

**CONTROL**



## PUBLIC-HEALTH MEASURES IN THE CONTROL OF POLIOMYELITIS

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In 1891, Medin<sup>46</sup> described the Stockholm poliomyelitis epidemic of 1887, but at first little attention seems to have been paid by public-health authorities to the possibility that poliomyelitis was an infectious disease. In 1907, Wickman<sup>66</sup> clearly stated that poliomyelitis was a contagious disease spread both by typical and abortive cases and by healthy carriers. Landsteiner,<sup>40</sup> by transmitting the disease to monkeys, proved its infective nature conclusively, and from then onwards publications urging public-health measures to control the disease began to appear with increasing frequency. Flexner<sup>25</sup> advocated quarantine measures which should include both the patient and his attendants, on the grounds that the disease was spread by healthy carriers. He regarded a quarantine period of from three to four weeks as necessary, and emphasized the importance of the safe disposal of nasal, buccal, and other excretions. He also suggested the possibility that flies might be important in the spread of the disease. In the years which followed, numerous publications re-emphasized and expanded these recommendations to include notification of the disease, quarantine of the family, placarding, fumigation, investigations into milk-, water-, and food-supplies to ensure their safety, prohibition of public gatherings, especially of children, postponement of the opening of schools, and restriction of movement into and out of an infected area.<sup>1, 21, 28, 48</sup> Such measures were more or less strictly applied until about two decades ago. There was little or no objective information on the results achieved which would enable an assessment to be made of their value in a given epidemic. Indeed, the general verdict of health officers was that the measures were of very little value, since epidemics of poliomyelitis became more common and more extensive, and in a given epidemic it was not possible to demonstrate that they had had any certain effect. During the last twenty years the enthusiasm with which these measures have been applied has waned, and recently in many countries only lip-service has been paid to the fact that poliomyelitis is an infectious disease. It is true that there have been reports of encouraging results in small outbreaks,<sup>23</sup> but generally most health officers have had little faith in the value of general public-

health measures in poliomyelitis control, although they might apply them for the important purpose of allaying public anxiety.

A great deal of new information has come to light in recent years, and it may be as well to re-examine the place of public-health measures in the control of poliomyelitis in terms of current thought on the epidemiology of the disease.

Important though the advances in knowledge are, there are still many points which remain obscure or unproven. It may therefore be permissible to indulge in some speculation when these uncertainties affect the possible application of public-health measures.

### THE EPIDEMIOLOGY OF POLIOMYELITIS AS IT INFLUENCES THE APPLICATION OF CONTROL MEASURES

The only manifestation of infection with poliomyelitis virus which causes concern is the occurrence of clinical symptoms—particularly paralysis—due to involvement of the central nervous system. If symptomless alimentary infection, or even the minor illness, were the only results of infection, the virus would still be of interest to the epidemiologist but it would have no public-health importance. The history of poliomyelitis suggests that in the first half of the 19th century the clinical disease was rare and largely restricted to infants, while epidemics were almost unknown. However, towards the end of the century—perhaps, as suggested by Burnet,<sup>18</sup> the Stockholm epidemic of 1887 may be taken as the beginning of this phase—a change became evident: epidemics began to appear, and have irregularly occurred since on an increasing scale in more and more countries.<sup>10, 62, a</sup> There are now few countries in the world which have not experienced an outbreak at least as large as the 1887 Stockholm epidemic. The phrase “the incoming tide of poliomyelitis” used by Stowman<sup>62</sup> gives an admirable picture of the situation: the shore is being increasingly covered and the waves are growing larger. If the tide were to change and recede to its position before 1887, there would be little need for action by health authorities.

#### Some Theories Regarding the Causes of the Changes in the Behaviour of Poliomyelitis

The reasons for the progressive change in the behaviour of poliomyelitis since the end of the 19th century cannot yet be said to be fully understood.

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<sup>a</sup> See also the articles by Paul and by Freyche & Nielsen on pages 9 and 59 of this monograph.

However, there is evidence on which working hypotheses may be based. Epidemics appeared after the role of defective sanitation in the spread of intestinal infections began to be appreciated, and they occurred first in those countries in which the new ideas were being most rapidly and effectively applied; that is to say, in those countries in which standards of living were being rapidly improved and in which, as a result, many changes in the pattern of social behaviour were taking place. As other countries have attained similar standards so they too have suffered epidemics of increasing severity. In parallel with this change in the incidence of disease there has been a change in the age-groups predominantly affected; this was observed first in North America, Scandinavia, and Australia, the same regions which first experienced poliomyelitis in epidemic form,<sup>17, 22, 45, 49</sup> the tendency being for paralytic poliomyelitis to appear in progressively older children, adolescents, and even adults. An interpretation of these observations is that poliomyelitis infection is spread in a way that is restricted by the hygienic measures and the changes in social behaviour associated with the attainment of higher living standards; but it is not eliminated, so that primary infection is often merely delayed until an age when the results of infection tend to be more severe and hence more often recognized. There is now a considerable body of evidence to suggest that this interpretation is at least partially correct, although it is difficult to explain all observed facts in this way alone. (These facts are reviewed in other articles in this monograph, notably those by Paul, Gear, and Sabin (see pages 9, 31, and 297), and the appropriate references may be found therein.)

There is yet another explanation, which may also be true since the two explanations are not mutually exclusive (see particularly the article by Sabin); that is that the poliomyelitis viruses may undergo changes which render them more liable to invade the central nervous system and cause paralysis. By analogy with the behaviour of other viruses, such changes would presumably involve mutation. This may have occurred once only, the mutant having been spread elsewhere by man, or, perhaps more probably, mutation may take place from time to time in a number of different places. In the latter event, the varying intensity and severity of epidemics may be connected with several possible mutations associated with various changes in the characteristics of the virus. At present, this explanation must be regarded as tentative, although there is good evidence to establish mutability of virulence of the poliomyelitis viruses in the laboratory, and Sabin & Steigman<sup>50</sup> have produced epidemiological evidence in its favour. However, it certainly provides a possible explanation of epidemics difficult to explain otherwise.<sup>b</sup>

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<sup>b</sup> For example, the 1947 outbreak in Great Britain was at the time by far the most severe ever recorded there, and it cannot easily be explained in terms of any recent change in the host or in the environment.

A change in the neurotropic properties of the virus might occur within the country involved, or the virus might be imported from another country where its altered properties need not necessarily have been apparent should the state of immunity of the population have been sufficiently high. Much research is needed before this explanation can be fully accepted, and success will depend on the development of techniques for the detection of such strain differences in the virus.

### Control of the Primary Infecting Agent

The importance of solving this problem is obvious. If changes in the inherent properties of the virus have not occurred in nature, then it might theoretically be possible to change the calendar back to the early 19th century by ensuring that all infants received orally a small dose of unattenuated living virus,<sup>c</sup> and by ensuring that the virus was so widely spread that no one would escape early primary infection and repeated reinforcing infections. Some infants would undoubtedly develop paralysis, as they did in the 19th century, and deaths would occur, but the overall incidence of both paralysis and death would presumably be considerably reduced in comparison with present figures. However, such empirical methods cannot seriously be considered today.

If, on the other hand, changes in the inherent qualities of the virus have occurred under natural conditions, then it should be possible to induce them in the laboratory and to produce an avirulent virus. Recently, considerable progress on these lines has been made (see the articles by Sabin and Koprowski, pages 297 and 335). With strains of avirulent virus that retain their immunizing power, the safe reproduction of the natural process of immunization may become possible.<sup>d</sup>

### Control of Infection in the Environment

The approach to the control of poliomyelitis which has been considered so far is based on the principle of the widest possible distribution of the infecting agent secured in such a way that immunity is produced in the

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<sup>c</sup> A degree of protection during the first few months of life is derived from maternal antibodies, which were presumably consistently present in the 19th century. Similar protection might be ensured today by injection of immune globulin before administration of the virus.

<sup>d</sup> There is a theoretical danger in this method. A virus that has undergone mutation in the laboratory during cultivation under artificial conditions might, if passed by a natural route in its natural host, regain its virulence. Poliomyelitis virus administered orally is excreted in the stools if alimentary infection results, and this continues for a few days or weeks. Thus, the opportunity might exist for passage of the virus to other persons. The practical importance of this danger will need careful study. The possibility is discussed in the contribution by Koprowski.

host but clinical symptoms develop rarely or not at all. A more conventional approach would be aimed at reducing the amount of infection in the environment. This approach has been practised in the past, as already indicated, and although the general opinion has been that it has been ineffective, in fact it seems to have achieved a limited measure of success. The reason for this apparent contradiction is that in assessing the results achieved by hygienic measures in the past only measures applied in times of epidemics have been considered, whereas similar hygienic measures—although not specifically directed against poliomyelitis—are being continuously applied in well-developed countries. In these countries, which have high standards of living and good hygiene conditions, the results of serological and virological examinations suggest that there is now less poliomyelitis infection than in countries where conditions are more primitive. Unfortunately, although there may be less infection there is more clinical disease, so that as far as poliomyelitis is concerned hygienic measures seem to have done more harm than good.

### **The Influence of Social Factors**

It is, however, by no means certain that improvements in sanitation and hygiene are wholly responsible for this change. There are other factors to be considered involving changes in the biological environment of the infecting agent brought about by changes in social behaviour. For example, if, as is quite possible, or even probable,<sup>14, 17, 27</sup> poliomyelitis infection is predominantly transmitted during personal contacts between children, especially in areas where spread resulting from defective sanitation is reduced, then the age at which the maximum number of infections would occur would be expected to depend on the age at which the number and intensity of such contacts between children were sufficient to make exposure probable. In communities with well-developed social systems of the type associated with Western civilization, families tend to be smaller and external contacts between young children tend to be fewer and more closely supervised than under some other social systems or in less well-developed communities, particularly where there is overcrowding. The number of opportunities for exposure to infection is thus reduced until school age is reached. This hypothesis is supported by the observation that in rural areas, where the dispersed population reduces the frequency of child-to-child contacts, poliomyelitis tends to attack children who are older than those infected in urban areas, where there is overcrowding.<sup>49</sup> It might therefore offer an additional explanation of the present tendency, observed particularly in countries with the Western type of social system, for school-age groups to be most seriously affected; and since poliomyelitis infection produces clinical disease both more frequently and of a more serious nature in older

children than in infants, an increase in the number of recognized clinical cases would result. Thus delayed primary infection due both to improved hygienic practices and to a reduction in child-to-child contacts in the younger age-groups may act by producing more clinical disease in older age-groups. The change in age structure of the population must, of course, also be taken into account.

It is an alarming thought that while, by improving environmental sanitation and hygiene and raising living standards generally, the health worker has in many parts of the world practically eliminated a number of serious epidemic diseases he may at the same time have created a new one. Of course, the credit balance is strongly in favour of hygienic measures and high living standards, but we cannot be satisfied until we have eliminated any deleterious effects of our interference with natural processes and retained only the benefits. There is a warning here of general application: any large-scale disturbance of the ecology of natural processes may be followed by unexpected, and often undesirable, side effects.

