Polio Immunization Policy in the United States: A New Challenge for a New Generation

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Abstract: The primary reason that live poliovirus vaccine is recommended in the United States today is because it may immunize contacts who have not otherwise chosen to be vaccinated. This policy places contacts at risk of paralysis from an untested, unlicensed "spread virus" vaccine and places infants at risk for an unproven, theoretical benefit to others, not themselves. The licensed killed poliovirus vaccine provides equivalent protection to those vaccinated, with no risk to recipients or contacts. The preceding analysis by Hinman, *et al*, is an interesting exercise in modeling, but many of their assumptions are open to question. Their sweeping conclusions are not justified by the type of analysis performed, which should yield an overall assessment of a decision environment not a single optimal choice. No measure of perceived social consequence or patient attitude is included, although this is of central importance today. Their report lends an aura of credibility to one conclusion, but this credibility is illusory at best. The major social issue today is not which vaccine to use, but how should polio immunization policy be evaluated. (*Am J Public Health* 1988; 78:296–300.)

Introduction

The changing epidemiology of poliomyelitis in the United States and the accumulation of experience with use of killed and live poliovirus vaccines have led to reevaluation of poliomyelitis immunization practices in many sectors. It is important to distinguish the facts that are now available from statements or misconceptions that have arisen out of debates in the past, which were frequently acrimonious and misinformed. Many health care professionals still believe that inactivated poliovirus vaccine is in some way ineffective compared with attenuated live poliovirus vaccine although more than 25 years of experience have led to the conclusion that either vaccine can be used effectively to control wild poliovirus disease.

In 1977, a special committee of the Institute of Medicine/National Academy of Sciences evaluated poliovirus vaccines:

> "Workshop participants and committee members were unanimous in their view that IPV and OPV are surprisingly comparable in their immunizing capability, the persistence of immunity induced, and their demonstrated ability to reduce the incidence of poliomyelitis to the vanishing level."[1]
>
> "The committee concluded that when properly used, either vaccine is highly effective both in preventing disease and in reducing circulation of wild virus in the community."[2]

The growing realization that the beliefs of a quarter century ago are no longer valid has also been reflected in other international meetings and publications.[3–10] With the recognition of the equivalence of the two vaccines in most respects, social, ethical, and practical issues have become important considerations. During the last generation the social and environmental milieu in the United States have changed, as well as the state of scientific knowledge. Wild poliovirus disease has been eliminated and the live poliovirus vaccine is responsible for essentially all domestically-arising paralytic poliomyelitis. The public has become more concerned about the release of potentially dangerous substances into the environment, including genetically selected or altered microorganisms. Litigation by injured parties has become a factor to consider in childhood immunization programs. Patients have demanded more involvement in decisions regarding their own health care.[11]

In view of the need for a clear understanding of the issues by health care professionals and the public, the report by Hinman and his colleagues[12] in this issue of the *American Journal of Public Health* is disappointing. Although their analysis is of some interest, their interpretation results in clouding of the issues, rather than clarification. They appear to have relied on some of the widely repeated but unfounded assumptions of 25 years ago, and the potentially very powerful decision analysis technique has been misapplied. Although ostensibly analyzing all questions relevant to the use of poliovirus vaccines, these authors did not consider the issues that truly lie at the heart of the situation in the United States today.

Decision Analysis Technique

Hinman, *et al*, have presented their work as an application of the decision analysis technique and concluded that it supports the continued primary use of live poliovirus vaccine in the United States. The principal purpose of decision analysis, however, is not to provide a single optimal patient management strategy, but rather to explicate the competing issues in a problem domain.[13,14] The benefit of a decision model is the capacity to ask "What if?"—to vary the values in a model to determine whether the optimal decision changes. This process is called sensitivity analysis and is an essential component of explicating the tradeoffs in a decision situation. By appropriate graphic or computer representation of the data, threshold analysis (which was not done by Hinman, *et al.*) determines the break-even value of a given parameter and these decision thresholds are useful for understanding when one or another action is optimal.[13,14]

Hinman, *et al*, place an inappropriate emphasis on what they refer to as the "base case." Although they use this set of assumptions as a starting point for their sensitivity analysis, it is no more valid than any other set of assumptions. Sensitivity analysis does not test the "base case" or make a more definitive statement about it than about any other variation of the model parameters: this so-called "base case" is not the same thing as a null hypotheses in standard statistical analysis.

