Interview with Dr. Albert Sabin  
January 19, 1974  
Washington, D.C.

Q: The last time we met we spoke of your early work in the purification and concentration of polio virus. This was followed with a subsequent work on polio virus that is of some interest. The title of the paper was "The Nature of Skin Reactions Produced by Heat and Activated Polio Virus" and it appeared in the Archives of Internal Medicine in June of 1933. One of the people who participated in this study or indeed might have even introduced the subject for study was Claus Jungeblut. I wonder if you could tell me how you first met Claus Jungeblut and what your impressions of him were.

A: I have to begin with a comment or introduction by saying that my involvement in this particular study on the nature of skin reactions produced by heat and activated virus people did it without poliomyelitis was not done something subsequently to the work on purification of the virus but actually was a cause for my undertaking the work for purification of poliomyelitis virus, which was all done in 1931, the year I graduated from medical school and continued to work on experimental coccyclin infection during the summer months before starting my internship and during the period, mainly the summer months of 1931, when New York City was struck for the second time by the largest epidemic of poliomyelitis in its history, in which there were, I believe, more than 6700 paralytic cases of the disease. I did not meet Dr. Jungeblut, in connection with this work at least, as far as my recollection goes, until I had to meet him
because of this work. I may have met him casually or been introduced to him during meetings at experimental, the Society for Experimental Biology and Medicine, but it wouldn't mean anything. He was already an important professor at Columbia University, and I was a medical student. So that, because I'd only just graduated from medical school. But our association began in an interesting manner and the only reason I think that it's worth retelling here is not that the work itself was so important, but it bears on two basic principles in the pursuit of research, the modus operandi, and also on some of the factors that applied to doing the research on human beings in 1931 and now.

The issue is simply this: Shortly before this epidemic began its explosive aspect in New York City, Dr. Jungeblut had published two papers suggesting, or in his judgement, the results justified the conclusion that in monkeys as well as human beings who had had a previous attack of an infection of poliomyelitis virus, there was an allergic reaction to the virus which worked on monkeys, was in the form of an accelerated febrile reaction upon reinoculation with poliomyelitis virus. Those survived with paralytic infection in the first place. Then this observation led him to preparing heat and activated extracts of the spinal cord of monkeys containing polio virus. This was the only source of polio virus at the time. And comparable extracts from the spinal cords of uninfected monkeys, that is uninfected with polio virus. Heat in activating them and then using these extracts
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for skin tests in polio convalescent children as well as normal controls. Using the word normal in its usual sense and this led to his publication of his conclusion that there were allergic reactions in polio convalescent children after injection of heat and activated poliomyelitis virus.

My chief at that time, because I was still working in the Department of Bacteriology at New York University Medical School, was Dr. William H. Park, to whom I owe so much for my entry into--interruption. My chief at that time, Dr. William H. Park, I was still working at the Department of Bacteriology, New York University Medical School, brought to my attention this work of Dr. Jungeblut's and pointed out how important it would be if we actually had a skin test that would be able to tell us which children, which persons, are susceptible to polio and which are immune. Because it would become the basis for work that was then in the process of planning of the possible preparation of the next against poliomyelitis, in the light of this horrible epidemic that was taking place in New York. I had never worked with the virus before. I didn't know polio went into a one end of a polio virus infected monkey from another, or anything about the virus. I agreed with Dr. Park that it was a very important problem and that if Dr. Jungeblut's conclusions were indeed correct, then it would provide an important tool for the further studies of various aspects of the polio problem that might ultimately be
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helpful for its prevention. It is for that reason that I accepted Dr. Parks' suggestion to repeat, or try to repeat Dr. Jungeblut's work, not because I didn't have anything else to do. To make a long story short, I did what Dr. Jungeblut had published and I failed to confirm his results in the sense that I found that with the virus that I used and with the controls that I used that sometimes normal, extracts of normal monkeys' spinal cords would give the same, and certain extracts of polio infected monkeys' spinal cords would not, and it was really a question of what constituted a negative control.

