Dr. Albert Sabin  
Charleston  
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Q There may have been a real transmission, although I am not aware of the proof because they used a strain of recent human origin that may have produced a high enough viremas. Just mechanical transmission might have been sufficient to do it. The failure to repeat the experiment again, as we know in passage in monkey brains, the virus change. The inability of Flexner to reproduce it could be explained for the same reason, or because it really wasn't transmitted in the first place.

The reason for taking it up again in 1940 is a totally different one. The reason was that for the first time, polio virus was demonstrated unquestionably in the stools of human beings, who had polio and also in non-paralytic polio by the work of Paul and Trask, and therefore it became, and it certainly was confirmed by others, and then it became necessary to know whether or not biting flies, blood-sucking flies, but flies who feed on human feces might become sufficiently contaminated with human virus, whether or not it multiplied in them, to deposit it then on food that would be eaten by susceptible persons. And to thus transmit the disease to a person without having, who had not had human contact with another carrier, with another individual. That was the reason for undertaking that.

Now, even though we knew, and Dr. Ward and I undertook our study, that Paul and Trask were just doing that. They were doing it in a rural area, and in a camp where the possibility for open privys, and the possibility of human infected feces being
to the flies was obvious. You could look at it. You could see the flies landing on human feces. But Dr. Ward and I were studying epidemics in Atlanta and Cleveland, in very nice housing projects where this obvious thing was not available. I think the maximum flight range of such flies is perhaps only two miles. We looked within the areas, to where they could get it. That's why we made a more concerted effort on the urban conditions. It was to our surprise that we found so many of the collections for flies to have polio virus in them. Let me just refresh my memory.

I say the first specimen of flies to yield a virus has a rather interesting history. You see that history is the site where the trap was set was a government housing project consisting of modern, clean thoroughly screened and hygienic homes situated on a hill in the center of Cleveland. There was a special brick enclosure for the garbage cans, all of which was covered. Two children who developed poliomyelitis on August 7 and August 9 respectively were admitted to City Hospital on August 11 from one of these homes. Investigation on August 16th revealed that two of the four siblings had been ill for one day, with signs and symptoms suggestive of abortive polio and that between August 7 and 13 seven other children in the homes facing on the same yard had minor illnesses compatible with the diagnosis of abortive poliomyelitis. There was also the story that about a month before, early in July, there were only a few cases of polio had been reported in Cleveland, after a severe storm, the
sewage overflowed, ran down the street and some of the children became contaminated in the course of play. There were so few flies about that it hardly seemed worthwhile to set out a trap. However, about 500 flies, not identified as the species, and they were mostly *musca domestica* and *fornio regina*, caught between August 16 and 18, mind you that's about a week after onset, yielded the virus upon inoculation of a cynamolgous monkey.

The second positive result of the Cleveland specimen were the flies caught in the back yard of a private home, many miles from the first site, at the other end of town, in which three children of one family developed polio between July 25 and July 30. Two were removed to the hospital on July 30, and one on August 4. The home was fairly clean inside, with suitable toilet facilities. But it housed seven other siblings, two of whom had had questionable minor illnesses. Four other children in adjacent homes have histories of having had minor illness compatible with abortive polio. Incidentally, I would like to interject I think this was also--this was a family group studied by Tommie Francis and his associates. Now, there were open garbage cans in the neighbor's back yard, and many flies were present, a larger number of flies were caught in the trap.

Well, then in Atâanta specimen, was collected between July 3 and 31st representative of who, caught in two places. One more or less in the center of town, and the other the outskirts.
The point was, and then we isolated still another one, because I find a footnote—"Since this paper has been submitted for publication we have demonstrated the presence of polio virus in two additional specimens of flies caught in two different regions of Cleveland during August 9, August 12."