Sensitivity analysis of a decision tree model should yield a pattern whereby one can determine which decision would be optimal under which set of circumstances, and it should answer the question: "Which parameters or which assumptions have the greatest influence on the optimal choice?" The

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Editor's Note: See also related Different Views, pp 291, 301, and 304 this issue.

TABLE 1—Numbers of Cases of Paralytic Poliomyelitis in Four Countries that Have Used only Killed Poliovirus Vaccine

	1974	'75	'76	'77	'78	'79	'80	'81	'82	'83	'84
Finland	–	–	–	–	–	1	–	–	–	–	9*
The Netherlands	–	–	–	1	110‡	–	–	1	–	1	1
Sweden	–	–	1	3‡	–	1	–	–	–	–	–
Iceland	–	–	–	–	–	–	–	–	–	–	–

*Type 3 outbreak associated with poor type 3 immunity due to use of low potency vaccine.[18]
‡Outbreak in unvaccinated subpopulation.[16,19]

authors' interpretation of the sensitivity analysis is that there would be more cases of paralytic poliomyelitis and more susceptibles remaining in the population if killed poliovirus vaccine were used. A more appropriate interpretation is that the two most critical parameters in the model are the risk of exposure to wild virus and the number of susceptibles immunized by contact exposure to live poliovirus vaccine. Variations of other model parameters have a much lesser effect on the outcome.

Exposure to Wild Poliovirus

What of the authors' assumptions about exposure to wild virus in the United States if killed poliovirus vaccine were used? They have chosen to draw their conclusions using data from The Netherlands (which they call the "IPV base") rather than data from Sweden and Finland or evidence from several countries, including the United States, that there is no difference in the degree to which killed and live poliovirus vaccines control the circulation of wild poliovirus ("OPV base"). In spite of the authors' implications, there are sound scientific reasons for considering these alternate assumptions to be at least equally valid.

The authors state that Finland and Sweden have "relatively homogeneous population[s] with a more limited pattern of immigration and travel to polio endemic areas." This kind of statement about the Scandinavian countries has been repeated in the literature until many have assumed it to be true, in spite of disclaimers by both Finnish and Swedish authorities and published evidence to the contrary.[15–17] In fact, immigrants form approximately 1 per cent of the total and 10 per cent of the child population in Sweden; and more than 20 per cent of the total Swedish population travel to Mediterranean countries, Africa, India, or Sri Lanka each year (M. Böttiger, personal communication). More than 12 per cent of the total Finnish population travel annually to Mediterranean countries or the Far East.[17]

No sociological data have ever been presented to support the oft-stated belief that Finland and Sweden are somehow so different from the United States that their poliomyelitis experience is not translatable. To the contrary, comparison of the actual experiences reveals striking similarities: in the 20 years after vaccination was introduced, the decline in incidence of paralytic poliomyelitis due to wild poliovirus was remarkably similar in all three countries (see Figure 7 in reference 3). The effect of live poliovirus vaccine during the second half of this period in the United States did not differ from that of killed poliovirus vaccine during the first half in the United States or the entire period in the Scandinavian countries. In both the United States and the Scandinavian countries, there was a decrease in the rate of decline as the incidence became low, reflecting both the relatively smaller role of the herd effect when more than half of susceptibles are immunized and the continued presence of pockets of wild poliovirus in less well vaccinated subpopulations.[3]

Hinman, *et al,* state that they believe The Netherlands experience of repeated introductions of poliovirus is more applicable for the United States than is the Scandinavian experience. They neglect evidence for repeated introduction of poliovirus into Finland and Sweden.[16–18] The major difference between The Netherlands and these Scandinavian countries is in the distribution of their unvaccinated populations. In The Netherlands there are relatively large collections of susceptibles in some Protestant denominations that refuse immunization.[19] There is not necessarily any greater incidence of introduction of virus into The Netherlands; it is simply made visible by the existence of these pockets of susceptibles that allow virus to become established and an outbreak to occur when virus is introduced.