### Isolation and Quarantine

However, the adverse effect of hygienic measures on poliomyelitis may well have been partly the result of faulty or incomplete application. A logical consideration of recent advances may enable us to apply them with more hope of success. The relevant facts will be found in other articles in this monograph and in the first report of the WHO Expert Committee on Poliomyelitis,<sup>68</sup> and only a brief summary is needed here.

Poliomyelitis is a highly infectious disease. The virus appears to enter the body by the mouth, either in the course of intimate association with infected persons who are shedding the virus in pharyngeal secretions or in faeces, or as a result of environmental contamination (including food and water) with infective faecal material directly, or indirectly by flies which have fed on contaminated material. The relative importance of these methods of spread evidently depends on the environment. The great majority of infected persons show no clinical evidence of infection. Its presence in them can be detected only by isolation of the virus in the laboratory or, indirectly, by demonstrating a rise in serum antibodies. The ratio of symptomless infected persons to clinically diseased patients varies at different ages<sup>47</sup> and under different circumstances. In epidemic areas, as already indicated, it appears to be less than in endemic areas; a ratio of 100 to 1 has been suggested for the former<sup>20, 38, 61</sup> while in endemic areas, judging by the early age at which the great majority of children can be shown serologically to have been infected<sup>32, 50, 57, 58</sup> and the comparative rarity of clinical disease, the ratio must be much higher, perhaps 1,000 to 1 or even more.

In these endemic areas in tropical and subtropical regions, poliomyelitis is, in general, still behaving in the pattern of the 19th century. There are only relatively few cases of clinical disease among very young children, and epidemics are rare and small in size when they do occur. Since there are many much more pressing problems in these areas, it is doubtful whether at the present time much effort can or should be expended on the control of poliomyelitis. It may nevertheless be anticipated that the situation will change in the future as social and hygienic conditions improve and, as described in the articles by Gear and by Freyche & Nielsen in this monograph (see pages 31 and 59) there are signs that this is already happening in some parts of the world.

In epidemic areas the number of inapparent infections, and probably also the proportion of inapparent to overt infections, varies greatly at different times. In times of severe epidemics inapparent infections may be numerous. During a severe urban epidemic, for example, with an incidence of clinical disease of 100 per 100,000 population, on the basis of a 100 to 1 ratio it is possible that there may be as many as 10,000 inapparent infections per 100,000 population. Too much cannot be expected from hygienic measures in such circumstances. However, in other circumstances the number of silent infections, and probably the proportion also, may be much less.<sup>51, 63</sup> There is increasing evidence that early in an outbreak, in inter-epidemic periods when only isolated cases occur, and even during some outbreaks in highly developed communities, infection may be rather sharply restricted to close associates of clinical cases.<sup>15, 16, 26, 47, 52, 53</sup> The proportion of silent infections will be dependent on the number of close associates of each case; and in rural areas and isolated communities, especially, the number is likely to be relatively small. Under these circumstances the systematic application of hygienic measures would seem to be more hopeful. Thus, while, on the one hand, experience has shown that during extensive epidemics the application of measures such as isolation and quarantine in the area involved does not lead to detectable results, even though presumably a number of infections and some clinical cases are prevented; on the other hand, if such measures were applied early in the poliomyelitis season before an epidemic has developed, both in areas where cases have occurred during inter-epidemic periods and in rural areas among relatively isolated communities, they might be expected to have a real effect on the incidence of the disease during the subsequent epidemic season. This may be regarded as a logical extension of the application of general sanitary measures which, as already noted, seem to have reduced the amount of poliomyelitis infection in highly developed communities, even though they have not hitherto been applied specifically against poliomyelitis.

### The Essential Role of Active Immunization

However, as has already been pointed out, should these measures be effective, for every clinical case prevented a natural inapparent immunizing infection would also be prevented in a much larger number of other persons. Primary infection in these persons would therefore be further delayed and when it did occur—which at present seems almost inevitable—the effects would tend to be more serious. In the absence of specific control measures, such as active immunization, it would seem, therefore, that as far as poliomyelitis is concerned hygienic measures may actually be undesirable—a conclusion already reached on historical grounds.<sup>e</sup>

Furthermore, since there is a possibility that different strains of poliomyelitis vary in their tendency to invade the central nervous system, it would be essential to know in a given epidemic whether or not the responsible virus was particularly neurotropic. If it were strongly neurotropic, clearly every effort should be made to restrict its spread. If it were not, a larger number of persons might benefit in the long run (assuming no prospect of artificial immunization) if its spread was unchecked and they were permitted to experience a natural immunizing infection; for if it could be ensured that the inevitable primary infection was with such a strain, a smaller proportion of persons would develop paralysis. Unfortunately, there is at present no way of knowing whether or not a given strain is particularly neurotropic. The incidence of paralytic cases gives no information on this point without corresponding information being available as to the incidence of inapparent infections. There appears to be little reason to assume on epidemiological grounds that neurotropism is necessarily linked with any special tendency of the virus to spread.

Fortunately this quandary is to some extent resolved by recent developments in research on artificial immunization in poliomyelitis. These are discussed in the articles by Sabin and by Koprowski (see pages 297 and 335). If, as seems probable, a satisfactory method of artificial immunization becomes available, using either an avirulent or an inactivated virus vaccine, hygienic measures to limit the spread of virulent virus would appear to be a necessary part of the control programme. It might, of course, be argued that, if immunization is effective, subsequent natural exposures would be advantageous since they would act as reinforcing doses. This might be true in the majority of instances, but heavy exposure

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<sup>e</sup> In order to avoid any possible misunderstanding, it is stressed that this statement refers here to *poliomyelitis only*, although it may also apply to certain other diseases with a similar ecology. The essential significance of hygienic measures in the control of many diseases of far greater importance than poliomyelitis has been abundantly proved, and they must be continued whatever effect they may have on poliomyelitis.

to a virulent strain or to a strain of an aberrant immunological type might overcome the resistance of a proportion of those persons immunized. Also, it is not yet known what effect active immunization will have on the prevalence of the poliomyelitis virus. If as a result the virus has difficulty in establishing itself in the alimentary tract—and the work of Koprowski suggests that this is possible (see page 335)—it may become relatively scarce within a country, as occurs with the diphtheria bacillus when the proportion of immunized persons is sufficiently high. “Reinforcing” infections would then take place infrequently and, as immunity waned, the population, particularly adults, would once more be in danger, especially from the introduction of virus of high virulence from outside. It would therefore seem to be as wise to control subsequent exposures to the virus antigen by the application of hygienic measures and by the administration of reinforcing doses of vaccine as it is to control the primary experience.

It is concluded, therefore, that as procedures for active immunization become available, they should be supported by measures designed to reduce the amount of infection in the environment.

### **The Future Place of the Laboratory in Poliomyelitis Control**

The possible value of isolation and quarantine under certain circumstances has already been mentioned, but it is evident that their value would be enormously increased if it were practicable to detect the presence and duration of infection in a given individual. This has been technically possible for many years, but the older techniques, based on the isolation of the virus in monkeys, have been too expensive and time-consuming for routine use. The developments which have followed the introduction of tissue-culture techniques (see the articles by Enders and by Rhodes et al. (pages 269 and 237), promise to provide a means whereby such routine examinations may become practicable. At present, the techniques are hardly sufficiently developed, and it is certain that the number of laboratories with trained staff able to carry them out is totally inadequate to permit of any general application of the procedure. However, progress is rapid and it may be anticipated that practical procedures will become available in the near future. It is clear that, if they are to be applied, many more laboratories will be needed and many more virologists must be trained. Such development of laboratory services will necessarily take a number of years and should therefore be begun without undue delay or the health officer will be unable to take advantage of the new techniques. It would seem logical to develop such increased virus laboratory services as part of a public-health laboratory service, especially since the public-

health importance of a number of other virus diseases is becoming increasingly apparent as bacterial diseases come under control. Existing research laboratories should not be swamped with routine work for, if they are, progress will cease. However, if developments take place as envisaged, it may become as normal and as easy for the health officer of the next generation to trace infection with poliomyelitis virus as it is for him to trace typhoid carriers today.

### **Control of Non-Specific Factors Influencing the Incidence of Paralysis**

The first method of approach to the control of poliomyelitis already discussed is directed essentially towards control of the characteristics of the primary infecting agent—the dissemination of virus of low virulence. The second is directed towards a reduction in the amount of natural infection in the environment, accompanied by the production of an active immunity in the host by artificial immunization. There is yet a third method of approach directed entirely towards the host, consisting of the control of factors affecting the susceptibility of the host, other than specific immunity, which alter the frequency or the severity of paralysis following infection.

Numerous non-specific factors have been suggested as predisposing to or precipitating paralysis. They include age, genetic factors, physique, endocrinological disturbances, pregnancy, nutrition, other infections, trauma, tonsillectomy, dental extractions, certain injections, over-exertion, exposure, and exhaustion from various causes. The evidence for the part played by some of them is incomplete; the influence of others is well-established. The effect of age at the time of infection has already been mentioned. There is no longer any doubt that on the average children aged between one and five develop paralysis after infection less frequently and less severely than older children, and that the severity of the disease, particularly the incidence of bulbar poliomyelitis, increases further in adolescence and adult life.<sup>41, 47, 49</sup> The position regarding infants under one year of age is less clear. In some countries serious disease is being recorded more frequently than formerly in very young babies,<sup>30</sup> an observation which may be related to the fact that a number of mothers in highly developed countries have no antibodies to transmit to their children and possibly to the fact that generally in these countries a smaller proportion of infants are breast-fed and for a shorter period; poliomyelitis antibodies can be detected in human milk if they are present in the mother's serum, although their importance is still obscure. The change might also be related to an increase in the incidence of infection in adults in these countries, of whom

a smaller proportion are solidly immune. Parents may therefore more often infect their very young children who have few other opportunities for exposure. Apart from the general deductions which have already been drawn from this effect of age at the time of primary infection, these observations make it clear that in highly developed communities it is not justifiable to assume that very young babies are protected by maternal antibodies; the relatively low incidence among them may be more a function of the reduced opportunities for exposure. Furthermore, in these communities adults should not lightly expose themselves to infection, since one in ten or more may still be fully susceptible. That this is a real danger is borne out by the increasing number of both nurses and parents who have been infected and have developed severe paralysis while nursing children who may have suffered only a mild or abortive attack. Since it may be anticipated that the proportion of susceptible adults will tend to increase in the future, it may become necessary to measure the state of immunity of nurses about to undertake the care of poliomyelitis patients, as is often done for tuberculosis and some other infectious diseases.