The mystery to us was in areas with modern sanitation, not rural areas like the Trask and Paul were studying. There too, we would find flies contaminated with polio. Frankly, I do not wish to speculate at this time where these flies picked it up, whether these flies became contaminated during the period in one instance where the sewage had overflowed, and was still contaminated, or whether in the other places, where there was no such history, they got it from somewhere else, the point was it is a fact. Subsequent, very careful studies by Dr. Melnick and others show there is no multiplication of polio virus in flies.

So there was a reason for doing it, for reviving it as you indicated, in 1940, which was quite different from the one pertaining in 1912. And basically, while it is conceivable under certain conditions that flies could be an additional medium for transmission, the conclusion with the basic chain of transmission is really person to person—remains valid, and interestingly enough, in later years, which proved it ideomologically, when the orally administered live polio vaccine was administered on a sufficiently large number of persons, it was possible to break the chain of transmission and to eliminate paralytic virus from very large areas where such studies were made. The mystery
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still remains where the flies got it. Dr. Ward's subsequent experiment with feeding the bananas to chimpanzees proves that it is possible to transmit polio even to chimpanzees, much less susceptible than a child by contaminated food.

When I recall my experiences in places like Egypt and in places like India, and many other places where you almost have to just chase the flies off the faces of the children in order to see them, or where they have to move the flies off their food in order to be able to eat what they eat. Sure, they could play some role in transmission. Later, in a subsequently in 1947, when I was at the clinic in Berlin, and I had an opportunity to study just epidemiologically the incidence of polio cases in the parts of Berlin occupied by the United States, the British and French forces, and the part occupied by Soviet forces. Under no barriers to crossing from one to the other, the much higher incidence in the Soviet parts of the city thought correlated with the fact that flies were eliminated extensively by spraying in the zones not occupied by the Soviet Union. While the Soviet Union did not permit such spraying, and the flies were much more numerous, and moreover, there were, food was hard to come by, and many people were growing tomatoes and other vegetables in flower box in little patches of garden which was fertilized by human feces, so I saw the possibility that maybe flies could play, an ancillary role, that I think that's the end of the story of the flies, and that's why no more has been done on it.
Q In doing the research, at the time, did you have difficulty, great difficulty in getting animals to work with?
A The animals which we used mostly. At the time there were two periods. There was the period when I worked at the Rockefeller Institute, and then in Cincinnati. We used either rhesus monkeys. In Cincinnati, we used cynamologous monkeys. They were very cheap by comparison, six, seven dollars apiece. I had enough monkeys at the Rockefeller Institute. Maybe Dr. Flexner's interest was so great that somehow or other, they were brought in. I never lacked for monkeys for any of the experiments. In Cincinnati, by that time the National Foundation was already established and functioning, and I think it had a special division to help with the importation of monkeys. I don't remember opening farms, and there were all sorts of places--
Q Okatie Farms--that was later.
A Maybe it came later. At any rate, the point was that while the numbers were not very large, I had no trouble getting enough monkeys.
Q You got them then from dealers?
A From dealers.
Q Did you ever get any from Frank Buck, the man who brought them back alive?
A I think Trefflich was right on him. Good recollection of him.
Q One of the important questions that you've brought was the whole problem of criteria for polio infection. When are you faced with a polio? The reason I bring this up is would
it be accurate to say that as late as 1940, '41, clinicians were still making mistakes in diagnosis of polio?

A There are two problems in that. Because the realization even with the technology available at the time, that there was so-called abortive polio, and non-paralytic polio, as well as paralytic polio was such that doctors were diagnosing non-paralytic polio as well as paralytic polio. As a matter of fact, public health reports reported everything together. You had 40,000 cases of polio that included almost 50% non-paralytic polio. To say that the clinical diagnosis of non-paralytic polio is an absolute impossibility became very clearly established within a few years of about ten years when it became evident that we had Coxsackie viruses and Echo viruses, among other things that were able to produce the clinical manifestations of non-paralytic polio, which was basically an aseptic meningitis, that was not associated with paralysis, although in occasional cases, some of the Coxsackian and Echo viruses produced paralysis occasionally.