Then there's a choice, and this is where we come to the longer-range interest rather than the significance of the work itself. Because it involves an issue which is a constant issue, will remain an issue for scientific workers whether it be medicine or anything else. The issue being, what do you do when you repeat the work of another scientist, because the conclusions are very important. You say to yourself "This is a very important, if true" and you fail to get the same results. You fail to repeat. Do you go ahead and publish the results of your studies and say "I'm very sorry, we have to conclude that we cannot repeat these data. Our conclusion is that perhaps Dr. Jungeblut didn't use enough controls, normal monkey cords to realize that he couldn't draw the conclusions that he did, period." What happens then?

What happens then is you have two reports in the literature. One, by a highly respected microbiologist which says yes; and
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another one by, the work was done by me, not by the respected Dr. William H. Park. A young man who just got out of medical school says "No!" So you weigh an elephant and a mouse. Is that the way to reach conclusions? And to me, already at that early stage I felt that was the wrong way to do it. So after discussing it with Dr. Parks, my suggestion that I go to Dr. Jungeblut, show him my data and ask him what was wrong, and see in what way the things that he did, or the agents that he used or strain of virus that he used might be different from what I used. That is how I really first met Dr. Jungeblut.

He did appreciate very much the courtesy of my not publishing these data but coming to discuss it with him. Accordingly we arranged to carry out some tests together. He would prepare new extracts. I would prepare extracts following his. We would do tests on the same children of the convalescent, polio convalescent home, not only for those coming in from the new epidemic but accumulated from previous years and controls. And then, when the work was finished and Dr. Jungeblut agreed that his observations could not be repeated, that there were other explanations for them, he joined as co-author in this publication with Dr. Park and myself so that the aspect of controversy was removed from it. And it isn't important now as to what was the real factor. Basically, it was that, for some reason, even normal spinal cords give such reactions, and that while it definitely did not exclude the possibility that there might be an allergic response to certain
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polio viruses components in convalescent animals, or human beings, the evidence on which this conclusion was based, was not valid. And it was also in the attempt to get away from other potentially skin toxic factors that led me to the work, the first work, on the purification of polio viruses by the methods of Wistathter which proved so effective in purifying many enzymes which we discussed the other time.

Now what is the other aspect of this particular episode. It has a wider significance. To me it is that if such a study was proposed at the present, or even in recent years, because it involves carrying out tests on human beings, children and adults, it would have to pass a so-called Human Experimentation Review Committee in the institution that supported your work of course at that time didn't have to apply for a grant to carry out this work. But at any rate in the institution in which you worked and certainly if you got support from the National Institutes of Health, they insist on it. These are the regulations. What do they insist on? They insist on evidence that the procedure that you are going to use will yield first of all, information of importance, of significance and not some petty question being elucidated. They would also insist on proof that the answer cannot be obtained in any other way. But above all, they would insist on safety of the materials that you are using and would insist that the material that you are going to inject in human beings has been very thoroughly tested to have at least the least
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possible probability of infecting some susceptible person with the virus and causing the crippling disease or even death.

Well of course all of this was absent in 1931. What did we do. What did Dr. Jungeblut do? What did I do? We inactivated our virus preparations at 65° centigrade. Surely it was the temperature we had reason to believe would inactivate polio virus. We showed that it inactivated polio virus. But how did we reach this conclusion. The number of monkeys with which we could work was small. We tested about two, three monkeys. We held the material before it was injected into any human beings to make sure that the monkeys which received the virus in their brains remained healthy. But how many monkeys did we lose? Very few. And when you go out and test twenty, forty, sixty human beings the possibility that a small amount of virus, which was not detectible by merely innoculating two, three monkeys, could still be there and could produce a crippling disease in one of the persons was certainly... This conclusion could not be made on the basis of the tests we had. So that, in the present era, such experiments would be forbidden, and in my judgement, rightly so. We certainly had the experience of 1954 I think it was or 55 when the first use of the Salk formula in an activated virus was prepared by the Cutter Laboratories--the famous Cutter Incident. And there they didn't use just two, three monkeys. They used larger numbers of monkeys and the tests. Well, it's unquestionable whether all the monkeys really remained well
but that's another chapter. But at any rate, it was passed. The monkeys remained well. But when it was innoculated in larger numbers of children there was no question that there was a residual virus that was not detected by merely innoculating ten monkeys or so and it produced crippling disease in many persons. So that this is not merely hypothetical. It was potentially danger. The subsequent experience bore out the dangers that were encountered. And these, I say, are the two interesting side lights of this particular work.

