POLIOMYELITIS PROBLEMS FROM AN EPIDEMIOLOGICAL POINT OF VIEW[1]

By

PETER M. HOLST, M.D.,
Professor of Epidemiology

I have accepted the invitation of the President to read a paper at the opening of this conference with great pleasure, not only because it is an honour to speak in such an assembly, with so many prominent Scandinavian colleagues present, but also because it allows me an opportunity of pointing out some of the problems which constantly arise during the daily treatment of poliomyelitis patients in my department.

Poliomyelitis first became a social problem with the great epidemics during the first decades of our own century. These epidemics brought the disease to the forefront and stimulated an intense research which was initiated on a very high level by the Norwegian *Chr. Leegaard,* but where otherwise the Swedes carried out the pioneer work.

Epidemiologically the disease is most characterised in the 20th century by a series of epidemics which especially in the last 15 years have steadily become more frequent and intense.

TABLE 1

Average number of poliomyelitis cases per year. Norway.

	Cases	Dead
1904–05	515	61
06–10	167	22
11–15	620	97
16–20	119	22
21–25	180	25
26–30	148	26
31–35	175	30
36–40	264	30
41–45	743	122
46–50	615	78
51	2217	

[1] Paper read at the Orthopaedical Congress in Oslo, 13.6.1952.

Similar developments have taken place in a number of other countries, for instance in the U.S.A., where 42,000 cases were reported in 1949, twice as much as the average number for 1942–48. In Europe the average number for 1948–49 was approximately 20,000 cases.

Statistical information on the disease however is unfortunately very incomplete.

It depends upon a number of factors and doubtful sources of information which so far have been difficult or impossible to check.

Where medical aid is unsatisfactory, we have of course correspondingly unsatisfactory information concerning the appearance of the disease. Until recently we believed that poliomyelitis was to be found preferably in the temperate zones, and especially in the western half of the globe. Now it is our opinion that the disease is probably just as widespread in tropical lands and in Asia. The disease appears over the whole surface of the globe. No country, no race escapes untouched. Its victims are found just as easily in the African jungle and on the pampas of South Americans amongst the Eskimos in Greenland and around Baffin's Bay. The prophylaxis and treatment of poliomyelitis has in reality become a world problem.

Another and still more important reason for our incomplete data about the disease is that its manifestations are often so slight that, generally speaking, they cannot be diagnosed. Under our conditions, certainly the great majority of paretic cases are diagnosed and reported. But when pareses disappear almost as quickly as they have come, and above all when no pareses develop at all, as in the non-paretic cases, statistical information will be very imperfect. The frequency of the non-paretic forms seems to vary greatly from place to place. During the 1934 epidemic in Denmark only about 15 % of approximately 4000 cases reported had pareses. Here in Norway we have never experienced such a large proportion of non-paretic cases. The Oslo epidemic of last year was quite exceptional since we admitted, in addition to 283 paretic cases, 156 cases of serous meningitis which may certainly be regarded as belonging to the poliomyelitis category.

Furthermore as far as the clinical diagnosis of the non-paretic cases is concerned we lack the means to distinguish between these and other, etiologically quite different forms of virus meningitis. Even if a virus laboratory is at one's disposal the differential diagnosis is not yet so advanced, I think, as to possess a practical significance. An example will illustrate our difficulties in this respect.

Simultaneously with the poliomyelitis epidemic during the summer and autumn of 1951 we had, as I mentioned above, an epidemic of

serous meningitis in Oslo. This is an extremely unusual occurrence, which has never before been recorded in Oslo. Since serous meningitis is not subject to medical report we do not know the actual number of cases but only the number of those admitted to hospital. The two epidemics followed an almost completely parallel course from week to week. Also the age distribution of these patients exhibited a striking resemblance.

The correspondence between the two epidemics was on the whole so great that we have no reason for doubting, on the basis of our present knowledge, that the serous meningitis was in actual fact poliomyelitis.

