyelitis, is related and the Incubation Period calculated. /

calculated.

CONCLUSIONS:

1) The Incubation Period of poliomyelitis is difficult to estimate. This is so for various reasons.

(a) Variability of type of onset of the disease itself.

(b) Variability in the character of the disease.

(c) Paralysis is the exception after infection with poliomyelitis virus.

(d) In the absence of paralysis, virus isolation or other experimental proof of infection is necessary for accurate diagnosis; this is difficult and costly.

2) The Incubation Period in the outbreak of poliomyelitis in Malta in 1950 - 51 ranged from 10 to 32 days but the period 10 to 23 days is likely to be a more accurate estimation.

III. THE MODE OF SPREAD OF POLIOMYELITIS VIRUS AND THE RELATIONSHIP OF MINOR CONCURRENT ILLNESS TO THE OUTBREAK

The mode of spread of poliomyelitis is still the subject of heated argument. There is a great weight of evidence in favour of transmission by ordinary human contact which was the original conception of Wickman (1913) and to which studies of the excretion of virus (Howe et al. 1945, Pearson et al. 1945, Pearson and Rendtorff 1945, Brown et al. 1949, Casey et al. 1950) have given confirmation. The work of Caverly (1924), Casey et al. (1945), McFarlan et al. (1946) and Sweetnam 1948 also supportsthis concept. Further, in a detailed investigation of poliomyelitis in South Australia, Southcott and Crosby (1949) were able to eliminate transmission by animal, bird, or insect victors, and all but enteric and contact routes of transmission, from their considerations. In the northern hemisphere near the Arctic circle contact with healthy carriers was thought to be the main mode of spread in an outbreak in the Chesterfield area of Hudson Bay (Moody and Adamson 1949).

In an epidemic in Lincolnshire in 1950, Clarke (1951), because of the occurrence of multiple cases in households, the spread of infection along bus routes and frequent history of previous contact with an infectious case, was of the opinion that the outbreak was transmitted by personal contact.

Spence (1949) observed an outbreak of poliomyelitis in Eccles /

Eccles in 1947. He found evidence of carrier to case contact between abortive and frank cases and he concluded that human contact played the major role in the spread of the infection. In addition, he thought that there were several factors which seemed to favour droplet spread.

In many other diseases evidence of this order has been accepted as conclusive proof if its validity, yet there have been many objections to it in relation to poliomyelitis. The basis of these objections has been the fact that the seasonal incidence of poliomyelitis is the opposite to that of the contact diseases as a group.

Aycock, Lutman and Foley (1945), however, thought that although the seasonal curve of poliomyelitis was the exact opposite of the upper respiratory contact group, this fact in itself was enough to suggest that it was not due to the operation of a different and unrelated mode of spread, but to the operation, in reversed order, of the same mechanism which determined the seasonal curve of the group of upper respiratory diseases in general.

In a study of a widespread outbreak of poliomyelitis in Kansas in 1946, Wenner and Tanner (1948) found evidence for a simultaneous seeding of virus throughout infected families, which suggested a common vehicle rather than a person. This could be linked with Bower's belief (described below). The spread /

spread of infection by means of milk has already been mentioned, and waterborne spread is not a likely one in civilised countries, although Hargreaves (1950) described an outbreak of poliomyelitis in Cornwall in which there were 20 cases in the city of Truro. In this outbreak the high age group of the patients, their sporadic distribution in the city, the lack of history of contact between the cases and the sudden cessation of the outbreak after superchlorination of a contaminated water supply suggested water dissemination of the virus.

Maxey (1949) however stated that although a large number of epidemiological studies of poliomyelitis have been made during the past half century, up to the present no convincing evidence has been presented that pollution of a community water supply was responsible for indirect or widespread exposure of consumers to infection with virus.

The role of flies in the dissemination of poliomyelitis virus must be considered in this discussion. As Sabin (1951) says, "the problem as regards the role of flies in poliomyelitis is not whether or not the infection can be transmitted without them, and not whether or not outbreaks can occur without them, because it is established, as in the case of dysentery, that flies are not necessary to account for either". Flies were reported by Trask et al. (1943) to be capable of transmitting the virus mechanically, and Ward et al. (1945) /

(1945) observed that flies could deposit sufficient virus on food to produce a carrier state in chimpanzees; and that food exposed to flies in an epidemic area was contaminated with enough virus to produce infection in chimpanzees who ate it. This indicates the potential danger to humans of food and drink contamination. Clearly, flies are not necessary for the spread of the dysenteric diseases or poliomyelitis, but this does not mean to say that flies are unimportant under certain circum-:stances, at certain places and at certain times of the year, in the dissemination of poliomyelitis virus. Sabin (1947) studied the first large epidemic of poliomyelitis in Berlin: because of an unexpected outbreak of malaria the preceding year, it so happened that just before the beginning of the 1947 poliomyelitis epidemic was suspected, somewhat more than half of the city was sprayed with D.D.T. from the air and from the ground, and the remainder was not so treated because the Russian authorities refused to carry out such preventive measures. Two months after the single spraying of D.D.T. the poliomyelitis attack rate was twice as high in the untreated Russian sector as in the remainder of Berlin, i.e. 65 per 100,000 as compared with 32 per 100,000. In Sabin's opinion the filth fly must be regarded in the same light as the human carrier during poliomyelitis epidemics, and that whether or not flies, by serving as additional non-human carriers, may play an important /

important role in the initiation of an epidemic, the large number of human carriers, who have become seeded with virus by the time a large urban epidemic has reached its peak, is unquestionably itself capable of maintaining dissemination on a large scale.

The incidence of the dysenteric infections is favoured by conditions of warmth; i.e. it is higher in tropical and subtropical than in temperate and cold climates and in the latter it is higher during the warmer seasons than during the colder. Conditions of warmth also favour the growth of the fly population. Conversely the incidence of upper respiratory infections, which are spread mainly by droplets, is higher in cooler than in warmer climates. Poliomyelitis in temperate climates occurs characteristically in the summer and autumn, when conditions are least favourable for the spread of disease by droplets, and certainly most favourable for the spread by faecal contamination.

The main factors which make dysenteric infections more common in summer are these:

1) Increase in the fly population.

2) Greater opportunity for faecal contamination of the fingers because of fewer clothes worn by adults and children.

3) Food and drink is more frequently exposed to the open and hence to flies.

One cannot ignore the role which may be played by flies in the spread of poliomyelitis virus in certain circumstances of climate, hygiene and time.

The early experimental work on the transmission of the virus in rhesus monkeys led to the conclusion that virus was trans-:mitted by droplet infection and that access to the central nervous system was gained through the olfactory bulbs. When however it was shown that the olfactory bulbs were not affected in human beings it was assumed, by some, that the respiratory and nasal mucosa and the naso-pharynx served as the sites of entry, multiplication and exit of the virus; and many epidemiologists have stuck to the idea that the hypothesis of respiratory transmission best explains the pattern of spread of poliomyelitis (Anderson 1947). Modern studies have, however, effectively ruled out the olfactory mucosa as a portal of entry of the virus by the demonstration that the olfactory bulbs in fatal human cases do not show the characteristic lesions (Sabin 1940, Howe and Bodian 1941) or contain virus (Sabin and Ward 1941); nor is there evidence that virus proliferates in the olfactory mucosa (Sabin and Ward 1941).

What are the facts regarding the sources of poliomyelitis virus and the potential routes by which infection can be trans-:mitted? Sabin (1949b) in his analysis of recent publications, indicated that the alimentary tract was the one system outside the /

the C.N.S. in which virus was regularly found, and the same author in his Bela-Schick Lecture (1944) showed that studies on fatal cases indicated that in any one patient the virus may or may not be found in the washed tissues of the oropharynx although it was invariably found at one or other level of the alimentary tract. Furthermore, it is now well established (Schabel et al. 1950, Brown et al. 1949) that virus can be detected in the stools in 70 to 90 per cent. of cases during the first week of illness and in about 50 per cent. in the 3rd and 4th weeks after onset. This can be done not only in frank cases of poliomyelitis, but also in children with a sub-clinical or minor illness type of infection. Moreover, Sabin (1951), in his "Analysis of differing interpretations and concepts in the transmission of poliomyelitis virus" concluded that the available evidence indicated that the virus, which is occasionally found in the throat of patients and healthy carriers, did not ordinarily reach the outer environment by droplets from the mouth or nose.