Q: So you would say, in a sense, that the design of the experiment itself is reflective of the amount of knowledge that one had at the time of viruses, in part.

A: It also is reflective of the investigator. If the investigator is really intent on double-checking his data and expanding his observations before he reaches a conclusion that because I did so and so, therefore, then you don't run the danger that Dr. Jungeblut had run into. But Jungeblut ran into that danger because of inadequate controls. This ultimately came out.

Q: You know there was one other thing about this innoculation. It was very, very painful to give. And people who were innoculated complained of the pain. There seems to be a kind of willingness to do the experiment.

A: What is your question?

Q: The question is, wasn't Jungeblut or yourself brought up by the fact that there was pain in just giving the innoculation.
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A: Well, the pain was a very transitory thing because certain salt solutions were used. And when they were eliminated, there was no pain. That was not a big point. I mean, the pain, when you stick the needle in, inject the stuff, that's a minor matter. I think that the more important matter, here, is whether or not the tests were justified. And I would say that on the basis of justification with the knowledge of that time, and here is where your comment comes in, one would have said, yes, the knowledge that we would obtain by these tests can not be obtained in any other way, you might say, why not do it monkeys first. See if monkeys give skin reactions. That was not done. All Dr. Jungeblut did was to record the observation that there were febrile reactions. Well, the monkey's disease was in a considerable sense regarded as artificial. And there is always the possibility that whether or not you've got a positive result in the skin in monkeys, it wouldn't tell you anything. And that if the original observation were correct that you had a test for determining immunity or susceptibility to poliomyelitis, it was a very important thing. Therefore the criterion of importance and the inability to get an answer for human beings in any other way applied. The only thing that did not apply was the method of ascertaining the safety of the materials that were used. At that time we were too naive.

Q: Now, this period of research leads to so many things. One of the workers who was working on polio in Dr. Park's
laboratory at the time was a young Canadian named Dr. William Brebner. Could you tell me how you first met Brebner?

A: Well I first met Brebner when Dr. Park recruited me to join the polio investigation that was going on under his direction in the Department of Health laboratories, because that was where Dr. Brebner was working. Dr. Park had recruited him and had worked in St. Louis after working in Canada and had joined Dr. Park and was working on poliomyelitis. His job was to isolate viruses from patients with paralytic disease in this epidemic. Because we had the same chief, and that's how we first met.

Q: Now, on October 22, 1932, while Dr. Brebner was handling a monkey he was bitten by the monkey, and subsequently became ill. The reason I attack the problem this way is that it seems to me when one discusses scientific matters, sometimes one overlooks the individuals involved, the promise that they had at the time and we hardly even remember their names. I'd like to recapture that human element and what becomes the discovery of a new virus, namely "B" virus.

A: Well, Dr. Brebner was 29 years old October of '32. He was obviously a very bright, intelligent person, dedicated investigator. One could predict that he had a great future ahead of him. He came from an academically distinguished family in Toronto. He would have moved fast and had all the advantages. He was handsome. He was polished. He was
intelligent. And as a matter of fact, there was gossip that Dr. Park was grooming him as a possible successor. And then he was bitten by a monkey. When he was bitten by a monkey, he at first didn't pay any attention to it, but later some blisters developed on the finger where he was bitten. They ascended. I won't go into the clinical details, but at that time, I was serving my internship on surgery at Bellevue Hospital. I had already had my six months of pathology and I was now doing six months on surgery. Really, I can't remember now why he was admitted to surgery, whether it was because I was there and I asked the chief to admit him or because there was a traumatic, there was a wound, he was bitten. At that time, really, he didn't know because basically all he had at the time he was admitted was this lesion on his finger and the lymph nodes in the area were enlarged. He had no central nervous system manifestations. So, that's why he didn't go to neurology or somewhere. Actually, it was a surgical thing. As a friend, and we were friends, I had an opportunity not only to watch him during the course of his illness during which within, oh, let's see. He was admitted on October 28th and by November 4th he developed urinary retention and sensory disturbances that are called hyperaesthesia in the lower extremities. And on November 5th he had a flaccid paralysis of the lower extremities, but also sensory disturbances in them. On November 6th it began to ascend--the paralysis ascended to involve other muscles, and with another day, on November 8th. I remember this vividly. He had his upper extremities involved and the following day, on November 9th,
he had respiratory paralysis and died.

