Little-Known Facts About Poliomyelitis Vaccinations

Mass vaccinations against poliovirus, also in conjunction with intramuscular antibiotic injections and vaccines given against a range of diseases, resulted in significant levels of paralysis especially among young children.

Part 2 of 2

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Outbreak in Oman (continued)

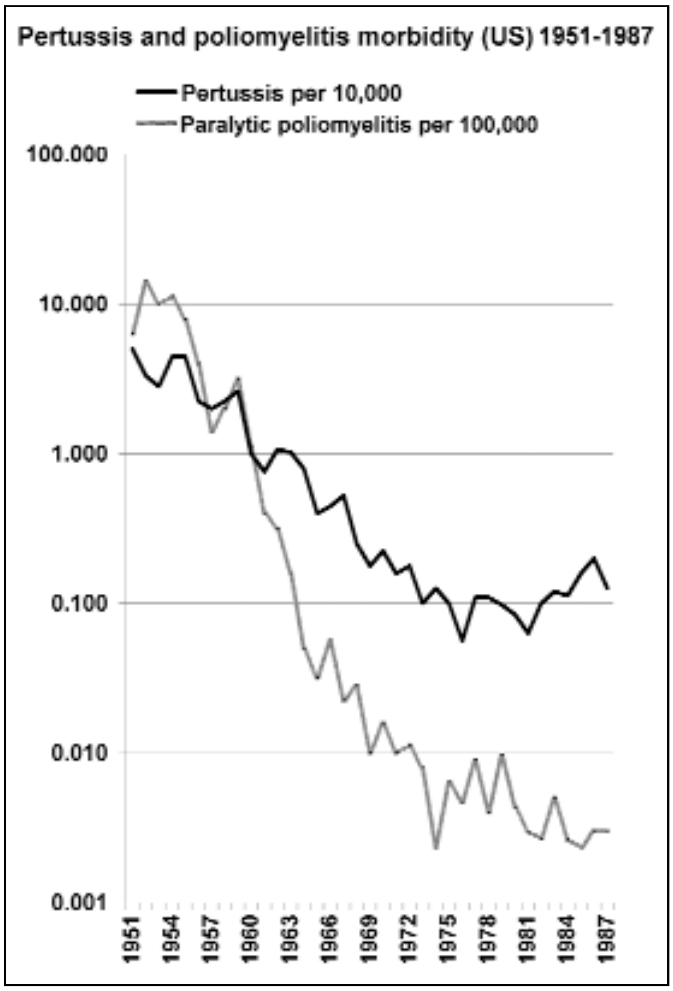
Sutter *et al.* (1991) also wrote: "Among the most disturbing features of the [paralytic poliomyelitis] outbreak [in Oman] was that it occurred in the face of a model immunisation programme and that widespread transmission had occurred in a sparsely populated, predominantly rural setting." This represents further evidence that vaccination caused the outbreak. The vaccinators had travelled into sparsely populated communities.

Sutter *et al.* (1992) reviewed vaccination records for 70 children aged 5–24 months with poliomyelitis and from 692 matched control children during a poliomyelitis outbreak investigation in Oman. "A significantly higher proportion of cases received a DTP vaccine injection within 30 days before paralysis onset than did controls (42.9% vs. 28.3%). The proportion of poliomyelitis cases that may have been provoked by DTP injections was 35% for children 5–11 months old." They concluded that their study confirmed that "...injections are an important cause of provocative poliomyelitis. Although the benefits of DTP vaccination should outweigh the risks of subsequent paralysis, these data stress the importance of avoiding unnecessary injections during outbreaks of wild poliovirus infection."

