In Coventry in 1957, a year in which 141 paralytic cases were admitted to the local isolation unit, six cases of acute encephalitis-like illness were admitted at the height of the poliomyelitis incidence, with a good recovery in each instance. In two of them the faeces were examined for poliomyelitis virus, and type 1 virus was found in both. All six were pyrexial, with meningeal signs in all except one and C.S.F. changes consistent with poliomyelitis in all save one other. Two of these patients were girls aged 8 and 3 years who presented fully conscious but with ataxia—in one of them limited to the left arm and leg. Another girl of 3 was also conscious but exceedingly irritable, and showed violent generalized clonic spasms especially affecting her eyes, face, and head. Any attempt to sit her up caused her to scream and the movements to increase, but when she slept the spasms disappeared. Two boys aged 6 and 10 presented in stupor; one of them lay quietly on his side, but if disturbed resisted violently with extreme hyperpnoea and the development of carpopedal spasm in a matter of minutes—which passed off just as quickly when he was left alone. The sixth case was that of a business man of 35 who had episodes of confusion in which he performed ridiculous acts—for example, trying to post a bundle of his belongings in the ward kitchen. It is a reasonable clinical supposition that “non-paralytic” cases of the above type occurring in the midst of a poliomyelitis epidemic are in all probability examples of “polioencephalitis.”

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St. Luke's Printing Works in Old Street, London, which was built as a hospital in 1784, was sold in January by the Bank of England for over £1m. Charles Dickens, who visited the hospital in 1852 (see *Journal*, 1951, 2, 1518), describes how it was founded as a new hospital for the insane by “half a dozen gentlemen” who were perturbed to see that the hospital at Bethlem was incapable of receiving and maintaining the great number of “melancholy objects of this sort who apply for relief.” The hospital's first home was a building called The Foundry (in what is now Tabernacle Street), where cannon had originally been cast and where later John Wesley preached. In 1784 George Dance, the architect, designed the new St. Luke's, the building in Old Street, which opened two years later. Since Dance's first important work had been the rebuilding of Newgate Prison it is hardly surprising that the new hospital had a massive and rather forbidding appearance. The Old Street building ceased to be a hospital in 1917, but during its long life it admitted 25,569 patients, of whom 11,077 were discharged cured. The hospital's name and purpose is perpetuated in the St. Luke's Woodside Branch of the Middlesex Hospital. In the nineteenth century the hospital's visitors included, besides Charles Dickens, Charles Lamb, who was one of its governors, and Mrs. Elizabeth Fry.

EPIDEMIOLOGY OF POLIOMYELITIS*

BY

G. W. A. DICK, M.D., D.Sc., F.R.C.P.Ed.
The Queen's University of Belfast

It is now well established that there are three types of poliovirus—types 1, 2, and 3—and that type 1 is responsible for about 80% of all paralytic infections. Although these types are immunologically distinct there is evidence which indicates a degree of relationship between them, and this may be of importance clinically. It must be remembered that paralysis is not a common outcome of infection with a poliovirus, for, as is well known, infections with all types of poliovirus are a common experience; usually there is an alimentary infection which is clinically either inapparent or unrecognizable. It has been shown that infections at a clinical or subclinical level generally result in resistance to further attacks.

Transmission of the Virus

The virus is spread by person-to-person contact, and children are probably the most important disseminators of virus in the community. The reason for this is that virus multiplies better in the alimentary tract of children than of older persons, and the children therefore excrete much greater quantities of virus and are thus more infectious.

There is no evidence that animals or birds enter into the natural history of the disease. Recently there was a report from the West of Scotland which suggested that budgerigars might play a part in the transmission of the virus. Dr. D. S. Dane and I have so far been unable to infect budgerigars with any of 12 different strains of poliovirus.

After infection, virus is found in the throat secretions of a proportion of individuals for a few days and in the faeces for weeks or months. In addition to the fact that children excrete more virus than adults we have also some evidence suggesting that there is a family pattern of virus excretion. Thus the children of some families seem to excrete more virus than those of other families. There appear to be “hot” families in whom the virus multiplies well and “cold” families in whom virus multiplies poorly. This familial susceptibility may be of importance in the pathogenesis of the infection and also in the spread of virus in the community. The precursor of paralysis is viraemia, and it may be that, in those families in whom virus multiplies well because of its greater multiplication in the gut, there is more chance of the virus getting into the blood stream and thence to target areas in the C.N.S. where the damage is done. This does not mean, of course, that everyone who gets viraemia will become paralysed. The time relationship between the pathogenetic events and the clinical picture is shown in Fig. 1.