The fact that poliomyelitis virus can be recovered from the throat in nearly 50 per cent. of patients within 3 - 5 days of the acute onset of the disease (Howe, Bodian and Wenner 1945; Wenner and Tanner, 1948; Pearson and Brown 1947) does not necessarily mean that the throat and mouth are the main portals of exit or that respiratory spread is common. An analogy with typhoid fever is here drawn: typhoid bacilli have been reported to /

to be present in the mouths of approximately 50 per cent. of typhoid patients by Purjesz and Perl (1912) and by Gould and Qualls (1912) yet no one will deny that the route of infection in this disease is alimentary.

In the entire literature of poliomyelitis there is only a single report, made by Flexner and Amoss (1919) in which the virus was said to have been recovered from the nasal mucosa of one of three fatal cases tested. Nor with all the possibilities for aspiration, has the virus been demonstrated unequivocally from the trachea or lungs (Sabin and Ward 1941, Sabin 1944).

The view of the relative unimportance of the respiratory hypothesis in the spread of poliomyelitis is supported by Bower (1950) who reported that during four years when 6,000 patients were treated in Los Angeles County Hospital, doctors, nurses and attendants did not wear masks unless they themselves had a cold (the mask was worn only for the patient's protection) and none contracted the disease. Had droplet infection been the main mode of spread of the virus one would have expected some secondary cases to have arisen in the staff in so large a series of patients admitted in the acute phase of the disease. In fact, Bower believes that the main method by which the disease is spread is by contact with objects which are contaminated through having been handled and infected by carriers or patients.

Presumably Bower meant infected with virus derived from faeces /

faeces but objects could just as readily have been contaminated by oral or pharyngeal secretions.

There are, then, three main views on the mode of spread of poliomyelitis virus. These are:-

Respiratory - (droplets).

Alimentary - (intestinal - oral circuit).

Alimentary plus the non-biting flies.

Neither the respiratory nor the alimentary hypotheses attempt to account for the fact that 90 per cent. of the cases and most of the epidemics occur during the late summer and autumn, except for the assumption that the hot weather makes the individual more prone to have the paralytic form of the disease. The alimentary plus the non-biting flies hypothesis regards the predominance of the disease during the late summer and early autumn as being due to greater dissemination of virus by flies contaminated with infected human faeces. According to this view, small outbreaks can occur in the absence of flies, and epidemics can continue, at least for a time, after the advent of the cold weather has caused the flies to disappear, because of the large number of human carriers that are established by the initial widespread dissemination of virus.

It is of fundamental importance to establish the mode of spread of poliomyelitis virus if the correct measures of prevention of the disease are to be formulated.

The /

The climate of Malta is subtropical with an average annual rainfall of 20 inches; the bulk of the rain falls between September and March. The summers are hot and almost rainless with a high relative humidity in the months of July, August and September.

The 1950 outbreak of poliomyelitis began in May, was at its height in August and September, and tailed off thereafter to end in February, 1951. Most of the cases, then, occurred during the hot months of the year when the conditions for transmission by faecal contamination of food, drink or hands were most favourable, and transmission by droplets least favourable. The summer of 1950 was unusually hot and it was my impression that flies, always abundant in summer, were even more so than during the previous or the following summers.

The incidence of gastro-intestinal infections (diarrhoea, gastro-enteritis and bacillary dysentery) in 1950 was appreciably greater among servicemen than during the previous summer when there were no cases of poliomyelitis in Malta.
Figure I shows the weekly admissions to hospitals (Naval and Military) of:

1) Gastro-intestinal Infections.
2) Short term fevers.
3) Upper respiratory infections (Tonsillitis, Pharyngitis and common colds) and
4) Poliomyelitis.

The /

The number of gastro-intestinal infections admitted shows an irregular rise to its peak in October; the sudden fall coincided with the onset of the cooler wet weather and the fall in the numbers of flies. This is what one would expect to happen in a country with the kind of hygiene and climate of Malta.

Can the respiratory hypothesis be invoked to explain the transmission of poliomyelitis virus in the Malta outbreak of 1950? Figure I shows a rising number of upper respiratory infections from June to a peak in August 1950; thereafter the number of cases falls to reach a lower level in late September. The Military and Naval records in Malta show that upper respiratory infections were commoner by one third in the summer of 1950 than in the previous summer, when there was no poliomyelitis. It seems probable that some of the cases diagnosed as one or other of the upper respiratory infections were in fact unsuspected abortive poliomyelitis, and it may be argued that such cases were likely to spread poliomyelitis virus by droplets. A large outbreak of measles, one of the diseases in which transmission by droplets and droplet nuclei is not disputed, began in February 1951 when the poliomyelitis outbreak had ended.

The peak of the admissions of upper respiratory infections occurred three weeks before the peak of poliomyelitis. This,

I /

I think, could be interpreted as showing that many of the cases of upper respiratory infection were in fact abortive poliomyelitis, i.e a carrier wave preceding the main poliomyelitis wave, and also that the mode of spread in the early weeks of the epidemic may have been by droplet infection. This carrier wave preceding the main wave, was suggested by McFarlan et al. (1946) to explain the mode of spread in the Mauritius outbreak of 1945.

Only six of my cases complained of sore throat at the onset and only three had coryza. I do not know how many had had upper respiratory infection preceding the onset of recognisable poliomyelitis.

Ingalls and Aycock (1951) studied the unpublished records of an outbreak of poliomyelitis in a boys' school in 1936 where there was a preceding outbreak of upper respiratory infection. During the period 1st to 20th May 1936, 41 per cent. of boys who later developed poliomyelitis had upper respiratory infection while only 19 per cent. of the others in the school had such an illness. They concluded that these distributions carried a definite implication that upper respiratory infection was one of multiple factors in the determination of the disease. These facts, and the conclusions drawn from them, support the respiratory hypothesis in the spread of poliomyelitis virus.

The mode of spread of the virus in the seven members of the hospital /

hospital staff (table III) who were in contact with known cases is difficult to determine. The staff wore gowns and masks, but not gloves, while in the poliomyelitis wards. They were impressed with the importance of washing the hands and taking all precautions necessary to limit the spread of the alimentary infections. I do not think that the precautions which were recommended were inadequate, nor is there reason to suppose that insufficient heed was taken of them. Was there, then, a heavy contamination of the fomites, the patients, or the wards by virus derived from either faeces or from oro-pharyngeal secretions? Lieut. D. had no bowel action while in hospital nor was he expelling virus from the oro-pharynx, in any obvious way. If it is assumed that he was the source of infection of several cases, then virus must have come from either faecal contamination or from droplets or droplet nuclei. I think that the former is the likely mode of spread because minimal contamination of the fingers or hands is easily acquired, especially by nursing attendants, and such contamination is all that is required to set up infection in a non-immune individual.

SUMMARY:

The mode of spread of poliomyelitis is not clearly known. The main hypotheses are concerned with droplet infection, the intestinal oral circuit and the role of flies in conjunction with /

with faecal contamination. The importance of establishing the mode of spread is stressed. The circumstances of the spread of virus in the outbreak of poliomyelitis in Malta in 1950 - 51 are discussed.

CONCLUSIONS:

1) The precise manner in which virus is transferred from one person to another is not clear.

2) Contact, direct or indirect, with a case or carrier is essential for the spread of poliomyelitis virus.

3) Contact may be direct through intimate personal relationships with cases or carriers, or indirect through food, drink or feeding utensils contaminated with faeces by fingers or flies.

4) Droplet infection may play a part under certain conditions.

5) Droplet infection probably operated in the early weeks of the Malta epidemic of 1950 - 51 but the intestinal-oral route of infection is the more important means of maintaining an epidemic over a period of months.

6) Medical and nursing attendants should wear efficient masks for the first few days of a patient's illness. Precautions for the disposal of faeces, the sterilisation of feeding utensils and the elimination of flies should be as for the enteric diseases /

diseases and should be maintained for at least 4 weeks. Thorough washing of hands after contact with a patient is essential and is more important than mask wearing.