The fact is that previous injections of other vaccines (such as those containing a pertussis component) causing provocation paralysis was described in the 1950s (for instance, McCloskey, 1950). So, the situation in Oman was just another example of the phenomenon of provocation paralysis. However, time and again, mass vaccination programs have ignored this important fact and continued causing suffering and disability to children all over the world. Another important well-known fact is that the significant majority (65 per cent) of recipients of any vaccines actually get the disease which the vaccines are supposed to prevent, after the first dose (Hedrich, 1933). Hedrich studied outbreaks of measles for 30 years in the Baltimore (USA) area. He established that when about 63 per cent of susceptibles get measles, an epidemic stops. Strebel *et al.* (1992) wrote that vaccine-associated paralysis in recipients of OPV usually occurs after their first dose. In Oman (and elsewhere), those who became paralysed after the first dose were simply excluded from efficacy calculations as unvaccinated or such vaccinations "were not counted".

Sutter *et al.* (1993) published an article on another outbreak in Oman after the post-vaccination polio outbreak of 1988–89. For obvious reasons I cannot quote the entire article, so I highlight certain sentences which reflect the observed reality. The authors wrote: "Investigation of the outbreak suggested that its occurrence was due to several factors, including accumulation of children susceptible to poliomyelitis due to a reduction in overall immunity levels from exposure to wild poliovirus in 1987–1988, suboptimal efficacy of trivalent oral poliovaccine (OPV), provocation

poliomyelitis from antecedent injections with DTP vaccine, and participation of fully vaccinated children in the chain of transmission... A total of four laboratory-proven cases occurred in 1991. The first two cases occurred in the Batinah region in March 1991 (44- and 49-months-old children), both of whom received four doses of OPV. Two additional cases (25 and 30 months old), all after 5 doses of OPV, occurred in August and October 1991 in adjoining Eastern and Interior regions. The same genotype of wild type 3 poliovirus was isolated in all of them." They concluded that the experience in Oman indicates that uniform implementation of the present WHO strategy "may not be sufficient to interrupt transmission" and that several additional doses of OPV to all children may be needed. (Obviously, no doses would do the trick.)



Paralytic poliomyelitis outbreaks in Romania

According to Strebel *et al.* (1994), although poliomyelitis due to wild virus infection had virtually disappeared from Romania, with no cases having been documented between 1984 and 1989, vaccine-associated paralytic poliomyelitis was reported at very high rates for over two decades. In November 1990, to decrease the risk of vaccine-associated paralytic poliomyelitis, oral poliovirus vaccine produced in Romania was replaced by imported oral vaccine produced by a Western European manufacturer. The overall risk of vaccine-associated paralysis in Romania was 14 times higher than the "reported" risk in the USA. However, the risks of recipient vaccine-associated paralysis relating to the first dose of oral vaccine were similar for the Romanian and imported vaccines.

The word "reported" is crucial due to a chronic endemic underreporting of any "vaccine-preventable diseases" after the introduction of mass vaccination, which consequently seemingly improves the efficacy and masks the real risk. All this is further compounded by a new definition of the disease poliomyelitis introduced after mass vaccinations were started in the 1950s and 1960s. The classical definition of poliomyelitis is "a disease with residual paralysis which resolves within 60 days"; the new definition is "a disease with residual paralysis persisting for more than 60 days". Since only less than one per cent of the cases develop a residual paralysis persisting for more than 60 days, the new definition "eradicated" the vast majority of cases in which paralysis resolved within 60 days as *not* being poliomyelitis. Strebel *et al.* (1994) wrote: "Cases are confirmed if they meet the following definition: an acute illness characterized by flaccid paralysis which is compatible with the clinical presentation of poliomyelitis in the acute phase and residual neurologic deficit 60 days (or later) after the onset of paralysis."