Q: They brought in neurological consultants when he began having these neurological signs. One of the consultants they brought in was really one of the great neurologists of the time, Foster Kennedy.

A: And Dr. Friedman.

Q: And Dr. Friedman. Now what was their reaction to the disease. Did they try to make--

A: Well, it was an ascending myelitis. As a clinical, neurological syndrome there it was. But how to associate it with the monkey bite, to associate it with the peculiar lesions that he had, nobody could really say because nobody knew anything about it.

Q: What was the reaction in the lab. Here is one of the prominent workers in the lab and he dies. Did people in the lab become afraid of working. Did they associate the illness with the monkey bite?

A: At that time, you know, it was very difficult to say. Nobody could say that it had anything to do with the monkey bite. I mean, people develop ascending myelitis all the time without reference to a monkey bite. All right, he had lesions at the site where he was bitten. As a matter of fact, as the disease developed, it was more of a question of whether he hadn't contracted polio. Because, what did he have. He had urinary retention. Alright, you sometimes get urinary retention. That is an early sign of polio. He had the sensory disturbances. Things start off. Polio very often begins with a peculiar
sensory disturbance which almost feels like a sunburn. And then when he developed flaccid paralysis of the legs, my God, but even before that, Dr. Brebner himself insisted that he be given 20cc convalescent polio myelitis serum. That shows what he was thinking about. So that, as the disease developed, the only thing to really interfere with the diagnosis of polio was the fact that there were, is a little more sensory disturbance than you see in polio myelitis. And this was disturbing. And that's why a definite diagnosis of polio myelitis could not be made, but it could have been polio myelitis. It could have been something else. One of the things that it could not be was, if I remember correctly now, I am trying to remember whether the Guillan Barré Syndrome was ruled out by the cerebral spinal fluid findings. But at any rate, that it could possibly a new type of infectious disease was not really considered by anybody. I mean, how about those damned vesicles. How about the lymph nodes. Oh, all right you could have two things you see, you have a bite and an infection. And then he also gets polio. Maybe, while he was working with polio through this wound polio virus got in somehow. So, actually, it was not discovered that it was not polio until there was an autopsy, until an autopsy was done.

Q: Now the autopsy was done by Thomas Gonzales who was the deputy chief medical examiner and then there were two other people associated with the autopsy. One was Lewis Stephenson who is a prominent pathologist and another pathologist named Irving Graeff. So, it was regarded with some seriousness.
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A: Irving Graeff was actually the resident on pathology. He was my immediate. It was under him that I served as an intern on pathology before and I did six months of pathology right there where Gonzalez--where that famous group which subsequently became The Institute of Forensic Medicine under Dr. Helpern. But it is a very vital period. The place looked like the devil. But I myself did 400 autopsies during the six months that I served on pathology. So, these were people with whom I had been intimately associated and they all studied this, and the only conclusion that they came to was that it was not poliomyelitis. On this they all agreed.

Q: They also said that the findings that they had were not characteristic of any virus disease in particular.