The possible effect of genetic factors,<sup>6</sup> nutrition, and physique will not be discussed in detail. Genetic factors are beyond the control of the health officer. The possible effect of nutrition is briefly discussed by Gear in his article (see page 31). If there is an effect, it would seem that malnutrition or an unbalanced diet has a favourable effect. However, direct evidence is lacking and there are other more convincing explanations of the observed facts. As far as physique is concerned, the observations of Draper<sup>24</sup> have not been confirmed by others.<sup>42</sup> Other observers have commented on an apparent increase in the severity of paralysis among athletic types. This may, however, be related more to the tendency of such persons to undertake severe exertion (see later paragraphs) than to their physical constitution per se.

The possibility that endocrinological disturbances may affect the incidence of paralysis derives support from laboratory observations that adrenocorticotrophic hormone (ACTH) and cortisone increase the susceptibility of certain laboratory animals.<sup>60</sup> However, apart from endocrinological changes associated with pregnancy and a suggestive rise in the incidence of the disease about the age of puberty, there is little convincing evidence that this happens in man. It is reasonably well established that poliomyelitis may run a severe course during pregnancy.<sup>5, 8, 19, 37, 44</sup> Whether this is due to endocrinological or other changes is not known. There is a difference of opinion as to whether or not the seriousness of the disease varies according to the duration of the pregnancy. There is some evidence<sup>12</sup> that deaths occur most frequently in the last trimester. The foetus is generally not affected, although abortion may occur, and there is evidence that the infant may be infected at or shortly before delivery.

It is clear that care should be taken to reduce the possibility of exposure of pregnant women in epidemic times. Under some circumstances it might be wise to administer a prophylactic dose of gamma globulin.

The role of other infections in predisposing to paralytic poliomyelitis is not well substantiated.<sup>39</sup> Such an effect would be difficult to prove, especially since most of the infections which have been incriminated are common diseases of childhood. Association by chance would therefore be expected to occur not infrequently. There is perhaps more evidence incriminating pertussis than most other diseases. Nevertheless, on general principles it would seem wise to bear the possibility in mind and to take special care to limit the spread of other infectious diseases when poliomyelitis is epidemic. Another good reason for this is that the early diagnosis of poliomyelitis which is so important may be confused if other diseases are prevalent at the same time.

If the word "trauma" is used in a broad sense covering injuries such as a blow, a fall, a fracture, surgical operations, certain injections, and over-exertion, fatigue, and exhaustion, there is no doubt that it has a pronounced effect on the appearance of paralysis following poliomyelitis infection. The best and perhaps the most important example of this is that exertion at the time of the major illness, when signs of central-nervous-system involvement have already appeared, may cause very severe and extensive paralysis.<sup>36, 56</sup> This is fully discussed in the article by Russell (see page 137), to which reference should be made for further details. The avoidance of this effect is an important public-health measure in poliomyelitis. This may be illustrated by the observation that the case-fatality-rate in patients admitted to hospital was nearly three times higher in patients transported long distances—average of 85 miles (135 km)—than in local patients.<sup>13</sup> Following infection, the avoidance of all exertion may often make all the difference between a non-paralytic and a paralytic illness, and if central-nervous-system involvement has already occurred, it may be a life-saving measure.

In epidemic times it may be justifiable to apply this principle more widely. In infants the minor illness may escape notice altogether and the first suspicion of illness may be aroused only when the major illness begins. Complete rest is then imperative. In adolescents and adults a diphasic illness is less common than in children and the onset of the major illness may be more insidious.<sup>35</sup> Such patients often fight against the illness and try to "work it off", with disastrous results. A public-health measure of real practical value is to educate not only medical practitioners but also the general public in these facts, though it is not easy to do this without accentuating the apprehension with which the appearance of poliomyelitis in epidemic form is viewed.

There have been many reports that bulbar poliomyelitis occurs with undue frequency in persons who have undergone tonsillectomy or adenoidectomy within the previous month.<sup>2, 4, 7, 9, 29, 33, 64</sup> There have been a few reports denying the association, but the general opinion is that the risk is very real and that elective operations should not be performed when poliomyelitis is prevalent. Some evidence has been produced that persons without tonsils, even though they may have been removed years beforehand, are more liable to bulbar poliomyelitis.<sup>65</sup> The association of bulbar poliomyelitis with dental extractions has also been observed.

Other forms of trauma such as blows and fractures have been associated with the appearance of paralysis in the injured limb. A special form of trauma has recently been incriminated, namely, the effect of certain injections.<sup>3, 34, 43</sup> Since the publication of the original papers on this subject, a number of both contradictory and supporting reports have appeared. Space does not permit of a comprehensive review, but it may be said that there is now good evidence that in times of epidemic poliomyelitis intramuscular injection of the adsorbed combined diphtheria-pertussis prophylactic is followed within a month by paralysis in the injected limb more frequently than would be expected by chance. Whether the same prophylactic injected subcutaneously has the same effect is uncertain, since this route is not often used because reactions tend to be more severe. However, Rhodes<sup>54</sup> has found no evidence of an increased incidence of paralysis following the subcutaneous injection of a fluid (not adsorbed) combined diphtheria-pertussis prophylactic. No convincing evidence incriminating other injections has been produced, with the notable exception of arsenicals, bismuth, and mercury.<sup>55</sup> There is highly suggestive evidence that these heavy metals, injected intramuscularly, not only tend to precipitate paralysis in the injected limb, but actually increase the frequency with which paralysis develops. This is a particularly important observation since hitherto it has been uncertain whether injections merely cause localization of a paralysis which would have occurred in any case, perhaps in another site, or whether they might precipitate paralysis in a case which would otherwise have been non-paralytic. It is still not known whether the adsorbed diphtheria-pertussis prophylactic acts in the same way, but clearly this must now be considered a definite possibility.

Fortunately the use of intramuscular injections of these heavy metals has decreased considerably since the advent of penicillin, and there is evidence that penicillin does not act in this way,<sup>31</sup> although this requires further investigation.

However, the possibility of serious interference with diphtheria- and pertussis-immunization campaigns as a result of these observations rightly causes much anxiety. Much capital has been made out of these incidents

by the opponents of vaccination procedures, and sensational reports have appeared in the press totally neglecting the great benefits of properly conducted immunization programmes, which vastly exceed any drawbacks, both in lives saved and in illness prevented. Nevertheless, this is small consolation to the parents of a healthy child who develops paralytic poliomyelitis after an injection. There are various ways of reducing this danger. The first is to avoid the use of the adsorbed combined prophylactic during times of prevalence of poliomyelitis. As recommended by the WHO Conference of Heads of Laboratories Producing Diphtheria and Pertussis Vaccines<sup>67</sup> and endorsed by the WHO Expert Committee on Poliomyelitis at its first session,<sup>68</sup> in times of severe epidemics it may be wise to suspend temporarily all immunizations in the locality affected. If the epidemic is of minor severity the use of the adsorbed combined prophylactic should be avoided, but immunization with the separate prophylactics may be continued. It would appear from the findings of Rhodes<sup>54</sup> that the use of fluid (not adsorbed) combined vaccine by the subcutaneous route is not accompanied by undue risk. Bousfield<sup>11</sup> advocates early immunization as a solution since the incidence of poliomyelitis in infants under six months is low. This might well be expected to reduce the risk during primary immunization; many authorities, however, consider that after early primary immunization an additional reinforcing dose is needed in the second year as well as the usual one at the age of school entry. Other precautions must be taken to reduce the risk at these times.

### SPECIFIC PUBLIC-HEALTH MEASURES IN THE CONTROL OF POLIOMYELITIS

The above review of the epidemiology of poliomyelitis as it affects the application of public-health measures for the control of the disease gives the general background on which the measures to be adopted should be based. Clearly the actual measures to be applied in a given country will depend on the epidemiological circumstances in that country.

Public-health measures are particularly indicated in countries in which poliomyelitis occurs in the form of severe epidemics. Where the disease is still largely endemic, there is little need for extensive public-health measures, and, indeed, they would not be likely to have much effect. As to the specific measures which might be applied in the epidemic areas, it would appear that the first report of the WHO Expert Committee on Poliomyelitis contains as clear and concise a statement on the subject as can be made in the light of present knowledge. The relevant paragraphs of the report are therefore reproduced below.

## Control Measures <sup>i</sup>

### Introduction

From the time when poliomyelitis was first recognized to be an infectious disease until about 1930, various types of control measures were applied in an endeavour to check its spread. However, none of the methods used appeared to be successful, and so in recent years the common attitude of health authorities has been that the general control measures usually applied to other infectious diseases are of little avail in poliomyelitis. This idea has received support from the pronouncements of certain authorities that, at the time of an epidemic, there are many hundreds of persons with inapparent infection for every case of paralysis. Although this may be true in very extensive and severe epidemics, virological studies of certain communities have indicated that the virus has been found mainly in the intimate associates of the paralytic case. For this reason, it appears possible that some reduction in the number of paralytic cases may be achieved by quarantine measures centred particularly around the first paralytic cases occurring in a community.

Institution of control measures will probably have an even greater chance of reducing the number of paralytic cases in isolated rural or island communities. Here not only may measures be exerted against an individual infected household, but it should also be possible to prevent the entry into an apparently healthy community of individuals from infected localities.

Many attempts have been made in the past to use convalescent serum as a prophylactic or therapeutic measure in poliomyelitis, without any conclusive evidence of its efficacy having been obtained. Improved methods of the fractionation of plasma have been introduced and it is apparent that antibodies against poliomyelitis, as well as against many other infections, are concentrated in the gamma-globulin fraction of pooled adult human plasma. Experiments in monkeys and chimpanzees, and to a lesser extent in man, have shown that if this gamma-globulin is given before exposure paralysis may be prevented. Thus, this material, which is now in short supply, may be a useful measure of control under special circumstances. <sup>g</sup>

A more promising method of control is the possible use of prophylactic vaccines <sup>h</sup> which are not yet available. However, the results of numerous experiments in primates and a growing experience from the experimental use of vaccine in man indicates the probability that a poliomyelitis vaccine may become available to the health officer in the not too distant future.

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<sup>i</sup> With the exception of reference 1, footnotes in this section are the responsibility of the author and did not form part of the committee's report. — ED.

<sup>g</sup> See the article by Hammon (page 357).

<sup>h</sup> See the articles by Sabin and by Koprowski (pages 297 and 335).

The various methods of control of the disease will be discussed under separate headings.

### Measures to reduce the spread of infection

#### *Notification of cases*<sup>i</sup>

All available aids, both clinical and laboratory, should be used in an attempt to make a definite diagnosis. Cases considered to be poliomyelitis should be notified as either non-paralytic or paralytic.<sup>j</sup> Although the diagnosis of non-paralytic poliomyelitis is less reliable than that of paralytic poliomyelitis, the figures so obtained, along with those of mortality-rates, permit of some estimate of the severity of an epidemic, of a comparison to be made with data from other epidemics, and of an evaluation of the validity of the reporting. A patient is considered clinically to have poliomyelitis for purposes of notification if the symptoms and signs correspond with the following descriptions:

#### (a) *Non-paralytic poliomyelitis*

An illness characterized by fever, headache, vomiting, sore throat, listlessness, stiffness of neck and back; pains in the back, neck, trunk, or limbs, and hyperaesthesia; cerebrospinal fluid changes are usually found. The diagnosis is often strongly supported by epidemiological evidence; for example, known contact with a paralytic case or residence in an epidemic area.