Additionally, cases were defined as "vaccine-associated" if there was no direct evidence of wild poliovirus infection and if there was a positive history of recent exposure to oral poliovirus vaccine. This definition is interesting in that it highlights the fact that vaccine-caused paralysis acquired very high significance. Just in case someone may think that vaccination eradicated the wild poliovirus in the environment (as claimed by the vaccinators), note that natural infections with wild poliovirus resulted in the development of natural immunity without paralysis. The outbreaks of paralysis were directly connected with the mass administration of a variety of vaccines, starting with smallpox and continuing with diphtheria, tetanus and, especially after World War II, all the other vaccines. A great number of articles have been published about "provocation poliomyelitis", meaning "provoked by prior injections with a variety of vaccines".

Even in the case of Romania, Strebel *et al.* (1995) wrote about intramuscular (IM) injections within 30 days of immunisation with oral poliovirus vaccines as a risk factor for vaccine-associated paralysis. They wrote: "In Romania the rate of vaccine-associated paralytic poliomyelitis is for unexplained reasons 5 to 17 times higher than in other countries. Long ago it was noted

that intramuscular injections administered during the incubation period of wild-type poliovirus infection increased the risk of paralytic disease (a phenomenon known as 'provocation' poliomyelitis). We conducted a case-control study to explore the association between intramuscular injections and vaccine-associated poliomyelitis in Romania.

"Of the 31 children with vaccine-associated disease, 27 (87 percent) had received one or more intramuscular injections within 30 days before the onset of paralysis, compared with 77 of the 151 controls (51 percent) (matched odds ratio, 31.2; 95 percent confidence interval, 4.0 to 244.2). Nearly all the intramuscular injections were antibiotics, and the association was strongest for the patients who received 10 or more injections (matched odds ratio for more or equal 10 injections as compared with no injections, 182.1; 95 percent confidence interval)."

So, the risk of paralysis was strongly associated with injections given *after* the oral polio vaccine, but not with injections given *before or at the same time* as the vaccine. However, in all cases, OPV was given simultaneously with DPT vaccine.

Interestingly, as reported by Strebel *et al.* (1995), the timing of the onset of paralysis, with IM injections given after DPT and OPV, was 9–30 with a median of 16 days (the highest risk being at 8–14 days, 15–21 days and 22–30 days), and 0–7 days and 15–21 days with DPT and OPV injections given before the onset of paralysis. This reflected the phenomenon of critical days as discovered and defined by Scheibner (2004).

The same situation of poliomyelitis occurring in fully vaccinated children, usually straight after mass vaccination drives, has occurred in many other countries, both developed and developing.

Poliomyelitis epidemic in The Gambia

Otten *et al.* (1992) and Deming *et al.* (1992) reported on the epidemic in The Gambia of poliomyelitis associated with type 1 poliovirus involving 305 cases (estimated 1986 population of 768,995) from May to November 1986, following a six-year non-epidemic period with only five reported cases. The highest attack rate was in one-year-old children: 394 cases per 100,000 of population. The national attack rate was 40 per 100,000 of population. A vaccination coverage survey showed that 64 per cent of one- to two-year-old children were vaccinated with at least three doses of trivalent oral polio vaccine at the beginning of the epidemic. Fifty-seven cases became paralysed more than two weeks after a national mass vaccination campaign, in which 95 per cent of children aged one to seven years old were reported to have received a dose of trivalent oral polio vaccine. The authors concluded that the mass vaccination campaign may only have been partially successful in ending the epidemic.

Wyatt (1987) addressed another well-known problem of provocation poliomyelitis caused by injections of DPT together with OPV in The Gambia. This phenomenon was addressed in the above section on Romania.

Paralytic poliomyelitis outbreak in Namibia

Van Niekerk *et al.* (1994) described an outbreak of paralytic poliomyelitis in Namibia. They wrote: "The last confirmed cases of poliomyelitis in Namibia had been reported in 1988. However, between November 8, 1993, and January 7, 1994, 27 cases of paralytic poliomyelitis were confirmed in the country. The outbreak was limited to the south health region; at least 80% of infants in this region have received four doses of oral poliovaccine (OPV) by the age of 1 year. The patients ranged from 13 months to 12 years; 24 were younger than 5 years. Of the 26 patients whose vaccine status was known, 14 had received four doses of OPV, 6 had one or two doses, and 6 no vaccine." The normal health services and hence vaccination in the north had been severely disrupted by a long war. Interestingly, the authors reasoned that since due to poor vaccination efforts the wild poliovirus was circulating freely in the northern health region, children developed solid immunity to it (without developing paralysis).