IV. RELATIONSHIP OF ACTIVITY IN THE EARLY STAGES OF POLIOMYELITIS TO PARALYSIS

There is, as yet, no specific remedy for poliomyelitis, but recent advances have given hope for the future. Hammon et al. (1952) have shown that injection of gamma globulin may help to prevent poliomyelitis, and that if it is given in the week before the onset of the disease it may prevent paralysis. The ability to grow the virus by means of tissue culture (Enders 1951) may lead to the development of an attenuated virus vaccine for active immunisation. Unfortunately these methods of prevention are not yet sufficiently advanced for general application and, because of this, our efforts to limit the spread of infection by general methods of hygiene, and to limit the extent of paralysis by early diagnosis and treatment, are still the only means of lessening the effects of the disease. It has been known for many years that treatment in the early stages of the illness by rest and sedation seemed to offer the best means of limiting paralysis. Thus, Limper et al. (1931) found that exhausting exercise preceded the onset in seven out of a series of sixty cases; Tebbutt and Helms (1933) in an epidemic in New South Wales in 1931 - 32 made similar observations, while de Rudder and Petersen (1938) reported two local outbreaks of poliomyelitis in a group of rural schoolboys, 13 to 19 years of age, shortly after a two-day athletic contest. Six /

Six paralytic and thirteen abortive cases developed among the participants while only three abortive cases occurred among non-participants from the same school. It was not until 1947, however, that Russell (1947) clearly demonstrated the correlation of activity, in the early stages of the illness, and the degree of paralysis, and in 1949 the same author stated that complete physical quiet from the onset of meningitic pains will seldom fail to prevent serious paralysis, while strenuous or even moderate physical activity at this stage is highly dangerous. Hargreaves (1948) analysed thirty cases of poliomyelitis which occurred in Cornwall in 1947 - 48, his findings supported those of Russell. In America this was followed up by Horstmann (1950). She studied 411 patients with poliomyelitis from three epidemics and presented data indicating that the percentage of patients who continued physical activity after the appearance of symptoms of the major illness was considerably greater in the paralytic group than in the non-paralytic. No correlation was demonstrated between physical activity during the first phase, or minor illness, and the final outcome. The critical time as far as physical activity was concerned seemed to be the first 24 to 48 hours of the major illness. There was no correlation between activity immediately preceding onset and paralysis. Albrecht and Locke (1951) studied the physical activity from three days before to a number of days after /

after the onset of illness in 200 patients in New York State. They concluded that death and severe paralysis might be attributed in part to the degree of physical activity in the meningeal stage (Russell's pre-paralytic stage) in adults, but not in children. Levinson, Milzer and Lewin (1945) attempted to obtain experimental proof of the importance of activity during the Incubation Period of poliomyelitis by subjecting monkeys (Macaca mulatta) to exhausting exercises, in this case swimming, after inoculating them with poliomyelitis virus. It was found that the monkeys developed a more severe and higher incidence of paralysis, than controls.

Russell (1952) has reviewed his own and Horstmann's data on this subject. There is no doubt that the relationship of activity at a crucial phase of the disease is of great significance in prognosis. This is likely to remain so until some means of active immunisation, akin to that provided against yellow fever, is practicable.

In the service cases in Malta in 1950 there was a remarkable correlation between activity in the pre-paralytic stage of the disease and the severity of paralysis. Nine cases had a severe degree of residual paralysis and two other cases died. Six of the nine cases who developed severe paralysis were admitted to hospital four or more days after the onset of symptoms and all six had a striking history of activity in the pre-paralytic phase. /

phase. Of the patients who died, one was a child of six years who was not admitted to hospital until the third day of illness and the other, Lieut. D., has already been mentioned.

The following are brief case notes of the six severely paralysed patients:

Case 1.

Housewife. Mrs. D. Age : 26 years.

Day 1: Severe frontal headache but nevertheless went picnicking and bathing; out in open all day.

Day 2: Headache continued, accompanied by nausea, anorexia, fever, generalised body pains. Went to bed and was treated symptomatically at home until day 5 when the fever subsided and the patient felt generally better. Noticed weakness of both legs. This was ascribed to the fever which she had had and she was allowed to sit up out of bed. Her general condition improved but weakness of legs did not, and when admitted to hospital on day 8 she had severe paralysis of both legs and slight weakness of the right arm.

Case 2.

Petty Officer. Age : 32 years.

Day 1 - 2: Complained of occipital headache, nausea and dizziness but continued to work.

Day 3: Still unwell but did not report sick.

Day 4: So ill that he could not go to work but refused to have /

have a doctor visit him in his own home. He preferred instead to make a journey of four miles, which included crossing the Grand Harbour by ferry-boat and a good deal of walking, to see a doctor. Admitted to hospital, developed severe degree of paralysis of both arms, legs and trunk.

Case 3.

Naval Nursing Sister. (Case I in table III). Age : 30 yrs.

Day 1: Felt unwell with anorexia and moderate headache.

Day 2 - 3: Pain in thighs and stiffness of the legs; continued to work.

Day 4: Symptoms continued; sacral pain and severe headache.

Day 5: Admitted to hospital; developed severe paralysis of both arms and both legs.

Case 4:

Airman. Age : 20 years. Keen athlete. Well built muscular man, good morale.

Day 1: Headache, vague generalised pain but engaged in wrestling bout.

Day 2: Headache worse, pain at back of neck and in lumbar region.

Day 3: Reported sick for first time because of increasing pain in the back, neck and thighs. Put to bed in station sick quarters but not strictly supervised.

Day 4: /

Day 4: Admitted to Military Hospital; widespread paralysis of arms and legs.

Day 5: Respiratory paralysis; respirator required for six weeks; very severe residual paralysis of arms, legs and trunk.

Case 5.

Girl. Age : 4 years.

Day 1: Sudden onset of fever, irritability, backache, headache. Treated symptomatically until day 3 when fever settled and child was allowed to move freely about the bed although still far from well. Parents noticed clumsiness of movements and child's difficulty in sitting up.

Day 4: Increasing weakness of limbs noted by parents but still child was not strictly supervised or immobilised.

Day 5: Admitted to hospital. Severe paralysis of both arms, both legs and trunk; neck and spine stiff. Child very ill manifesting extreme irritability, restlessness, emotional disturbance, and disturbance of sleep rhythm.

Case 6.

Lieutenant, Royal Navy. Age : 22 years.

Day 1: Vaguely unwell; slight backache.

Day 2: Still not feeling well but played game of hockey.

Day 3 - 4: Continued to work although complaining of headache.

Day 5: /

Day 5: Felt much worse; complained of headache, backache, fever. Treated symptomatically in quarters. Noticed slight weakness of legs.

Day 6: Unable to walk. Admitted to hospital. Neck and back stiff; severe flaccid paralysis of both legs.

The converse, that adequate rest from the onset of symptoms will prevent serious paralysis, was also strikingly demonstrated in the series studied. Of twelve members of the hospital staff who developed poliomyelitis no less than ten returned to full duty within six months. Of the two members of the staff who did not do so well, one was case 3 above who did not report sick early, and the other had a relapse with increased paralysis of the legs in the fifth week of illness. Nine other cases, who were admitted to hospital soon after onset, had paresis of all four limbs; they all made a rapid recovery. This may be attributed to adequate rest early in the illness.

Why does activity at a crucial phase of the disease increase the likelihood of severe paralysis? Since the extent of paralysis depends on the extent of damage to the anterior horn cells, the cause cannot be a local muscular one; but if it is assumed that the anterior horn cell can be affected by its peripheral connections, then muscular activity could so influence the peripheral connections as to cause damage to the nerve cells. This /

This is supported by the work of Hydén (1943) who showed that intense muscular work in the experimental animal depleted the protein of the anterior horn cell. Before this, Howe and Bodian (1942) showed that the susceptibility of the anterior horn cell to poliomyelitis virus was altered by section of (i.e. damage to) the peripheral nerve originating in these cells. This may be related to the effect which tonsillectomy has on the onset and form of the disease, but is also relevant in this discussion. Harrington (1951) studied 100 unselected cases of paralytic poliomyelitis in servicemen and found that five had a significant history of trauma which preceded by a few days the appearance of paralytic poliomyelitis. In each case the paralysis was in muscles closely related to the site of the trauma. He discussed two possibilities to explain this. Firstly, that local injury opened up some pathways of infection to the spinal cord, and secondly that the effect of local trauma was to alter the spinal cord cell physiology in such a way as to reduce resistance to the virus which was already established in the cord (Bodian and Cumberland 1947). Does muscular activity also bring about a change in the anterior horn cells, rendering them more susceptible to attack by the virus of poliomyelitis? This seems a reasonable assumption. Horstmann (1950) asks in discussing this question: is the patient impelled to activity as a result of the disease process which causes irritability and restlessness: /

restlessness: is back pain only relieved by walking about? This was not so in my cases, because patients in the pre-paralytic stage felt ill and were mostly impelled by this feeling to stay in bed. The adult patient who is likely to become severely paralysed is either a stoic or one with a high sense of duty. In either case he tends to minimise his symptoms and to remain active in spite of feeling unwell. This was certainly the case in four of the six severely paralysed cases detailed above on pages 34, 35, 36 and 37.

