Interview with Dr. Albert Sabin
December 8, 1973

Q: ... curve ball, Dr. Sabin. If you had had your choice of, would you have gone to NYU? Was it easy to get into a medical school in 1927, 1928.

A: It was not easy, but I went to the New York University Medical School because it was a natural for me. I don't think I applied anywhere else. It was a natural for me because during my pre-medical studies at the Washington Square College of New York University, I had, as I have indicated before, established what was for me an extraordinary liaison with the Department of Bacteriology, which was located at the Medical School. For one year prior to my admission to medical school, I was already working, exploring, living a life of dreams more than activities in all off hours in that medical school. I wanted to stay there. I wanted to be able to continue. When I say now that I don't think I applied to any other medical school, I can't be absolutely certain that I--but I have no recollection of applying anywhere else, or what I would have done if I had been rejected. All I remember is that is where I wanted to work, because that's where I already had an opportunity to carry on laboratory work which I had hoped to continue during my medical school days because I would be repeating many of the pre-clinical sciences that I already had at dental school. I hoped to have time to work in the lab. That's where I wanted to work because I was given an opportunity there.
Q: Now, the other thing is that today, when we look at the costs of being a medical student, they are astronomical compared to, let's say '27, '28, but at that time, I think the general cost for a year at NYU was $488.00 for tuition and some other minor matriculation fees that came to about $512.00. Five hundred twelve dollars was a lot of money.

A: Well, it was a lot of money for me. I think it was perhaps proportionately no more in 1927, which is what, 46 years ago, than let's say $2500, or whatever it is, $3000 would be now.

When I consider, when I went to work at the Rockefeller Institute as a staff member, that my salary for the first year, 1935, was $2200, which is one-fifth of what a technician gets now. Great changes in values of monetary, dollar values have taken place in the last 45, 50 years. So it was a lot of money and I worked during summers and then I was helped by scholarship and of course, I worked to help support myself.

Q: You know, today, if you go to a medical school, you see very fine dormitories. Where did medical schools live '27, '28?

A: They lived in rooming houses around the school, nearby. I am trying to think, during the periods when I wasn't working at Harlem Hospital in order to make a living. I remember living for a short time in a place where other medical students lived within walking distance of 26th and First Avenue. Very soon thereafter, I got a job at Harlem Hospital, where I used to go after school hours to work and to study and to live and to eat.
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I had a long commuting on the subway because Harlem was quite a distance from one hundred and some odd street from 26th and First.

Q: You know, we think of expenditure of time. Now a medical gets into an automobile.

A: That's another thing about Bellevue Hospital's medical school, the university medical school at the time. As I recall it now, and I can refresh my memory by looking through the yearbook, I think, more than 50% of the students were residents of New York, so that they commuted back and forth from home. Many came from New Jersey. Some lived nearby. There were a few fraternity houses that had some medical students. But by far the largest number probably just were commuting back and forth from home.

Q: So it's a commuter school. Would it be fair to say that essentially the people who went to this school were second generation sons and daughters of immigrants?

A: For a very large proportion, yes. For others, no. As consulting the yearbook would show. But I would say probably for the majority, yes. For example, one of my close fellow students was one who's second generation Italian. We went to high school together in Patterson, New Jersey. His people were Italian immigrants, a really warm atmosphere in their home where his name was Joseph Mott and maybe it was Motto, but it was Joseph Mott. I used to go to their home and have a very warm feeling.
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So this is one example. I think this was true of many of the Jewish and Italian students that were my classmates.

Q: Were there many women in the--?

A: Well, there were four. Of course, I remember most of all the most spiritual physically beautiful one, Francis Holmes. I suppose everybody was in love with her. I am not sure about everybody, but I certainly had a crush on her, having to work very hard and not being very attractive, I had no girlfriends. I remember sitting in that ampitheatre type lecture room where we used to take our lectures. I invariably would choose the seat on the other side where I'd be able to look at her. Sometimes I looked at her more than I listened to the lectures. I don't remember if she was from North Carolina or South Carolina. I think she taught school before she went to medical school. She was really, she looked to me like an absolute angel. Years later, she married ultimately a surgeon, a gynecologist, I don't remember and lived in California. I think they had two children. During the mass vaccine trials that were carried out in Los Angeles, I think it was 1963, which was, you see, a long time after our grad school. I had to speak to a large audience in Los Angeles, there was Frances Holmes, still the same beautiful figure, still the same beautiful face, although with gray hair now. There she was and came running down the aisle. She knew I had a crush on her. I went back home with her, met her family and she comes to mind more than any of the other three girls.
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Q: Do you know if any of these girls continued their medical careers after getting out of--?

A: I think they all did. Certainly Frances continued even while she raised a family. Frances Holmes continued to do medical work. Another one who was MacFayden and who married a classmate, Netter. MacFayden was from North Carolina. I guess Frances was from South Carolina. She continued her medical work, but pretty soon became a columnist, because Frank Netter became so successful as a medical artist that he lived high on the hog. They had many children together. She became a medical columnist which I think she continued for some time. I think the others also continued Rianovskly who married somebody who was in the class ahead of us--Lockwood Lief. And then there was another girl. I don't know what she did, but I think they all continued in medicine.

Q: How were they accepted? Were they accepted with ease? Did the male students have any objections really to their being there?

A: No. I think it sort of--it was nice to have them around. Besides, it wasn't easy for a girl to get into medical school in the first place, they were very good, so they commanded respect on the basis of their ability. Secondly, each of them had her own specific kind of charm. I can't speak for everybody else, but I think it was nice. It certainly was nice. They are admired. There is no question about it.

Q: Dr. Sabin, I take it the pre-clinical years of medical were no great difficulty for you. Would it be fair to say that
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it was almost a repetition of what you'd had in the dental school before?

A: Yes. Yes, it was. Nevertheless, I had to do quite a bit of studying. It wasn't quite the same. Besides, you know, you forget. You learn, you forget, you learn, you forget. But I must say, I didn't find myself apply myself very hard. I never tried to be an excellent student, to make excellent grades. I don't know, I must have flunked one of the exams too as far as I recall. I only did what I had to because I already had my mind set on things that I wanted to do, in the laboratory, that was all.

