A Using these freshly obtained sera from about forty multiple sclerosis patients and a comparable number of sera that I had frozen from the Chillicothe volunteers, I carried out a neutralization test in mice in which the neutralizing capacity of both sera were tested by a procedure in which the number of mouse infective doses that could be inactivated by multiple sclerosis sera was compared with that which could be inactivated by the normal sera. I would like to say at this point here that the results of that tests were completely in accord with the observations of the Soviet investigator. Namely that the multiple sclerosis sera neutralized and the normal sera, none of them that I had stored about -20° centigrade for different periods of time from the volunteers at Chillicothe, didn't. Now stop here because--

At this point I would like to read into the record the summary of the work that we then did in the laboratory with introduction. It is contained in my report to the N.F.I.P. for the period of July 1, 1957 to June 30, 1958. And I give the background. In 1946 Margoulus Soloviov and Shublatza. Shublatza is his wife. It isn't my friend Soloviov, reported the isolation of two identical strains of virus from two patients with acute disseminated encephalomyelitis and expressed acute disseminated encephalomyelitis is another name for multiple sclerosis in the acute phase. And expressed their belief that this virus was also the cause of multiple sclerosis because the blood of patients with these diseases neutralized the virus while the blood of healthy people and the patients with other
diseases did not neutralize it. Just as we had been able to confirm. A formulaized preparation of rodent brains infected with this virus. The reason I say rodent brains here is because I think it was also rats as well as mice. Infected with this virus constitutes the vaccine that has been used for treatment of multiple sclerosis in the Soviet Union. This was 1956. Incidentally it is still (inaudible). Already in 1954 Shumblotza and Guydomovich, one of her coworkers reported that this virus which they were using as a vaccine against multiple sclerosis was antigenically closely related to rabies. But on the basis of the sera neutralization tests still believed it to be the cause of acute disseminating encephalomyelitis and multiple sclerosis. My own tests with this virus which was given to me by Dr. Shublatza in 1957. I am sorry. It is not 1956. Show the following.

(1) Quantitative serologic tests showed no difference between this virus and that of ordinary rabies. So it was not a question of antigenic relationship but it was rabies. Histologic studies showed that it produced negrebody in the brains of mice characteristic of rabies virus but no demilenating lesions. I was ultimately unable to confirm the findings of the Russian investigators that the blood of patients with multiple sclerosis can neutralize this virus but also found that the blood of healthy people has the same effect provided the sera from the patients and healthy persons are stored in a similar manner. The neutralization by both types of sera is not caused by antibody I discovered then. But by a non specific factor that deteriorates even on prolonged
storage in a deep freeze at -20° centigrade and is completely 
destroyed by heating at 56° for 30 minutes. So here I should 
interject what I actually did when the first test confirmed 
the Soviet observation. I said my God, now what are the 
differences between the sera from the multiple sclerosis patient 
and those from the volunteers in Chillicothe other than the 
fact that these had multiple sclerosis and these did not. And 
like a detective, you know, that is the way we have to work 
in this field also. I went down the different things and I 
came down to the fact that the sera that I got from the 
multiple sclerosis patients I obtained were fresh and the 
others had been stored for six months or something like that 
at -20° centigrade. It shouldn't make any difference to 
ordinary antibody but I said now how am I going to do this. 
We lined up the staff of the lab, you see. We all bled 
ourselves, and I had ten sera, freshly obtained the same way 
as multiple sclerosis, did a simultaneous test and then there 
was no difference. Freshly obtained, normal sera did exactly 
the same thing and they did exactly same thing against this 
Russian virus and against rabies virus. So that my conclusions 
were that the virus used for the preparation of the multiple 
sclerosis vaccine is identical with that of rabies and has no 
ediologic relationship to multiple sclerosis. I did not 
publish this data.

Q You must have told the Russians.