Mere physical activity at a crucial stage cannot be the only factor which determines the severity of the illness. As in other infections, dosage, virulence, and strain of virus, host susceptibility and immunity, as well as mental and physical activity, must interplay in determining the pattern of the disease for each individual. There is probably no disease which shows such a variation in pattern from individual to individual in a more striking fashion than poliomyelitis. In some patients the picture is of severe irreversible damage from the onset causing either death or severe paralysis, and at the other extreme is the patient with mild or minimal invasion and destruction. The factors which cause such variation are clearly multiple, and it is reasonable to assume that some of the factors are concerned with nerve cell metabolism; activity at a crucial stage in the disease is possibly one such factor which might result /

result in the complete destruction of anterior horn cells which might otherwise have received lesser and reversible damage.

SUMMARY:

The evidence that activity at a crucial phase of poliomyelitis bears a relationship to the severity of paralysis is reviewed. This relationship was found to exist in six out of nine severely paralysed patients in the 51 service cases in Malta in 1950 - 51. The causes of this relationship are discussed.

CONCLUSIONS:

1) The virus of poliomyelitis may be present in anterior horn cells 24 hours before the onset of paralysis. Undue muscular activity or trauma at a crucial stage of the disease in some way alters the cell-virus relationship. This renders the cell less resistant to the virus and brings about cell damage which may be irreversible.

2) Until some form of active immunisation is discovered, the best means of limiting paralysis in poliomyelitis is the avoidance of physical exertion in the pre-paralytic or meningeal phase of the disease.

V. THE CLINICAL FEATURES OF THE OUTBREAK

The clinical features were not in any way unusual but certain aspects were of interest.

(a) Main Symptoms:

It is a well known observation that the prodromal symptoms of acute poliomyelitis are not characteristic and that they vary in different epidemics. In this series gastro-intestinal symptoms, as expressed by vomiting and diarrhoea, occurred in the following percentages; vomiting 29 and diarrhoea 21.5. Sore throat was not very conspicuous and occurred in six cases. Vertigo, with or without fainting, was the initial prodromal symptom in 16 cases, headache in 32 cases, and muscle pain in 21 cases. (See Figure II).

The explanation of the prodromal symptoms is a matter of speculation. The old view was that the prodromal symptoms were due to a systemic reaction to the presence of the virus, while the central nervous system manifestations only became apparent when the blood - C.N.S. barrier was overcome. This has very little support now as there is pathological evidence of the presence of the virus within the central nervous system at the prodromal stage. Such symptoms as headache and muscle pain would be explicable on the same mechanism that is responsible for their presence in the pre-paralytic and paralytic stages. The meningeal reaction might explain the headache, while the explanation /

explanation of muscle pain remains obscure. In view of the frequent and early involvement of the vestibular nuclei and the reticular formation in the brain stem (Bodian 1947), the occurrence of vertigo or fainting is not surprising, and one might surmise that gastro-intestinal symptoms may also be due to the early involvement of the central stations of the vegetative system in the hypothalamus. Every patient manifesting one of the usual prodromal symptoms was regarded as a suspect, kept in bed, and sedated. Daily, or if necessary more frequently, clinical examinations were carried out, particular attention being paid to the central nervous system. In this way it was possible to make a presumptive diagnosis in the pre-paralytic period. This is thought to be the reasonable attitude to take during an epidemic. An objection to this policy is that many patients, who do not eventually develop poliomyelitis, will have been subjected to unnecessary rest in bed for a few days. Does this matter? Quite apart from the possibility that even a proportion of these patients may be cases of abortive poliomy-:elitis, I think that it is only in this way that activity can be avoided and paralysis limited. Figure II shows the incidence of the main symptoms on admission to hospital.

(b) Main Signs:

As is generally known, the outstanding clinical finding in poliomyelitis /

poliomyelitis is weakness or paralysis of various muscle groups. Muscle weakness was detected in 41 cases on admission (figure III). The detection of muscle weakness is an important diagnostic finding which differentiates poliomyelitis from other acute infectious meningeal processes.

In discussing the development of paralysis in the early stages of poliomyelitis two important experimental and pathol-:ogical findings are relevant.

(i) The virus is demonstrable within the grey matter of the spinal cord sometime before the onset of clinical paralysis - 24 hours at least (Bodian and Cumberland 1947).

(ii) Clinical evidence of manifest paralysis does not appear until the damage to the anterior horn cells has reached a certain relatively high degree. This leads one to believe that in every case, between the initial prodromal symptoms and the onset of manifest paralysis, there is a "pre-paralytic" period of variable duration where slight muscle weakness is present but only detectable by a very careful examination of all voluntary muscles. In the non-:paralytic type, the process appears to be arrested at this stage. The variability in the duration of this period would appear to be responsible for the difference /

difference between "infantile" and the "adult" types described by Horstmann (1949).

In a number of cases the degree of paresis found on admission was slight. Although a febrile patient may show some loss of muscle power, when one limb was found to be weaker than its opposite, however slightly, one was able to make the correct diagnosis at once, without waiting for specific muscle groups to be picked out. In many instances, weakness of the anterior neck muscles or of the abdominal muscles was a prominent early sign. The value of weakness of the anterior neck muscles as an early sign, especially in children, is one that was described many years ago, (Peabody, Draper and Dochez, 1912), but its value needs to be re-emphasised. The bulk of motor loss in most cases occurred during the first ten days of the paralytic stage, but it was found that it was not safe to assume that no further loss would occur after the temperature had returned to normal. One of the cases had a reactivation of the disease in the fifth week of illness. Watkins (1949) reported involvement of new segments and of greater weakness of those already affected in 10 - 20 per cent. of a series of cases at the end of the third or fourth week of illness. /

illness. This he attributed to sudden renewed activity of the virus, and he supported this view by reporting that examination of the cerebro-spinal fluid at this time revealed a greatly increased protein content and occasionally a mild cellular reaction.

Neck rigidity was found in 37 cases and requires little comment, except to stress its frequency. Once it is detected, poliomyelitis should be thought of and the patient handled appropriately.

Generally speaking an assessment of tendon reflexes did not help in diagnosis. They were often brisk in the pre-paralytic stage but sometimes normal or depressed; importance was placed on them when a difference in the two sides was found.

Muscular pain and tenderness can be very troublesome and may go on for much longer than is often supposed. In most cases in this series, pain and tenderness subsided within ten days but in a few it lasted much longer in spite of treatment with rest and locally applied heat. The type of "spasm" noted was, in most instances, a heightened response to stretching or increased myotatic irritability; it was often accompanied by pain.

(c) Cerebro-spinal fluid: /

(c) Cerebro-spinal fluid:

Cerebro-spinal fluid (C.S.F.) was examined in only 19 of the cases. It is perhaps unfortunate, from a statistical point of view, that all were not so examined, but it was considered unnecessary to perform routine lumbar puncture for the following reasons:

(i) Where a patient was admitted in the paralytic stage, lumbar puncture for diagnostic purposes was clearly unnecessary.

(ii) It was considered, if not actively harmful, at least harrowing for the patient to subject him to lumbar puncture in the acute stage of the disease, when rest is essential, if the diagnosis was already clear on purely clinical grounds. Lumbar puncture was thus only done where, at the time, there was an element of doubt in the diagnosis.

Scrutiny of the results of the C.S.F. examination (table IV) shows the following points of interest.

(i) The cell counts ranged from 2 to 560 per cubic millimetre. This conforms with the findings reported by Anderson (1949), who found counts ranging from normal to 584 cells per cubic millimetre in 196 cases examined in an epidemic in Utah in 1945. Fraser (1913) stated that the C.S.F. in poliomyelitis might be /

be entirely normal in as many as 12 to 15 per cent. of cases, and might remain normal even until paralysis or death occurred.