Q: Were there any pre-clinical teachers that you found attractive to you?

A: Yes, many. Some of them I had before because the dental and the medical school shared them. But the first one I was exposed to was the professor of anatomy. Harold Dickinson Senior. I remember him very well. He was a Britisher who came to the United States. He was from Durham, also a fellow of the College of Surgeons. He was a man who did a great deal of research in anatomy. I'll never forget his opening lecture. He came in. He looked at this bunch of young faces eager to start on their careers. I don't recall his words exactly, but the sense of it stuck in my mind forever after.

He said, "You can't imagine what a relief it is for me to come and talk to you. I've been struggling with my research problems." I think he worked on the lymphatic system and made
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important contributions. And there were lots of frustrations. I came to know that myself later. He says "to break away from them and come to talk to you young people, eager to have something that I can impart." He gave me a gratification which I wasn't getting from my research work. So he made very good impact, philosophical impact, particularly on me. Anatomy during those years was basically taking home a box of bones first. Then the long hours of the section over stinking forminalized body and it was studying things by rote. Because at that time anatomy was not directly related to the problems that a doctor of medicine would have to encounter. If during the period of study of anatomy you could have the meaning of the particular part of the body, and certain problems that present themselves in disease and not only in surgery, the study of anatomy would be much more exciting. I think such changes have now come about in many medical schools.

Another very vivid figure was the professor of biochemistry. The professor of biochemistry was a man by the name of Mundell. He was a very imposing figure. I don't know whether he was originally Danish or what. But he was a colorful figure who made his lectures in biochemistry come alive because he wasn't merely trying to transmit a bit of knowledge as if it came as sort of a revelation. He was a man who during his years of training and study had come to act with the leading biochemist of that time, it was Germany in Europe, and always he would have anecdotal stories that related to something that we were studying, personal experiences. I remember listening to his lectures with
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a great deal of interest. And looking forward to them. The laboratory work was also interesting, although it was a repetition because there were people whom I'd come to know before, the people in the laboratory was a man by the name of McTavish who was the first assistant of Mondell's. He was a professor of mine at Washington Square, whom I came to know

And the other one was Dr. Gettler. Dr. Gettler was, held down a job as the chief toxicologist for the city of New York and he had innumerable suicides and all sorts of things that he'd have to be taken care of in the coroner's or medical examiner's office. You see the city of New York. His job was to do the chemical detective work. He was a man I admired, also because he was a very straightforward man. What sticks out in my memory, what comes back now, was that because I-- by questioning, I came a little closer to him. He was investigating the death of a number of people who worked in the watch making industry. They were painting dials, you know, with radioactive--with radium. How the devil did they get the radium in? He did the detective work and he found that they had to put on with very fine brushes, and they would stick the brushes in their mouth to point them up. I remember, I saw the first aural radiography, how he detected it in their bodies. He showed how the ash produced it on a radiograph. I have a very pleasant recollections of Gettler and Mondell.

Q: When did Keith Cannon come to--
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A: Keith Cannon came immediately after I had my biochemistry. He came, not immediately after, actually he came in 1930. So I was already in my senior year, entering my senior year. Because I graduated in 1931. I met him in a very odd way. As he arrived, he was making the rounds of different labs. I was already working in the lab upstairs. Biochemistry was below. He came up at a time when I was the only one working in the lab in bacteriology. He didn't know me from Adam. He came up, introduced himself. He was a young man, only 37 when he became professor. But 37 to me was already old at that time because I was quite young. He just stuck his hand out and he said, "I'm the new man in biochemistry. Who are you?"

Well, we got to know each other that way. Then in subsequent years, when he retired as professor of biochemistry at New York University and became of course, the director of the medical science division of the National Research Council. We had a lot of contact, because especially later, I was to be chairman of the Committee on Tropical Health for the National Research Council. We worked together in subsequent years. But it was a relationship which was established this way in 1930. He was not one of my teachers, but he was one of the brilliant biochemists—another Englishman incidentally. He got Paul Histrainey (?) in London, from the University who came to the United States. That was the brain drain at the time.
Another man who was really a great man, as he was subsequently acknowledged to be, because I came during a transition of physiology. Physiology then was a second year course, although it was begun by somebody whose name I don't remember now. It was Homer Smith who was the new professor. A very young professor. I think he was thirty when he was made professor of physiology. He was a most stimulating man because he certainly, his work in the physiology of the kidney, even then was already well known. He became professor, yes, in '29 when I was a junior. He was only 34 years old. He came from Hopkins. No, I think he got his training at Hopkins, his degree at Hopkins, Doctor of Science, and then he was professor of physiology at the University of Virginia. He was brought our school. He was an outstanding man. He certainly didn't glue in stature as a professor. He came at age 34. His lectures were stimulating. Of course, he became the father of the school of physiologists. Among them, James Shannon who really revolutionized. I am talking about Jim Shannon. Who helped to revolutionize biomedical research in the United States as the first really and most active member and director of the National Institutes of Health. I shouldn't say that. I should say as the director of the National Institutes of Health, under whose leadership and great influence, the National Institutes of Health grew from a tiny organization into perhaps the outstanding biomedical research
establishments in the world.

Q: But essentially as a student, you weren't exposed to Homer Smith.

A: I was exposed to his lectures. I mean, I didn't become interested in physiology. But certainly, it was wonderful to have him, although I had physiology before in dental school, it was a revelation to have it again from Homer Smith. And then also, the laboratory work was quite different.

Another person who apparently held a double, let's say appointment, which was not uncommon then, in medicine and physiology was Norman Jolliffe, who had made extraordinary contributions to nutrition. Norman Jolliffe, even though there were years between us, we became very good friends in subsequent years.

Q: Now, you know, when you look at the--

A: That isn't all. Because really, I want to say that there was a remarkable--I am sticking now to pre-clinical--

Q: Yes.