A I told them but it had absolutely no effect on them 
whatever. And then my friend George Dick also got this virus 
and as I said the question about being related to rabies was
raised by Shumblotza the Russians themselves but they overlooked it because of these neutralization. They thought well why shouldn't it be--and then he publishes in I think paper in the Lansing saying it is rabies virus and there is no question that it is rabies virus and that is all. And I forget what evidence George Dick knew of my results. Why didn't I publish it? I have had long standing, now obviously I was at a stage of working with my Soviet colleagues in which I wanted a good relationships of collaboration to continue. But the habit which I developed very early when I couldn't confirm somebody else's work was to go to the investigator whose work I couldn't reproduce, show him my results and ask him what did I do that was different. What did I do, what might I have done wrong. And let me mention the instances where that happened. In 1931 when at Dr. Parks' request I tried to repeat the work on skin tests that Jungeblut published it. He couldn't get the same result. I went and worked with Jungeblut at that time and we published a joint retraction because to me it was much better than create a controversy. I was very happy about it. You remember that later on Simon Flexner while I was at the Rockefeller Institute came to me again with a publication of Jungeblut's who was professor of microbiology in Columbia. He said my, look, my God and he was really all excited. I never saw Simon Flexner so excited. He says, and it was published in the Journal of Experimental Medicine he said if Jungeblut is right that these doses of vitamin c can protect monkeys against the development of paralysis after they are infected starting the treatment soon after infection as possible, he
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says that would be a tremendous discovery. Please Albert he didn't order me. He said please, please Albert. I don't think he said. Please Dr. Sabin. He was always very formal. Take time out. You have an excellent—you have all the monkeys he says and if you need more out of my own budget and Hassel Popo—or whatever his name was—his own technician. He says will help you. You find out whether this is correct. And if it is correct I think we went over that at one time. He said we should really know. This is an important discovery that needs to be—well I did not confirm Jungeblut's observations and again I went to Jungeblut and I said what did I do wrong. And as you remember we did an experiment together in his own laboratory it couldn't be confirmed when I did the infecting but then he would not agree to a joint publication. So, following this example that I had set myself I went and I said, look, the whole basis for your calling this a multiple sclerosis virus as having an epidemiologic relationship even though you say it is related to rabies depends on these neutralization tests. Here is my explanation for the neutralization tests. Please, repeat the work yourself. Take the serum but make sure that your normals and the others are stored the same way, are equally fresh or inactivated. Nothing ever happened. And frankly I did not want to start up a controversy on multiple sclerosis. And besides I was busy with too many other things at that time to sit down and write and so I never published that work. And actually I am not sure whether George Dick in his publication mentioned as a private communication the explanation that I found for this.
Q Well we could always look that up. But you became intrigued with the problem of multiple sclerosis and you did some little work trying to see the relationship between herpes virus and multiple--

A This may be a good time to mention this, because while we were working with herpes virus particularly in relation to the development of the vaccine against polio because I had isolated a strain of herpes virus from a child who died with encephalitis about that period with very unique properties. And in studying the lesions and intracerebrally inoculating mice and also with "B" virus in mice I observed areas of demilinization in the white matter. And this incidentally was also found in the first patient, Brebner, who died of it. So I said to myself is it possible that patients infected with herpes simplex virus who do not develop an encephalitis see this is herpes simplex now. Not "B" virus. That it invades the central nervous system. It might. This is all hypothetical since then it has been proved that it can occur. And that the virus could remain dormant in the ali-go-den-dra-gle-a and then because why the aligodendraglea.