This represents the sequel of events in a paralysed child. Essentially the same picture occurs in the asymptomatic infection but with little or no invasion of the C.N.S.

If a child becomes infected it is a common experience for all members of the family to be also infected. This occurs whether or not any of the family is showing signs or symptoms of infection.

*Read to the Section of Medicine at the Annual Meeting of the British Medical Association, Birmingham, 1958.

The relative importance of oropharyngeal virus and faecal virus in the transmission of the disease has not yet been established. It is my own belief that faecal-oral transmission is perhaps the commonest method of spread from child to child and to other members of the family. The important factor in the spread of virus is close personal contact.

As I have said, there appear to be "hot" families who are good disseminators and "cold" families who are poor disseminators of virus, and it appears that the spread of the virus in a community may take place as shown in Fig. 2.

It may be thought somewhat academic to discuss the spread of a virus the effects of which are gradually disappearing as a result of vaccination. My reason for mentioning it is that this type of spread may occur in other infectious diseases in which there may also be good and poor disseminators of the infectious agent.

FIG. 1.—Time relationship between pathogenetic events and clinical picture in paralytic poliomyelitis.

FIG. 2.—Diagrammatic representation of the spread of a virulent strain of poliomyelitis in a community.

Control of the Disease

This, then, brings us to the question of control. It is not possible to prevent the virus passing round the community. It may, however, sometimes be possible to avoid infection with a virulent strain by preventing contact of children with paralytic cases and their siblings.

Poliovirus is excreted in its largest amounts before the onset of paralysis, and the period of infectiousness is of relatively short duration (Fig. 1). If a child in a family is paralysed it is most probable that not only will the paralysed child be excreting a large quantity of virus but so also will the siblings. It therefore seems reasonable to quarantine the siblings for about one week—that is, during the period of expected high virus excretion. Unfortunately, in the majority of cases this will be closing the stable door too late.

So far as live virus vaccines are concerned I do not consider that any strains so far developed are satisfactory for wide-scale trials. In all studies which have been made with oral attenuated live virus vaccines it has been found that after feeding with these attenuated strains the virus multiplies in the gut and, like naturally occurring strains, is excreted in the faeces. Also like naturally occurring strains, these live vaccine viruses can spread from person to person. Furthermore, although the vaccine viruses which have been given by mouth are avirulent, they show an increase in virulence after multiplication in the gut of some people, and the excreted vaccine viruses cannot always be differentiated from the less virulent naturally occurring strains.

We have some evidence that naturally occurring strains passing through a community may alter in virulence. There is a wide variation in the virulence of polioviruses being excreted by healthy children, and while there is doubt about the stability of these naturally occurring strains there must also be doubt about the stability of excreted vaccine viruses which can spread from person to person.

May I say here that, although poliomyelitis has been more intensively studied than any other virus over the past ten years, we as yet have no idea why epidemics occur. Epidemics do not seem to be due to changes in the virus strains in the community. As Dr. Ritchie Russell has said, the paralytic case-fatality rate is now apparently uninfluenced by the nature of the epidemic. We have laboratory evidence which indicates that viruses recovered from paralysed individuals during a year of low incidence of the paralytic disease cannot be differentiated in the laboratory from epidemic strains.

Conclusion

We must conclude, then, that there is a host change which plays a large part in determining whether or not an epidemic will occur. It is not clear what this host factor is that permits a virus, which may be silently passing through a community and immunizing it one year, to produce an epidemic the next year. We have no idea why the virus should multiply better in some families than in others or why a particular child should become paralysed or even why poliomyelitis outbreaks are seasonal. If we only knew what the host factors were in determining whether the outcome of infection with a poliovirus was paralysis or a clinically unrecognizable immunizing infection we might be on the road to preventing the paralytic disease without using the formalized Salk type of vaccine.