Therefore, one can say that by and large, the diagnosis of non-paralytic polio was an impossibility—a clinical diagnosis—it still is. You can’t do it. The probability may vary. During an epidemic, you may have more or less, but it doesn’t matter because we find that later, even during an epidemic of poliomyelitis, there are other viruses spreading too—Echo and Coxsackie viruses. Now, as to the clinical diagnosis of paralytic poliomyelitis—I would say that probably in 90% of the cases, particularly during
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an epidemic, there is no danger of a mis-diagnosis. But, unless very careful criteria are used for the clinical diagnosis, it is possible to make mistakes to a varying degree. For example, to make a diagnosis of paralytic poliomyelitis in the absence of an increased number of white cells, about ten or even more, in cerebral spinal fluid, as it is—we know now, at least at least on the basis of my own experience—that there is a 90% probability that if a patient with flaccid paralysis that looks like polio is seen during the first week after onset of the paralysis, and he has no increase of white cells—that is pleocytosis of the cerebral spinal fluid I would say there is a 90% probability that it is not polio. There are things like Guillain Barre syndrome, there are certain motor neuritis cases there are sometimes even the possibility that multiple sclerosis can start in a way to confuse the issue. We certainly had that problem a great deal during the oral polio vaccine trials, and also during the Kild Vaccine Trials.

The figures that I find particularly revealing were those that came from a study on which I collaborated in Mexico City during the period after oral polio vaccine was introduced, although it had not in those years been used very extensively in Mexico City, but one of my associates, Dr. Manuel Lamos Alvarez, who worked with me from about 1953 or '54 to '57 and then worked in the Children's Hospital in Mexico City undertook a very interesting study. That was that every child with a diagnosis of poliomyelitis, with flaccid paralysis extensively fit polio, had died was autopsied and a complete histological examination was made, and he obtained
materials from both the nervous system and from the stools for isolation of polio virus. One of the interesting things that happened in that. Of course, we already had some impact of certain number of children having been protected by the oral vaccine which was given, so there was an increase in the relative proportion of other things that ordinarily might be present in lower incidence. But nevertheless, approximately 50% of the fatal cases over a period of five years. The numbers I don’t remember exactly. Turned out on histological examination not to be polio. Furthermore, on further analysis of the histology in which then I joined in, it was discovered that only a few—and this is out of about 25 that turned out not to be polio—only a few turned out to be the usual Guillan Barre syndrome. They followed a pattern that you might have recognized clinically. But the others started with paralysis anywhere in the extremities, and really mostly, not always, rapidly progressed paralytic disease. There were two identifiable categories, histologically.

One was the category that, in the first place, it was non-inflammatory. There was no inflammation. There was no question of separating it from polio and also polio virus was not isolated, or any other virus. But the neurons were effected in one group which I christened cytoplasmic neuroneopathy, and that referred to the fact that we found all the nerve cells—they were there, but the cytoplasm was complete abnormal. The nucleii were shoved to one side. The Nissl substance was mostly gone. It was not a secondary reaction to injury in the
peripheral nerve roots, because we examined the nerve roots and they were mostly normal. So it was a primary thing which was attacking the cytoplasm or the motor neurons enough to lead to death without any inflammatory response. The interesting thing, also, was that they had no peer cytosis. They had no white cell increase, increase of white cells in the cerebral spinal fluid.