#### (b) *Spinal paralytic poliomyelitis*

Signs and symptoms of non-paralytic poliomyelitis with the addition of partial or complete paralysis of one or more muscle groups, detected on two examinations at least 24 hours apart.

#### (c) *Bulbar paralytic poliomyelitis*

Signs and symptoms of non-paralytic poliomyelitis with involvement of the cranial nerves and/or medullary centres.

#### *Isolation of the patient*

It is established practice in some countries for patients to be isolated for 1-3 weeks from the onset of the major illness if paralytic, or from the onset of symptoms in non-paralytic cases. Periods of isolation longer

<sup>i</sup> In many countries the reported incidence of poliomyelitis is known to be much lower than the actual incidence; there is an urgent need for improvement in case notification.

<sup>j</sup> Modern views on the epidemiology of poliomyelitis suggest that valuable information as to the trend of the disease within a country may be obtained from a study of the changing age-incidence within the country. It would therefore appear to be useful to record the incidence of the disease not only as paralytic or non-paralytic cases but also by age-groups. Ideally, the age-groups should be: < 1, 1-2, 3-4, 5-9, 10-14, 15-19, 20-29, and then in 10-year age-groups; or, if this is impracticable: < 1, 1-4, 5-9, 10-14, 15-19, 20-29, 30+.

than three weeks may be considered advisable under special circumstances, since excretion of virus in faeces may continue for several weeks.

When conditions permit, isolation of the patient in his home should be considered. If the patient is removed from his home, it should be to a hospital or unit for infectious diseases, a special hospital for poliomyelitis patients, or an isolation unit (one or more rooms) in a general hospital.

Suspected cases who are removed to hospital should preferably be isolated from known cases of poliomyelitis until the diagnosis is confirmed.

At some future date, it may be possible to determine the periods of isolation for individual patients by using tissue cultures as a means of detecting the presence of virus in the faeces.

*Concurrent disinfection.* Throat discharges and faeces are infectious and should be disposed of as quickly and safely as possible. Soiled articles should be promptly disinfected by heat. Patients should have individual bed-pans unless immediate cleansing and sterilization by heat is possible.

All those attending the patient should be instructed that the disease is highly infectious and that they must practise maximum hygienic precautions (e.g., those precautions which would normally be adopted in attending a case of typhoid fever). Hand-washing before and after handling the patient is essential. Nurses need not be isolated but, where it is practicable, should not attend other patients while caring for acute poliomyelitis patients.

*Terminal disinfection.* A hospital isolation unit (or room), after being used for poliomyelitis patients, and before being opened to receive other cases, should be washed thoroughly with soap and water.

Patients should not be moved to an orthopaedic ward or hospital until the locally approved period of isolation is complete. Poliomyelitis convalescents may still be excreting virus in the stool, and therefore should not associate for 6-8 weeks from the onset of the disease with other orthopaedic patients or others in swimming-baths for rehabilitation or pleasure. If possible, poliomyelitis patients should have completely separate rehabilitation units.

#### *Measures regarding contacts*

*The family.* Family and intimate associates, especially children, should be considered as probably infected. Children with familial or intimate exposure should be confined to their homes for 21 days, avoiding over-exertion. Adults need not be confined, but should refrain from over-exertion and should observe maximum hygienic precautions; they should refrain from association with children other than their own, and should avoid intimate contact with adults. They should not handle foodstuffs

served outside the family. Any associates who do not feel well should go to bed and a physician should be consulted.

*Day-nurseries and nursery schools.* Numerous investigations have demonstrated a very high infection-rate in infants associated with paralytic cases in such institutions. If a case occurs, nursery schools should be closed and the staff, all the children, and their siblings should be treated in the same manner as family associates. The parents of such children should observe maximum hygienic precautions and refrain from over-exertion.

*Residential nurseries, schools, and children's camps.* If a case occurs in such a community, the other residents should be kept under observation for at least 21 days and instructed to avoid over-exertion; no new children or adults should be introduced. It should be remembered that if the residents are dispersed to their own homes they may seed the virus in a number of presumably unaffected communities.

#### *Measures regarding the community*

The public should be instructed in the probable modes of spread of the disease, and advised to take the following precautions in epidemic periods :<sup>k</sup>

1. Wash hands frequently, especially after defaecation and before eating.
2. Protect food from flies, and thoroughly wash uncooked food, such as fruit and vegetables.
3. Avoid intimate associations (shaking hands, common eating utensils, communal towels, etc.) with members of a family in which a case of poliomyelitis has occurred within three weeks.
4. Treat all febrile illnesses with caution; bed rest or at least the avoidance of over-exertion for a period of a week is advisable.
5. Avoid over-exertion, particularly if not feeling perfectly well.
6. Unnecessary travel into or out of communities where the disease is prevalent should be discouraged.
7. In the presence of a severe local epidemic it would be wise to delay opening schools after the summer holidays, but normally schools need not be closed nor public gatherings forbidden. Swimming-pools with adequately chlorinated water need not be closed, but should not be overcrowded. Unchlorinated pools should be closed.

<sup>k</sup> It should be remembered that in some countries the appearance of epidemic poliomyelitis is viewed by the public with an alarm quite disproportionate to the real dangers. Over-publicity can easily exaggerate these fears and may sometimes result in the population's taking all sorts of extravagant precautions. Firm handling by public-health authorities combined with judicious educational measures should help to avoid this.

If, in the future, isolation and quarantine applied on a national level should prove to be of real value as methods of prevention of epidemics, some countries, particularly those where the general level of immunity in the population is low, might be inclined to apply quarantine on an international level as well. The committee feels that, at present, restrictions of international travel would not be justified and recommends that the developments in this field be closely followed by the proper authorities.

### Measures to reduce the incidence of paralysis

It has already been mentioned that a number of factors may predispose to or precipitate paralysis. Some reduction in the incidence of paralysis may be expected to result from attention to the following principles :

1. Elective operations for the removal of tonsils and adenoids should not be carried out during epidemic prevalence of poliomyelitis.

2. The activity of persons suffering from an illness in which there is reason to suspect poliomyelitis should be restricted for a week, preferably by rest in bed.

3. Persons in intimate association with a case of poliomyelitis should take the minimum amount of exercise during the period in which symptoms might be expected to develop, that is, between 5 and 21 days after exposure. Fatigue from any cause, including travel, should be avoided.

4. With regard to immunizations and injections, of which mention has [already] been made, the following extract from the report of the WHO Conference of Heads of Laboratories Producing Diphtheria and Pertussis Vaccines<sup>67</sup> is endorsed by the committee :

'The conference feels that the effectiveness of diphtheria- and whooping-cough-immunization campaigns should be disturbed as little as possible by the fear of subsequent poliomyelitis. Immunization against diphtheria and whooping cough should normally be continued during the poliomyelitis season ; but if the disease should assume serious epidemic proportions in any given area, all immunization should be temporarily suspended in that locality. If, in the opinion of the local health authority, the epidemic is of minor severity, then immunization with diphtheria and whooping-cough vaccines may be continued, but the use of adsorbed combined vaccine should be discouraged.'

5. It seems advisable to suspend during epidemics of poliomyelitis the large-scale use of intramuscular injections of an irritant character, for example, organic arsenicals and heavy metals.

6. In view of the possibility that the skin may be contaminated with poliomyelitis virus, before administering an injection, cleansing with tincture of iodine is recommended, and separate heat-sterilized syringes and needles should be used for each patient.<sup>1</sup>

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<sup>1</sup> Alternatively, if individual syringes cannot be used, devices designed to prevent contamination of the nozzle of the syringe may be considered, e.g., that devised by Professor R. Gispen (see *Lancet*, 1952, 2, 171).

## REFERENCES

1. Amesse, J. W. (1912) *Pediatrics*, **23**, 741
2. Anderson, G. W., Anderson, G., Skaar, A. E. & Sandler, F. (1950) *Ann. Otol. (St. Louis)*, **59**, 602
3. Anderson, G. W. & Skaar, A. E. (1951) *Pediatrics*, **7**, 741
4. Anderson, J. A. (1945) *J. Pediat.* **27**, 68
5. Aycock, W. L. (1941) *New Engl. J. Med.* **225**, 405
6. Aycock, W. L. (1942) *Amer. J. med. Sci.* **203**, 452
7. Aycock, W. L. (1942) *Medicine (Baltimore)*, **21**, 65
8. Aycock, W. L. (1946) *New Engl. J. Med.* **235**, 160
9. Aycock, W. L. & Luther, E. H. (1929) *New Engl. J. Med.* **200**, 164
10. Biraud, Y. & Deutschman, S. (1935) *Epidem. Rep. L. O. N.* **14**, 207
11. Bousfield, G. (1951) *Lancet*, **1**, 1028
12. Bowers, V. M. & Danforth, D. N. (1953) *Amer. J. Obstet. Gynec.* **65**, 34
13. Brahdry, M. B. & Katz, S. H. (1951) *J. Amer. med. Ass.* **146**, 772
14. Brown, G. C. & Ainslie, J. D. (1951) *J. exp. Med.* **93**, 197
15. Brown, G. C., Ainslie, J., Gilliam, A. G., Zintek, A. R. & Francis, T., jr. (1952) *Amer. J. Hyg.* **55**, 49
16. Brown, G. C., Francis, T. & Ainslie, J. (1948) *J. exp. Med.* **87**, 21
17. Burnet, F. M. (1940) *Med. J. Aust.* **1**, 325
18. Burnet, F. M. (1946) *Virus as organism: evolutionary and ecological aspects of some human virus diseases*, Cambridge, Mass.
19. Cobb, S. W., Stuart, J. & Mengert, W. F. (1953) *Obstet. and Gynec.* **2**, 379
20. Collins, S. D. (1946) *Publ. Hlth Rep. (Wash.)* **61**, 327
21. Craster, C. V. (1916) *Trans. Amer. Ass. Stud. infant. Mort.* **7**, 187
22. Dauer, C. C. (1948) *Amer. J. Hyg.* **48**, 133
23. Deeny, J. & MacCormack, J. D. (1946) *Lancet*, **2**, 287
24. Draper, G. (1932) *Amer. J. med. Sci.* **184**, 111
25. Flexner, S. (1911) *Amer. J. Dis. Child.* **2**, 96
26. Francis, T., jr. & Brown, G. C. (1948) *J. infect. Dis.* **82**, 163
27. Francis, T., jr., Krill, C. E., Toomey, J. A. & Mack, W. N. (1942) *J. Amer. med. Ass.* **119**, 1392
28. Frost, W. H. (1910) *Publ. Hlth Rep. (Wash.)* **25**, 1663
29. Galloway, T. C. (1953) *J. Amer. med. Ass.* **151**, 1180
30. Geffen, D. H. & Tracy, S. (1953) *Brit. med. J.* **2**, 427
31. Greenberg, M., Abramson, H., Cooper, H. M. & Solomon, H. E. (1952) *Amer. J. publ. Hlth*, **42**, 142

