A So that it was not a question of taking a biologically different strain or an antigenically variant. It was the same thing except that it didn't have the neurovirulence as the parent Mahoney virus. It was available.

Q The thing that I basically cannot understand: all of these things are available. There is a virulent Brunhilde strain that Enders developed that the English had used. Would it have stopped the vaccination program for any great period of time?

A Well it might have stopped it for a month, two months or postponed it for a month, two months, until you carried out, until you made formalized vaccine from the attenuated Mahoney let's say or attenuated other strains and compared it for potency in monkeys.

Q So that is the time we are really talking about?

A What was the date of that congressional hearing again?

Q In June of 1955.

A Alright. Now you must recall that during that era July, August, September were the months of high incidence of paralytic polio. And in all fairness, those who wanted to go ahead without multiplication thought that more good would be done by starting because vaccine had already been prepared, filtered and so on by the time it got out—to start using it in 1955 right away, rather than postponing it that some could be protected. Actually, there was not
much effect in 1955. I mean the incidence of polio was still pretty high despite this. But I think that this was a dangerous decision. It was a decision furthermore that could have been made—that well, we will do what we can with what we have but at the same time we will start tests to determine whether or not substitution of an attenuated virus for the virulent Mahoney would make a difference. But there were so many really sub-surface factors that enter into this. You must remember that there was a group of people there around Basil O'Connor, Tom Rivers who is St. Paul, Jesus Christ, who felt that any variation from the dogma that was laid down for the field tests was heresy. That it was in a sense a criticism—a criticism which could not be tolerated. It reminds you so much of fanatical religious dogmas. There was a good deal of that. There was a good deal of that, that any variation from the dogma was in effect a criticism of the people in charge and they couldn't tolerate the criticism. You can see that it was malicious. Suggestions were malicious

Q Alright, I—

A And I take issue here with those of my respected colleagues Horstfall—I don't know. You can't say how much the fact that they were sort of scientific sons of Tom Rivers—had an impact—

Q Well you were a scientific son of Rivers—

A Horstfall and Smardell and others who joined and I remember it was Cullen McCloud who joined with Tommy
Francis said no chance. I cannot go along with whatever thinking was there because while they could have said alright now let's do what we can right away to see if we can prevent as many paralytic cases as possible—if this is the logical place. But at the same time begin those tests which cannot take more than two or three months at the most to see whether substituting an attenuated strain or the most virulent Mahoney virus would be equally, would provide equal potency.

Q You know why I raised this question. I raised this question because there were modifications in the production plans. There were modifications in filtration, in particular, and those modifications had to be put in anyway.

A And they weren't tested. For example, if they had been tested, they would immediately have found as I have already said that the modification of filtration greatly reduced the potency. So what they did was to pass the lots on minimum potency. Well minimum potency is not quite what it was in the field trial. You see, fallacious thinking is not necessary something that may not be used by very respected scientists. Only time shows who is fallacious and who is not. It is nothing new.

Q Now, it has now been close to a quarter of a century after, almost a quarter of a century after—would you modify your criticism in any way? Do you see things now that you didn't see during this heat of battle?
A Well the things that we discussed I think have proven that those who thought the way I did—or—I mean this group—I was perhaps the most vocal, but I certainly wasn't alone—proved to be right. As regards going ahead with the most extensive use of killed virus vaccine until something else became practical or feasible, I was on record in print that I was all for it. So I don't see any reason for retracting. So I see no reason to change.

Q In other words, you would stay by your reviews that you made in '58 and subsequently of Salk vaccine. But it is a matter of record that you urged the use of Salk vaccine.

A Exactly right.

Q Okay. That's—

A And what's also on record if you want to look it up because I don't remember the exact wording in the Lascroll (?) Award Lecture that I gave in 1965 I have a paragraph in which I indicated how very much was gained from the use of Salk vaccine during the years when something else was not available. But I did deplore and I continue to deplore the tactics that were used to misrepresent things subsequently. During the mass campaigns the tactics that were used by the National Foundation to prevent such mass campaigns, communities engaged in it. Actually the record shows that after five years or so of use of Salk vaccine in the country there was the very desirable and laudable reduction from
let's say roughly a hundred paralytic cases on the average per annum per million total population to twenty-five per million, that the introduction of the live virus vaccine in the United States ultimately reduced it to three suspicious ones—not even definite ones per hundred million, that the change is quite extraordinary and yet the National Foundation tried to block it because again there is that peculiar mentality that anything that might represent some sort of criticism of what was done was just unbearable.