A: Because I've already spoken about bacteriology. Because I owe more to Dr. William H. Park, the professor of bacteriology than to any other professor in the medical school. But, I would say that next in line certainly the man who greatly influenced me and about whom I already talked was the professor of Pharmacology, George Barkley Wallace.
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He actually had been a professor for quite some time. He became a professor at age 30. But he was a professor at Bellevue Medical School since 1905. He'd already been a professor for something like twenty years. So that he was an extraordinarily wonderful, wonderful person. As I have indicated before, because he gave me opportunities to work in his laboratory and gave me human guidance as well as scientific guidance. We published together. I had a very special, warm feeling for George Barkley Wallace. So, you see, all in all I haven't mentioned everybody. But, they were a good group of professors. You see, I think, from the very beginning. I am talking only about the pre-clinical ones. It was a stimulating atmosphere.

Q: Now, the pathology department was run by a fellow named Alex Fraser. Did you do much work with Fraser at all?

A: No, not with Fraser. Fraser actually left no impression on me in the department of pathology because I don't know that he did very much. It was a course in which I was interested, myself. If you ask me now there was a name. I am trying to remember, and I can't remember who was the second in command.

Q: Was it Simmers?

A: No, that came later.

Q: Was it Dammon?

A: Simmers I worked with. No. But there were two assistant professors who helped me. One, well, not who helped me, but they were good, and I became good friends with them.
One was Curphey C-U-R-P-H-E-Y. The other was Dammon. Because they were really close to the students. And what was pathology at the time? Pathology was really microscopy. I took a great interest in that because I felt this was going to be one of my tools. Curphey and I became very good friends. Of course the neuropathologist was a very colorful man. His name was Globus. I greatly enjoyed studying neuropathology. I subsequently had to use neuropathology as one of the tools of my work. I never forgot the things that I learned from him.

Then Simmers was actually the professor of gross pathology where hundreds of autopsies were done a month. You see every month there were hundreds and then of course I was to serve an internship in pathology and got to know Simmers very well because I worked side by side with him.

Q: But as a student you went across to Bellevue for example, for your pathology.

A: Not across. What do you mean, across? Because the microscopic pathology was right in the building. But the gross pathology was at Bellevue Hospital. But we only observed. I didn't get to do autopsies until my internship when I had six months on pathology and did 400 autopsies myself during the six months, not counting the time I spent on surgical pathology and all others. It was the most concentrated experience in pathology that one could have in six months. I got to know Douglas Simmers because he was the boss and problems in diagnosis and so on and so on. I got to know Douglas Simmers quite well.
So, the pathology training came in very hand for my subsequent career. I must say that I spent a lot more time subsequent to what I had in dental school pathology than in some of the other things.

Q: Well, when one looks at the clinical years, one sees an array of clinical professors of extraordinary ability. People like Warren Colman, Wycoff.

A: I'll stop you right there. I would say undoubtedly Wycoff who then became Dean and was head of the service at Bellevue Hospital when I interned. He was an outstanding cardiologist. He had a group of people around him who did research, clinical investigation in cardiology. He was really outstanding. Then, of course he was professor of medicine also. There were other professors of medicine. The whole department of medicine was very research oriented, with Elaine Ralli and diabetes; and Bill Goldring in problems of the kidney.

The atmosphere was as much of a scientific medicine, a scientific medical research atmosphere as I think we might have found anywhere. The facilities were not as good. They were working under difficult conditions. But they were stimulating, very highly stimulating group.

Q: But it was clinical research mostly.

A: The professors of medicine, professors of surgery, should do clinical investigation. When you say it was clinical research mostly I think one must understand, and this is something that I
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came to understand very early. What is real clinical research? Clinical research is research carried on by person who is trained as a physician, but who has acquired a basic scientific discipline, one or more basic scientific disciplines to use in the investigation of problems of disease. This is what it still is, and should be. Because, while there are aspects of life sciences that can be carried out very well by Ph.D.'s, the basic clinical investigation is best done by people who have had a training in medicine either before their Ph.D. or after their Ph.D. It is the same thing, before. Or without a Ph.D. because the classical clinical investigator in those years, and I think the best still do it now. If they didn't have good basic science training, where they could actually use it, before they studied medicine, they would quit. Usually after their internship. And then go off for two or three or four years and not do any clinical medicine at all. They'd either do biochemistry or physiology or pharmacology or microbiology or whatever was going to be their tool for study. And then come back to the clinic.

I think that not enough of that is done now. I think these training grants at the present time are hurting (telephone). I think at the present time on the training grant system, where people assume after a residency or an internship or during the residency or internship, are sort of taken on as apprentices in a clinical investigation. I don't think that's a good way. I think they should stop their clinical work, all their responsibilities, and go and become a good biochemist. You
cannot become a good biochemist or a good virologist or a good--
almost anything in basic science that you have to use as a tool--
while you are taking care of patients, while you are doing that.
You have to concentrate. I've had people go through my
laboratory in exactly the same way.

Dr. Robert Channock, who had excellent training in
pediatrics, finished his residency at the University of Chicago,
and he came to me in Cincinnati and he had no patient responsibilities
at all. He just worked in virology. Dr. Robert Ward, one of
my first ones in Cincinnati, finished his residency in pediatrics
at Johns Hopkins. He had wonderful opportunity at Johns Hopkins.
But, he was interested in virology. His boss had a certain
respect for me. He sent him. He came to work with me. Although
he was a first class pediatrician and then became a professor
of pediatrics at New York University, University of Southern
California, where he now. He did nothing, nothing but virology.
That's the way, that's the way, the best clinical investigators.