32. Hammon, W. McD., Sather, G. E. & Hollinger, N. (1950) *Amer. J. publ. Hlth*, **40**, 293
33. Hayes, M. B. (1953) *J. int. Coll. Surg.* **20**, 350
34. Hill, A. Bradford & Knowelden, J. (1950) *Brit. med. J.* **2**, 1
35. Horstman, D. M. (1949) *Amer. J. Med.* **6**, 592
36. Horstman, D. M. (1950) *J. Amer. med. Ass.* **142**, 236
37. Horstman, P., Ipsen, J. & Lassen, H. C. A. (1946) *Nord. Med.* **30**, 807
38. Howe, H. A. (1949) *Amer. J. Med.* **6**, 537
39. International Committee for the Study of Infantile Paralysis (1932) *Poliomyelitis. A survey...*, Baltimore, Md.
40. Landsteiner, K. (1908) *Sem. méd. (Paris)*, **28**, 620
41. Lenhard, R. E. (1950) *J. Bone Jt Surg.* **32-A**, 71
42. Levine, M. E., Neal, J. B. & Park, W. H. (1933) *J. Amer. med. Ass.* **100**, 160
43. McClosky, B. P. (1950) *Med. J. Aust.* **38**, 613
44. McGoogan, L. S. (1932) *Amer. J. Obstet. et Gynec.* **24**, 215
45. MacLean, F. S. (1950) *N.Z. med. J.* **49**, 652
46. Medin, O. (1891) In : *Verhandlungen des X. Internationalen Medizinischen Kongresses*, Berlin, **2**, Abt. 6, 37
47. Melnick, J. L. & Ledinko, N. (1953) *Amer. J. Hyg.* **58**, 207
48. Molner, J. G. (1949) *Amer. J. Med.* **6**, 628
49. Olin, G. (1952) *Epidemiologic pattern of poliomyelitis in Sweden from 1905 to 1950*. In : *International Poliomyelitis Congress, Poliomyelitis : papers and discussions presented at the Second International Poliomyelitis Conference*, Philadelphia, p. 488
50. Paul, J. R., Melnick, J. L. & Riordan, J. T. (1952) *Amer. J. Hyg.* **56**, 232
51. Paul, J. R., Salinger, R. & Trask, J. D. (1933) *Amer. J. Hyg.* **17**, 601
52. Pearson, H. E., Brown, G. C., Rendtorff, R. C., Ridenour, G. M. & Francis, T., jr. (1945) *Amer. J. Hyg.* **41**, 188
53. Pearson, H. E. & Rendtorff, R. C. (1945) *Amer. J. Hyg.* **41**, 164, 179
54. Rhodes, A. J. (1953) *Canad. med. Ass. J.* **68**, 107
55. Rosen, L. & Thooris, G. (1953) *Amer. J. Hyg.* **57**, 237
56. Russell, W. R. (1947) *Brit. med. J.* **2**, 1023
57. Sabin, A. B. (1947) *J. Amer. med. Ass.* **134**, 749
58. Sabin, A. B. (1951) *Amer. J. publ. Hlth*, **41**, 1215
59. Sabin, A. B. & Steigman, A. J. (1949) *Amer. J. Hyg.* **49**, 176
60. Shwartzman, G. & Aaronson, S. M. (1953) *Ann. N.Y. Acad. Sci.* **56**, 793
61. Stocks, P. (1932) *J. Hyg. (Lond.)* **32**, 219
62. Stowman, K. (1947) *Epidem. vital Statist. Rep.* **1**, 114
63. Sweetnam, W. P. (1948) *Brit. med. J.* **1**, 1172
64. Toomey, J. A. & Krill, C. E. (1942) *Ohio St. med. J.* **38**, 653
65. Weinstein, L., Vogel, M. L. & Weinstein, N. (1954) *J. Pediat.* **44**, 14

66. Wickman, I. (1907) *Beiträge zur Kenntnis der Heine-Medin'schen Krankheit (Poliomyelitis acuta und verwandter Erkrankungen)*, Berlin
  67. World Health Organization, Conference of Heads of Laboratories Producing Diphtheria and Pertussis Vaccines (1953) *Wld Hlth Org. techn. Rep. Ser. 61*
  68. World Health Organization, Expert Committee on Poliomyelitis (1954) *Wld Hlth Org. techn. Rep. Ser. 81*
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## INDEX



## INDEX

*The figures in bold type indicate the page numbers of articles*

- Abortive poliomyelitis, *see* Diagnosis
- Adrenocorticotrophic hormone, increasing susceptibility, 383
- Africa, epidemics, 34-39  
strains isolated, 54-55  
wide dissemination and low incidence, 80, 309-310  
*See also under names of individual countries*
- Africa, North, incidence among immigrants, 61  
*See also under names of individual countries*
- African Bantu children, incidence, 52-53
- Aga respirator, *see* Respirators
- Age, as factor in development of paralysis, 15, 382-383  
-shift in incidence, 12, 33-34, 46, 61, 104, 375, 377-378  
*See also* Immunity, transmission of maternal
- Alaska, epidemics, earliest, 60  
incidence, 1950-3, 77, 79  
serological studies, 23, 302, 305, 307
- Albumin-globulin ratio, 50-51, 161
- Algeria, average annual notifications, 1931-53, 84
- Alimentary tract as route of infection, 16-17, 27, 315-316, 330-331, 338, 346, 351-354, 378
- Alkali reserve, 160  
*See also* Hyperventilation
- America, Central, epidemics, 41
- America, North, age-shift, 375  
epidemics, earliest recorded, 60-61  
incidence, 1921-53, 76-77  
mortality-rates, 76  
nutritional factors, 50-51  
poliomyelitis-like illness, 238  
*See also under names of individual countries*
- Anaesthetists, role in treatment, 141, 158
- Angola, average annual notifications, 1931-53, 84  
epidemics, 34, 35  
incidence, 36, 82  
among immigrants, 61  
seasonal, 37
- Animals, experimental, apes and monkeys, avirulent variants in, 325-331, 335-336  
cost, 218, 299-300  
development of antibody, 315-319  
earliest use, 373  
immunization, with gamma globulin, 358, 360-361  
with inactivated virus, 290-291, 321-323  
isolation and identification of virus, 240-241, 256-260  
oral infection, 17, 18, 302, 315  
re-infection, 308-309  
virus-neutralization tests, 248  
cattle, virus-neutralizing substances in, 303  
rodents, methods of virus passage, 241  
mouse encephalomyelitis virus, 217  
mouse immunization, with gamma globulin, 357, 360-361  
with inactivated virus, 290, 321-323  
with passively introduced antibody, 318  
virus neutralization tests, 248-249
- Antibody, and viraemia, 340-341  
as index to previous infection, 302-303  
complement-fixing, 247-250, 253, 323-324  
development, in animal experiments, 330-331  
in human experiments, 323-325, 330, 361-362

- Antibody, (*continued*)  
 incidence of different types, 303-306  
 induced experimentally in animals, 315-319  
 levels, after gamma globulin, 361-362  
   after immunization, 340-346  
   and resistance in normal populations, 306-309  
 response to clinically recognized infection, 297-303  
 surveys, 23-26, 359-360  
 virus-neutralizing, 247-250, 253, 269, 287-288, 300-303, 304, 323
- Antigens, tissue cultures as source of, 288, 290-291
- Arctic, Canadian, Hudson Bay epidemic, 23, 320  
 incidence of antibody, as compared with that in other regions, 304  
 serological studies, 23, 302
- Arthritis, mistaken for poliomyelitis, 121
- Asia, incidence, 39-42, 84-85, 309-310  
*See also under names of individual countries*
- Assay, of viral infectivity, *see* Virus of virus-neutralizing antibody, *see* Antibody, virus-neutralizing
- Ataxia, mistaken for poliomyelitis, 124-125
- Atelectasis, 130, 150, 151, 153-154, 170, 173, 174
- Attenuated strains, *see* Variants of virus
- Australia, age-shift, 26, 375  
 case-fatality, 1921-53, 82, 99, 101  
 epidemics, 31  
   earliest recorded, 61  
 incidence, 1921-53, 80, 81, 99, 100, 101  
 mortality, 1921-50, 82  
 poliomyelitis-like illness, 238
- Austria, case-fatality, 75  
 incidence, 1921-53, 68-70, 73, 74  
 mortality, 1921-50, 74
- "Avirulent" variants, *see* Variants of virus
- Azotemia, 176, 177
- Bacterial infection, 174, 201
- Baffin Island surveys, *see* Arctic, Canadian
- Bang respirator, *see* Respirators
- Bangkok, incidence, among immigrants, 61  
 outbreaks, 1952, 85
- Basutoland, average annual notifications, 1931-53, 84  
 incidence, 36
- Bechuanaland, average annual notifications, 1931-53, 84  
 incidence, 36
- Bed, inverted-V, 148, 149, 170  
 ordinary poliomyelitis, 143  
 rocking, 191-192, 198  
 tilted for drainage, 149, 150, 169, 170
- Belgian Congo, average annual notifications, 1931-53, 84  
 epidemics, 34, 35, 36  
 incidence, 36  
   seasonal, 37  
   1931-53, 83, 84
- Belgium, case-fatality, 1921-50, 75  
 incidence, 1921-53, 70, 71, 73  
 mortality, 1921-50, 74
- Bennet positive-pressure attachment, 188
- Biochemical aspects of bulbar and respiratory poliomyelitis, 159-161
- Bombay, incidence among immigrants, 61
- Bornholm disease (epidemic myalgia), mistaken for poliomyelitis, 125
- Bovine colostrum, virus-neutralizing, 303
- Brazil, epidemics, earliest recorded, 61
- Breast-feeding, and immunity, 51, 310, 311-312, 382  
*See also* Immunity, transmission of maternal
- Bulbar poliomyelitis, management, 148-151, **157-211**  
*See also* Diagnosis
- Bulgaria, case-fatality, 1921-50, 75  
 incidence, 1927-49, 70, 73, 75
- Canada, age-shift, 26  
 cases, 1933-52, 11  
 case-fatality, 99, 101  
 incidence, 1921-53, 76, 77, 98, 99, 101  
   by age and sex, 96-98  
 mortality, 1931-53, 76, 77  
 paralytic and non-paralytic case-notifications, compared, 85-87  
 serological studies, 2  
 strains isolated, 246
- Carbon dioxide retention, *see* Hypercapnia
- Carriers, *see* Spread
- Case-fatality-rates, **59-106**  
 correlation with morbidity-rates, 98, 104