Here in Oslo quarantine is a part of the poliomyelitis treatment. As a rule this means isolation in hospital, for 3 weeks after the onset of the disease. Oslo has a fever hospital with about 400 beds, i.e., one bed to every 1000 inhabitants. But as in so many of the older hospitals we very much lack single rooms, where the patients can be isolated individually. A case of serous meningitis must be individually isolated as long as the etiological diagnosis is questionable. When no pareses occur, I cannot exclude the possibility of a different etiology and, quarantine must therefore be continued in an individual room. Owing to the deficiency of single rooms, after a few days I have usually been compelled to send the patients home even though I was convinced that almost all cases of serous meningitis were in reality poliomyelitis cases. In this way I have knowingly and wilfully deprived medical statistics of a large number of poliomyelitis cases. The official report about the occurrence of the disease in Norway during 1951 will therefore remain incorrect until the end of time.

But from an epidemiological point of view it does not matter even if we do succeed in recording these cases as well. The research of recent years has made us aware that the clinical forms of the disease comprise only a small fraction of the actual total of infections.

With the aid of virological methods, animal experiments and the compliment fixation test it has been proved that during an epidemic infection is practically omnipresent. A great number of persons are infected, but they do not fall clinically ill. We now calculate that for every certain clinical case there are at least 100 sleeping infections.

Even if research has solved several of the old problems it has by way of exchange created new ones. New barriers hem us in, where we had hoped to find a clear prospect.

Poliomyelitis does not originate from one distinct virus. We know of at least 3 different types of virus which can produce the disease. Besides these, which are generally named Lansing, Leo and Brunhilde,

others are probably in existence. Which type or types are the cause of the disease here in Norway we do not yet know. Another problem which so far has also been wrapped in mystery is the immunity question. Although we are acquainted with various cases of re-infection we consider that infection will more often than not give immunity. Is this immunity specific to the type or the genus? In other words, will infection with Lansing virus provide immunity against Leo and Brunhilde as well, or is there a risk of becoming ill again if infection occurs through another type?

I have already mentioned that there is a general susceptibility to poliomyelitis. No age group and no race escapes. But susceptibility varies greatly between age groups. And it seems to differ with the passage of time. The disease is no longer such an outstanding childhood affection as before. In some places older age groups are just as strongly or even more strongly attacked than children in their first years. In Sweden *Kling* has calculated that the poliomyelitis morbidity for children up to 5 years was 180 per 100,000 in 1911, but only 60 in 1936. Here in Oslo, however, we observed last year that the age group 0–5 years is far more vulnerable than the older age groups. The age group distribution of Oslo in 1951 in this respect almost corresponds with the curve from Stockholm, Gothenberg and Malmö during the period 1911–13.

TABLE 2

Age distribution of the patients. Oslo 1951.

	Poliomyelitis	Serous meningitis
	283 cases	156 cases
0–10 years	48.1 %	64 %
11–20 „	12.6 %	11.3 %
21–30 „	18.7 %	14.8 %
31–40 „	15.4 %	4.5 %
> 40 „	5.2 %	6.4 %

During the very same epidemic the disease makes an attack of greatly varying intensity in different places, and even within the same city boundaries considerable variation can often be witnessed. We had a typical example of this during the Oslo epidemic of 1951.

This morbidity cannot be explained by differences in age distribution amongst the population of the respective parishes of the city, nor by differences in housing conditions.

Our knowledge of the spread of infection has been greatly increased thanks to the research of the last few decades in, amongst other countries, Sweden and the U.S.A.

The patient is an important supply centre for the virus. Even before

TABLE 3

Poliomyelitis in Oslo, 1951.

Showing the number of paretic cases per 10,000 inhabitants distributed amongst the parishes within the old city boundaries.