Now, perhaps when you use the term clinical investigation,
there is, or there was still, at the time, the tradition of the
great tradition. You studied clinical manifestations without
using basic sciences at all. This was the British tradition
that turned out very very good clinicians who could smell a case
of typhoid across the room. But it didn't advance medicine to--
the way it advanced subsequently. Now, we had those also at
Bellevue because we were in a transition period. But, for example,
at the time I was there, the professor of medicine was Robert Carlisle, a magnificent man of the old school. A great clinician, but, the people in the department of medicine who were really making important advances were, of course, John Wycoff who was only an associate professor and people like Ralli that I mentioned. In addition to that, when I mentioned Bill Goldring, there was Bill Asher Pell. There were just any number of people who really were using basic science to advance medicine.

Q: Dr. Sabin, I'm surprised that you haven't mentioned Warren Colman.

A: I tell you very frankly, he just didn't leave an impression on me. I have to refresh my memory.

Q: He did the metabolic studies on typhoid fever with DuBois, very early. Now, it may be that--

A: How early?


A: That's it, that's it. He was already all burned out.

Q: Um huh.

A: He was already all burned out. So that he was not doing any work, and while he probably gave some rounds and we would make rounds and so on, he was not (sentence incomplete). The others that I mentioned were the young people. They were the avant garde. He had done something, but he stopped. You know, everybody has a half line.
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Q: Now, here you are, your interested in research, you are in your clinical years. How do people who are straight clinicians, who are also professors, people like Harlow Brooks or people like Sayre or orthopedic surgery. Or Howard Fox in dermitology. How did they--

A: Don't forget Stewart and Wright who were great surgeons of the old school.

Q: How did they react to young bucks who have this other tradition and who may not be doing their class work as efficiently as the others who are being trained essentially as clinicians.

A: Well, I would say that most of the people with whom I had more contact didn't know me from a hole in the wall. Perhaps the most relevant, the event that I remembered what you just said came more during my internship than during my school years. Very often I flunked exams because I didn't study very hard, for the facts. But, it was John Wycoff. I wasn't working in his field at all, but who was very good to me by giving me this opportunity to serve my internship at Bellevue Hospital. It was something to be sought for. But then, while I was doing my internship and working very hard, particularly after I'd come on on medicine, because I spent a year on medicine, after pathology and surgery, I sometimes apparently didn't attend to my duties too well.

He once called me into his office and he said, "Albert, I want to talk to you about the fact that, while I have the greatest admiration of what you are doing on the side." Because at that time I was working on pneumonia patients and developing
the Neufeld reaction and its application to the practical--tool. He said, "While I have the greatest admiration for these things which you are doing--" Also, you see, I was involved in studying the "B" virus because I'd just isolated the--

Q: We'll get to that later.

A: Alright, the point was that in medical school days, while I was a student, nobody bothered. But later on, it was (sentence incomplete). Again, it required understanding. But then it didn't require understanding. I either passed my tests or I didn't. Of course with some of the people, as a student, they knew I was doing other things. I established a relationship and it became more of a, like colleagues rather than student and instructor or professor relationship.

Q: I wonder if, while you were a student, you were exposed to really an extraordinary psychiatrist at Bellevue named Menas Gregory.

A: I'll tell you the extraordinary psychiatrist Menas Gregory gave us lectures, but he is not the man who left an impression on me. Maybe for those who, let us say, who were--who knew psychiatry better, he was an extraordinary psychiatrist. He didn't leave the impression, the great impression in psychiatry. Menas Gregory, I would doubt that he is remembered now. But the man who is remembered who was a research professor of psychiatry, is Paul Schilder.

Paul Schilder left an indelible impression of psychiatry. When he would take you around to see patients with his falsetto
voice, when patients would mock him, "You sissy you, for God's
sake you stinking so and so, etc, and etc." His reactions
would be--he immediately used that as a handle. "Why did you
call me a sissy?" Then his classes on hypnotism, you were
actually hypnotized during the process he was trying to tell
you. No, I tell you the one person during my school years who
was then a great psychiatrist later who left an impression on
me was Paul Schilder. As far as Gregory was concerned, he
might not have been there.

Q: Did any of the surgical staff leave an impression?
A: It came more as a subsequent relationship. Of course,
the professor of surgery was a most colorful figure. He was
a great surgeon, again, out of the school of the old school of
surgeons, Stewart. (pause) of surgery was George David Stewart.
He looked every bit the professor of surgery. He was an
imposing figure. I think he came from somewhere up north in
Canada. At any rate, he had been a professor for something--
since 1914. He really didn't come into surgery until 1930, '31
so you see he'd already been a professor for a long time.

He had as his right man Arthur M. Wright. He was
another very excellent surgeon. Now, there were others on
the staff. There was one, Dr. Standard, with whom I became
very close as a person.

Q: Did he ride a bike when you knew him? You know, Sam
Standard is the only man in New York who rides a bike.
A: Frankly, that aspect I wasn't aware of. There's John Mulholland and I am talking about people with whom I developed then a very good relationship. I'll never forget one special situation when I was a senior medical student. I was presenting a case for George Stewart, a great man. As I described the natural history of the disease of this man who subsequently died, I think I ended up—I think I used fairly literary language—because of my background. My language had to be literary because it was acquired, not at my mother's knee, but (laughter). And he stopped and he said "Sabin, you know that was a beautiful presentation you gave. Your English was very superb, the best I've heard in a presentation." But why did you have to spoil it by saying it that he petered out?"

Well, at any rate, the point was that we had these associations during clinical rounds and sessions. Then of course, I spent six months on surgery during which I came to know Arthur Wright, who then became professor of surgery. Actually, the paper that I published on "B" virus disease was with Arthur Wright. Not because Arthur Wright did anything. He took care of "Bill" Brebner who died. I felt, as a student, that I wanted to put his name on. He appreciated it. I had, somewhere in my files, subsequent correspondence from Arthur Wright while he was still professor. Then when he retired he became emeritus. He taught me things when I was an assistant in surgery that I didn't forget. It was great respect. As a matter of fact,
experimental surgery at that time was only just being established as a separate activity. As a matter of fact, I think they brought in somebody from pharmacology. But they were good surgeons. Very, very good group. I have very fond recollections of them.

Q: Dr. Sabin, as a medical student, how many babies were you expected to deliver?