- Case-fatality-rates (*continued*)  
 definition of term, 98-100, 303-306  
*See also under names of individual countries and regions*
- Cerebralia, *see* Symptoms, cerebral
- Ceylon, average annual notifications, 1946-53, 84  
 incidence, by age, 41  
 seasonal, 41
- Chile, incidence, 1931-53, 78, 79  
 mortality, 1931-53, 78, 291
- China, incidence of antibody, as compared with that in other regions, 212-216  
 outbreaks, 10, 33, 310-314
- Chlorination of polluted water, 230-231
- Chronic phase (phase of sequelae), 118-120
- Climatic and seasonal factors in spread, *see* Spread
- Clinical classification, 161-168, 201-202  
*See also* Diagnosis; Notification of cases
- Clinical management, **109-211**  
 hospital, 140-142  
 isolation and quarantine, 140, 378-379  
 nursing, 142-154, 168, 196-198  
 of respirator cases, 151-153, **157-211**  
 pre-hospital, 137-140
- "Combined" bulbo-spinal paralysis, treatment, 151, **157-211**
- Committee on Typing of the National Foundation for Infantile Paralysis, Inc, New York, USA, 241, 248
- Complement-fixation test, antigen for, 250, 288-289
- Complications, described, 118-120, 161-166, 174-177  
 treatment, 141, 142
- Constipation, 154
- Contact, *see* Spread
- Contact prophylaxis, with gamma globulin, 365-367
- Contamination, environmental, *see* Spread  
 of culture, 278, 281
- Control of poliomyelitis, immunological aspects, **357-370**  
 public-health and epidemiological aspects, **373-394**  
*See also* Public-health notification; Spread
- Cortisone increasing susceptibility, 22, 383
- Costa Rica, incidence, 1931-51, 77, 79  
 mortality, 1931-50, 79, 80
- Coxsackie viruses, and poliomyelitis, 125, 215, 216, 217, 251, 252
- Cross-immunity, *see* Immunity
- Cuba, incidence, of antibody as compared with other regions, 304  
 of poliomyelitis, 1931-52, 77, 79
- Cuff-tube, 153, 158, 172-173, 184, 189
- Cuirass respirator, *see* Respirators
- Cyprus, average annual notifications, 1936-53, 84
- Czechoslovakia, case-fatality, 1921-50, 75  
 incidence, 70, 73
- DEBRÉ, ROBERT & THIEFFRY, STÉPHANE, **109-134**
- Deglutition, impairment, 130, 145-146, 163-164, 177  
*See also* Stomach-tube
- Denmark, case-fatality, 1947-50, 93, 99, 102  
 Copenhagen 1952 epidemic, 60, 158-159, 165, 174  
 incidence, 1911-53, 63, 66, 73, 74, 75, 93, 99, 102  
 by age, 89, 90  
 by sex, 90, 91  
 nutritional factors, 50-51  
 paralytic and non-paralytic case-notifications, compared, 85-87  
 therapeutic results, 1934-44, 1952, 207
- Desiccation of virus, 226-227
- Diagnosis, clinical, **109-134**  
 differential, from other diseases, 121-125, 140, 201-202, 237-238  
 difficulties of early, 137-139  
 in experimental animals, 259-260  
 laboratory, **237-267**  
 methods, electrodiagnosis, 117-118  
 electromyography, 118  
 muscle-paralysis classification, conventions, 117  
 physical examination, 144  
 serological, 120-121, 247-250, 252-253  
 spinal-fluid examination, 120-121, 132-134  
 virus isolation, 241-247, 285-287  
 of abortive poliomyelitis, 14-16, 131-133  
 of bulbar poliomyelitis, 128-131, 145-146, 148, 388

Diagnosis (*continued*)

- of inapparent poliomyelitis, 14-16, 131-133
- of non-paralytic poliomyelitis, 14-16, 131-133, 388
- of spinal paralytic poliomyelitis, 388
- terminology, 14-15, 109-110, 117, 122, 131-133, 137, 388
- Dietetic factors in spread, *see* Spread
- Disinfectants, *see* Virus, resistance and sensitivity
- Disinfection, 233, 389
  - See also* Spread, contact; Virus, resistance and sensitivity
- Draeger respirator, *see* Respirators
- Dulbecco culture, *see* Tissue culture
- DUNCAN, DARLINE, *see* RHODES, A. J.
- Economic and social factors in resistance to infection, 203, 305-306, 375, 377-378
- Egypt, average annual notifications, 1931-53, 84
  - epidemics, 10
  - incidence of antibody, as compared with other regions, 304, 305-306, 311
  - insect carriers, 314
  - serological studies, 23-26
- Electrodiagnosis, *see* Diagnosis
- Electromyography, *see* Diagnosis
- Electron microscopy, 220, 222-225
- Electrophrenic respirator, *see* Respirators
- Emergency treatment of respiratory insufficiency, 186
- Emerson rocking bed, *see* Respirators
- ENDERS, JOHN F., 269-294
- Endocrinological disturbances increasing susceptibility, 22, 383-384
- England, cases recorded, earliest, 9
  - epidemics, earliest, 10
  - therapeutic results, 207
  - See also* England and Wales; United Kingdom of Great Britain and Northern Ireland
- England and Wales, case-fatality, 75, 87-89, 92, 99
  - incidence, 62-63, 64, 73, 92, 99
  - morbidity by age and sex, 87-89, 90-91, 92
  - mortality, 1921-50, 74

England and Wales (*continued*)

- paralytic and non-paralytic case-notifications, compared, 294-296
- See also* England; United Kingdom of Great Britain and Northern Ireland
- Engström respirator, *see* Respirators
- Epidemicity, 60-61
- Epidemiology, 9-106
  - influencing application of control measures, 374-386
  - See also* Spread
- Eskimos, *see* Alaska; Arctic, Canadian
- Europe, age-shift, 12, 26
  - epidemics, earliest, 10, 60
  - trends in incidence, 62-75
  - See also under names of individual countries*
- Europe, central, trends in incidence, 68-70
  - See also under names of individual countries*
- Europe, northern, trends in incidence, 63-67
  - See also under names of individual countries*
- Europe, southern, trends in incidence, 70-72
  - See also under names of individual countries*
- Europe, western, trends in incidence, 70
  - See also under names of individual countries*
- Far East, attack-rates in American troops, 33
  - incidence of antibody, as compared with that in other regions, 310, 312
  - See also under names of individual countries*
- Fatigue, role in infection, 21-22, 138, 197-198, 382-384, 390-391
- Finland, case-fatality, 75
  - cases, 1933-52, 11
  - incidence, 1921-53, 65-67, 73
  - decrease, 74
  - mortality, 1921-50, 74
- Fixed-cell (fixed-fragment) tube culture, *see* Tissue culture
- Flask cultures, preparation, *see* Tissue culture
- France, case-fatality, 75, 95, 97, 102
  - cases, 1933-52, 11
  - incidence, 1921-53, 70, 71, 73, 97, 102

- France (*continued*)  
 mortality, 1921-50, 74  
 notifications by age and sex, 93-96
- French Cameroons, average annual notifications, 1931-53, 84  
 epidemic, 80
- French Equatorial Africa, epidemics, 34, 35  
 incidence, 36
- French Morocco, average annual notifications, 1931-53, 84  
 incidence of antibody, as compared with that in other regions, 304, 305-306, 311
- French Oceania, epidemics, of measles and poliomyelitis, compared, 16, 47  
 of poliomyelitis, 46-47  
 incidence by age, 46
- French West Africa, incidence, 36
- FREYCHE, MATTHIEU-JEAN & NIELSEN, JOHANNES, **59-106**
- Gamma globulin, cost, 369  
 limitations, 139, 363-368  
 producing immunity, in man, **357-370**, 384, 387  
 in monkeys, 319, 358  
 in rodents, 357  
 standardization, 363  
 substitutes, 363
- GARD, SVEN, **215-235**
- Gastric dilatation, 154, 169
- GEAR, JAMES, **31-58**
- Genetic predisposition, *see* Predisposing factors in infection
- Geographical distribution and incidence, 13, 31-48, **59-106**, 309-313, 375  
*See also under names of individual countries and regions*
- Germany, case-fatality, 1921-50, 75  
 incidence, of antibody, as compared with that in other regions, 304, 310, 314  
 of poliomyelitis, 1909-53, 68, 69, 73  
 mortality, 1921-50, 74  
 notifications by age, 93, 95
- Gilbert and Ellice Islands, epidemic, 80
- Glass-ware, preparation for culture, 242, 261
- Gold Coast, average annual notifications, 1931-53, 84
- Greece, case-fatality, 1921-50, 75  
 incidence, 1931-53, 72-73
- Greenland, epidemics, 79
- Guam, infection-rates, 307, 309-310
- Guatemala, mortality-rates, 1931-50, 80
- Guillain-Barré syndrome, differentiated from poliomyelitis, 122-124, 238
- Gullberg positive-pressure attachment, 181, 182, 188
- Haemorrhagic diathesis, 177
- HAMMON, W. McD., **357-370**
- Hawaii, case-fatality, 1921-53, 82  
 epidemics, 10, 49  
 incidence, 1921-53, 80, 82  
 racial, 49  
 mortality, 1921-53, 82
- HeLa cells for tissue culture, 243, 278-280, 282-283, 287
- History of poliomyelitis, 9-13
- Hospital organization, 140-142
- Hot packs, 147, 196
- Hudson Bay epidemic, *see* Arctic, Canadian
- Human experiments in immunization, 309, 323-325, 337-346, 347, 358-360, 361, 364-367
- Hungary, case-fatality, 1921-50, 175  
 incidence, 70, 73  
 mortality, 1921-50, 74
- Hyperaesthesia, 144
- Hypercapnia, 159, 161-163, 168, 174-175
- Hyperpyrexia, 176, 177
- Hypertension, 176
- Hyperventilation, 175
- Hypoventilation, 174-175
- Hypoxia, diagnosis, 151, 159, 161-162, 168, 174-175
- Iceland, case-fatality, 1926-50, 75  
 incidence, of antibody, as compared with that in other regions, 304  
 of poliomyelitis, 1924-52, 67, 68  
 mortality, 1924-52, 67, 74  
 poliomyelitis-like illness, 238
- Immune-serum globulin, *see* Immunity
- Immunity, active, 51-52, 361-362  
 and persistence of antibodies, 340-341  
 and vaccination, **297-334**  
 as factor in determining incidence, 51-54  
 cross-, 318  
 passive, 51, 318-319, 341, 345-346, **357-370**