Another category turned out to be something that I called nuclear neuronomapathy because the cytoplasm of the neurons, the motor neurons, was alright. The initial substance was there. It had no abnormality, but there was something wrong with the nucleus. When we used the silver stain looking for certain changes in the routes, and the tracts, it turned out that these degenerated nucleii had a terrific affinity for the silver, and they stained black. So, they stood out. So, I called these nuclear neuronomapathy. Something, we don't know what, attacking the nuclear area of the neurons here, or the cytoplasm in the other category, enough to kill them. Interestingly enough, when I started to look through such material and in Cincinnati, when I got back, I found one patient who, for example, had similar lesions of nuclear neuronomapathy. To the best of my knowledge, more of that has been found in Mexico City. This was published in the Journal of the American Medical Association. I forget whether this was '69 or '70 now I don't know of any follow up work by others. But that taught
me more of a lesson more than any other of my clinical experiences
that the diagnosis of poliomyelitis may be correct on a clinical
basis to a certain level, under certain circumstances, but it can
certainly not be made with absolute certainty. There was even
one instance where we isolated polio virus from the stools of a
child that died, but there was no polio in the central nervous
system at all. If you had come up and caught with a patient
like that, and you didn't have an autopsy, and you said, "here
is a child with paralytic disease. I isolated polio from the
stool, it must be polio." The probability, but you can have
polio virus in the stools and something else affecting the
nervous system.

Q When I raised that question, I thought that you would
grab in your bit and talk about your work with Charley Ehring
on infectious polyneuritis.

A The point is that the case of infectious polyneuritis
that I studied with Dr. Ehring presented no problem of differential
diagnosis with poliomyelitis at all. They were clean-cut cases
of infectious polyneuritis which no self respecting neurologist
in adults would have a problem differentiating from poliomyelitis.
It is quite a different thing. However, during the period when
we were doing all autopsies on fatal cases, we at that time, 1940
or thereabouts, there was a general impression that infectious
polyneuritis of Guillam Barre syndrome, it was the same thing,
was practically unknown or very rare in children.

I remember a child being rushed in a hundred miles from
Portsmouth, Ohio and I think it was Portsmouth, and rapidly put into
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a respirator. We hardly had a chance to get any specimens at all, it was during an outbreak of polio incidentally, in Cincinnati and there. The child died within a short while. As we were doing sterile autopsies, we did one on this child also. It turned out that this child died of infectious polyneuritis, or the Guillam Barré syndrome. There was no time to do a spinal tap on this child. It had to be put right into the respirator. But I have seen patients, autopsies who had the Guillam Barre syndrome and who did not have the increase in protein in the cerebral spinal fluid. Alright, they didn't have the cells, the increased cells, but they didn't have the protein. Unless the process involves routes where you are down at the bottom, you don't get any. This was another lesson years ago, but the work with Dr. Ehring on infectious polyneuritis was specifically to see whether or not some infectious agents could be isolated. It was interesting because it gave me an insight into this disease which came in handy later. And which led me at least to the hypothesis that we were dealing in infectious polyneuritis with a condition in which a bacterial infection, either in the upper respiratory tract, usually, or in some other tracts, perhaps in the eteric (?) tract. Liberated exotoxin, that had a certain affinity for certain nerve routes, peripheral portion of the neurons. Now, I was influenced by that to a considerable extent because of my experience with the model systems studies with mice, and pleura pneumonia like organisms in which I was able to demonstrate in the culture
an exotoxin which was responsible for the specific lesions produced in the central nervous system, very specific affinity just for the lower pole of the cerebral. Extraordinary

And I said to myself that if a pleura pneumonia like organism can produce an exotoxin with such specificity that it will just take the lower pole of the cerebellum, why couldn't other organisms produce again toxins of extraordinary specificity, some that will take the brachial plexus, others that will take the lower extremities and I don't think that the picture has advanced very far since then.

I still maintain this hypothesis.

Q That is gorgeous. This is exactly what I wanted from you. At this point, what I would like to do is to talk about your going away to work with the neurotropic virus commission, and eventually going to war.

A Let me, may I interject a few things?

Q Sure.