Gamle Aker	1.6
Gammelbyen	2.5
Kampen	3.5
Grønland	3.7
Markus	3.9
Frogner	4.3
Jakob	4.6
Petrus	5.3
Fagerborg	5.8
Vår Frelser	6.0
Uranienborg	7.0
Torshov	8.2
Paulus	8.4
Trefoldighet	8.5
Sagene	10.3
Vålerenga	14.5

the clinical symptoms are manifested, the virus can be demonstrated in the faeces of 40 % of the patients. According to newspaper information *Bodian* has established the presence of virus in the blood 2 weeks before the disease makes itself known. If this is confirmed we must change our view about the incubation period of the disease. Moreover we know that virus can be excreted through the faeces for a very long period after the beginning of the acute stage. Even if it will more often than not quickly disappear from the upper air passages secretion from the nose in the meantime can carry infection for more than 2 years after the onset of the disease. We must assume that similar conditions also prevail amongst other individuals who are infected even if they do not reveal clinical symptoms of the disease. Furthermore we know that the virus is very resistant and that it can live for a considerable time outside the human organism. It has been found in sewage water both before, during and after an epidemic. Under these circumstances it is not logically possible to think of limiting an epidemic by making use of the very old quarantine measures to which the public health authorities still cling. All our experience argues that the disease does not spread from contact with the clinically sick.

Many believe in transmission of infection through the faeces. They think that the virus in the faeces will infect drinking water and foodstuffs, and that the best defence against the disease may be a very advanced nutritional hygiene. But our poliomyelitis epidemics lack the signs which otherwise denote an epidemic through food infection. I am

convinced that the disease is generally transmitted by personal contact with a carrier of the virus. And such contact cannot be avoided in modern society. Therefore prohibition of public meetings, closing of schools, etc., will be of no avail. It is eyewash which the authorities maintain in order to calm the public. The attempts made to counteract the disease by active immunisation have been followed with interest but these have not hitherto produced practical results.

Our attempts to influence the disease in its acute stages have not been crowned with success. The serum therapy has been a great disappointment, and even if what the newspapers inform us is correct, that *Bodian* has been able to influence the course of the disease during the incubation period, it is hard to understand how this can prove of much practical significance. Antibiotics have no effect upon the poliomyelitis virus.

Our therapy therefore is completely symptomatic, but none the less of great value for many of the patients.

The first task of importance is, if possible, to reduce the mortality rate of the disease, which comprises 10–20 % of the paretic cases. These fatalities are due in the great majority of cases to respiratory paralysis of bulbar or spinal origin. By means of artificial respiration life may often be prolonged and in some cases the patient may be so much benefited that he will obtain the chance to take up again a more or less normal life. In order to achieve this, various types of mechanical respirators have been constructed. Also in this field, the Swedes have led the way.

The results of the respiratory treatment have not fulfilled our expectations, however. Many of the patients in the respirator avoid a certain amount of suffering, and this is a great advantage. For many, life is prolonged to some extent. But the majority of our respirator patients have died in the respirator. Those who are forced to remain in the respirator for months or years fare particularly badly. Of the few who can finally breathe without external aid, only a fractional number recover to such an extent that they can again enter upon a fairly normal life. The remainder are doomed to live as invalids and are usually very susceptible to infection of the air passages. The mortality rate of about 140 patients treated in the respirator in my department was approximately 85 %. In recent years laryngological aid has been employed to an increasing degree in the treatment of these patients. We too have had remarkable evidence showing that tracheotomy and bronchoscopy with aspiration can help some of these patients. But fundamentally I do not believe that laryngology alone can improve the prognosis of these patients.

As far as other aspects of the treatment are concerned Norway has long been numbered amongst the under-developed countries.

Until 1936 a rational and expert treatment was practised only at the neurological department of the Rikshospital and in the Foundation for the Crippled. Moreover, since these institutions only had an extremely limited capacity, patients were as a rule referred to the general internal medical departments, if indeed they received any treatment at all. Owing to the Oslo epidemic of 1936 the city authorities had to create a special department for poliomyelitis treatment in Ullevål hospital. Here patients for the first time in Norway obtained specialist neurological treatment in the acute stage of the disease. After this the interest in poliomyelitis treatment steadily grew. A national association was formed to combat poliomyelitis and this now has more than 10,000 members. The association now owns and administers 2 special institutes and several more of the same type are planned and partly under construction.