Table 1 shows the poliomyelitis experience in countries that have used killed poliovirus vaccine exclusively and demonstrates that the "IPV base" calculated by Hinman, *et al,* from The Netherlands' data, which is presented by them to be an 11-year average experience, is strongly biased by a single outbreak.[19] The applicability of such a number is arguable at best.

The Delphi survey of experts apparently lends credibility to the assumptions used by the authors in their modeling. The results of the survey are given in a different form than the authors' estimates, however, so it is necessary to convert them to make a comparison. It then becomes clear that the survey data do not agree with the authors' assumptions for the risks of exposure to wild virus in either the IPV or OPV "base case."

The annual risk of infection in susceptibles if killed poliovirus vaccine were used in the United States ("IPV base") is calculated by the authors to be 0.00215 using rates of paralysis in The Netherlands. If, like the authors, one ignores the uneven distribution of susceptibles in The Netherlands and translates this risk to the generally more evenly distributed susceptible population in the United States, then the risk of exposure to wild virus in the nonsusceptible population should be similar, even though such exposure might not lead to infection and would not be manifest as paralytic disease. Assuming a total US population of 230 million, the authors' estimate suggests that there would be 494,500 persons exposed to wild poliovirus in the United States each year. The median value estimated by the participants of the Delphi survey was 100,000 persons exposed to wild poliovirus annually if killed poliovirus vaccine were used, with estimates of five of the nine panelists in the range of 10,000 to 100,000. Similarly, the authors estimate the current risk of exposure to wild poliovirus in the United States ("OPV base") to be 0.0001121, which suggests that a total of 25,800 persons are exposed to wild poliovirus each year. The median value estimated by the participants of the

Delphi survey was 5,000, with estimates of five of the nine panelists in the range of 1,000 to 10,000. There is thus a five-fold difference between the median estimates of the Delphi panel and the assumptions used by the authors for exposure to wild poliovirus in the United States.

Much might be learned by evaluation of a table of the individual responses in the survey of experts, rather than a summary of the median response. The uncertain usefulness of this statistic rather than the mean or mode is demonstrated by the expected outcomes of the model under conditions of the Delphi estimates (Table 5 in Hinman, *et al.*[12]): there is no difference between the median estimates and the "worst case" estimates, although the "best case" estimates result in a much smaller number of polio cases. Even in the summary data presented, which exclude the two largest and two smallest responses, it is apparent that there is a very wide range of opinions among the panelists regarding killed poliovirus vaccine.

The value of a decision analysis is strongly dependent on the acceptability of the underlying assumptions. Not only is there a discrepancy between the authors' assumptions and the opinions of the Delphi panelists, but several of the authors' specific assumptions and calculations are open to question.

Hinman, *et al,* calculate the current risk of exposure to wild poliovirus in the United States based on 10 epidemic, 13 imported, and 8 cases of endemic nonvaccine-associated polio. They make the assumption that each case represents 200 infections in susceptibles and then calculate the number of susceptibles infected with wild poliovirus in the United States each year. One might legitimately debate whether the same paralysis-to-infection ratio (0.5 per cent) ought to apply to the epidemic and "endemic" cases in the current epidemiologic situation in the United States. Nevertheless, there is no question that an imported case of paralytic poliomyelitis does *not* represent 200 infections in susceptibles in the United States: if anything, it represents 200 infections in the country in which the exposure occurred. Inclusion of imported cases in the calculation almost doubles the estimate of the risk of exposure to wild poliovirus in the United States. Hinman, *et al,* use these same 13 imported cases a second time to calculate an independent risk of exposure to wild poliovirus in foreign countries.