A: The obstetric training was, of course, an extramural one. You were assigned to one of the many services in New York City. I don't remember. I must have delivered. I don't know. During the period of service. I must have delivered 75, 100. I guess that was about in 1930. It was during my senior year, because we graduated in '31. Oh, there are so many, many colorful events that suddenly come rushing to mind.

Q: I wish you would--

A: When I think how many are walking around the streets of New York that I might have delivered. Middle aged people.

Q: I wonder whether you had come into contact with Austin Flint at all. He was sort of the last of a generation of the last generation of several generations of distinguished professors of obstetrics.

A: Very very distant. Very distant. He didn't have certainly any close relationship. As a matter of fact, I'm not at all sure whether he ever appeared to the students at all. I think that what was done. The people who were taking care of
obstetrics were other people at all. As a matter of fact, I
don't remember that I ever saw him. I don't think he came.
I think it was Dr. Rice who was the associate professor who
gave us the lectures and early instruction. I don't think I
ever saw Austin Flint. I saw his photograph and I think that's
all.

Q: Well, the one department we really haven't spoken of,
which is really special and central to your development of
course is the department of bacteriology. While you were a
student, he was still alive, he was second in command, was
Charles Krumwiede. I wonder if you would tell me something of
your relations with Krumwiede.

A: I would say that there were practically none. Because,
if it had been left to him, I wouldn't have had the chance.
He turned me down. Krumwiede was never around in the department
of bacteriology. He was second in command to Park at the
division of laboratories of the New York City Department of
Health. We had almost no contact at all. He gave some lectures.
It was not that personal relationship that had developed between
Dr. Park and myself. So I would say, it wasn't that I held a
grudge against him or he against me or that he held me in low
regard. But because of that initial exposure, when I went
asking for a place. And he was absolutely right. As I have
pointed out before, I probably would have done the same.
He lacked a certain human quality. And so subsequent, there
were no relations.
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Q: Was it difficult? After all, was it-- Could you differentiate that Department from the New York City Health laboratory?

A: No. Because the people who were really responsible for the teaching of bacteriology, not so much infectious disease, because that became clinical, but bacteriology. These people were holding the really important jobs, dealing with medical and public health bacteriology in the division of laboratories of the city of New York. They would only come in. They were all part time people. They did their day to day work, their day to day chores. Whatever research they did, they did over there on Sixteenth Street by Willard Parker, which was the infectious disease hospital. They would just come in for lectures. So that the people who were on the spot, except for Klosterman who had a Master of Science degree. He was neither fish nor fowl. I'm sorry. He was a very nice man, but he was neither fish nor fowl. They were all visiting. But by God, they were right on the firing line. You see.

When they would come and give lectures, they gave lectures that had immediate relevance to the practice of medicine, to the practice of public health, to the problems of infectious disease, and that is now unfortunately lacking. Molecular microbiology or microbiology is a life science of tremendous importance, but there is such a thing as medical microbiology which is important by itself. Most medical schools in the
United States now have professors of microbiology who don't know a disease from a hole in the wall. They know the last little detail of some metabolic reaction, or something else. Which is fine, they ought to be in the University life sciences. I speak with feet in my voice. I rose here. Because I think this a great deficiency in our medical schools now. I think that people who teach medical microbiology should know disease. Infectious disease are still a tremendous problem not only in the United States. I mean, not only in the large part of the world that is undeveloped where infectious diseases are still the first cause of all disease problems. But even in the United States it's of tremendous importance and we don't have people who know beans about infectious disease in the part of microbiology--most of them--not all. Obviously, there are exceptions. But for the most part, the selection of a professor of microbiology in a medical school is on the basis that has very little to do with medical microbiology.

Q: So in a sense this was a great plus.

A: It was a plus. They were not full time, but that was the period. They were able to bring. They would come right from the front of action. It's a little bit like the political scientist or the economists at Harvard, who are rarely at Harvard or somewhere else. But by God when they would come and talk to the students, they came back from dealing with the actual problems. They brought life. I am not at all sure that in all things full time activity, which is divorced from the
problems, current problems of importance is necessarily the only way. I'm not saying that (sentence incomplete).

Q: Now, in a sense, you've described your days as a medical student, and I think if one were to use traditional criteria, one would say you were not a very good medical student. Were you ever worried where you were going to take your internship? What kind of internship could you hope for?

A: Alright. What's your question?

Q: The question is this: did you, in a sense, know that you were going to go to Bellevue. When I look at the places that your contemporaries go to, they go to the Brooklyn Jewish Hospital; they go to Mount Sinai Hospital; they go to a hospital in Patterson, New Jersey. But Bellevue, you know, that's the prize. That's like being appointed the Osler intern at Hopkins.

A: I'll tell you, I don't remember now how many places I applied to for an internship. I remember only two. One that I lost and one that I made. Of course, I applied to Bellevue Hospital because I was close to the laboratory and I wanted to be able to continue there. For the same reason that I chose the medical school, you see. I also applied to Boston City Hospital because Boston City Hospital, which was being run by Harvard Medical School in the same way as the Third Division only of Bellevue Hospital is being run by New York University was a place where infectious diseases were being investigated in an extraordinary way. And of course they had a great reputation. I remember going up for an interview there. One of the people who really has become sort of a father figure,
although he is only five years older than I or thereabouts, namely Maxwell Finland interviewed me. And very sympathetically in the hope that I would come on as an intern there. This was at the Thorndike Memorial Laboratory. But, somebody else—I didn't quite make it. So naturally, I was delighted when I was notified, and quite early, that I would have an internship at Bellevue Hospital. Of course, Dr. Wycoff was the boss. Although we didn't have much contact, I think he had, sort of sub-clinical I call it, sub-clinical respect for the things that I was doing. Not as a student with remarkable grades, but for the way I was mixing, you see, the mix of routine study and dedication there to work.

Q: Did you take a straight internship?