(ii) There was no correlation between the severity of illness or extent of paralysis and the magnitude of the cell count. (Compare, in table IV, cases No. 3 and 44 who died, with cases No. 29, 33, 35, 37 and 41 all of whom made a rapid recovery). Thus the C.S.F. findings were not a reliable guide as to the presence or to the severity of the disease in this outbreak.

(iii) The protein was elevated above a normal of 45 milligrams per cent. in all but two cases and this elevation was found to be a more helpful sign in diagnosis than the cell count.

(d) Retention of Urine

Ten cases developed retention of urine in the acute paralytic stage. The use of "Prostigmine" or Carbamyl-choline Chloride ("Carbachol") was sufficient to make the patient pass urine in all but two cases, when a catheter was passed, because one or other of these drugs was ineffective. It was necessary to set up tidal drainage of the bladder in one case, but the patient regained bladder function after 5 days.

(e) /

(e) Vasomotor disturbance

Vasomotor disturbance, manifesting itself as coldness of the extremities, was noted in three cases. In all three there was a marked diminution of pulsation of the vessels of the feet, and the skin was seen to be pallid, and cold and moist to the touch. It is possible that involvement of the hypothalamus may be responsible for this feature. Affections of the hypothalamus, brain and brain stem, are also possibly the explanation for the encephalitic symptoms that are sometimes encountered in poliomyelitis.

SUMMARY:

The main symptoms and signs are recorded and the causation of some of them are discussed.

CONCLUSION:

There was nothing unusual in the pattern of the symptoms and signs.

VI. PREGNANCY AND POLIOMYELITIS

Pregnancy may be a predisposing factor in the onset of paralytic poliomyelitis but views on this differ. Bradley and Gale (1948) studied this aspect of the 1947 epidemic in Great Britain and came to the conclusion that the incidence of poliomyelitis was not higher in pregnancy than the expectation among the population of child bearing age. Aycock (1941), who has repeatedly emphasised the importance of the role of variable susceptibility, reviewed the subject of pregnancy associated with poliomyelitis and found an actual increase in the incidence over the anticipated frequency. He pointed out that the endocrine changes occurring in pregnancy might be important factors in predisposing pregnant women to poliomyelitis. Shaefer and Shaw (1949) have stated that aside from the purely endocrine changes, the stresses and strains of pregnancy may predispose to the susceptibility of poliomyelitis. This may well be related to the "fatigue factor" (see Chapter IV). Weinstein et al. (1951) also came to the conclusion, in a study of 1,770 patients in Massachusetts, that pregnancy increased the susceptibility to clinical poliomyelitis. Fox and Belfus (1950) in a 6 year study of 717 patients, of whom 33 were pregnant, came to the same conclusion.

In Malta, during the period May - December 1950, 663 wives of servicemen either became pregnant or were delivered of a child. /

child. Of these women, four developed poliomyelitis; one in the second month, two in the fourth month, and one in the seventh month of pregnancy. They all had normal pregnancies and labour and were delivered of normal children. There were 7 other cases of poliomyelitis in adult women in my series but only one of these was married. Thus eleven of my patients were adult women of whom five were married; four of these were pregnant at the onset of their illness. This gives prima facie support to the argument which states that pregnancy is a factor in the onset of poliomyelitis.

CONCLUSION:

Pregnancy, either because of endocrine changes, or through unusual stress, is a factor in the predisposition to poliomyelitis.

VII. TREATMENT

There is still no general agreement on the best means of treating limb and trunk paralysis. The traditional methods of resting muscles in relaxed postures for many months have been revised, especially in America, where Mead (1950) has advised active exercise from the earliest days of the disease and has used progressive resistance during short periods of hospital treatment. In this country Russell (1952) has failed to confirm that deliberately induced fatigue does harm to paretic muscles in the recovering stages of poliomyelitis. The resting of weak muscles ceases to serve any useful purpose after the early weeks of the major illness, as the spinal cord cells affected by the virus have probably either disappeared or recovered (Howe and Bodian 1942) within one month of the onset of the major illness.

In Malta in 1950 each case was seen, as early as possible after diagnosis, by an orthopaedic surgeon who thereafter supervised the progressive stages of orthopaedic treatment. The basic considerations underlying the principles of treatment were these:-

(i) Damage to the motor neurones is known to take place very rapidly in the earliest phases; extensive damage is already present before the onset of manifest paralysis, and evidence of irreversible degeneration /

degeneration or even neuronophagia may be present within 24 hours of the appearance of paralysis. Motor neurone destruction may continue to take place during the acute phase of the disease (Bodian 1947).

(ii) The only method of proven value in limiting the extent and severity of the paralysis is physiological rest in the early and pre-paralytic stages (Russell 1947, 1949).

(iii) On general principles, it seemed reasonable to assume that rest in the early stages of the paralytic phase might possibly limit the extent of permanent neuronal damage. Strict proof of this is difficult to obtain from this series but it seemed to be a reasonable assumption.

(iv) Mere rest in bed, as usually understood by the average patient, is not enough; it is essential to provide adequate sedation and to enforce a limitation of motor activity.

(v) Recovery is a spontaneous process, when it occurs, and some degree of it almost always follows the acute stage.

(vi) Every patient who was admitted complaining of one or other of the prodromal symptoms was strictly confined to bed and carefully observed. As soon as it became apparent that a patient was in the pre-paralytic phase /

phase of poliomyelitis he was told to limit his movements and to rely solely on the nursing staff for his slightest requirement. A limb which showed any degree of muscle weakness was immobilised in the position of election in a padded wire splint of light construction. Further to increase the amount of rest the patient was given continuous sedation; but this was of course not done in any case showing the least sign of respiratory difficulty. Once the diagnosis was established, routine daily examination of motive power was omitted.

Muscle 'spasm' was treated by the application of local heat, either by heat-cradle or hot-packs. Passive movements were carried out daily but no attempt was made to stretch a muscle that was in 'spasm'. The type of 'spasm' noted was, in most instances, a heightened response to stretching; it was often accompanied by pain. In view of this, and of the uncertainty as to whether the myoneural junction is at all affected in poliomyelitis (Buchthal 1949), it was considered inadvisable routinely to stretch muscles without preliminary relief of hyperexcitabil-:ity.

There has been much discussion of the term 'muscle spasm' /

spasm' in poliomyelitis. It has often been used to describe various types of muscle disfunction. Pohl and Kenny (1943) considered muscle spasm to be a decisive factor in the course of poliomyelitis. Pollock et al. (1949) denied the existence of muscle spasm, which they defined as a state of sustained involuntary tetanic contraction of muscle, except the muscular spasm associated with the meningeal reaction in the meningeal stage of the illness, but Bouman (1947) explained 'spasm' by assuming a replacement of the single neuron reflex arc, normally responsible for myotatic activity, by multineurone arcs possibly associated with the disappearance of inhibitory impulses. Mitchell (1952) tried to resolve the controversy by describing the various clinical find-:ings which are collectively described as 'spasm'. He considered that the limitation of straight leg raising and shoulder abduction were due to pain arising from residual inflammatory changes in the region of the dorsal roots or meninges. With this I am in agreement since such limitation is observed quickly to resolve soon after the acute phase of the illness is over. Mitchell also considered that the greater resistance to stretch found occasionally in muscles /

muscles such as the biceps and the calf muscles could not be explained by a guarding action alone, although pain always co-existed. In the series here observed this sort of resistance to stretch, accompanied by pain, was observed in three cases, as follows.

The first, (case 4 on page 35) was nursed in a Both respirator for 6 weeks and selective positioning of the upper limbs, which were severely paralysed, was clearly impossible; spasm of the flexor-adductor groups of the arms occurred.

The second, a girl of 4 years (case 5 on page 36) developed resistance to stretch of the calf muscles; this was attributed to the difficulty in getting co-operation from the patient during physiotherapy in all stages of the illness, and the development of continued overaction of a stronger over a weaker muscle group.

The third was a Royal Army Medical Corps nursing orderly who developed increased paralysis of one limb in the 5th week of illness, long after the acute stage. In all these cases the pain occurred only on active or passive movement of the affected limbs.

Muscle spasm was not a problem except in the three cases mentioned above; this is the normal finding in cases /

cases treated from the onset with rest, sedation, selective positioning of affected limbs and passive movements.