A I am interjecting here because of your use of the word going away to war. The work with the neurotropic virus commission did not require going away. Most of the members of the neurotropic virus commission of the board for investigation of the influences and other epidemic diseases in the army to involve a certain number of commissions included investigators who continued to work in their own laboratories. That participation in that activity did not require my going away from the laboratory. It was another decision in connection
with this work that did take me away from my laboratory.

Q  Well, if one looks at the neurotropic commission or indeed, the advisor to the army at this time, on medical matters, one finds almost a predominance of people who have what we might call a route to Johns Hopkins, a route at the Rockefeller Institute, or the route at Yale. It almost seems to be a club. Would it be fair to characterize?

A  I think, of course, this is largely true. I knew it was documented. One of the reasons the explanations for it is that when people are called upon to work a group who working in a certain field are much more likely to call in the people they know. Now, I had been at the Rockefeller Institute before, but I wasn't at the time that I went to work--I became a member of the Neurotropic Virus Commission. But my associations with John Paul, who was the elected, who was appointed chairman of that commission. John Paul of Yale, by Francis Blake of Yale. And Francis Blake of Yale was appointed because, being Stan Hope, Bayne-Jones of Yale had gone in as deputy chief of preventive medicine in the army. You see, you call on people you know, and with whom you've worked for many years. I was a member of the club indirectly. Now, Dr. Hamman who became a member of the virus commission was never at the Rockefeller. He never--he was initially, I think, still out in California, San Francisco if I am not mistaken. And later only moved to Pittsburgh, so he would be an exception. I think Dr. Hamman was in with us from the beginning. Dr. K. F. Meyer was in San Francisco, and
he had neither Rockefeller nor Yale nor Hopkins associations. But if you'd say predominantly, you are correct. But I think the choice was basically made, not on the basis of the buddy system, but on the basis, I think, of selecting people who were known who had certain expertise, and who had been working in the fields.

Q If one had to say--
A But the Yale orientation, you're right. I think there is no question that the very important and fortunate fact that B. J. as he was affectionately known, or Bayne Jones, was appointed deputy chief of preventive medicine. The surgeon's general's office of the army led him to rely on people he knew predominantly. But before, even though Francis Blake of Yale was the chairman, there were other people like--it's true, they most were from the Hopkins or Rockefeller. Avery was on the board. Maxcey from Hopkins was on the board. Dochez, and one maverick was Perry Pepper from Philadelphia, from the University of Pennsylvania. But I would say that in all instances, they were really top people. No matter--I don't think other very good ones were left out, because they were not members.

Q I didn't mean to imply that good ones--
A Really, they were extraordinarily fine and competent group of people that Bayne-Jones had gotten together.

Q You mention that there were a number of such commission. If I am not mistaken, there was a measles-mumps commission,
A respiratory disease, and special influenza commission. All the different things that presented health problems to our armed forces.

Q Now, what was the mission of the neurotropic virus commission.

A Very simply stated, although I don't remember the exact words that they used. It was to first of all, be prepared to deal with any problem in the armed forces that might be caused by neurotropic viruses. This actually included predominantly the fear of outbreaks of mosquito-transmitted encephalitis viruses, like Western equine encephalitis in some of the training camps.

Q Was it--

A Okay. Potentially, ultimately, Japanese Encephalitis. St. Louis Encephalitis. I mean, these were the main diseases. So the idea was to have a group that would have the expertise to go to any place where there was a potential epidemic, to begin to carry out studies on vaccination, so that if there were a problem, we would be ready to go. Also, although one couldn't be exactly certain whether they'd be neurotropic viruses or not, any virus disease which did not fall within the specific commissions, like influenza or respiratory disease, and particularly if it was insect-transmitted, it was also within the mission of the neurotropic virus commission. That is why, in my own capacity we worked on our laboratory before I went into uniform, on Western equine encephalitis, on Japanese bee encephalitis--vaccines.
That was vaccines. We--then--completely taken out. I hadn't yet completed my course in officers' training when I was flown out to Egypt--

Q  Before you go to Egypt, how much of a threat really was there of encephalitis. I know there was an epidemic in Canada about 1942.