This interesting and important information was repeated by Biellik *et al.* (1994), who wrote: "In late 1993 a poliomyelitis epidemic occurred almost exclusively in residents of the south health region [the area that was highly vaccinated]. We speculate, therefore, that endemic wild poliovirus circulation continued uninterrupted in Angola and the two north regions of Namibia across the well-travelled border since 1989, when cases were last reported. Although OPV coverage was fairly low in northern compared with southern Namibia, a higher proportion of northern children may have been protected, at least to type 1, by natural immunity, thus suppressing epidemics. In 1993 OPV3 coverage among infants aged less than 1 year was higher in the south than the north. However, evidence suggests that a substantial pool of susceptibles,

especially among children aged 1–3 [years], was created when [vaccine] coverage was low, and the apparent interruption of wild poliovirus circulation limited the acquisition of natural immunity [in the well-vaccinated southern health region]."

The same situation of poliomyelitis occurring in fully vaccinated children, usually straight after mass vaccination drives, has occurred in many other countries, both developed and developing. The difference was in the truthfulness in reporting.

Mechanics of vaccine-associated paralysis

There is more than one aspect to the mechanics of vaccine-associated paralysis. One of the most important suspects is increased neurovirulence associated with a single nucleotide change in a noncoding type 3 poliovaccine genome.

Evans *et al.* (1985) wrote: "Most of the small number of cases of poliomyelitis which occur in countries where Sabin's attenuated poliovirus vaccines are used are temporally associated with administration of vaccine and involve polioviruses of types 2 and 3. Recent studies have provided convincing evidence that the Sabin 2 and 3 viruses themselves may revert to a neurovirulent phenotype on passage in man [meaning babies] ... a point mutation in the 5' noncoding region of the genome of the poliovirus type 3 vaccine consistently reverts to wild type in strains isolated from cases of vaccine-associated poliomyelitis. Virus with this change is rapidly selected on passage through the human gastrointestinal tract. The change is associated with a demonstrable increase in the neurovirulence of the virus."

Not only has mass polio vaccination *not* eradicated paralytic poliomyelitis, it has caused a number of outbreaks of paralysis directly linked to the administered vaccines.

Inherent problems with inactivation of viruses (including those contaminating polio vaccines) were already known as early as 1961 and 1962.

Gerber *et al.* (1961) described inactivation with formaldehyde which is subject to asymptotic factor, meaning that within about 40 hours most viruses are inactivated but afterwards there is a viable residue of live viruses indefinitely.

Fenner (1962) described reactivation of animal viruses: "It is still a common practice among medical men [and women] to speak of 'killed' and 'live' viral vaccines, and the everyday meaning of the terms is clear enough. But, as I shall demonstrate, virologists now recognise a variety of situations in which 'killed' virus may multiply and produce new infectious virus. They have therefore discarded the term 'killed' and adopted the word 'inactivated' to replace it. Even 'inactivated', however, is used in a restricted sense; it refers to the loss of viral infectivity—that is, to the inability of the virus particles to multiply and produce a new infectious virus in susceptible cells, when these cells each receive only single particles of the inactivated preparation, and no other virus particles or derivates thereof." More recently, inactivation has been used as a method of studying the structure and function of viruses. "This approach received its principal stimulus from the discovery that inactivation was sometimes reversible." (Fenner, 1962)

Little attention has been given to viral intracellular reactivation. Multiplicity of reactivation of UV-irradiated influenza virus was demonstrated in 1951, and cross-reactivation was shown to occur with the same virus in 1956 and 1961. Recently-irradiated vaccinia virus was shown to undergo both multiplicity and cross-reactivation (Fenner, 1962). (In my opinion, this shows the fallacy of irradiating food: irradiated bacteria are only temporarily weakened and revert to their original virulence.) In 1936, Berry and Dedrick (quoted by Fenner, 1962) had already demonstrated that some rabbits inoculated with a mixture of heat-inactivated myxoma virus and active fibroma virus died of myxomatosis. These data emphasise the dangers of injecting dubiously "inactivated" or "non-genetically" reactivated viruses.