A: Actually, it was one of the most fortunate things that an internship that I was given did not start within after I got out of school, but started January 1st 1932. That gave me six months to work in the lab. There were many things I was then working on problems of pneumococcus infection and mechanism of disease and mechanism of death and all sorts of other things that I wanted to have full time. So this was ideal for me.

Besides, I think it changed my whole life.

Q: In what way?

A: I'll tell you how. If my internship would have started July 1st, then I would have been immediately involved in such hard work that when the epidemic of polio appeared in July in New York City, William H. Park couldn't have come to me and said,
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"Now, look, I think you should. I strongly recommend that you begin some studies on polio, this and that and the other thing." And I would never have become involved in polio. I might have done other things. I don't know. My life would have been, but it certainly would have been different. While I was working in the laboratory right after getting out of medical school, this huge epidemic broke out. My good old friend, Dr. William H. Park, was of course, in the midst of responsibility in dealing with it.

I didn't know beans about viruses. I never worked with a virus. Of course, virology was still in its infancy then actually. So, he came to me with a very specific question--always specific talk. Here we are, this was the next biggest epidemic after 1916, was developing in a tremendous way, and he said that Professor Jungeblut, Claus Jungeblut, Columbia University, just published a paper saying that he's got a skin test which will differentiate between those who are susceptible and those who are immune to polio. That's very important, why don't you have a look at it and see."

Well, one of the first things, when I looked at that, I said, "He's dealing with crude monkey spinal cord extract. I should, of course, do exactly what he did. But I'd like to have a pure polio virus preparation." So here comes in another interesting interplay of events.

I'd worked earlier with Dr. Sobottka about whom I've already spoken, a really first-class biochemist, trained with Willstätter and we were working side by side in the laboratories there at the medical school. Now he had a great influence on me because he
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transmitted knowledge to me that I might not have had time to read about. So one of the things, obviously, that was going on in Willstätter's laboratory, was the utilization of alumina gels for the purification of enzymes, so they could study enzymes. As a result of that, the question arose in my mind "Why not use the sieve," because at that time we had no idea about what viruses really were. I said "Why don't I try using the alumina gel technique that Willstätter used for purification of enzymes to see if I can purify polio virus" So I asked Sobottka to teach me everything that he could--that he'd learned in Willstätter's laboratory about the use of alumina gels, and he did.

I went to work concurrently with working on the skin test problem, to purify polio virus by alumina gel, and it worked. Now getting back--because alumina gel c very specifically, unlike other gels, absorbed polio virus out of a tremendous _______ of the spinal cord suspension. Then on elution of a certain ph gave a very pure thing, and how did I know? I had to get to work with monkeys. You see, because the only way I could measure the activity and the changes and all that--well, I had to get to learn how to work with monkeys, with polio virus in monkeys. Therefore, my life was actually changed because my internship didn't start until January 1st, 1932.

Q: You know, I think you ought to say something at this point what the armamentarium was of laboratories at that time to deal with viruses. You know, today you have Tiselius apparatus,
you have an ultracentrifuge. You didn't have any of this for purification of concentration of viruses.

A: Incidentally, the first use of the ultracentrifuge came when I went to the Lister Institute later. At that time, medical virology as planned virology (end of A side of tape).

BEGINNING OF SIDE B

A: ... studying or pursuing my curiosity by what was it that killed animals and human beings with pneumococcus infection. I was naturally intrigued by the papers that Parker published by himself, with McCoy and so on. A certain toxic substances that he obtained by the anerobic autalysis of concentrated pneumococcus suspensions. The toxic principles in these anerobic autalyses were capable of producing necrosis when injected into the skin and death associated with marked pulmonary lesions when it was injected intratracheal into guinea pigs.

Naturally, the question here was, and then this was followed up, before I take the question, because this is important. Parker and an associate of his by the name of McCoy then reported in 1929 the production of a potent antitoxic serum in horses that was able to neutralize these toxic effects in guinea pigs. I was intrigued, not just by this phenomenon because to me, and this is something, if I think back now, was always the issue of when you have an interesting phenomenon that you study, what is its role in the natural infection. Infection, whether it be in
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an animal or in man. So the question was, how do we find out whether or not this toxin that was produced by experimental procedures and for which this potent antitoxin is available? Did it play a role in human disease? Was this perhaps the way people died, despite the fact that you gave them serum and you had no more pneumococci in the blood, they went ahead and died anyway. Or studies that I had made on rabbits already where you could prevent the invasion of the bloodstream by bacteria but this huge lesion that you produce went on and killed the rabbits.

That is why I became interested, and also the question that if it really played a role, then you'd be able to use this to help man. See, I mean, was this merely a nice laboratory exercise or did it have potentialities? That is why I designed a series of experiments to determine, in the laboratory, whether or not this actually played a role, and discovered that it played no role. How? Because I was able to control the bacteremia, so called invasion of the bloodstream with anti-pneumococcus serum, not the antitoxin, and the so-called skin pneumonia quote, the Goodner Model ("Goodner Model") where injection of pneumococci into the skin of rabbits produced the huge lesions, just like the consolidation of the lung, or the pneumococclyn in the lung, and then I tried to see whether using this potent antitoxin, I could prevent death. And it didn't prevent death. Now, as I see it, my conclusions were, since the anti-pneumotoxic serum failed to modify the course of pneumococcus infection as shown in the mice and rabbit experiments, it seems fair to assume that the
anerobically produced toxins are probably products primarily of the enzymatic changes occurring in avetrotolisis (?) and play no part in the course of natural infection. It was just one step on the way of exploration to the causes of death from a bacterial infection, and with all the work, wonderful work, that's been done in the intervening years, I say intervening years because I see that this paper was published in the Proceedings of the Society for Experimental Biology and Medicine in 1931, the year that I was out of medical school. I did the work as a medical student.

In the forty some odd years that have intervened, we still do not have answers of what is the cause of death, or serious disease from certain bacterial effects.

Q: The next problem you posed for yourself is the way anti-pneumococcyl serum actually works, and whether it works the way people thought it worked. I wonder if you would tell me something about these experiments.