- Immunity (*continued*)  
 produced by gamma globulin, 319, 357-370, 384, 387  
   by immune-serum globulin, 341-346  
   intramuscularly, 330-331  
   orally, 330-331, 335, 337, 338-346, 351-354  
 role of economic status in, 305-306  
 tissue-resistance, 316  
 to re-infection, 316-319  
 transmission of maternal, 42, 51-54, 302, 311-312, 345-346, 376, 382-383  
*See also* Antibody; Immunology
- Immunization, active, 380-381  
 of man with living virus, 335-356  
 passive, 357-370  
*See also* Antibody; Immunity; Immunology
- Immunology, 297-370  
*See also* Immunity; Immunization
- Inapparent infection, antibody response, 301-302  
 ratio, to clinical disease, 15, 378-379  
*See also* Diagnosis
- Incidence of poliomyelitis, since 1920, 59-106  
 trend towards increase, 31, 61-85, 374-375
- Incubation phase of disease, *see* Minor illness
- India, earliest cases recorded, 9  
 incidence, 40  
 outbreaks, 10, 33  
 in troops, 61
- Inoculation, monkey, 256-260  
*See also* Vaccination; Vaccines
- Insects, role in spreading poliomyelitis, *see* Spread
- Intramuscular injection of heavy metals, *see* Predisposing factors in infection
- Invasion (pre-paralytic) phase of disease, *see* Major illness
- Iran, average annual notifications, 1936-53, 84
- Iraq, average annual notifications, 1936-53, 84
- Ireland, case-fatality, 1921-50, 75  
 incidence, 1920-53, 63, 64, 73  
 mortality, 1921-50, 74
- Irradiation sensitivity, 222
- Island epidemics, 42-48  
*See also under names of individual islands*
- Isolation of patients, 139-140, 388-389
- Israel, average annual notifications, 1946-53, 84  
 epidemics since 1950, 85
- Italy, case-fatality, 1921-50, 75, 99, 101  
 earliest cases recorded, 9  
 incidence, 1921-53, 70-71, 72, 73, 101  
 mortality, 1921-50, 74
- Japan, average annual notifications, 1946-53, 84, 85  
 case-fatality, 1948-53, 85  
 development in epidemicity, 41  
 incidence of antibody, compared with that in other regions, 205-207, 212-216, 303-305, 310-314  
 of poliomyelitis, by age, 41  
 in immigrants, 61
- Jordan, average annual notifications, 1951-53, 84
- Kenny treatment, *see* Hot packs
- Kenya, average annual notifications, 1931-53, 84  
 incidence, 36  
 increased, 82  
 1931-53, 83  
 outbreaks, 35
- Kifa respirator, *see* Respirators
- KOPROWSKI, HILARY, 335-356
- Korea, epidemics, 10  
 incidence, of antibody, compared with that in other regions, 303-305  
 of poliomyelitis, in immigrants, 61
- La Réunion, epidemics, 82
- Laboratory methods in diagnosis, 237-267
- LASSEN, H. C. A., 157-211
- Lebanon, average annual notifications, 1936-53, 84
- Lung physiotherapy, *see* Physiotherapy
- Lyophilization of virus, 226
- Madagascar, average annual notifications, 1931-53, 84
- Major illness (invasion; pre-paralytic, phase), 137-139, 110-113

- Malta, epidemics, 10, 42-43  
 compared with other island, 47-48  
 incidence, by age, 42-43  
 racial, 42-43  
 local immunity, 313  
 nutritional factors, 50-51
- Manila, incidence, 85
- Manual bag ventilation, 158-159, 183-187,  
 188, 189, 201  
*See also* Positive-pressure intratracheal  
 ventilation
- Mass prophylaxis, with gamma globulin,  
 364-365
- Mauritius, average annual notifications,  
 1931-53, 84  
 epidemics, 10, 43-44  
 compared with other island, 47-48  
 incidence, by age, 43  
 racial, 44, 49  
 seasonal, 37, 43-44  
 1921-53, 82
- Measles and poliomyelitis, 46-47, 201,  
 357, 365-366
- Media for tissue culture, 243-244, 249,  
 262-265, 271, 277-278, 279-280, 281,  
 288, 289
- Mediterranean, Eastern, epidemics in  
 troops, 61  
*See also under names of individual  
 countries*
- Medium No. 199, *see* Media for tissue  
 culture
- Meningitis, differentiated from polio-  
 myelitis, 140, 238, 251  
 in non-paralytic poliomyelitis, 132-133  
*See also* Diagnosis, differential
- Meningoradiculomyelitis, mistaken for  
 poliomyelitis, 122
- Metabolic factors in respiratory polio-  
 myelitis, 159-161
- Mexico, mortality-rates, 1931-50, 80
- Millikan oximeter, 159
- Minor illness, 109-110, 137-139  
*See also* Diagnosis
- Monaghan respirator, *see* Respirators
- Monolayer cultures, 247
- Morale, methods of maintaining patient's,  
 139, 142, 146, 169, 186, 196, 198,  
 200, 201, 202, 206
- Morbidity-rates, 59-106  
 correlation with case-fatality-rates, 98-  
 104
- Morbidity-rates (*continued*)  
 definition of term, 98  
*See also under names of individual  
 countries and regions*
- Mortality, in life-threatening poliomyelitis,  
 207, 208  
 in patients with respiratory insufficiency,  
 202-205
- Mortality-rates, 59-106  
*See also under names of individual  
 countries and regions*
- Mouse encephalomyelitis virus (*Poliovirus  
 muris*), 216
- Mozambique, average annual notifications,  
 1931-53, 84  
 incidence among immigrants, 61
- Myelitis, mistaken for poliomyelitis, 122
- Netherlands, case-fatality, 1921-50, 99,  
 102  
 incidence, 1921-53, 70, 71, 73, 99, 102  
 mortality, 1921-50, 74
- New Zealand, 31  
 case-fatality, 1921-53, 82, 99, 102  
 epidemics, earliest recorded, 61  
 incidence, 1921-53, 80, 81, 82, 99, 102  
 mortality, 1921-53, 82
- Nicobar Islands, epidemics, 46  
 compared with other island, 47-48
- NIELSEN, JOHANNES, *see* FREYCHE,  
 MATTHIEU-JEAN
- Nigeria, average annual notifications,  
 1931-53, 84
- Non-paralytic poliomyelitis, incidence, 312  
*See also* Diagnosis
- Non-return respiration valve, 186-187,  
 188, 196, 197
- Northern Ireland, case-fatality, 1921-50,  
 75  
 incidence, 1920-53, 63, 64, 73  
 mortality, 1921-50, 74
- Northern Rhodesia, average annual noti-  
 fications, 1931-53, 84  
 incidence, 36  
 among immigrants, 61  
 racial, 37, 39  
 seasonal, 37, 39  
 outbreaks, 35
- Norway, case-fatality, 1921-50, 75, 99  
 epidemics, earliest, 10, 60  
 incidence, 1905-53, 65, 66, 73, 99

- Norway (*continued*)  
 mortality, 1921-50, 74  
 paralytic and non-paralytic case-notifications, compared, 85-87  
 therapeutic results, 1936-45, 207  
 Notification of cases, paralytic and non-paralytic, compared, 85-87  
 recommended, 70, 104-105, 388  
 variability, 59-60  
 Nyasaland, attack-rate among Europeans, 37  
 average annual notifications, 1931-53, 84  
 incidence, 36  
 outbreaks, 35  
 Oceania, trends in incidence, 80  
*See also* French Oceania; *and under names of individual countries and territories*  
 Okinawa, incidence of antibody, compared with that in other regions, 303-305, 310-314  
 Osteomyelitis, mistaken for poliomyelitis, 121  
 Oxford Inflator, 152-153  
 Oximeter, 151, 152  
 Palestine, incidence, 39-40  
 Panama, *see* America, Central  
 Paralysis, diaphragmatic, 204-205  
 distribution, 114-115  
 effects, 119-120  
 facial, 43, 128, 130  
 spinal, phases in development, 109-116  
 Paralytic ileus, 176, 177, 195  
*See also* Stomach-tube  
 Paralytic poliomyelitis, *see* Diagnosis  
 Pathogenesis, 16-19  
 PAUL, JOHN R., 9-29  
 PAYNE, A. M.-M., 373-394  
 Pertussis predisposing to paralysis, 384  
 Peru, mortality-rates, 1931-50, 80  
 Philippine Islands, attack-rates, in American troops, 33, 61  
 average annual notifications, 1936-53, 84  
 epidemics, 10, 33  
 wide dissemination and low incidence, 309-310  
 Physiotherapy, 119, 144, 153, 169, 173, 196, 358  
 Poland, case-fatality, 1921-50, 75  
 incidence, 70  
 Polioencephalitis, diagnosis, 162  
*Poliovirus hominis*, *see* Virus  
*Poliovirus muris*, 218-219, 224  
*See also* Virus  
 Polyneuritis, mistaken for poliomyelitis, 122  
 Polyradiculoneuritis, *see* Guillain-Barré syndrome  
 Portals of entry and exit of virus, 16-17, 18  
 Portugal, case-fatality, 1921-50, 75  
 incidence, 1921-53, 72, 73  
 mortality, 1921-50, 74  
 Positive-pressure intratracheal ventilation, 183-194  
 indications for use, 147, 171  
 Postural drainage, 149, 150, 158, 169, 181, 195  
 Posture in bed, 143-144, 148, 169, 170  
 Predisposing factors in infection, 21-22, 320, 382-385  
 Pregnancy, increasing susceptibility, 22, 383-384  
 Public-health measures for control, 373-394  
 commonly recommended, 373, 377  
 recommended specifically by WHO, 386-391  
*See also* Spread  
 Public-health notification, recommended clinical criteria for, 388  
 Puerto Rico, epidemics, 10  
 incidence, 1931-53, 47, 79  
 mortality, 1931-50, 79, 80  
 Pulmonary oedema, 175-176, 177  
 Quarantine, 373, 378-379, 387, 391  
 Racial factors in spread, *see* Spread  
 Recovery, medullary, 199  
 muscle, 119  
 respiratory, 199, 201, 205  
 Resistance, racial, to poliomyelitis, 49-50  
*See also* Immunity  
 Resistance, viral, *see* Virus, resistance  
 Respiration unit, 141-142  
 Respirators, adjustment and handling, 151-153, 179-183, 200-201  
 Aga, 190-191, 196, 197  
 Bang, 189-190, 191, 192; 193, 194  
 cuirass, 182-183