A  The problem that armed forces have to deal with are certain unknowns. In other words, you have to deal with the potential without having any hard data what might happen. Nobody could predict what might happen. If you would bring together from different parts of the country, young men with unknown immunity, let's say to Western equine encephalitis--encephalomyelitic virus, in an area which might be edemic--there were certain areas known to be in California, and around the Northwest, around Canada. Under conditions in which the mosquito control might not be optimum and how much of a problem--there was no way of knowing. So you had to be ready for it. Ready to diagnose it quickly, that was problem one. Ready to follow through with epidemiologic study. In other words, have a team ready to go! And don't go out in the country searching for somebody with expertise after it occurs. But foresee potential possibilities and be ready to deal with it.

Q  Alright, now you said two things. First, diagnosis, then epidemiological studies, and third, which you mentioned,

A  Be prepared to deal with it.

Q  To deal with vaccine.
A vaccine, or mosquito control etc. So those are the basic responsibilities. Now, when World War II came to an end, we came, we got an idea of how exaggerated the potential danger was, but don't forget the United States eventually expected to be in Asia. It was known that there was Japanese encephalitis. It was known, at least in experimental animals, that St. Louis encephalitis didn't protect against Japanese encephalitis and vice versa. And what would happen, I mean, there were documented epidemics involving thousands of people in Japan, people born and raised in Japan. What would happen, we bring in half a million Americans without any immunity whatsoever. Alright. Those are problems that had to be faced.

Q Now, one of the--

A They didn't turn out to be big ones, but they had to be faced.

Q Now, if you are going to work in making a diagnosis, certainly you would have to have neutralization tests after--

A There were various ways. At that time, what one had available, were neutralization tests and complement fixation tests.

Q Was there agreement?

A Also, on isolation of the virus, in which you would have to--isolation of the virus would be encephalities, was mostly in fatal cases. Suspected clinical cases you would have to take one specimen of blood early after onset and another one later, and then show that antibodies developed for a particular virus.
Complement fixation—was tested. These are two different antibodies, Ecologic antibodies and complement fixing antibodies are towards different components of the virus. Sometimes both may respond well. Otherwise, only one. With complement fixation you may have a little more crossing. Of course later, I introduced, I brought in the hemaglutinating antibodies into the business. But during World War II, we had just neutralization and complement fixation.

Q The reason I raised the question is that there are very interesting debates that break out after criteria for doing neutralization tests—Webster and yourself, for example, had fierce arguments about this.

A Refresh my memory.

Q Webster wanted to proceed by something called the Reed Muench formula. There is a question of what was positive, what was negative, what was equivocal in the neutralization—

A You are beginning to deal now with nitty-gritty things that are very important when we get down to the specific business. The Reed Muench was hard to get a 50% end point. It was a method of getting a 50% end point. It was necessary for establishing certain standards, that a method be used for calculating the end point. Because when you take your first serum specimen for neutralization test when a patient already has clinical manifestations—he already has some antibody. What you have to demonstrate is the increase in quantity of this antibody. In order to do that, you have to use a
quantitative technique. So, among--there are many things--
tiny things that you have to deal with, and the neurotropic
virus commission had to deal with that. I remember writing
criteria--we used to have discussions, guide-lines to be used
and so on. That's par for the course which has to do with
technical problems.

Q  But the technical problems basically--
A  They were all--they had to be discussed in full, there
had to be agreement, and very, very good--it was a good way
of proceeding. In other words, things were aired pretty well.
Submitted to criticism.

Q  Okay. One of the things that you work on is making
vaccines against St. Louis Encephalitis virus and Japanese bee
encephalitis virus. As you look back, what is the importance
of this experience for you?