It is one of the gleo cells of the white matter. Because the aligodendraglea have the function of maintaining the milant sheaths in good health so that for every little segment of milant you have several aligodendraglea who have the function for maintaining that milant and if you destroy those that milant is destroyed. I said wouldn't it be wonderful. Not wouldn't it be wonderful. It becomes absolutely necessary to test this hypothesis. But how do we test this hypothesis. Well, a young
Italian by the name of Messori came to work with me around that time and went right after this business. And the immunofluorescent technique was already being used a great deal. And we wanted to see whether or not in the, we could localize herpes virus by especially potent sera which I had in good supply because this was part of the immunization work against "B" virus. If we could localize lesions by immunofluorescence in sections because patients with multiple sclerosis sometimes died in the acute phase. Most of them, more than 90% die of something else after chronic, progressive disease. So I trained Messori in the immunofluorescent technique. We cut sections. First we studied it in mice and it turned out to be absolutely beautiful. But you could see exactly where the herpes virus had done its work by immunofluorescence. But the technique used at that time was one in which you had to have fresh material, freeze it. I froze the sections, do the immunofluorescent sandwich technique, etc. And it was evident that if I wanted to study material that was available in banks, pathological material in blocks that have already been fixed in formulant and then for imbedding in parafin had to go through all sorts of liquid solvents which has an effect on the envelope (?) of at least the virus envelope material of herpes virus that if I was going to be able to utilize this accumulated material and also human herpes encephalitis material which I had blocks of and stored. It was necessary to find out whether or not this reaction that we could show so beautifully in mice when frozen sections were made could (a) be shown after fixation in formulant and
the answer was yes. Beautiful. It didn't make any difference. But the next big question was, supposing we put it through all the lipid solvents and everything that is necessary for imbedding in parafin would it still be demonstrable because presumably you put solvents and the alcohol, it would just kill those antigens. And lo and behold it did. We could take blocks of the same material, the one side by side and after all of this you could cut a section and serial sections from the parafin the blocks and show beautifully where herpes virus was. And incidentally the localization of herpes virus was well controlled with other viruses and with normal beautiful specificity. Where the herpes virus was located by immunofluorescence there weren't even sign of inflammation at all. And this is a little bit what you find in the plaques and the lesions for multiple sclerosis. You find plaques and particularly whether they are the younger ones or the older ones, you may find no information around them at all. So the next question was could we demonstrate this in sections with human herpes encephalitis. And we had just had as I say a young boy died of encephalitis. I isolated virus from his brain and as a matter of fact the virus was in minute amounts in his brain. Because by the time he died he already had so much antibody in his blood I don't know whether that was the reason or whether it had already disappeared. And inflammatory lesions were very scarce. So that and this had already been in blocks for some time, in parafin blocks. But at any rate, we cut sections and got the most beautiful specific immunofluorescence for herpes virus in this proved case of herpes encephalitis. I think these were
nice exciting results and so it showed that even after it has been in blocks for months or a year I forget the details now. But you could still do it. It was extraordinary. So then I got blocks from the Army pathological museum. The armed forces pathological museum, institute, armed forces pathology institute because there were a number of deaths from herpes encephalitis among soldiers and some of the blocks were four years old, six years old and so on. And I found that even after it had been in the box for years I could cut the section and show the specific lesions. And moreover, in areas in which the ordinary pathologist would never suspect because I would cut serial sections on one of them do the immunal fluorescence and on the other do an ordinary stain and there was the ordinary stain that showed nothing in the cells that you could, that a pathologist would suspect that the virus was in them. There were other areas in which there was a good inflammatory reaction but there was no virus there.

So I said well now the stage is set. Now we have a tool by which I can grow and this was Dr. Zimmerman the great neural pathologist who had a wonderful collection of pathological material in blocks from multiple sclerosis patients dying in various stages of disease. And I think I obtained material from fourteen cases of multiple sclerosis. Cut the sections, we stood on our head. Beautiful multiple sclerosis plaques with, without inflammatory reaction. Not a smell. Not a smell. And so I had to draw the conclusion that by methods that made it possible to detect herpes virus in patients who died of herpes encephalitis, it was not possible to demonstrate herpes. Then I realized that the immunofluorescent technique is not the most
sensitive technique and I still didn't discard the hypothesis that a virus like herpes could remain in perhaps aligodendraglea preferably because the neurons, nerve cells themselves are not effected in multiple sclerosis. It is only the aligodendraglea and then the white matter is the thing that happens with it. And as a matter of fact I still maintain that there are other newer techniques for testing this hypothesis. Because reactivation may not necessary lead to complete synthesis of those antigens that are picked up by this method so it must be special antigens that reduce the alcohol and acetone and all the liquid solvents that go through for getting blocks of parafin. So I thought it only shows that by this technique I cannot show herpes virus. And then there is an interesting footnote. My old friend Dr. McCallum whom I got to know in England when I worked there in 1934 had an ongoing interest. He was then and still is at Oxford University in England and he was so intrigued by report of these findings at a neurological congress in Amsterdam. He was so intrigued by the possibility of demonstrating herpes virus that he also had a lot of material from patients who died of herpes encephalitis and he immediately went to his laboratory, repeated the same thing that I said exactly. I sent him the technique that I was using, and he couldn't confirm the observation. He said Dear Albert. I got it Dear Albert letter which said that somewhere in the--probably in my files if it were anybody else but you I would just publish something and say I don't know what Albert Sabin and Messori did, but I certainly cannot confirm it with material I have in England. But he said since it was you, and
since I have great respect for your critical treatment of data please tell me what did I do wrong. So I asked him about the serum that he used obviously. The assumption that a herpes, rabbit that a rabid herpes antisera, a potent is an antiserum; is it antiserum; is it antiserum. It is all the same. Is rubbish. There is a tremendous multiplicity of antigen in herpes virus which I have learned in my studies in attempting to vaccinate. And I said frankly you used a very potent rabbit antiserum but there is only one way to settle this. I will send you some of the antiserum that I used, that I prepared in a special way for hyperimmunizing rabbits. And you repeat your tests simultaneously, using your potent rabbit antiserum and my antisera and see what happens. And when he did that, sure enough, that is what happened. When he used my antiserum he got exactly the same results that we did. When he used his antiserum simultaneously he did not. Now what does that mean. It means that there is a special antibody that resisted all of this. There is a special antigen that resists all of this treatment for which my serum added antibody and high titer and his did not. It was for that reason that I concluded that while I could not demonstrate the antigens in multiple sclerosis that I was able to demonstrate in patients that died of herpes encephalitis it was not conclusive proof against a potential role of a DNA virus like herpes and I would say it wouldn't necessary by herpes only. It could be avera cell exhausta virus that we know invades the central nervous system and remains dormant. And of course we know that now. Subsequent work by--stop it a moment.
Some years later Bob Good, while he was still a student actually did a wonderful experiment with herpes in rabbits because there are some strains of herpes that invade and kill rabbits with encephalitis and others that apparently invade and remain dormant. And then he had such rabbits and when he induced an antiphagocytic reaction in them the herpes virus was revived and they died of encephalitis. And I said, and I still think that one must consider new techniques that are available for determining whether or not when there are lesions activated, whether or not in the aligodendraglea there is an activation of activity, there may not even be destroyed, you see, which interferes with millant conduction. And I think possibly at the present time if one would use the new molecular biology techniques for the effect in let's say pieces of herpes virus DNA or Marcella's ostra DNA in the grey matter of patients that have plaques of multiple sclerosis I think it is still an open question. I didn't succeed. But it was a wonderful technique that brought up some new knowledge and I think if I hadn't gotten involved with those Russian tests and my curiosity aroused, well you know, then all of these serendipity things. You start working on one thing and you get excited about something else. This is to make clear that during all these years in the late '40s and the '50s that I was working on polio, although that was the main goal. I didn't completely shut off the curiosity that was aroused by other ways.