A: Well, as a pathologist at that time, that was all that could be said because, after all, there were certain things that you could consider, which were known and that had to be eliminated. So it was a neurologic disease with unusual pathological findings not only in the central nervous system but in the viscera. That was unique and puzzling. So the spleen, liver, oh, different things were collected to try to determine. First of all, despite the unusual findings, they could isolate polio virus. And if not polio virus, whether you could isolate something, something else. I remember that I myself, even though I was then working very hard as a surgical intern, I obtained some of the material myself and
innoculated monkeys, dogs, guinea pigs, mice, rabbits, by various routes. That was my involvement but actually I must say here that to the best of my recollection when I obtained this material I was not aware that anybody else had obtained material for virologic studies. Without my knowledge, I didn't know that at the autopsy, and I was not present at the autopsy, that some of the material was given to Professor Gay and his group from Columbia University. I was not aware when I undertook the studies for a possible role of the virus, purpose of the virus. But, they also carried out studies. You see Dr. Gay had an interest because since the epidemics of so-called encephalitis that became the prevalent after the big influenza epidemic after World War I, in 1918. He was chairman of a special encephalitis commission and he had a hypothesis that herpes virus was the responsible for encephalitis and because he had a lot more experience than I, so was his co-workers with herpes virus he knew that in rabbits at least herpes virus could produce such a disease in rabbits. And because there were vesicles on the finger, I think. I mean, we never talked about it much. Dr. Gay and I never met on this subject, I mean, after I had finished my work and it came to my attention that Dr. Gay and his associate Dr. Holden were also involved. But he had quickly evolved a hypothesis particularly after innoculating rabbits and isolating a virus with certain properties. For him it was pat. It was herpes. A well known human virus. And although it was never
proved before that it was responsible for disease in human beings as such, there it is. There is a virus that does the things in man that the herpes virus does; therefore it is herpes. I think that was his conclusion. I am trying to remember now whether or not he published on this--

Q: He and Miss Holden did publish on this. I have the--
A: I have to recall now. That was a long time ago, more than forty years ago. So, I don't recall how. I know we had correspondence about this. Ah, yes, I see now that in the footnote of my paper which was published in association with my professor and chief on surgery, Dr. Wright. He was a wonderful surgeon but--well, he saw the patient clinically, I suppose. I have a note and I say here that "through Dr. Josephine B. Neal Dr. Gay and Dr. Holden obtained from us some brain and cults from the human case." Now, this must have happened, although Dr. Neal says "No, that's not how it happened." But at any rate, this is what is on record, and then in a preliminary paper which was published in 1933--

Q: In _The Journal of Infectious Diseases_--
A: In _The Journal of Infectious Diseases_ and I am trying to think my paper appeared in February 1934 and it must have been submitted six months before at any rate. Well, this is something that I added, I think in the galley. They reported the demonstration of the virus having the properties of our "B" virus as here described and stated their belief on experimental evidence
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that this virus is identical with the human herpes virus. So that
the publication, their publication actually came out first. No,
it was a preliminary paper--they had a longer one--

Q: No, no, that was the only note they published.

A: Because it was "The Proceeding Society for Experimental
Biology in Medicine" 1933, and it was part of a number of other
cases because this was Case Four in a series of isolations that
they believed were isolations of herpes virus from human central
nervous system disease. Now there is no question now we know
that human herpes virus does invade the human central nervous
system, can cause fatal disease, subsequently as I myself isolated
it and it is quite possible that if I had known as much about
herpes virus as they did at the time. And I didn't approach it
completely fresh without any prejudice, that I would have jumped
to the conclusion that they did and that the existence of a whole
evolutionary tree of herpes viruses of which "B" virus of monkeys
formed part of the evolutionary tree that developed with primates
at a certain level, that was completely distinguishable as I was
then able to show from the human herpes virus. All that would
have been postponed many years. Sure, it ultimately would have
been discovered under different circumstances, but because I
think the reason I discovered it is because I was too ignorant.
If I had been as sophisticated about herpes viruses as they were,
I would have jumped to the conclusion "Well, this is herpes virus,
so what."
I pursued these studies not because I had a special emotional feeling about my friend Brebner who started off with the personal element, and I really wanted to get to the bottom of the cause of this disease. When I submitted this publication this paper for publication, to the Journal of Experimental Medicine, I called it the Brebner virus. The esteemed and I say beloved editor of the Journal of Experimental Medicine, Paten Rous who taught me many things about writing papers, at least got me to agree to use only the initial "B" instead of Brebner. Why I do not know now, but I had so much affection and love for the man that when he suggested that it just be called by the initial, I accepted his suggestion. I want you to know that "B" comes from Brebner, and not, as an indication that it is some sort of subvariety of herpes. "B" stands for Brebner, and I wanted to call the virus Brebner virus because I wanted to, for posterity, if it meant anything at all, for his name to be attached to this virus in memory of one of the hazards of this kind of biomedical research and in memory of a man who, by his dedication to his work, was removed from this world before he was even able to establish what he could do.