Even if there are still many poliomyelitis patients who have not received the special treatment desirable, and even if we often still see that our patients wish to be sent abroad sooner or later during their treatment, particularly to Denmark, because they believe that they will have a better chance of being cured there, nevertheless it is probable that we shall soon be able to say with a good conscience that also here we do everything that can be done for these cases.

Personally I have no qualifications for guiding the treatment of poliomyelitis past the acute stage. But I have had so many dealings with these patients, that I have formed my own opinion about the treatment. As this is not at all standardised and because I do not think that it ought to be standardised, I hope that I may be pardoned if I trespass on ground which, strictly speaking, is not my own.

Here in Norway we hand over the treatment in the first place to our neurologists. In Sweden it is the responsibility of the orthopaedic surgeons and in Denmark of the physical treatment experts.

Personally I think that a close and periodical neurological check of the patients is a conditio sine qua non, whatever the treatment. It is difficult enough to judge the effect of our therapy even with such a check. It is quite impossible without it. But in my opinion it is not necessary to master neurology in order to evaluate the functional disturbances which characterise poliomyelitis. Any doctor can learn this and it is especially easy for orthopaedic surgeons and physical treatment experts who in so many ways have contact with the physiology of the muscles. I therefore believe that poliomyelitis treatment can be administered equally well by orthopaedic surgeons and physical

experts as by neurologists. For me it is most natural that the physical experts should take charge of the treatment because they are perhaps the most familiar with the therapy which we employ during early treatment. But the question is scarcely of any great importance.

Neurological and orthopaedical control of patients must continue for years even with the slight cases which can be discharged early from the hospitals. If not, many unpleasant surprises may be experienced in the form of contractures when patients are only seen again after the lapse of a considerable time. From what I can understand this control is far more developed in Sweden and Denmark than in this country. It would be very interesting to hear what results this has produced.

It is of the highest practical and economic value to decide how much physical treatment the individual patient and individual muscle is to have. As I understand it, we have not proceeded very far in this field.

The general and obvious rule is that the treatment must be carried out with the greatest care. The muscles must not be over-strained. In practice the method followed at our hospitals is that physiotherapy is prescribed in individual cases. This means that the patient daily has only from 20–40 minutes' treatment. This has become a necessity in Norway, where physiotherapists are at a premium. But it is surely not a good principle: ideally, treatment should be extremely individual so that its duration, with doctor and physiotherapist acting in close cooperation, is increased to the maximum that can be borne. Similarly with exercises in water.

But when shall we end the treatment? or rather, when shall we end the physical treatment? This is a problem which is of great urgency in those hopeless cases where after some months one understands that the patient is doomed to lifelong invalidity, because so many of the most important muscles have been permanently deprived of function. These patients cling to hope more than anyone else as long as the treatment lasts. The problem is often sharply outlined when they seek help from the sick fund and other institutions, so as to obtain medical treatment abroad, treatment which we so often know will not achieve success.

But the question is also urgent where it affects orthopaedic aid to the sick. It is urgent in my own department, where we have dealt with many severe cases in the last 6–8 months, yet we have no facilities for removal to an orthopaedical hospital, where these patients in actual fact belong. Even if the patients have to wait perhaps a considerable time for the right moment to be operated upon, in my opinion the neurol-

ogist and the physical treatment expert respectively have more often than not by this time played out their parts in the treatment. Now is the time for the orthopaedic surgeon to take over complete responsibility for the patient.

Today all experts will agree that a planned and rational treatment can help many patients to a great extent and therefore this is of the greatest importance both for the individual and for the community. But there are very definite limits to what the treatment can yield. It can never revive a muscle when the motor nerve cells are destroyed. It can be definitely ascertained whether or not this is so after 6–8 months. Even in such cases the physical treatment should often be continued since it may be possible to train other muscles to take over the function of the paralysed one. But in other cases the destruction is so widespread that there is no possibility of our doing this. Again the paralysed muscle may have carried out such an important function that it cannot be replaced. If we, for instance, after 6–8 months establish that there is a complete paralysis of m. quadriceps femoris or of m. deltoideus, I personally think that there is not much point in persisting in physiotherapy with these muscles. In such cases we can only aid the patients with splints or along the surgical path.