Q This is, as I look at this thing, when I think for example of polio epidemics in the past that might not have been polio epidemics if they had had the techniques that
existed by the late '50s they might have classified them as coccacki A-9 or echo 10 or.

A May I tell you why not. Because the key word you used was epidemics. That while there are isolated cases that have been shown to have been caused by cocc sacki A7, by echo 6 perhaps by certain cocc sacki B viruses, they were always isolated cases. And an epidemic of poliomyelitis is not called an epidemic of poliomyelitis because 99% of it is non paralytic and only an occasional one is paralytic. An epidemic of poliomyelitis has a distinct characteristic. It is a characteristic in which probably 99% of the paralytic cases actually are caused by one or another type of polio virus. It is a clinical syndrome which although it has three different types of polio virus involved they are the major cause. It is quite unlike a problem which we are facing now with influenza for example influenza is a syndrome, a clinical syndrome that yes it is caused by influenza a virus and influenza b virus but the same syndrome is caused by so many other viruses that is quite unlike poliomyelitis. But in poliomyelitis I would say no. Real epidemics of poliomyelitis were polimyelitis. We can still identify that there was polio in Egypt thousands of years ago as you know by examining the beautiful records that were left of certain kinds of atrophy which nothing else produces you see. Polio has certain fingerprints so to speak that even without isolation of virus gives you an opportunity to have a reasonable--

Q Then I bespoke my--

A Epidemics. Not in individual cases. That is the difference.
Q Epidemics. Because there were several cases in 1959 at Willowbrook among children who had had four doses of Salk vaccine and--

A Several cases of what.

Q Of polio like diseases.

A I don't know of that. Where did you see that?

Q Oh, it is in your report.

A Oh. I am sorry. Start your question over again. In the light of the correction.

Q Dr. Sabin, I may have bespoke myself before by speaking about epidemics being misdiagnosed. But certainly by '56, '57 techniques for, in the laboratory for making diagnoses of viral disease had become so elegant that certain cases that had previously been clinically called polio turned out on examination not to be induced by polio virus. Could you speak for such cases.