Q One of the things I found of interest was your really looking critically at the question of associated illness with Salk vaccine. As a matter of fact, you are very—you defended Salk on some of the things that--

A I don't remember all the situations now. I remember pointing out that there are many things that can occur shortly or within a month or so after the administration of the vaccine that would be blamed on the vaccine but wouldn't be reasonable. But on the other hand, as I pointed out later I think that the position of the Public Health Service that not considering the possibilities of the small number of perhaps one per hundred thousand or one per million—that was really caused by the vaccine was not defensible. It was still guilty of it. They hide behind weasel words. There is We know now that this probably every possibility that there is as much Guilliam Barre after Salk vaccine as their is after killed influenza vaccine. And after killed influenza vaccine we know that one, that for every hundred thousand that receive the dose of killed influenza vaccine there was
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one case. One per hundred thousand of Guilliam Barre and personally I think that the balance of probability that the ten, fifteen or more cases that occurred in the year at the site of inoculation—the paralysis beginning at the site of inoculation are probably due to small amounts of virulent virus that couldn't be detected even when you test a hundred or a thousand ml of vaccine. But when you give it to a hundred thousand human beings with the most virulent virus, you do detect it.

Q Dr. Sabin,  
A Incidentally, one of the Canadian firms—Connolt that has continued to make killed virus vaccine still is using the Mahoney virulent virus.

Q What happens to personal relations revolving around a scientific debate like this?

A Well, there is a time when it is not, does not effect personal relationships when it is a matter basically of scientific principle that ultimately is established by time. But when it reaches a point where personal comments are made that are not for the resolution of the scientific question, they deteriorate then.

Q It's a grand pity.

A Well, I think history shows that there is a great deal of that. I have been amused over the years to see some of the plays about Pasteur, you know there are so many plays about Pasteur. My God some of them—of course
I mean Pasteur wasn't a very critical guy. But he thinks you see he was a fine chemist that even though he got Dr. Route to be with him and some of the decisions he made were open to criticism. And some of the conclusions are open to criticism. But nevertheless, the epitimous of some colleagues, some members of the Academy and all that was above and beyond the need for resolving points of scientific principles.

Q This question of criticism I find probably the most interesting thing. I remember when you were discussing the Russians, you made the point that basically they were good scientists but they didn't have critical apparatus.

A They were honor scientists.

Q Honor scientists.

A They were not good scientists. They were honor scientists who lacked a certain critical discipline, and that is a very important part of the scientific endeavor. It doesn't come easily. And it is not only Russian scientists. Many American scientists and others also don't have that critical discipline.

Q Where does that come from? Does it come from your upbringing?

A Well, it comes from a certain amount of fundamental training which is self training and other because the rules which of evidence must be applied in scientific evaluations can certainly be no less than those that have to be followed in the law. Some evidence is never admitted in court and
also should not be admitted in the scientific evaluation either. And yet it happens.

Q Well, I have one other thing to question you about and that has to do with some of your later work on toxoplasmosis, particularly the work that you did with Harry Feldmann. I don't know how Harry Feldmann came to work with you and I don't know what you precisely did with Harry Feldmann and I wonder if we could deal with that now.

A Well, while I was still at the Rockefeller Institute in 1937, '38, when I was doing other things, I encountered this unknown parasite which I began to try to identify and it turned out to be toxoplasmosis I was fascinated by it because here was a large parasite that was an obligate intracellular parasite like the virus was--couldn't multiply outside of the cell--at that time we didn't know enough about viruses to realize the different mechanisms of intracellular parasites. But at any rate, I continued to work with it. The next thing that happened was that certain congenital diseases which killed infants were reported from Columbia and looking at it I got the feeling that what they were seeing and didn't realize was actually toxoplasmic infection transmitted in vitro and then when I came to work in Cincinnati in 1939 and was working on all central nervous system disease in children I very quickly found two cases of encephalitis in older children that turned out to be--so I continued to work on this as a side occupation sort of moonlighting effort on
the role of toxoplasmic infection and manifestations in human beings and how to be able to diagnose the discretion during life.

Q How does one make an early diagnosis of toxoplasmosis?

A Well at that time you see first, you couldn't cultivate it and I had developed a test in rabbits. You couldn't make an early diagnosis because except for certain instances where you had a very generalized disease in adults—because the mothers who gave birth to infants with congenital disease were perfectly alright. Well you would have to demonstrate it actually post mortem. It was all done post mortem. And when I had developed a technique of perhaps getting some data, some tissue and inoculating rabbits, inoculating mice that there was a way of proving infection. But as I continued, I was trying to develop a test outside the body and here again is how things connect up. I mentioned Pasteur and his work. You can trace a line one to the other. When I was still working with pneumococci I developed this test with metholyn blue in which I added serum to the pneumococci and metholyn blue and watched the swelling of it and you could see it under the microscope very fast. So in working with toxoplasma I wondered what would happen if I would add serum to the toxoplasma and then added some
methyln blue. This is going way back there. And I saw a very unusual phenomenon which then had to be worked out because it was quite complex and that was that when toxoplasma in suspension let's say, in the peritoneal extra data (?) that we had at the time were alive, free, they would take up the dye and the material in the scitoplasma became very brilliant. But after they reacted with specific antibodies and particularly in the presence of certain accessory factors--they had to have accessory factors--a change happened. The scitoplasma no longer took up the dye. Very remarkable. Well that had to be worked out very carefully and at that time Harry Feldmann who had been working at the Walter Reed asked to come to work with us. He actually came to work with me (inaudible)

Isolate measles virus. This was '48. And so I put Harry Feldmann on to work with me on this and step by step we worked out from the initial basic observation a practical useful test for the detection of antibody against toxoplasma which became the basis of such tests throughout the world and I think within the subsequent ten years there must have been two thousand publications of something on that. We published it together in 1949 in *Science*. 
Q You know I am interested here apparently Harry Feldmann came to you—probably recommended.