A  The problem was a special one. At that time, I never
thought that one--although I think now--that one of the ways
to really control, in an edemic area, control either St. Louis
encephalitis virus or Japanese bee encephalitis virus, the
vaccination of a population with a killed virus vaccine was
a practical approach. Now, whether, when you are dealing with
armed forces, when you have to--when you are in the face of an
epidemic, and you know that it is starting now, and that it maybe
will run two, three weeks, three to four weeks, you want to
find out whether or not, you can develop and produce a vaccine
which killed it quickly in a couple of doses a few days after
each other, whether you can produce enough of an immune response to at least protect part of your population. This had to do with vaccines like St. Louis or Western equine—we had no way of knowing whether in some area where there was a training camp, that you couldn't get an outbreak of St. Louis encephalitis. After all, there was a terrible epidemic in St. Louis in 1933, not so long before '40 when we began to deal with it. It was fresh on our minds. We had these big epidemics.

Alright, so the problem there was not just to take material and inactivate it, because that's kitchen chemistry. That's not science, although you have to have certain criteria, of assay, all this is technological. It has to be developed. Whether or not the antigenicity is lost by exposing it to the inactivating agent too long temperatures, but this is mechanics. That has to be done. But after you've found the best way to preserve most of the antigen, then you have to ask yourself the question, what is the quickest procedure by which I can produce a demonstrable immunity. Because you are dealing here with two factors. One was you've made a diagnosis of a couple of cases. There is an indication of an epidemic, and you want to vaccinate the rest. How do you do it? You don't have time to give one dose and a month later another dose, and a month later, another that's for the birds. That's no good for this kind of situation. So I approached my problem by saying, what can be done in a week? What immunity can you get within a week? So that when I tested animals, and when I tested student volunteers in this, I gave one
dose on day zero, and another on day three, and then see what they had within a week. How many, and what kind of a dose would I need to produce immunity within one week. Now, that's for doing it in the face of an epidemic. Actually, we were faced with that on Okinawa, in 1945.

The other one was, let's say you are going to send in a force into Japan, in an area that you know to be endemic, during a time of the year when the rice paddies are flooded, and the mosquitoes which transmit it are wide-spread, and you want to give it to them in advance. You want to protect them for a short period. In other words, for armed forces, where you need to protect persons for short period of time, quite different criteria are considered than for the protection of a population living in the area a lifetime. So, the only thing that I learned during this experience was how variable the antigenicity of formalinized vaccines could be when you enactivated it in the cold, or at 37°c, and the probability of giving rapid, of inducing a rapid immune response without waiting too long for another dose, etc. But I say from the point of view from lasting experience,

Q You made this--
A It certainly made me never to want to want to work with a vaccine like that against polio.

Q The vaccine that you used is a mouse-strain vaccine.
A Yes.
Q  Most of the testing that took place with both the St. Louis encephalitis and the Japanese bee encephalitis come out with untoward incident, except in one case.

A  Now wait a minute, I don't know what you mean by most of the testing, because there was different stages. What testing are you referring to? the human beings?

Q  The testing in human beings.

A  But in the first place, there are different types of testing. There is testing in small numbers, the purpose of which is chiefly to get the immune response, and therefore you can't expect very much. Then there is the use of it in larger numbers under actual conditions, because there were never, I mean, finally, the one vaccine that was prepared for army use and it was prepared by a commercial company under contract, under specific regulations, of public health service, it was monitored by public health service, they followed my protocol was Japanese bee encephalitis vaccine. We had enough vaccine made by the time the invasion occurred on Okinawa for about a thousand persons. It was not for general use. It was for use only by the army. The assay of that was done entirely in animals, and it was not a question of doing safety tests as you do with others. You give it to five thousand persons, and so on. That is not the way--

Q  I wasn't talking about safety testing. I was getting at something else. The tests that were done with the Japanese bee were done in five hundred inmates or so of Princeton State--
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A No, no, it was never Japanese. Encephalitis vaccine was never tested in persons. (prisoners).