Published orthodox medical literature has documented many outbreaks of paralysis connected with mass vaccination programs time and again and in many countries. In this article, I've only described a few examples, but it would be easy for the interested reader to search the existing literature for more examples.

Conclusions

Not only has mass polio vaccination not eradicated paralytic poliomyelitis, it has caused a number of outbreaks of paralysis directly linked to the administered vaccines.

These days, when a vaccinee develops poliomyelitis, it may not be called poliomyelitis; instead, it may be called viral or aseptic meningitis, ascending paralysis (Guillain-Barré syndrome), cerebral palsy (over 75 per cent of cases are not diagnosed at birth but after six

months) or other such names. According to MMWR (1997; 32[29]:384-385), there are 30,000 to 50,000 cases of aseptic meningitis every year in the United States. Considering that the vast majority (99 per cent) of the reported cases in the pre-vaccine era were non-paralytic and would have corresponded to aseptic or aviral meningitis, then vaccination has actually increased the incidence of poliomyelitis. In the pre-vaccine era, such high numbers only occurred in some epidemics. Now, such numbers occur every year, year by year. ∞

About the Author:

Viera Scheibner, PhD, born in 1935 in Bratislava in the former Czechoslovakia (now the Slovak Republic), is a retired Principal Research Scientist. Having studied medicine in 1953 and changed streams in 1954, she graduated in natural sciences in 1958 and was awarded a doctorate in this discipline (RNDR) in 1964 from Comenius University in Bratislava.

Before emigrating to Australia in 1968, she progessed to Senior Associate Professor (Docent) and lectured in biology, micropalaeontology and geology at the university. She had 35 scientific papers and one book published.

After arriving in Australia, Dr Scheibner took up a position as Research Scientist (Micropalaentologist) with the Geological Survey of New South Wales, Department of Mines (later renamed as Department of Mineral Resources). She retired as Principal Research Scientist in 1987, having published scientific findings in a further 47 papers and two books. In the late 1980s, Dr Scheibner was engaged in the study of babies' breathing with the Cotwatch microprocessor-based breathing monitor, developed in conjunction with her husband, Swedish biomedical electronics engineer Leif Karlsson (deceased in 1994). The findings with Cotwatch sparked her interest in the link between sudden infant death syndrome (SIDS) and vaccination, and resulted in her intensive research of orthodox medical literature into the dangers and ineffectiveness of vaccines which continues to this day.

Since 1990, Dr Scheibner has had numerous papers and letters published in peer-reviewed and other journals and has lectured and held seminars in Australia and internationally on vaccine dangers and ineffectiveness. Since 1996, she has provided over 100 reports and appeared as an expert witness for numerous court cases involving vaccine injuries and deaths misdiagnosed as physical injuries by parents and other carers, called "shaken baby syndrome".

Dr Scheibner is the author of *Vaccination: 100 Years of Orthodox Research Shows that Vaccines Represent a Medical Assault on the System* (1993; reviewed in NEXUS, vol. 2, no. 16) and *Behavioural Problems in Childhood: The Link to Vaccination* (2000; reviewed in NEXUS, vol. 7, no. 5). She has previously contributed five articles to NEXUS, most recently "Vaccinations and the Dynamics of Critical Days" (vol. 12, no. 6; first published in J ACNEM 2004; 23[3]:10-14).

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