Q: What I find really interesting in this paper is how you methodically attacked the question. You really don't come to rabbits as an experimental animal right off. There are a whole series of animals that you test the virus in initially. First you test it in monkeys, and then in mice, and guinea pigs and
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dogs and only after doing this do you come to the monkey. There is a kind of seriousness of purpose in this.

A: There is also--I don't know if you were about to make a point. I don't want to interrupt you.

Q: All right, go ahead.

A: But I can explain the reason for every experimental animal that I used to innoculate in this experiment. Rhesus monkeys, first of all, because one wanted to determine whether or not the monkey had polio virus. The rhesus monkey--the monkey is the only way to test it. And besides, having worked with rhesus monkeys I already had become a little expert myself in working on the purification of polio virus. This was my introduction to the virus field. I naturally used rhesus monkeys. Why dogs? My work in medical school--I worked on dogs in the study of the chill producing factor. And I had come to know how to work with dogs. And I have been available. Don't forget I had to work as an intern around the clock on surgery and work in the operating room and I used to do this in off hours. So I took whatever animals I could. I'd go across the street, you see, or I'd bring them up, I remember, no, not across it. Actually I brought them up, if I remember correctly, to the Pathology Department Building, up on the roof, where they had some place for animals. So, I had dogs. I had monkeys. Well guinea pigs, mice and rabbits. Of course rabbits I used in my pneumococcus infections studies in mice.
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And at that time, what was the tarian of the virologist? He's innoculate whatever animal he could lay his hands on and see if he could reproduce a disease, a disease in which you could eliminate certain bacteria and then transmit it in series and find out what you had. So that was the reason for using these animals. Now, interestingly enough, in the first publication, monkeys, yielded, the monkeys that I had available failed to react. In a sense it was good because it made it possible to say "Well, polio virus, we didn't have, obviously polio virus." But in a sense it was misleading because when I took the virus with me to England because in 1933 I left to work at the Lister Institute. I discovered that the special pathogenicity of this special strain of virus in monkeys was quite different and the monkeys they happened to have at the Lister Institute at the time for the monkeys we had in New York at the time. And that this became an identifying character for this virus. And the results that I reported on it that I obtained before, in New York, were what they were because as it ultimately turned out, monkeys were the natural hosts of this virus and some of them had immunity to this virus so that if you innoculate them with the virus, nothing happens. And if you happen to use other monkeys without immunity to this virus you get a terrible disease. You see? If you use an abnormal root. Because monkeys in nature, as we learned later, don't develop any serious disease, any more than the vast majority of human beings infected with herpes virus. Monkeys also have lesions
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in their mouth, in their throat, on their lips, in the throat, like human beings have. But, when you introduce this virus into the central nervous system of monkeys, well, as we described later, you get a tremendously severe disease. And when you do the same thing with ordinary human herpes virus, you don't. And then of course we went on to show that this--

Q: I don't want you to jump ahead. You found that when you introduced the virus into rabbits, it was almost 100% fatal. I think it was one case that escaped.

A: I don't remember now, but I know that it is.

Q: And so the latter part of your work was really working out a relationship of "B" virus to other viruses. You eliminated the fact that it was not similar to polio virus, rabies, or vaccinia. But it did resemble herpes virus, and you differentiated it from herpes virus as well.

A: Of course my concluding sentence in the first publication was "Although the relationship between the "B" virus and the virus of herpes must still be determined, by cross immunity tests, it has been shown to possess certain properties which warrant consideration of it as a distinct entity." Now this is where I say that if I weren't so green I probably would not have paid so much, let's say, emphasis on those slight differences and properties that we observed at that time and would just have let it go the way Gay and Holden said, "Oh, well, it just herpes virus." But because I was green, and I decided to study it further,
and to do cross immunity tests. And I took it with me to England. I was then able to show that it was a different virus and then to discover what subsequently became evident that it is a virus of monkeys and not of man. Then when it transfers--when it infects man, it is terrifically, and with great regularity fatal and then--I don't know whether this is the time to talk about it--then came stories that years before, some years before, maybe I forget, maybe five or so, the King of Greece was bitten by a monkey, and he died. I know I have been trying to track down the data how he died. I still haven't succeeded. Now, I have important friends in Greece and I think when I have nothing better to do, I am going to try to get them to see if they can fish out from the royal family archives the doctor's notations. But whether he died of this, we don't know. And then-- That was the only story of anybody dying of a monkey bite, and after that, after this paper was published, another doctor, a Canadian again working at Johns Hopkins was bitten by a monkey and--this was in 1934--my paper had just appeared and Warfield Longcope was Professor of Medicine there. Old Professor of Medicine had just read the paper and as he was making rounds--I remember these stories, I don't think it's recorded anywhere--he said to them "You know, there's something very similar about this disease that Dr. Henry is exhibiting with a report of Sabin and Wright that I just read in the Journal of Experimental Medicine."