A: Well, here again is the influence of my association with Sobottka. Sobottka, of course, had the job. It was a mission-oriented research project. That's why he was brought in to study the role of soluble specific substances, the carbohydrate, capsule substances of pneumococci. Because this was the forefront, the forefront of investigation of pneumococcus infection because Avery and Heidelburger (?) and Gobel and Sobottka was in with them. It was believed that this was basically that the material in the capsule determined whether an organism was virulent or avirulent, if it was rough, it didn't have too much of this
substance, it was avirulent. If it had a good capsule and the capsule was made up of this specific soluble substance, it was virulent. So the question that came to my mind, I say it was generally assumed, that the soluble specific carbohydrate of pneumococcus can neutralize the type specific protective action of antipneumococcus serum. Antipneumococcus serum worked by combining with this, and that's how it worked.

So, I undertook to determine whether antipneumococcus serum contained not only types specific protective antibodies which could be neutralized by soluble specific substance, but also other things. And, well, this work, again, was done during my medical school days because the paper in the *Journal of Experimental Medicine* on it was published the first of January, 1931, while I was still in medical school. Well, the conclusion was that the mutual relationship of the anticarbohydrate precipitins and the protective action of antipneumococcus serum to the soluble specific substance was the basis for investigation.

I'm going to read, if I may, just to refresh my mind, "The assumption is made that there exists in antipneumococcus serum types specific protective antibody which is distinct from the anticarbohydrate precipitins and is not neutralized by the soluble specific substance." This is the assumption that I—that had come from the experiments that I designed. "This assumption is based upon the following observations in experiments which were conducted primarily with Type One antipneumococcus horse serum."
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These were the experiments that I did. "One, lack of proportionality between the quantity of soluble specific substance added and the amount of anticarbohydrate precipitin and protective action neutralized. Protective capacity of specific precipitates is accounted for on the basis of the liberation of non-specifically absorbed protective antibody. Three, the soluble specific substance only partially neutralized the protective action of antipneumococcus serum envivol (?) and four, the types specific protective antibody remains in antipneumococcus serum after complete precipitation of the anticarbohydrate precipitin."

Really, all the other ammunition is perhaps irrelevant because once you take out everything, there was still types specific protective antibody. This residual types specific protective antibody was not neutralized by additional soluble specific substance, nor by absorption with heterologous pneumococci. It is definitely absorbed by the omoligus (?) whole pneumococcus. So from these experiments, that I carried out, I had to conclude that there was another types specific factor in the organism in addition to the specific carbohydrate that everybody assumed was the beginning and the end of—

Q: And you assumed that this was a non-antibacterial factor?
A: No, no, no, because the conclusion was that it was absorbed by the holmologous (?) pneumococci. Since it was absorbed by the holmologous (?) pneumococci, it must have been on the organism or in the organism. In other words, that the
pneumococcus in addition to having types specific soluble carbohydrate had something else that was types specific for which an antibody was present that gave the serum its protective capacity.

Q: Where did you go from there?
A: Oh, God, I'd have to refresh my memory.
Q: Yes. (tape shut off)
Q: Dr. Sabin, in June of 1942--19, let me change that.
On October 1st, 1932, the Journal of Experimental Medicine published, really, a very interesting paper of yours, and I would like to read the first paragraph of this paper, which in a sense states the problem.

"Despite the numerous clinical and experimental investigations on antipneumococcus serum its exact role in pneumococcus infection is still relatively obscure. The purpose of this communication is to report experiments which show that antipneumococcic serum contains an important therapeutic factor which is not antibacterial in character."

And I wonder if you would speak to the development of that work.

A: Well, this paper was actually a continuation of my basic interest in the mechanism of death and recovery in pneumococcus infection. To recall the theories and conceptions of the action of antipneumococcus serum at the time, it is necessary to point out that the way serum to be used for human beings was standardized was by measuring its capacity to protect mice against infection by a certain dose. So the mouse-protective potency was the measure of
the potency of the serum, or the purified globulin to be used for human beings.

I already had done some experiments which suggested that that may not be the whole story. This again, came from observation, clinical observation, as a student when I was working at Harlem Hospital. I saw patients who were treated with serum at a time when they had bacteremia. They had many pneumococci in their blood. Subsequent blood cultures showed that the bacteremia had disappeared. The serum was able to take care of it. But the patients went on and died. So here is another example of learning--this is what a real clinical investigator's function is. He observes the course of events, and then to see if whether the explanations are really--are compatible with the course of the disease in man. So I wondered whether or not there may not be something else. Now, how to investigate.

Dr. Goodman (?) had just joined. He came from Harvard Medical School. He was a Ph.D. Just joined the staff with Avery and the other group, this wonderful group of people who were working at the Rockefeller Hospital on pneumococcus infection, with the wards for pneumonia and so on. He developed an experimental model that was different from the mouse that was inoculated with a certain small dose of--a number of fatal doses--of pneumococcus. His model consisted of inoculating pneumococci under the skin. They developed a terrific edemous (edematis) lesion. It almost--it looked like a consolidated lung, except it was the skin and under the skin. It seemed to me that that was a model that could be used for answering some of the questions.
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I studied the course of the infection in this model and found that it, of course, there was a great deal of multiplication of the organisms right under the skin that formed this huge lesion. Also that there was a bacteremia. So in a sense, it was a little similar to the human disease, where you have consolidation in the lung and organisms in the blood. One of the first things I wanted to see was, whether or not if you just used enough serum to prevent the bacteremia, whether you could prevent death of the animal. And I found, interestingly enough, that that was not the case. As in human beings, you could eliminate the bacteremia, but the rabbits went on and died anyway. Mind you, this is allowing first of all the lesion to develop so it would be comparable to what you are doing in man. I always wanted to have something that is comparable. I didn't want to get a rabbit that I just injected and he has a little inflammatory lesion, because that's not how you get a patient in the hospital. So I wanted it to be bad. You see, just like the patient. And then, this then provided an opportunity to determine whether or not there was anything, other factors in the serum that were quantitatively different from its mouse-protective that might influence the outcome of this infection.