Another example of an assumption that is open to question may have less influence on the final results, but is indicative of the care with which assumptions have been made. The authors estimate that if killed poliovirus vaccine is used in the United States, the risk of exposure to live vaccine virus (resulting from importations of vaccine virus from other countries) would be 5 per cent of that if live vaccine were used. This means that the equivalent of 5 per cent of the hypothetical cohort of 3.5 million children, or 175,000 persons entering the United States, would have received live poliovirus vaccine. Immunization is not required for entry into the United States, so the only persons who have received live poliovirus vaccine are those who have been vaccinated by chance just prior to their arrival. To be conservative we can make the maximal estimate that all children entering the United States receive live poliovirus vaccine at the routine time and that the principal period for vaccine virus transmission to contacts is during the two months after the first dose. This means that live vaccine exposure (either as a recipient or a contact) is represented only by those who are two and three months of age, or approximately one-sixth of the entrants less than one year old. According to data from the US Immigration and Naturalization Service and the US Bureau of the Census, the total annual rate of entrance (immigrants, nonimmigrants, and illegal aliens) for those less than one year and those less than five years of age, respectively, is 53,618 and 268,089. One-sixth of the entrants less than one year of age yields a maximal rate of 8,936 live vaccine recipients entering the United States annually. Although it is clearly possible to debate some of the assumptions made in the present analysis, it is difficult to see how modifications could yield a value as great as 175,000 as proposed by Hinman, *et al,* a number that is equivalent to 65 per cent of all entrants less than five years of age.

Contact Exposure to Live Vaccine Virus

Efficacy of the Spread Effect

Hinman, *et al,* conclude that the number of susceptibles will be greater if killed vaccine is used because of the lack of the spread effect of live poliovirus vaccine: i.e., the transmission of vaccine virus from recipients to susceptible contacts. This conclusion is based on estimates of the risk of exposure to vaccine virus and the assumption that the "spread vaccine" has the same efficicacy as the vaccine administered to recipients.

The chance of exposure to vaccine virus is calculated from the number of reported cases of paralytic poliomyelitis among contacts, but it is risky to do so. Rather than being the "tip of the iceberg," contact vaccine-associated paralysis may represent a larger proportion of vaccine virus transmissions than has been assumed. The "spread vaccine" probably carries more risk of inducing paralytic poliomyelitis than the vaccine itself. Thus, the paralysis-to-immunity ratio for contacts is greater than that for recipients and the calculations by Hinman, *et al,* overestimate the number of susceptibles exposed to OPV and underestimate the number of susceptibles that remain in the "OPV base case."

It may not be appropriate to apply the same efficacy (98 per cent) to the "spread vaccine" as to the vaccine itself. The number of effective contacts is estimated from consideration of "sufficient closeness to permit transmission of vaccine virus," and it is assumed that transmission of virus results in an effective infection, i.e., one that results in paralysis and/or immunity. However, the amount of virus transmitted to contacts is very small compared with the amount of virus in a full dose of vaccine, which has been estimated to represent between 1 and 1,000 grams of infectious fecal excretions.[20] A small dose of "spread vaccine" is likely to have less immunizing efficacy than a full dose of the original vaccine.

Hinman, *et al,* state that their sensitivity analysis included variation of the risk of exposure to vaccine virus, but no results are given. Considering the range of possible assumptions, it would be useful to know how variation of assumptions about vaccine virus spread, the number of "effective contacts," and "spread vaccine" efficacy would affect outcomes of the model.

The belief that the spread of vaccine virus would augment immunization rates was first proposed theoretically some 30 years ago. In spite of constant repetition and its aura of common sense believability, this theory has yet to be demonstrated in practice and has been disproved in several settings. A variable amount of spread has been reported from different studies,[21–26] and Fox, *et al,*[23] concluded that spread may not be relied upon to immunize those susceptibles missed during a vaccination program. Despite evidence in Great Britain that attenuated vaccine strains have largely

replaced wild strains circulating in the population,[27] Codd and White[28] reported serologic studies that indicate the spread of vaccine virus does not significantly augment the immune status of a population. Evidence from a recent epidemic in Taiwan[29] also indicates that the benefits of live poliovirus vaccine are confined to those directly immunized.[8]

Discussion

The question raised by Hinman, *et al,* is certainly an important one and the model they developed is potentially useful. However, the data they apply are derived from questionable estimates and assumptions. They inappropriately draw conclusions from a sensitivity analysis, which is intended to present an overall assessment of a decision environment, not to identify a single optimal choice. They do not determine thresholds or present completely the sensitivity analysis they did do "in a format that provides the physician with clinical insight."[13] In contrast to articles that rely heavily on statistics, the authors provide no detailed technical discussion of the analytic and computational methods used: this has been cited as a common inadequacy of reports of decision analyses.[14]