SUMMARY

In Norway epidemics of p. have occurred with increasing frequency and number of sufferers since 1936. A peak was reached in 1951, when 2217 cases were notified. Oslo (population 400,000) had 283 paretic cases and 156 cases of serous meningitis, which must be supposed to have been p. In the epidemic of 1951 the age group 0–5 years had a comparatively higher morbidity than has been the tendency in many other places during the last few decades. The varying morbidity in the different parts of the city could not be explained.

In author's opinion public health authorities have no means of controlling the disease by quarantine measures, which ought to be abandoned as useless. Since 1936 the municipal hospital of Oslo has treated 140 cases of respiratory paralysis in respirators with a mortality of 85 %. Physical training of the patients has been supervised by neurologists, but should in author's opinion be left to orthopaedic surgeons comparatively early. Too much money, time and energy is spent in prolonged treatment of hopeless cases.

RESUME

En Norvège les épidémies de poliomyélite ont augmenté en fréquence et en nombre depuis 1936. Une pointe a été atteinte en 1951 où 2217 cas ont été déclarés. Avec une population de 400.000 habitants, Oslo a 283 cas de paralysie et 156 cas de méningite séreuse que l'on peut supposer avoir été des poliomyélites. Au cours de l'épidémie de 1951, la mortalité a été comparativement plus élevée dans les groupes d'âges de 0 à 5 ans que les tendances constatées en beaucoup d'autres endroits au cours des dernières décades Il a été impossible d'expliquer les variations de la mortalité dans les différentes parties de la ville.

De l'avis de l'auteur, les autorités de la Santé Publique n'ont aucun moyen de contrôle de la maladie par l'application de mesures de quarantaine et celles-ci devraient être abandonnées comme étant inutiles. Depuis 1936, l'Hôpital Municipal d'Oslo a soigné 140 cas de paralysie respiratoire dans des respirateurs, avec un taux de mortalité de 85 %. L'entraînement physique des malades a été surveillé par des neurologistes, mais devrait, de l'avis de l'auteur, être confié à des orthopédistes relativement tôt.

Il est dépensé trop d'argent, de temps et d'énergie au traitement prolongé de cas désespérés.

ZUSAMMENFASSUNG

In Norwegen sind seit 1936 Kinderlähmungsepidemien in zunehmender Häufigkeit und Stärke aufgetreten. Ein Höhepunkt wurde im Jahre 1951 erreicht, in welchem 2217 Fälle gemeldet wurden. Oslo (Bevölkerung 400,000) hatte 283 Lähmungesfälle und 156 Fälle von seröser Meningitis, die man als Poliomyelitis ansehen muss. Während der Epidemie in 1951 zeigte die Altersgruppe 0–5 Jahre eine verhältnismässig höhere Erkrankungsziffer als dies der Fall in vielen andern Gebieten während der letzten Jahrzehnte war. Die verschiedengradige Krankheitanfälligkeit in den verschiedenen Teilen der Stadt konnte nicht erklärt werden.

Gemäss der Meinung des Verfassers hat das öffentliche Gesundheitswesen keinerlei Möglichkeit die Ausbreitung der Krankheit mittels Isolierungsmassnahmen zu begrenzen. Diese sollte daher als nutzlos verlassen werden. Seit 1936 hat das Krankenhaus der Gemeinde Oslo 140 Fälle von Atmungslähmung in Respirator mit einer Sterblichkeit von 85 % behandelt. Die Übungsbehandlung der Patienten ist von Neurologen geleitet worden, sollte aber nach Ansicht des Verfassers an einem verhältnismässig frühen Zeitpunkt dem Orthopäden überlassen werden. Zu viel Geld, Zeit und Energie wird an die langausgedehnte Behandlung hoffnungsloser Fälle verschwendet.