- Respirators (*continued*)  
 disadvantages and dangers of use, 150,  
 179, 180-181, 182-183, 206  
 Draeger, 178  
 electrophrenic, 192-194  
 Engström, 187  
 indications for use, 169  
 Kifa, 181, 182-183  
 Monaghan, 182  
 rocking bed (Emerson), 191-192, 198  
 Sahlin, 182  
 Siebe-Gorman, 151  
 tank, 146-147, 151, 154, 170, 178-182  
*See also* Manual bag ventilation;  
 Positive-pressure intratracheal ven-  
 tilation
- Respiratory insufficiency, 143, 163, 177  
 chronic, 204, 205-206  
 evolution and regression, 202-205  
 mechanism, 125-128  
 treatment, 151-153, **157-211**
- Rheumatism, mistaken for poliomyelitis,  
 121
- RHODES, A. J., WOOD, W. &  
 DUNCAN, DARLINE, **237-267**
- Roller-tube culture, *see* Tissue culture
- Romania, case-fatality, 1921-50, 75  
 incidence, 1927-46, 70, 73  
 mortality, 1921-50, 74
- RUSSELL, W. RITCHIE, **137-155**
- St. Helena, epidemics, 44-46  
 compared with other island, 47-48  
 earliest recorded, 9-10, 44, 203  
 incidence, by age, 45  
 seasonal, 37  
 nutritional factors, 50-51
- SABIN, A. B., **297-334**
- Sahlin respirator, *see* Respirators
- Salvador, epidemics, 10
- Sanitation, as factor in spread, *see* Spread  
 measures to improve, 230-231  
*See also* Virus, resistance and sensitivity  
 to disinfectants
- Scandinavia, age-shift, 375  
 epidemics, 31, 60-61  
*See also under names of individual  
 countries*
- Scotland, case-fatality, 1921-50, 75  
 incidence, 1920-53, 63, 64, 73  
 mortality, 1921-50, 74
- Seasonal factors in spread, *see* Spread
- Sedimentation-rate, 221-222
- Serological diagnostic methods, *see* Diag-  
 nosis
- Serological-epidemiological, methods, 22-  
 26, 52-54  
 studies, 301-302, 303-314, 337-346, 359-  
 360
- Siebe-Gorman respirator, *see* Respirators
- Singapore, incidence, 85  
 among immigrants, 61
- Skin care, 197, 206
- Southern Rhodesia, attack-rate among  
 Europeans, 37  
 average annual notifications, 1931-53,  
 84  
 incidence, 36  
 among immigrants, 6  
 recent, 82  
 seasonal, 37  
 outbreaks, 35
- Spain, case-fatality, 1921-50, 75  
 incidence, 1921-53, 72, 73  
 by age, 96  
 mortality, 1921-50, 74
- Spinal-fluid examination, *see* Diagnosis
- Spinal paralytic poliomyelitis, 109-128  
*See also* Diagnosis
- Spread, carriers, experimentally-infected  
 animal, 317, 319  
 human, 13, 15, 18, 256, 307-309,  
 338-339, 340, 341, 344, 360, 373  
 insect, 19-20, 314, 373, 378  
 climatic and seasonal factors, 20-21,  
 37-39, 100, 203-204  
 contact, 13-16, 18-19, 27, 46, 140,  
 307-309, 352-353, 360, 364, 373,  
 377-378, 383, 389-390  
 dietetic factors, 50-51, 382, 383  
 portals of entry and exit, 16-19  
 racial factors, 37, 38, 49-50  
 reservoir of infection, 15-16, 62  
 sanitation, 19, 20, 27, 34, 39, 41, 53,  
 233, 305, 314, 373, 379  
 speed, 16  
*See also* Geographical distribution and  
 incidence; Predisposing factors in  
 infection
- Stationary culture, *see* Tissue culture
- Statistical problems in poliomyelitis epi-  
 demiology, 59-60
- "Sterile mutant", *see* Virus, mutations
- Stomach-tube, for feeding virus, 344  
 indicated in acute respiratory polio-  
 myelitis 169, 195

- Stools, preparation for culture, 260
- Suction treatment for respiratory insufficiency, 195
- Suspended-cell (suspended-fragment) culture, *see* Tissue culture
- Swaziland, incidence, 49
- Sweden, age-shift, 12-13, 26, 33-34  
 attack-rates, 94  
 case-fatality, 1921-50, 75, 99, 102  
 1905, 1911-13, 1925-34, and 1935-44, 94  
 epidemics, earliest, 10, 60, 373, 374  
 incidence, 1905-53, 65, 66, 73, 99, 102  
 by age and sex, 91-92  
 mortality, 1921-50, 287  
 nutritional factors, 50-51  
 paralytic and non-paralytic case-notifications, compared, 294-295  
 therapeutic results, 1934-45, 207
- Swine encephalomyelitis virus, 216
- Switzerland, case-fatality, 1921-50, 75  
 cases, 1933-52, 11  
 incidence, 1924-53, 69, 70, 73  
 mortality, 1921-50, 74
- Symposium on Applications of Tissue Culture Methods in the Study of Viral Infections, *see* Tissue culture
- Symptoms, 109-116, 128-133, 137-139, 168  
 abdominal, 112, 126  
 autonomic, 165  
 cerebral, 164  
 circulatory, 129  
 eye, 130, 165  
 of experimentally-induced poliomyelitis in monkeys, 259  
 pharyngeal, 113, 128, 145-146, 163-164  
 postural, 111-112, 143  
 reflex, 111-112, 147, 163  
 respiratory, 143, 146, 126-129, 130  
 skin, 143  
 speech, 126, 143, 145  
 sphincter, 147, 154, 112-113  
 spinal, 111, 128, 132, 138-139, 164  
*See also* Diagnosis
- Tahiti, epidemics of measles and poliomyelitis, compared, 16, 47  
 incidence by age, 46
- Tank-type respirator, *see* Respirators
- THIEFFRY, STÉPHANE, *see* DEBRÉ, ROBERT
- Throat microphone, 148, 149, 152
- Tissue culture, advantages, 251-252, 269-270  
 agents resembling virus in, 150-151, 252  
 explants, primary, used in, 270-278  
 fixed-cell (fixed-fragment) culture, 272-275  
 flask cultures, 244  
 roller-tube culture, 244, 272-275  
 stationary culture, 275  
 stock cultures, 278-283  
 suspended-cell (suspended-fragment) culture, 271-272
- Symposium on Applications of Tissue Culture Methods in the Study of Viral Infections, 284  
 techniques, 237-267, 269-294  
 trypsinized-cell culture of Dulbecco, 275-277, 292  
*See also* Virus, purification
- Tissues, choice and preparation for culture, 242-243
- Tobago, *see* Trinidad
- Tracheotomy, complications, 172, 202  
 indications for, 158, 170-172  
 technique, 172-173  
*See also* Positive-pressure intratracheal ventilation
- Trauma increasing susceptibility, 384-385  
 and gamma globulin, 367-368  
 dental extraction, 382  
 intramuscular injection, 46, 47, 138, 338, 382, 385-386, 391  
 limb surgery, 138  
 tonsillectomy, 138, 367-368, 382, 385, 391  
*See also* Predisposing factors in infection
- Treatment, *see* Clinical management; Control of poliomyelitis
- Trinidad and Tobago, mortality-rates, 1931-50, 80
- Tropical characteristics of poliomyelitis, 10, 13, 54-56, 61, 379
- Trypsinized-cell culture of Dulbecco, *see* Tissue culture
- Tunisia, annual notifications, 1931-53, 83, 84
- Uganda, average annual notifications, 1931-53, 84  
 incidence, 36  
 outbreaks, 35

- Ultrafiltration methods, 220-221
- Ultraviolet irradiation inactivating virus, 288, 291, 324-325
- Under-developed areas and poliomyelitis, 31-58
- Union of South Africa, average annual notifications, 1931-53, 84  
incidence, 36  
increased, 82, 83  
of antibodies, as compared with that in other regions, 308  
racial, compared, 37, 38, 49, 308  
seasonal, 37, 38, 39  
1931-53, 83  
outbreaks, 35  
strains isolated, 54-55
- United Kingdom of Great Britain and Northern Ireland, incidence, 1913-53, 62-63  
*See also* England; England and Wales; Ireland; Northern Ireland; Scotland
- United States of America, age-shift, 26  
case-fatality, 99, 101, 102  
earliest cases recorded, 9, 60  
epidemics, 31  
earliest, 10, 60-61  
urinary symptoms in, 112-113  
gamma-globulin field trials, 358-360, 366, 367  
incidence, 1921-53, 76-77, 79, 99, 101  
of antibody, compared with that in other regions, 303-314  
racial, 49  
mortality, 1931-53, 79, 80  
serological studies, 15-16, 21, 23-26, 301-302  
therapeutic results, 1940-52, 207
- Uraemia, 176
- Urine retention, 112-113, 147, 154, 176
- Uruguay, incidence, 1931-53, 78, 79
- Vaccination, and immunity, **297-334**  
*See also* Vaccines
- Vaccines, desirable characteristics, 346  
killed-virus, and live-virus, compared, 320-321  
in animal experiments, 321-323  
in human experiments, 323-325  
live-virus, **335-356**  
and killed-virus, compared, 320-321
- Vaccines (*continued*)  
polyvalent, 291  
produced by tissue-culture techniques, 269, 283, 290-291  
prophylactic, 387  
risks, 325, 346-348, 352-354, 381  
ultraviolet irradiated, 324-325
- Variants of virus, "avirulent" (attenuated), 325-331, 347, 348-350, 376  
*See also* Virus, antigenic variation
- Vasometer shock, 161, 163, 175, 176, 177, 196
- Viraemia, and passive protection, 319, 330-331, 360-361, 339-341, 350-351  
in natural infections, 351  
and predisposing factors, 21-22  
distribution in body, 17-18, 239
- Virology, **215-294**
- Virulence, definition, 325-326  
intensification in cotton-rat CNS, 335  
measurement, 326-327  
modification, 328-331  
mutability, 375-376  
of different strains, 313-314  
of dose, 314
- Virus, agents resembling, in tissue culture, 246-247, 252  
antigenic variation, 300-301  
classification, 215-217  
definition, 215, 216, 225-226  
identification criteria, 216, 220-221, 229, 232, 246, 260  
infectivity, 284-285, 344-345  
isolation, 239-240, 244-245, 253-260, 285-287  
morphology, 220-225  
multiplication, 289-290  
mutations, 203, 291-292, 327-331, 375  
pH stability, 228-229  
physical and chemical aspects, **215-235**  
precautions in handling, 253, 257  
preservation and storage, 225-228, 239, 255-256  
purification, chemical, 217-220, 290  
techniques, 22, 26, 218-220  
resistance and sensitivity to, desiccation, 226-227  
disinfectants, various, 230-233  
formaldehyde, 229-230  
irradiation, 222, 228, 291, 324-325  
organic solvents, 229  
temperature, 227-228

Virus (*continued*)

- shipping, 256
- sites of predilection, 279-280
- sources, 239, 253-254
- strains, Aycock, in serological surveys, 53
  - Brunhilde (type 1), 54-55, 216, 297
  - Lansing (type 2), in serological surveys, 52, 53-54, 216, 297
  - Leon (type 3), 54-55, 216, 297
    - illustrated, 299
  - Mahoney (type 1), illustrated, 298
  - MV, in serological surveys, 53
  - tropical, 54-56
- typing, 241, 246-247
- See also* Variants of virus

- Virus-cell interaction, 348
- Virus-neutralization test, 252-253
- Wales, *see* England and Wales
- Weaning from artificial respiration, 183, 199-202
- WOOD, W., *see* RHODES, A. J.
- World Health Organization, Expert Committee on Poliomyelitis, 240, 249, 253-260, 378, 386
  - recommendations for poliomyelitis control, 386-391
- Third World Health Assembly, resolution, 202
- Yugoslavia, case-fatality, 1921-50, 75
  - incidence, 70, 73