One of the most disappointing features of their analysis, however, is the lack of any measure of perceived social consequences or patient attitudes. The power of the decision analysis technique is that it is explicit: it forces consideration of all pertinent outcomes, it lays open the assumptions made about a problem, and

> ". . . it forces us to consider how patients feel about the quality of outcomes; and it allows us to come to grips precisely with the reasons why colleagues differ about actions to be taken."[13]

Hinman, *et al,* allude only in passing to the different public perceptions of vaccine-associated and wild virus cases. Their model considers all cases of polio as equivalent, without recognizing any difference in social and financial costs. Yet these differences are at the crux of the matter. By not considering them and by not recognizing how patients feel about the quality of outcomes, Hinman and his colleagues fail to come to terms with the moral and social questions that exist about polio immunization policy in the United States today. They fail to come to grips with the real reasons why colleagues differ about the actions to be taken.

What Are the Real Issues At the Present Time?

The principal difference between killed and live poliovirus vaccine is the transmissibility of live virus vaccine progeny. A special committee of the Institute of Medicine recommended continued use of live poliovirus vaccine primarily *because* of this effect: "the spread of immunogenic agents from vaccinated to nonvaccinated persons is maintained."[2] Hinman and his colleagues would appear to subscribe to this view. Melnick has pointed out, however, that "while the spread of live vaccine virus from the vaccinee to household and community contacts is considered by some to be an advantage, in that it may provide 'free immunization' to larger numbers of persons, the fact remains that the virus that spreads to the contacts is not a licensed vaccine . . . [and] would no longer pass the safety tests required of the vaccine itself."[30] Killed poliovirus vaccine "has the advantage of not introducing into the community any living virus that can spread in an uncontrolled fashion to persons other than those who have sought or agreed to receive the vaccine."[30]

The property of transmission of vaccine virus distinguishes live poliovirus vaccine from all other vaccines currently in use. The fact that immunization of contacts by virus spread is an *intended* effect of live poliovirus vaccine distinguishes it from all currently available medical substances and practices: no other drug, medical device, or therapeutic procedure has any effect, much less an intentional one, on someone other than the person being treated.

In 1960, Thomas Francis pointed out the special nature of the questions raised by use of live poliovirus vaccine:

> ". . . I would comment on a public health feature of the entire setup which live vaccine poses to the health officers of the community. Probably for the first time, it is proposed to introduce into a natural population an infectious agent for prophylactic purposes, an agent whose spread cannot be controlled by the responsible agency. In [the United States], at least, it would be interesting to know how this will be handled as a general public health procedure, because this is quite a new undertaking and a new problem which I think may have wide reverberations.[31]

The challenge posed by Dr. Francis remains with us today. In the 27 years since his observation, the issue has been handled in the United States by benign neglect—it has simply not been addressed. Yet it is the central question now facing us.

The primary reason that live poliovirus vaccine is recommended for use in the United States is not for any benefit to the person who receives it, but because it may immunize those who have not chosen to be vaccinated. This amounts to involuntary vaccination, using a vaccine that is known to carry a risk both for recipients and for their contacts. Such an effect might be acceptable if there were no alternative, but a licensed vaccine does exist that provides equivalent protection to those vaccinated, with no risk either to recipients or contacts.

Use of live poliovirus vaccine places infants at risk of paralysis for a theoretical benefit to others, not themselves, and places contacts at risk of paralysis from an untested and unlicensed "spread virus" vaccine without their knowledge or consent. Infants are paralyzed who would otherwise be protected. Contacts of all ages are paralyzed by a disease to which they would not otherwise be exposed: parents and grandparents, uncles and aunts, friends and strangers.

The issue we must address today is who should decide such a policy and in what forum. Is the risk to otherwise normal, healthy infants worth a theoretical benefit to those in the population who for one reason or another have not protected themselves? Is the danger to the susceptible population (which Hinman, *et al,* estimate to be only 2 per cent of the total) sufficiently great to warrant placing more than 95 per cent of our newborns at some risk of permanent paralysis? What is the social, emotional, or even financial value of 10 iatrogenic cases of paralytic poliomyelitis and how does it balance against the value of an equal, lesser, or greater annual number of cases caused by wild poliovirus? These are questions that place poliomyelitis immunization policy squarely in the public sector; it is not an issue to be decided solely by a single group, no matter how expert.