A Well as I have already mentioned, many laboratories were involved in such studies and I think there is growing evidence that occasional cases of poliomyelitis were caused by other enteral viruses. A-7 as I said. I find that in, during the period of 1957, the summer of '57 and on to '58 that in my laboratory we also carried out a number of studies and particularly to the point here there was an interesting situation of paralytic disease that had occurred in Houston, Texas among children who had had three or four doses of Salk vaccine. I had stool specimens from these and no polio viruses were isolated but a virus that when isolated in suckling mice and which had the behavior properties of coecssacki virus we studied very carefully
and spent a lot of time on it and found. I won't go into the
details because they are not pertinent. But this virus could
not be identified with any of the 19 coxsackie A viruses. The
antisera for them that were available at the time nor with the
five coxsackie B viruses nor with any of the echo viruses
because by that time we already had a tremendous number of
reagents. And I remember concluding at the time that this
would appear to be a new coxsackie A virus that is different
from all previously known coxsackie A viruses including the
additional types recently studied by Sickles who was working
with Dr. Dahldorf that are not pathogenic for monkey kidney
culture. The point is we went through a lot of studies and at
this time and I know I sent this virus I think to Dahldorf who
was still carrying on work. I do not remember now whether this
virus had been worked up as a new type of coxsackie virus. But
at any rate this is still another example that with all the
coxsackie viruses that we already do, the 19, which means
including A-7 and all the others. And with the five coxsackie
B viruses. Some of which had already been implicated, and the
various enteral viruses which we had sera certainly for echo 6
and some of the others. We isolate still a virus that had all
the properties of a coxsackie virus because in mice it did not
produce any lesions in the central nervous system. It produced
typical skeletal muscle micros of coxsackie A viruses with
some lesions in brown fat and it had scitopathogenicity but--
and it was difficult to carry out neutralization tests although
we got some evidence that by special techniques we could detect
antibodies against this virus, in the stools, in the patients
from whom paralyzed patients from whom the stools were obtained. This is an aspect of the work that was going on all the time. Now I have a note here in my report for that year that we also studied six fatal cases with evidence of involvement of the central nervous system. They were studied virologically and they had Salk. Some had Salk vaccine. Some had not. And one of these patients. There was a diagnosis of polio on it. And I said in one of these patients, Schooler, and this is the name of the herpes strain that I had studied so extensively and which others have studied so extensively. Who had had two doses of Salk vaccine earlier in the year. A clinical diagnosis was bulbar poliomyelitis. In my judgment the diagnosis was erroneous. But nevertheless this was the clinical diagnosis. Pathologically there were no lesions of poliomyelitis in this patient but rather those of a hepatone encephalitis unassociated with readily demonstrable intranuclear inclusions. I want to recall here that I am a pathologist. I was trained as a pathologist in Bellview Hospital doing 400 autopsies and subsequently all my work was based so that I studied these things myself. Nevertheless, I think this was a patient in Cincinnati and I had the material. And after a great deal of difficulty, a virus was isolated directly from the brain in young rabbit kidney tissue culture, directly from the brain of this patient. In monkey kidney culture nothing was obtained, you see. And ultimately proved to be herpes simplex virus and it was in the brain of this patient and the sections of this patient that I was able to demonstrate by the immunofluorescent technique the lesions of herpes virus actually that I mentioned before and this
strain became very useful in multiple studies. Not only in the attempt to develop a vaccine for "B" virus but also in subsequent studies on the role of herpes simplex in human cancer. So this shows side products which involve a great deal of work in the lab while the work on polio vaccine was going on. Let me give you another example.