Then a man was assigned to work it up, and I had heard that a
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virus had been isolated and all that, that all my attempts to track it down were nil. And so, for years, this one piece remained the one documented case of having an infection with a virus that is apparently of monkey origin, only slightly related to human herpes virus, only slightly related to a comparable virus in cattle, pseudo rabies etc., swine. And it was not until 1949, when I was then in Cincinnati and a young doctor working at Christ Hospital in Cincinnati was in Pharmacology in the Department of a very good friend of mine, Dr. Leon H. Schmidt. He was passing a stomach tube. His job was to give drugs by stomach tube to monkeys. He'd cut his finger in opening a penicillin bottle. You know, the aluminum thing. And apparently, he must have gotten this cut contaminated with material on the stomach tube that he passed down the stomach. And I never heard about it. But he acquired the disease and died. And I received a call at midnight from one of the people who knew about this story of "B" virus and Brebner, and they were doing this autopsy at midnight and they called me up. And he told the pathologist, "Look, you know, there is something about this which brings to mind the case which Sabin published in 1933." They called me up and I went out there to get material, and that was the second proved case of isolated "B" virus. This was in 1949 and was identical--the virus was identical with the one isolated--the original strain. And then another quiescent period. Then came the big push on the production of sulfa. And suddenly
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pharmaceutical companies all over the United States and in other places were having monkeys by the thousands. And they were being handled and their kidney cultures were being handled and then people began to die from "B" virus infection, until proper precautions were taken. And the requirements of guaranteeing monkeys for testing them for "B" virus--and this of course is still one of the standard requirements now--for testing polio virus, or anything produced in monkey kidneys. I don't know what the total number of fatal cases is now among laboratory workers, but the number is well beyond ten, twelve. But that's the human story of "B" virus, the biologic significance of it is that it is a remarkable example of viruses evolving along with the animal species, that you find it in all animal species and you can draw an evolutionary tree of comparable herpes viruses, probably from fish on up. Now this was the first indication, and simply because I was too green, too ignorant to accept the initial data and say,"Yeah, it's herpes virus."

Q: You publish in *Journal of Experimental Medicine* very distinguished Dr. Gay, who after all, had introduced Bordet's work to the United States, publishes a few months before in an opposite direction. And so there is a conflict. very, very early. Do you go to confer with anyone about this?

A: Well, in the first place, this publication appeared in 1933, I think either I wrote this paper just before this publication appeared. I don't know the month. I could look it
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up probably, but I notice that I submitted my paper for publication October 28, 1933. At that time, I was senior resident on medicine. I was working around the clock. I was also developing a new test for the quick typing of pneumococci in patients. I was doing all sorts of things. I had to write this on the side, you see, and I am quite sure that I didn't see the paper of Dr. Gay and Holden and the "Proceeding Science Experiment in Biology in Medicine" until I probably had already sent in the paper and had gotten--because in December, just about a month or so, two months later, I was on my way to England, you see winding up my work in the United States, going to England to work at the Lister Institute, and I put this in, put this footnote in as a footnote in a galley. Because this paper was published February 1st, 1934. I must say they published papers much faster then than they do now.

Q: I'm sure
A: November, December, January--publication in three months.

Q: Now, this is interesting. It goes to the Journal of Experimental Medicine. Knowing Paten Rous, Paten Rous would have sent it out to readers, and one of the readers undoubtedly was Rivers because you have a note thanking Rivers.