A Well I mean he had worked for Smardell. He had worked with others. I think I may have encountered him in working during my associations at Walter Reed but actually he had been involved during the War in work on malaria and other things. He hadn't worked on viruses.

Q You didn't put him to work on polio. That is the first thing. Why didn't you put him to work on polio?

A Well actually, we, the work on polio at that time was being pursued at a lower level and I already had people with whom I was continuing the polio work. This is a period right after the war. We were very much taken up with encephalitis, particularly Japanese encephalitis. You may recall that I went out to Japan and Korea and China in 1946 and again in 1947 there was a great concentration of work on Dengue and so on so the work on polio was less and when he came to work with me it was more to see if the measles virus could be isolated by certain techniques, in animals.

Q One of the things in reading one of your papers on—

A Incidentally, excuse me. Although I stopped work myself on toxoplasma I think as of 1950 or something like that. This has remained one of Harry Feldmann's main occupations for the rest of his career.
Q I know that and this was one of the things I was going to make comment about: that even though a scientific son leaves the laboratory, in essence he does not leave home.

A Well, if, if something, if he became involved in something that has relevance on problems that are world-wide and is not just a passing--

Q The thing that I find of interest is that when people would write to you about toxoplasma you would immediately refer them to Harry Feldmann.

A I said I didn't do it any more. My laboratory wasn't engaged anymore.

Q That is what you said, but you would also, you know, my laboratory is not engaged any more. Period. But then you would say Harry Feldmann at Syracuse University is now engaged in that.

A I think I said more than that because I had very high regards for Harry Feldmann and very warm personal relationship has remained over the years. So I could say that if you really want a critical evaluation, get Harry.

Q Yes, alright. There was one thing in one of your papers which amused me. You said you were guilty of a crime. Guilty of a crime of identifying a disease by guilt by association so if someone showed up with coreoretinitis, toxoplasma is the result of that, and I never thought of you the crime of guilt by association.
A Well I don't remember exactly. I would have to see the tests. But what happened was because we made available this diagnostic test and because the work which Harry and I subsequently did on a lot of clinical cases show that coreoretinitis in the congenital syndrome, children who are born with toxoplasmosis was almost invariable. We carried out an extensive study on patients with coreoretinitis--older ones--because coreoretinitis leads to blindness in the eye. It not an uncommon condition so that this test became very popular with clinicians, ophthalmologists, and so they would get a patient with coreoretinitis for which there would be no other explanation. They would do a test and if they found that it was positive, they would say this is toxoplasma coreoretinitis, without realizing that in that particular population group if they took anybody with coreo coreoretinitis or without retinitis they would have a 40, 50 percent chance of having it anyway, having a positive test because inapparent infection with toxoplasma was so widespread in the community that merely finding a positive test didn't mean that the syndrome that had many causes was necessarily caused by toxoplasma. And this was guilt by association. That simply because the person happens to have had an infection of toxoplasma some time in his life, that the coreoretinitis that he had was necessarily that. And as a matter of fact,
Harry Feldmann and I in certain studies of other congenital conditions that gave rise to coreoretinitis, we could quickly prove that there were other causes of coreoretinitis and that we had very rigid criteria by which to make a serologic diagnosis of congenital toxoplasmosis, which the ophthalmologists were not using, so I was guilty of contributing towards looking at an ideological diagnosis on the basis of guilt by mere association—as was done for many years when the Wasserman test for syphilis was used. You would have a condition you couldn't explain, you would do a Wasserman test—the Wasserman was positive, ipso facto, post hoc/propter hoc. But it was not otherwise I don't think I was guilty very often of incriminating certain agents by simple association where it could be established that it was producing widespread inapparent infection. Plus we had that problem with the tremendous number of enteric viruses—viruses in the intestinal tract—the echo viruses of which we discovered so many new ones in my work with Romulus Alvarez and the cosacky viruses. There was a problem created of having viruses which one had to use very, very strict criteria, strict groves of evidence to incriminate the cause of effect and relation.

Q You are mentioning strict criteria. One of the things that strikes me is that you would not accept a diagnosis of toxoplasma on morphological grounds alone.
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A Well there was a period when this was common. There was a common, or rather a high incidence of erroneous diagnosis on morphologic grounds. This particularly in sections, some other organisms may resemble toxoplasma. This was early in the game and that's why we insisted that the parasite actually had to be isolated by animal inoculation and then its relationship to the disease shown by other procedures.

Q Good. I think we will stop here.

A Stop here?

Q And--

END OF TAPE