Another child with a diagnosis of poliomyelitis in Nashville, Tennessee. Now mind you this was during the Salk vaccine days, not when they get live virus vaccine. But the same thing happened of course and continues to happen with live virus vaccine. That is different. Another child with a diagnosis of poliomyelitis in Nashville, Tennessee died after several months of severe illness and the pathological findings turned out to be those of Wilson's disease through pathural ventricular degeneration. No virus could be recovered from either the liver or the brain. In Wilson's disease you wouldn't expect it. But then we also and this was an interesting period. This is 1957. This is the era of Asian flu and people, some people also died of encephalitis. And I studied three patients with so called post influenzal encephalitis. One in Cincinnati and two in Milwaukee. And the pathological findings in all three were strongly suggestive of a primary viral infection. As I reported elsewhere, some of the lesions were like those of pharney colomos (?) disease. No influenza virus was recovered from any of them but in one instance, herpes simplex virus was isolated in young rabbit kidney tissue. That is the most sensitive for herpes, but not monkey kidney tissue culture. Sera obtained
during the acute phase and post mortem from the patient from whom the herpes simplex virus was recovered exhibited a distinct rise in antibody to both the Asian influenza and herpes simplex virus. I reported on the pathological findings here during the discussion at a meeting of the American Association of Physicians when David Rogers gave his report on six fatal cases of influenzal pneumonia you see. And I asked him whether he had encountered any cases of encephalitis in his studies so called influenza. And he hadn't and I reported my findings on this. Then I also say that during the course of that year virologic studies were carried out on two infants that died within twelve to thirty days after birth with clinical manifestations of gastroenteritis but no enteral viruses were recovered. But, we did have as part of the overall study of the role of enteral viruses. We had quite an organized activity because we studied for example together with Romulus Alvarez we carried out a very extensive study over a period of years to see whether various enteral viruses or I should say enteric viruses. It turned out coxsacki, adenal, echo were demonstrable with significantly greater frequency in summer diarrhea of which we used to get a lot still admitted to the Children's Hospital in Cincinnati then in comparable controls studied at the same time of the year etc. And we published a paper on that and those particular studies in a city like Cincinnati there was a definite association of high frequency of infection with these viruses. Another thing we did was, in '57, there broke out cases first of all in Cincinnati, we isolated cases with aseptic meningitis and with rash and again we studied them and found echo 9. And lo and behold a
little while later I get a call from a health commissioner in Milwaukee, Michigan that they are having an epidemic of an infectious disease with aseptic meningitis. They had one paralytic case but lots more aseptic meningitis, diagnosed non-paralytic polio and also many patients with a rash. And I went down and I made a very extensive study there together with Dr. Weigand who was working with me at the time and that it turned out to be an epidemic that involved about 40,000 people. And we were able to prove that it was such a virulent echo 9 virus that 85% of all those that had an infection had a clinically apparent infection. And it was cutting across many age groups so that there were many pregnant women. And we carried out a special study on the effect of echo 9 virus infection during pregnancy on incidence of congenital defects and disturbances in psycho-motor development subsequently because this was a joint study with, and we found absolutely nothing. The disease was very similar clinically to rubella but there were no—oh sure, you have an incidence of about 2% of congenital defects but studying very carefully, the relationship to the infection and control groups, no evidence, this went on for a couple of years because the infants who were born from mothers who had evidence of infection of echo 9 any time during the course of pregnancy even if they didn't show anything during birth they were followed up by a team. And the department of pediatrics there for psycho-motor disturbances, and we found no evidence. Now we also studied summer febrile disease with rash in Cincinnati as a regular thing and found in some instances echo virus infections and some none.
Q One of the things that I want you to comment before we finish today is the studies that you conducted at Willowbrook to see the effects of children who had had Salk vaccine, whether there was any interference with the--

A This came closer to the end I think this was during the period of already widespread field trials with polio vaccine and the question arose and there was considerable disagreements and arguments. You know these things go on during periods of great activity. Is it possible, and it was claimed that it was, that if you produce very high titers of antibody with Salk vaccine under conditions in which a natural infection might not have occurred to fortify to change the situation. That you would be able to get interference with multiplication of the virus in the intestinal tract. So that a very potent killed virus vaccine given many times and this is what Sven Gaard had been claiming and Salk was claiming this could really interrupt the chain of transmission of polio virus. In order to be able to test it, it was necessary to know many things. It was necessary to know that you were starting with a child that had no evidence of previous infection by absence of antibody. That is prior to giving Salk vaccine. And you had to make certain that during the course of the administration of Salk vaccine that it didn't pick up an inapparent infection with polio virus, you see, which would then give him a resisitance of the intestinal tract. And this of course obviously would have to be done in a closed population and with children who could be tested, selected for being either triple negatives or single or double negatives, etc. And then kept in isolation
and studied periodically to make sure that they didn't pick up whatever else they had, that they didn't pick up a natural infection with polio virus. And study them not only by isolation of virus but by antibody development. So the ideal place for that. Ideal not from the point of view of any enteral viruses being present because the ideal from the point of view of being able to have isolation units, frequent follow up was at the Willowbrook State Hospital where my cousin Dr. Saul Krugman had carried out such excellent studies on hepatitis, measles and so on and we decided to carry out a collaborative study on this. Incidentally this study was never published in detail although I mentioned it in one of my summary lectures (inaudible)