Considering the number of cases and the staff available to treat them, the scheme outlined is thought to have been rational. Although splinting of paralysed limbs is not now fashionable the results achieved, I think, were good, (table V). I think there is still a place for the kind of splinting described. It was by no means rigid in the sense that it was maintained without daily passive movements; and it was dis-:pensed with at the earliest moment and as soon as active movements were prescribed by the orthopaedic surgeon. Active movements, with support from sling and spring, were begun as soon as the acute phase of the disease was over, and continued, with gradual increase of activity, as long as the patient required them. It was not found expedient to force the pace with active exercises and certainly no muscle was made to act to the point of fatigue. In two instances, where by over-enthusiasm on the part of the patient, rather too much exercise was taken, the muscle improvement was halted, and in one case regressed, as judged by the weekly objective assess-:ment /

assessment of muscle power. This is not in keeping with Russell's (1952) finding that fatigue does no harm to paretic muscles.

The padded wire splints which were used in the acute phase of the illness were, in effect, an attempt to place the limbs in the position of comfort such as might also have been achieved, with less effect and the more certainty of loss of optimum position, with pillows.

The results of treatment are shown in figure IV. Other figures for early recovery of paralytic cases are not strictly comparable with this relatively small series. Seddon, Hawes and Raffray (1946) quote 31.4 per cent. of early recovery in a sample of 500 cases in the Mauritius epidemic of 1945 in which the age distrib-:ution was of the infantile type, and Lenhard (1947) found that 76 per cent. of 405 cases in Maryland made what he called a good recovery (table V).

SUMMARY:

The basis of the treatment of the acute stage of polio-:myelitis is presented and the meaning of muscle spasm is discussed. The results of treatment are shown and roughly compared with those of other epidemics.

CONCLUSIONS: /

CONCLUSIONS:

1) Although methods of treatment of the acute phase of poliomyelitis vary in detail, rest of paralysed muscles with daily intermittent passive movement is likely to give the best results.

2) Return to active movements, under expert supervision, as early as possible is advocated.

3) Muscle fatigue should be avoided at all stages of treatment, as physiotherapy too vigorously pursued may retard recovery of muscle power.

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TABLE I

DISTRIBUTION OF CASES IN THE THREE SERVICES

Service	Officers	Other Ranks	Wives	Children	Civilians Employed by Services	Total
Army	4	15	2	2	3	26
Navy	3	9	3	8	-	23
Royal Air Force	-	1	-	1	-	2
Totals	7	25	5	11	3	51

TABLE II

AGE GROUPS

Age Groups in Years	0-4	5-9	10-14	15-19	20-29	30-39	40-49	Total
Number of Cases	7	3	1	9	20	9	2	51
Percentage	13.9	5.8	1.9	17.6	39.2	17.6	3.8	100

TABLE III

CALCULATION OF INCUBATION PERIODS

Case No.	Date of onset of illness	Incubation Period (days)	Severity
1	16th August 1950	18	Severe
2	18th " "	20	Moderate
3	20th " "	22	Mild
4	20th " "	22	Mild
5	21st " "	23	Mild
6	28th " "	30	Non-paralytic
7	30th " "	32	Mild

TABLE IV

CEREBRO-SPINAL FLUIDS

Case No.	Day of L.P.	No. of cells per cubic m.m.	Protein (mgm per cent.)	Remarks
1	3	L.2 RBC 34	75	Moderate case.
3	3	L.3	120	Died within 36 hours of admission. Fulminating case.
4	4	L.5	50	No residual disability. Right arm principally involved. Mild case.
5	2	L.6	50	Right facial and lower limb weakness. Mild case. Full recovery.
7	6	L.12	100	Moderate case. Good recovery.
12	5	L.9	45	Non-paralytic.
24	6	L.9	50	Non-paralytic.
29	3	L.72 Few P.	60	Meningeal type with marked head retraction. Minimal weakness. No residual disability.
33	5	L.220 Few P.	50	Meningeal type. Non-paralytic.
35	5	L.560	170	Mild case. Minimal left leg and right facial weakness. No residual disability.
36	4	L.380 Few P.	120	Non-paralytic.
37	3	L.120 Few P.	55	Non-paralytic.

TABLE IV (Continued)

Case No.	Day of L.P.	No. of cells per cubic m.m.	Protein (mgm per cent.)	Remarks
38	3	L.3	75	Marked paresis of legs.
	9	L.3	45	Severe case.
39	2	L.52 Few P.	110	Marked paresis of legs. Severe case,
40	1	L.3	85	Paresis and weakness of legs. No residual disability. Mild case.
41	3	L.123	40	Meningeal type. Slight generalised leg weakness. Mild case with no residual disability.
42	3	L.3	65	Marked arm and leg weakness but no residual disability.
43	6	-	85	No residual disability. Mild case.
44	3	L.23	55	Died in mechanical respirator 3 days after admission.

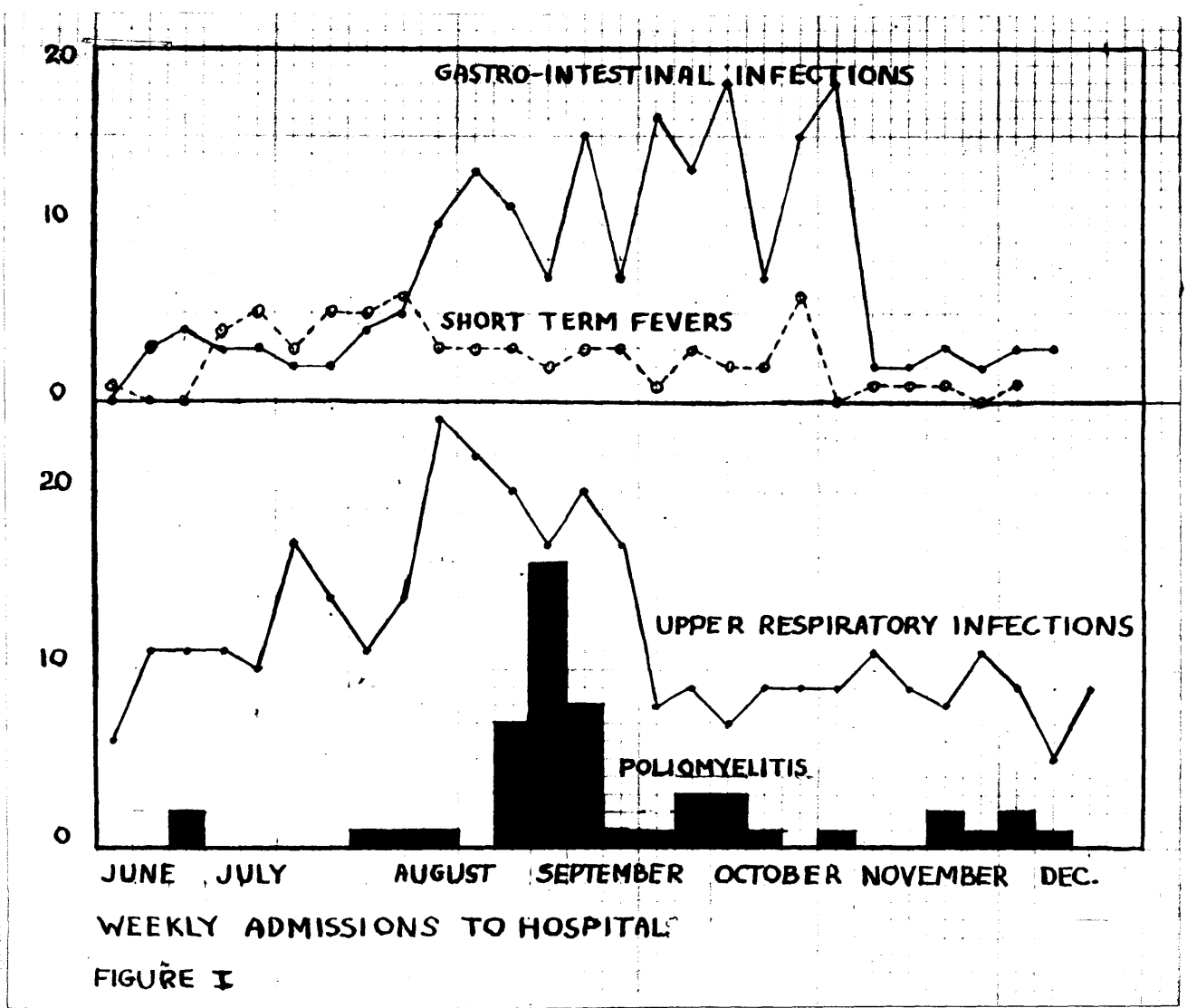
N.B. CHLORIDES AND SUGAR SHOWED NO DEVIATION OUTSIDE THE NORMAL RANGE.