By not coming to terms with these questions, advocates of current poliomyelitis immunization practices place themselves in the role of physician as *the* decision maker, as opposed to *a* decision maker when "patient and physician work as a team."[13] By failing to clearly inform patients, health care providers, and the public of the central importance of the intentional spread effect, they circumvent the

challenge posed by Dr. Francis almost 30 years ago. By repetition of unfounded or disproven litanies about the relative efficacies of killed and live poliovirus vaccines, by repetition of the comfortable cliches of the past, they cloud the real issues and fall victim to the suspicions voiced by Bodian in 1961:

> "Unfortunately, widespread misconceptions concerning the potentialities of both vaccines, published in scientific journals and the lay press, have made policymaking by medical and public health agencies difficult if not dangerous. An example is [a document recently approved by one medical organization] concerning the present status of poliomyelitis vaccination in the United States . . . This document contains assumptions concerning the effects of killed and oral attenuated poliovirus vaccines which in some instances are unproved, and in others have proved to be erroneous.
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> " . . .It is difficult to escape the suspicion that the policymakers have been misled by the widely held misconceptions concerning the evidence relating to the effects of both types of vaccine."[32]

The issue we must face as a society is not "Which polio vaccine should we use?" but rather "How should we go about choosing polio immunization policy?" The principal point for advocacy may be freedom of choice rather than use of a specific vaccine: as was the case 25 years ago, perhaps recommendations should describe the two available licensed vaccines and leave the choice to physicians and their patients. If a policy is to be followed that makes it difficult or even impossible for a physician or mother to choose a safer vaccine for an infant because the policymaker perceives a benefit to someone else, then that policy should be set by a public body in a public forum where the entire spectrum of views can be considered, including those of the individuals and families who are most affected.

It is clearly important that both the professional and lay public be well informed about the issues surrounding poliomyelitis immunization. The report of Hinman and his colleagues lends an aura of credibility to one conclusion, but this credibility is illusory at best. Many may be impressed with the power and apparent validity of a computer simulation, and if so the effect may be to obfuscate rather than to clarify the issues: such a result would be counterproductive. What is more needed today is elucidation of the social, ethical, and moral issues that lie at the root of the modern debate about poliomyelitis immunization policy.