A: I have a note thanking Rivers because I was so damned green in virology. Alright, I knew a little about polio virus, but I didn't know anything about any other virus. And you know, virology was still in the early stages and Rivers can be called in a real sense "The Father of American Virology." molecular
level or something. We didn't think about the way it was done. So naturally, I went to Dr. Rivers and I said, "Look at these sections; look at these data. Please advise me." Now, I don't remember now whether Rivers already knew of Gay's work or not. He probably didn't. I don't think that Rivers and Gay were very close so that they had frequent contacts. And probably when I showed Rivers my data, which was long before I submitted the paper, it was before the publication. So, Rivers certainly helped me a great deal in guiding the differentiation from other viruses and more or less educating me a little about viruses especially.

Q: So in a sense, there was no immediate counsel.

A: No, because I didn't know the other work at all. Nobody even told me they were doing anything. I didn't learn about it until later.

Q: Okay. Now, I'd like to leave off your scientific work and come to other questions. Here you've been in America for a little more than a decade, working really extraordinarily hard. I'm sure that by this time you knew that your career was going to be in science. Did your family know that your career was going to be in science? Or did they see you as--

A: My family played very little role in my life. I left home in 1923 to go to college in New York. And the decisions were always my own. So that they knew after I made a decision not to ________. My family was not involved in the decision-making process because they were neither in a position to help
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me financially nor to influence my course, except my uncle--
I exclude my immediate family. So that actually I had to make
my own decisions. It was pretty clear even while I was interning at Bellevue Hospital that my career was going to be in
investigative medicine, scientific medicine rather than in the practice of medicine. My chief had to call me in. Dr. Weicoff
who was chief of medicine and on the one hand bawled me out
because I was apparently not doing all the things that I should
do as a senior resident. And I think the great James Shannon
who is responsible ultimately for the extraordinary development
of biomedical research in the United States. He was my resident.

A: I didn't know that.

Q: He must have reported to Weicoff that this guy Sabin may be alright, but he doesn't pay enough attention to the patients
for whom he has responsibility. So Weicoff felt that, felt himself compelled to call me in and tell me that. Then, however,
Weicoff said "Now I'll take off my hat as Chief of the Medical Service."--end of side A

BEGINNING OF SIDE B:

A: worthwhile to go and work abroad, to see how other people work, and during that period, there were many publication coming
from France and England on the viruses. Particularly Levaditi
was one of the people at the Pasteur Institute who had for many
years already tried to implicate, even before Gay, implicate
herpes viruses in human encephalitis, in human central nervous
system disease. So when I applied for a National Council--
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National Research Council Fellowship, to work abroad, I named two institutions where I would like to work. One was with Levaditi at the Pasteur Institute and the other one at the Lister Institute because Dr. Park, my chief had friends there. The director, who subsequently became knighted--Ledingham, Sir Charles Ledingham was a friend of Dr. Park's. There was certain work going on there that special kind of work. This is where Dr. Rivers came in. Because work was going on at the Lister Institute at the time by Dr. Eagles which concluded that it was possible to cultivate the vaccinia virus in the absence of urine salts. In his inimitable style, Tom Rivers, who I'd gotten to know by seeking his advice about this "B" virus, said to me, "Albert, now you go there and you either learn how to grow vaccinia virus in the absence of living cells the way Eagles does it." Eagles, not Eagle, does it. "Or you show them what's wrong."

So, I said, "Okay." Now, the National Research Council Committee's selecting fellows at that time had as chairman a man whose interest in me I did not realize. I did not know. It was Francis Blake, from Yale University. The _____ of my work was based on my pneumococcus work that he followed. But they decided, at any rate, that committee decided that the Pasteur Institute was the place in which the work was not held--the results they were reporting was not held in very high esteem and instead of breaking up my one-year fellowship by working in
two places, they said, you can have the Fellowship but in one
place you go to, Lister Institute. And that's why I first went
to the Lister Institute to do what Tom Rivers asked me to do.
And of course that's another story. It turned out that the
man was wrong. But that is where E. W. Hurst--E. Weston Hurst
worked. And I owe a great deal to that man because I learned a
lot of neuropathology experiments, experimental neuropathology
from him. And because of the monkeys that he had and the help
that he was able to give me, I was able to carry out, complete
the studies on "B" virus, which gave it more meaning than when
I left New York.

Q: So fine, we'll stop at this point and next time, we'll
take up your stay at the Lister Institute.