L = LYMPHOCYTES P = POLYMORPHONUCLEAR.

TABLE V

COMPARISON OF DEATH AND MORBIDITY RATES PERCENT

	Malta	Mauritius	Maryland
Deaths	4	5.8	4
Poor recovery	18	19.8	20
Good recovery	78.4	74.4	76



WEEKLY ADMISSIONS TO HOSPITAL.

FIGURE I

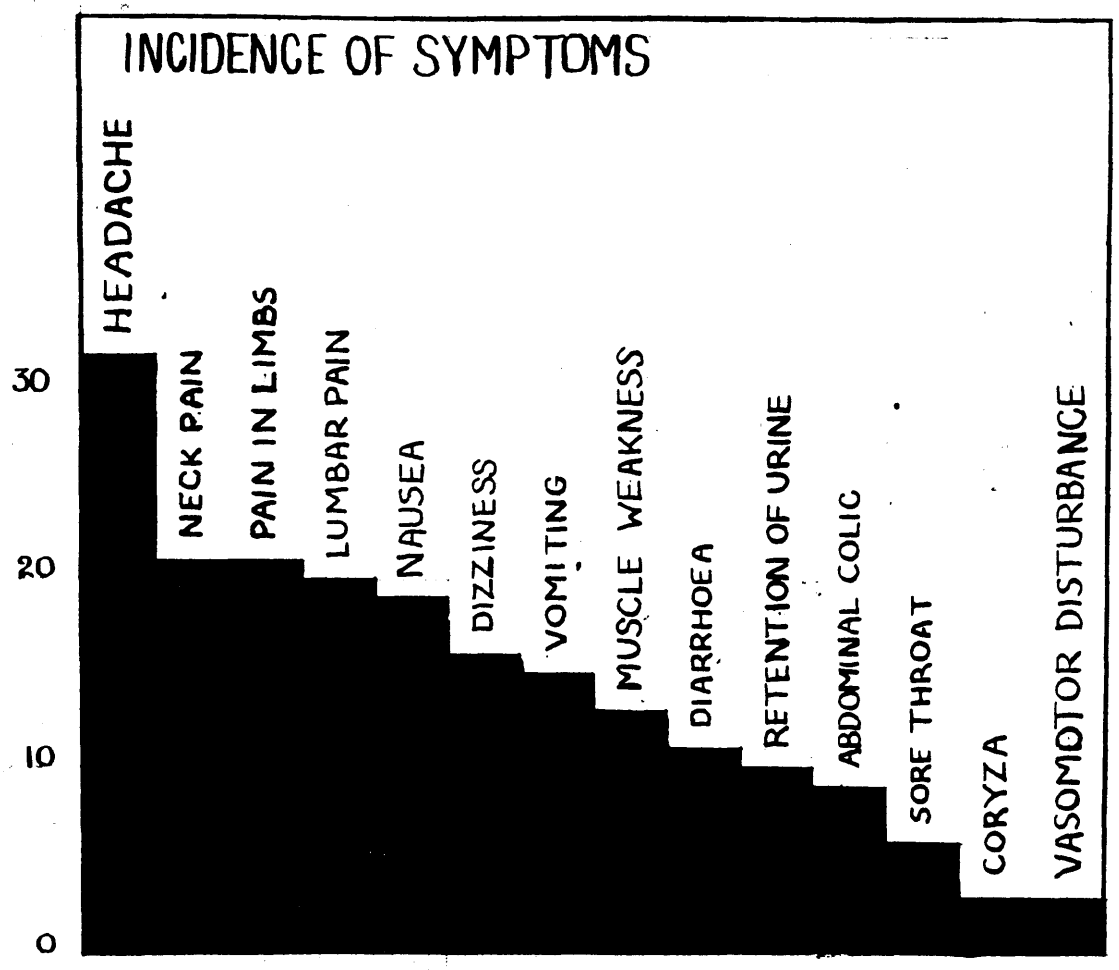

INCIDENCE OF SYMPTOMS
30
20
10
0
HEADACHE
NECK PAIN
PAIN IN LIMBS
LUMBAR PAIN
NAUSEA
DIZZINESS
VOMITING
MUSCLE WEAKNESS
DIARRHOEA
RETENTION OF URINE
ABDOMINAL COLIC
SORE THROAT
CORYZA
VASOMOTOR DISTURBANCE

FIGURE II

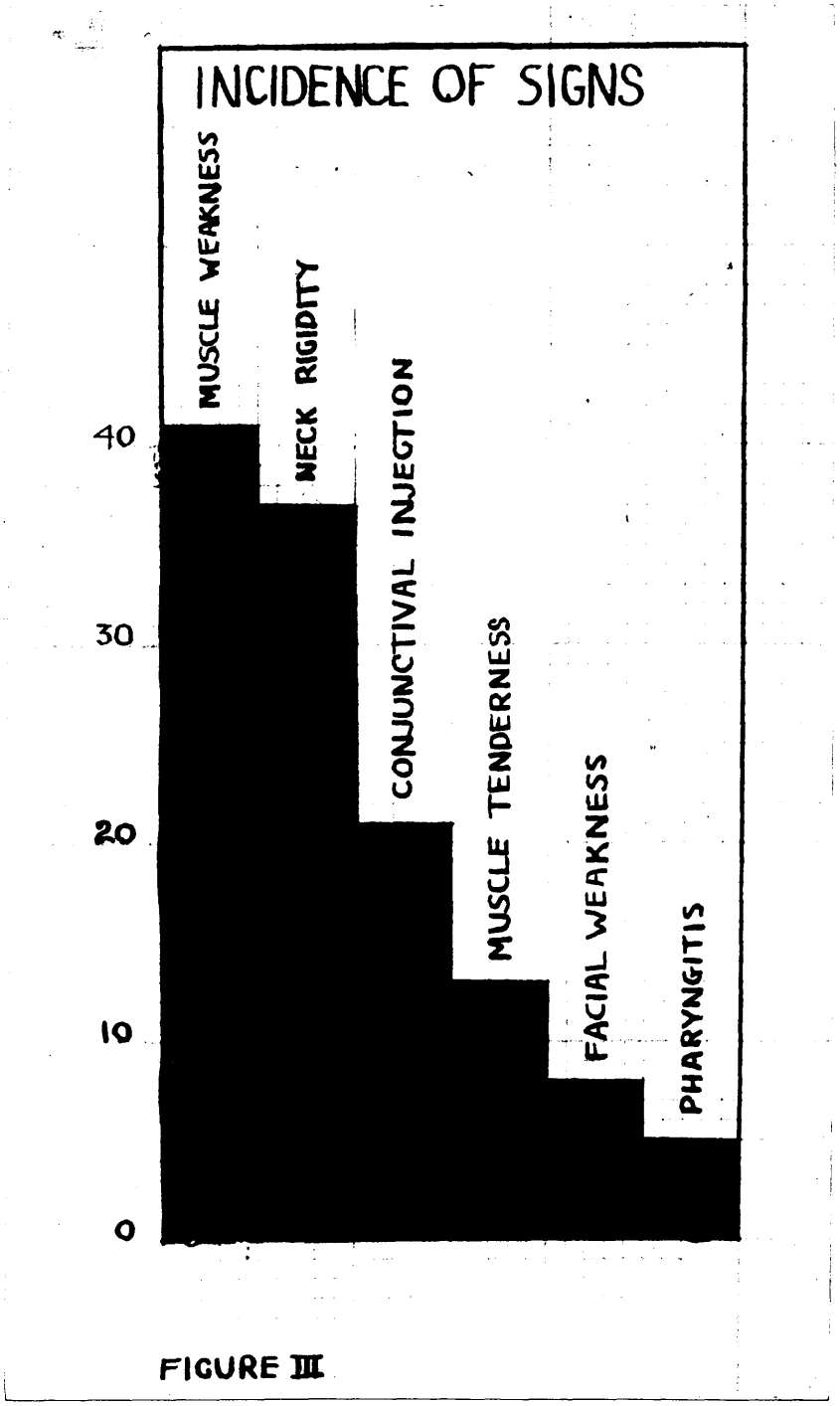

INCIDENCE OF SIGNS
MUSCLE WEAKNESS
NECK RIGIDITY
CONJUNCTIVAL INJEGTION
MUSCLE TENDERNESS
FACIAL WEAKNESS
PHARYNGITIS
40
30
20
10
0