What I did was to first of all, absorb out from the antipneumococcus serum the antibody that could be absorbed out with bacteria. Previously I had already studied the soluble specific substance, and was able to show that the soluble specific substance was not the whole antigen which combined with the antibody that provided protection. That there was something more
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specifically on the organism. So now I used the whole organism. Then, in experiments which were designed to give only just enough serum to prevent the bacteremia, but not death of the rabbits, I tried to add the absorbed serum which was now deficient in the mouse-protective capacity to see whether the course of death in rabbits could be prevented, the incidence of death. And it was. The conclusion from this, without going into too much detail, was that there was something else in the serum. Aside from the types specific thing, because I could show that the factor that remained was not types specific, that it would work equally—that it would play a similar role against type two and type three, and therefore became reasonable to conclude that the whole serum and particularly I studied serum produced by Dr. Bansohoff (?) where, in addition, to immunizing horses by just inoculating them intravenously with sedimented heat killed pneumococci broth culture, the free—the bacteria filtrates were used, that there was some other factor, which couldn't be absorbed out with the bacteria. That's why I called it a non-antibacterial therapeutic factor. Now, when I look back on some of the experiments, I didn't use—I find that I used rather a large number of rabbits. Like I used hundreds of rabbits in some of these experiments. I'm looking at the paper now, and I see in Table 4, for example, in the first paper in which the non-antibacterial therapeutic factor and standard pneumococcic serum, and just for control, I had 40 rabbits, of whom none survived.

Then I used a sub-effective dose of this serum by itself—31 rabbits, 29% survived. But then I move on to a sub-effective
dose of the serum with the absorbed type one serum, and 43 rabbits and 83% survived. Then I use a sub-effective dose of the type one serum with heterologous serum, type two, you see, and 70% survived. So, in this experiment, I see that I used close to 200 rabbits. The numbers were significant. That's just one, one experiment. Well, then, the work went on to see, again from the practical point of view, because the thing to do then was to use a certain part of the globulin, the portion that was precipitated when you increased the concentration of ammonia sulfate from 30% to 50%. I wanted to see whether we were losing some of the stuff in using the purified, and apparently we were. Then there was the second work in which an attempt was made to find out the distribution of this additional factor in different globulin factions of the serum. It turned out that it wasn't distributed the way the mouse-protective type specific, protective antibody was. Therefore, that there was a certain balance between the two that was required to get the optimum results and the conclusion from that was that obviously, when we use only the one kind of globulin from that serum, we were not getting everything out that was in it.

At any rate, it merely threw a little more light on what was going on, when an animal with pneumococcus infection, died without having organisms in his blood. It was a long way from explaining---

Q: Now, Dr. Sabin, these are obviously important papers to you and it's obvious that you spent, really, I can't see doing this kind of work under a 13 or 14 hour day. Now, you begin this kind of work. You have this six month period of grace, in essence.
But what happens when you have to take care of patients?

A: Alright, now let me perhaps, an interesting aspect of this will show—Let's take the first—well, there were two papers.

Q: Yes.

A: ... that were published on this subject. One I sent in on June 2, 1932. One June 2, to send it in on June 2, 1932, I must have done it during it—must have written this work up, you see, very carefully, and it takes a lot of time to write up a paper like that, because it is fully documented. I wrote it up during a very difficult period. What was I doing during that first six months of 1932? The first month, or some month in between, I forget whether it was the first month—it must have been the first month.

I was on ambulance duty. One month on ambulance duty. It was an extraordinary experience. In New York City, in one month on an ambulance duty at Bellevue Hospital, you see almost every conceivable thing. From some construction worker's having his legs broken because something collapsed on him, from somebody committing suicide, from somebody being cut to pieces with a bottle that's been broken in half and the ragged edges used to slash somebody up, from people dying in a coronary attack, from people with acute appendicitis—you really—it's the most concentrated experience.

Then I go on to pathology, and I do autopsies—autopsies from morning to night, I did 400 autopsies. It was during that period that I must have been writing—in addition to everything else, writing up this paper.
The second paper, I notice, which was published on January 1st, 1933, was submitted August 19, 1932. By then, you see, I was already on surgery, because the next—after pathology came six months in surgery. That was an all-round-the-clock job. That meant that instead of catching some sleep, I was writing, getting my data together, to present. So, it was one of those compulsive things where many things were combined. I don't think I was doing a bad job as an intern on pathology, you see, when I was doing all those autopsies. Because I used to stay up till midnight, studying the sections. Because internship in pathology at that time meant you did everything from beginning to end. You did the autopsy. You looked at all the sections in microscopy. You submitted your final report to your superior officer. Then you had to present it at clinical pathological conferences. Or when you were serving on surgical pathology, you'd have to be in the operating room and get the sections. And it was a round-the-clock activity. After you'd get five hours sleep, you were lucky, in addition to that, to write papers. It wasn't easy now that I think back.

Q: Not only to write the papers but to do the work.

A: Well, if you think about it. No, you see the point is, I was no longer working in the laboratory when I wrote something up, or finally submitted it for publication, on August, 1932, that was the work I had done between, let's say, the middle of June, 1931 and the end of the year, 1931. So, you see, the laboratory work, the experiments had been accumulated, but now they had to be written up. As a matter of fact, I wrote up much more work
during those years than I have done subsequently, because I have a Pandora's box of experiments that were never written up. I was more conscientious in writing things up then than I was subsequently. So it was a tough round-the-clock activity. It certainly didn't allow any time for any social life or anything else.

Q: Well,

A: Mind you, the other things I was doing then, when I got on medicine, because I notice publication in May, 1933, in the Journal of the American Medical Association about immediate pneumococcus typing using the Neufeld reactions that I was doing in addition to taking care of patients and learning all the clinical aspects of medicine, I was doing research on the side also.

It looks like it's no different now than it was then.

(chuckle)

Q: I think at this point, we can stop. It's really been a tough--