REFERENCES

1. Institute of Medicine: Evaluation of poliomyelitis vaccines. Vols 1 and 2. IOM Publication 77–02. Washington, DC: National Academy of Sciences, 1977; p 32.
2. Nightingale ED: Recommendations for a national policy on poliomyelitis vaccination. N Engl J Med 1977; 297:249–253.
3. Salk, D: Eradication of poliomyelitis in the United States. II. Experience with killed poliovirus vaccine. Rev Infect Dis 1980; 2:243–257.
4. Salk D: Eradication of poliomyelitis in the United States. III. Practical considerations. Rev Infect Dis 1980; 2:258–273.
5. Proceedings: International Symposium on Reassessment of Inactivated Poliomyelitis Vaccine, Bilthoven, 1980. Hennessen W, van Wezel A (eds): Develop. Biol. Standard., 1981; 47:364 pp. Basel: S. Karger, 1981.
6. Proceedings: International Symposium on Poliomyeliltis Control, Washington, DC, 1983. Horstmann DM, Quinn TC, Robbins FC, (eds): Rev Infect Dis 1984; 6(Suppl 2): pp S301–S600.
7. Editorial. Poliovaccine. Lancet 1983; 1:1022–1023.
8. Editorial. Polio reconsidered. Lancet 1984; 2:1309–1310.
9. Curry B: Polio: merits of two vaccines reviewed. *Los Angeles Times*, June 1, 1985; 1, 22, 23.
10. Proceedings, 19th Annual Symposium of the International Congress for Biological Standardization, Amsterdam, 1985. van Wezel AL, Hennessen W (eds): Develop. Biol. Standard., 1986; 65:284 pp. Basel: S. Karger.
11. Karzon D: Immunization on public trial. N Engl J Med 1977; 297:275–277.
12. Hinman AR, Koplan JP, Orenstein WA, Brink EW, Nkowane BM: Live or inactivated poliomyelitis vaccine: an analysis of benefits and risks. Am J Public Health 1988; 78:291–295.
13. Pauker SG, Kassirer JP: Decision analysis. N Engl J Med 1987; 316:250–258.
14. Kassirer JP, Moskowitz AJ, Lau J, Pauker SG: Decision analysis: a progress report. Ann Intern Med 1987; 106:275–291.
15. Gard S: Poliomyelitis in Sweden 1964–65. European Association for Poliomyelitis and Allied Diseases 1967; 11:54–55.
16. Böttiger M, Mellin P, Romanus V, Söderström H, Wesslen T, von Zeipel G: Epidemiological events surrounding a paralytic case of poliomyelitis in Sweden. Bull WHO 1979; 57:99–103.
17. Lapinleimu K: Elimination of poliomyelitis in Finland. Rev Infect Dis 1984; 6(suppl 2):S457–S460.
18. Kinnunen E, Hovi T, Stenvik M: Outbreak of poliomyelitis in Finland in 1984. Scand J Infect Dis 1986; 18:15–18.
19. Schaap GJP, Bijkerk H, Coutinho RA, Kapsenberg JG, van Wezel AL: The spread of wild poliovirus in the well-vaccinated Netherlands in connection with the 1978 epidemic. Prog Med Virol 1984; 29:124–140.
20. Gard S: Exit poliomyelitis—what next? Yale J Biol Med 1961/1962; 34:277–288.
21. Sabin AB, Michaels RH, Spigland I, Pelon W, Rhim JS, Wehr RE: Community-wide use of oral poliovirus vaccine: effectiveness of the Cincinnati program. Am J Dis Child 1961; 101:546–567.
22. Paul JR: The spread of attenuated polioviruses among household contacts. *In:* Poliomyelitis: Fifth International Poliomyelitis Conference, Copenhagen, 1960. Philadelphia: J.B. Lippincott, 1961; 359–367
23. Fox JP, Gelfand HM, LeBlanc DR, Potash L, Clemmer DI, Lapenta D: The spread of vaccine strains of poliovirus in the household and in the community in southern Louisiana. *In:* Poliomyelitis: Fifth International Poliomyelitis Conference, Copenhagen, 1960. Philadelphia: J.B. Lippincott, 1961; 368–383.
24. Zacek K, Adam E, Adamova V, Burian V, Rezacova D, Skridlovska E, Vaneckova N, Vonka V: Mass oral (Sabin) poliomyelitis vaccination: virological and serological surveillance in Czechoslovakia, 1958–59 and 1960. Br Med J 1962; 1:1091–1098.
25. Horstmann DM, Emmons J, Gimpel L, Subrahmanyan T, Riordan JT: Enterovirus surveillance following a community-wide oral poliovirus vaccination program: a seven year study. Am J Epidemiol 1973; 97:173–186
26. Rossier E, Phipps PH, Pepin O: Benefits and risks of attenuated polio vaccine strains in a community immunized with inactivated polio vaccine. Dev Biol Stand 1979; 43:179–185.
27. Cossart YE: Evolution of poliovirus since introduction of attenuated vaccine. Br Med J 1977; 1:1621–1623.
28. Codd AA, White E: Protection against poliomyelitis. Lancet 1977; 2:1078.
29. Kim-Farley RJ, Rutherford G, Lichfield P, Hsu ST, Orenstein WA, Schonberger LB, Bart KJ, Lui KJ, Lin CC: Outbreak of paralytic poliomyelitis, Taiwan. Lancet 1984; 2:1322–1324.
30. Melnick JL: Advantages and disadvantages of killed and live poliomyelitis vaccines. Bull WHO 1978; 56:21–38.
31. Francis T: Panel Discussion. *In:* Poliomyelitis: Fifth International Poliomyelitis Conference, Copenhagen, 1960. Philadelphia: J.B. Lippincott, 1961; 412–413.
32. Bodian D: Poliovirus immunization. Science 1961; 134:819–822.