FIGURE III

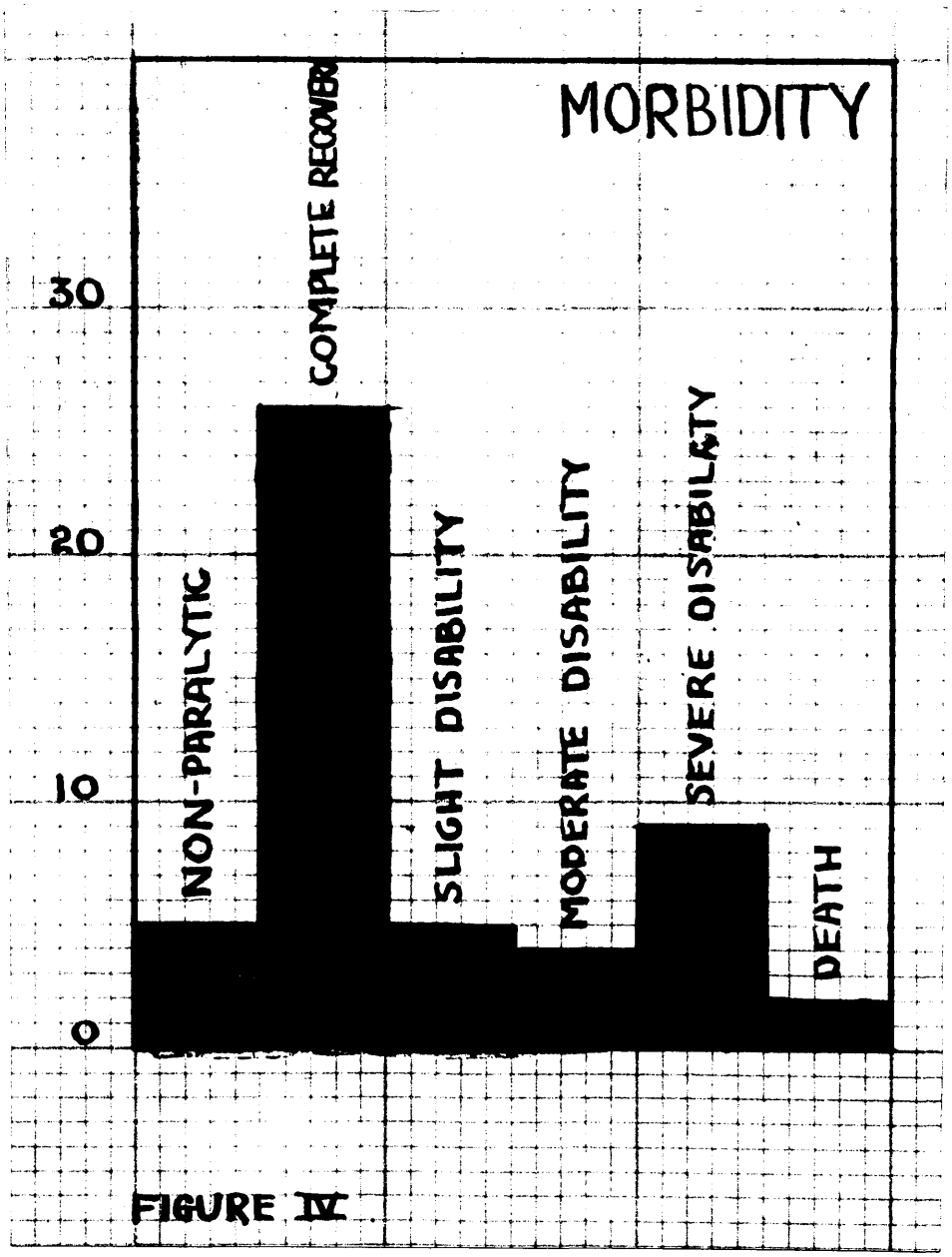
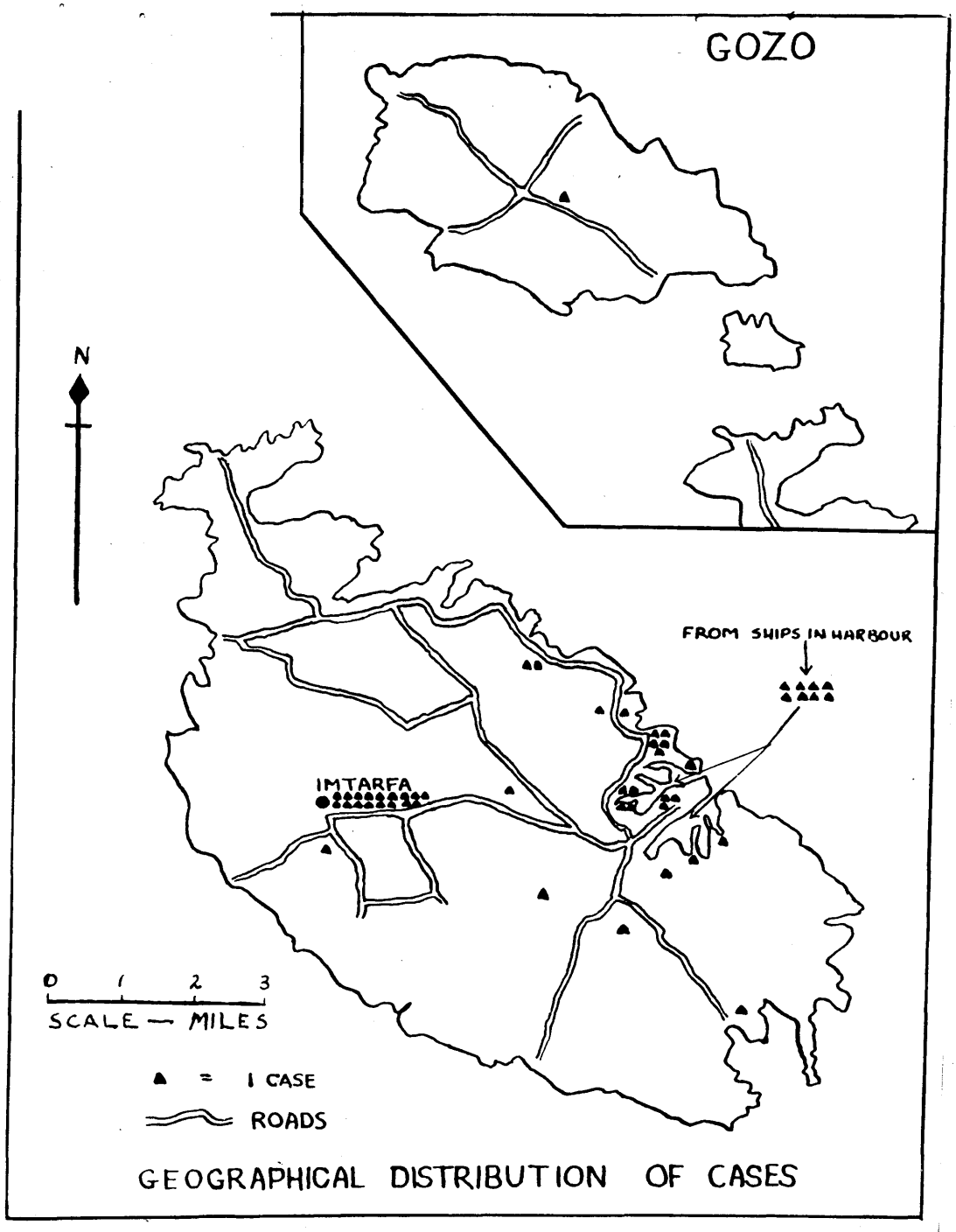

MORBIDITY
30
20
10
0
NON-PARALYTIC
COMPLETE RECOVERY
SLIGHT DISABILITY
MODERATE DISABILITY
SEVERE DISABILITY
DEATH

FIGURE IV



GEOGRAPHICAL DISTRIBUTION OF CASES