This is an interview with Dr. Henry Masur at the Clinical Center of the National Institutes of Health (NIH) on November 22, 1989. The interviewers are Dr. Victoria Harden, Director, NIH History Office, and Dennis Rodrigues, senior program analyst.

Rodrigues: Could you begin by telling us about your training and experience and how that led you to your involvement with AIDS patients?

Masur: Actually, I became involved with AIDS in a very indirect way. I had a fellowship at Cornell [University]. I trained under a protozoologist named [Dr. Thomas] Tom Jones, who is an expert on *Toxoplasma.* I was beginning to work on *Leishmania donovani*. Tom's major clinical interest was tropical medicine, and he thought that for somebody training in his laboratory, it would be useful to use some of the techniques he had developed with *Toxoplasma [gondii]* and *Leishmania*, and to apply them to a parasite at which no one else was looking. He thought that *Pneumocystis carinii* would be interesting because I was interested both in immunosuppressed patients and in tropical medicine. *Pneumocystis* was something that affected both immunosuppressed patients and patients in some developing countries, such as in orphanages following World War II.

At that point, there were very few people in the United States who were interested in *Pneumocystis*. When I started my fellowship in 1975, the CDC [Centers for Disease Control and Prevention] had just published a review of the occurrence of *Pneumocystis* in the United States from 1969 to 1971. There were only 70 cases a year documented in the United States. It was a very uncommon pathogen. Only three or four groups across the country were looking at it. There was not very much in the literature. So I started doing some *in vitro* work on *Pneumocystis* because Tom thought that it was an organism that would provide a means for me to establish my own identity but still use the approaches he had developed.

Over the next few years, I split my time between three different areas. I did some work with immunosuppressed patients because I was interested in the infectious complications of immunologically abnormal people. That also provided an opportunity to study some *Pneumocystis* patients, so I did all the consults for a big kidney transplant program at Cornell. I also was working in Brazil on *Leishmania*. Until about 1978, while I was going back and forth to Brazil, I did some laboratory work on *Pneumocystis*. From 1978 to 1980, I spent a fair amount of time in the field in Thailand and in Brazil, and some time in the laboratory in New York.

In 1979, after I had been in Brazil, I came back and was on the Cornell faculty when I attended the first patient who came into the emergency room with what turned out to be, after a long work-up, *Pneumocystis* pneumonia. It was clear to me, because I knew the literature very well, that it was very unusual for someone

previously healthy to walk in with *Pneumocystis*. At that point, we had studied the immunology of the organism, but there was not very much clinical literature. The only patients that one saw were either patients with previously recognized immunosuppression or, occasionally, in the developing world, one saw an epidemic of *Pneumocystis* in malnourished infants. When I came back from Brazil, however, suddenly we had this patient come in, and we worked him up very intensively. It was interesting. We did not know what to make of the fact that he had *Pneumocystis* pneumonia. By some simple immunologic parameters, he looked like he was very abnormal. We then looked to see who was interested in working him up with us. There was an immunology group under [Dr. Gregory] Greg Siskind at New York Hospital, which performed some preliminary work on the first patient. Then the person who was doing T- and B-cell analysis was [Dr.] Susanna Cunningham-Rundles, a well-known immunologist at Memorial-Sloan Kettering, and also Dr. Mary Anne Michelis. So, Susanna worked this patient up and, at that point, we thought we had a case report of something that would be interesting but not very important. We presented to Dr. [Robert] Good, who then was one of the world's most famous immunologists and who had recently come as the director of the Memorial-Sloan Kettering Medical Center. Although I guess one should not tell tales out of school, he ran a very imperious conference—a very regimented conference. We presented this case, and he said: "This is clearly a case of malnutrition. You should get hair clippings for zinc." He had never seen the patient. We did not think the patient looked that malnourished, but we were amenable to his advice. So, we said, "Okay." We sent off the hair clippings to test for zinc, and they came back normal. So, he said, "Send them to another lab. It must be malnutrition." So we did it again, and he said, "It's malnutrition." And that was the end of it. Clearly, we were not going to get very much help from him.

It is interesting how life is serendipitous. One of the people whom I have mentioned was working on putting this data together. Her work was delayed by a pregnancy, so, as a result, she was slow completing her portion of the case report, which would have been the first documented AIDS case, but would also have been buried in some obscure journal. Every month I would call her up asking about where all the immunologic data on this one patient was. But she dragged her heels so much that by the time she got the data together, we had seen two more patients. By then, we were planning to write about three patients. We thought that it might be a more interesting report. Actually, at that point, I called the CDC (which they subsequently denied), and I talked to the people who do *Pneumocystis* serology and asked them if they would do *Pneumocystis* serology [on these three cases] because this was very interesting. These three cases were unprecedented. The people in the serology laboratory were not interested. At that point, I was sufficiently naive and I did not realize that the CDC was a big place and that talking to the serology laboratory was not talking to the epidemiology

people. So, they did not want to do anything more than just run the serologies. I said, "It would be very interesting to give us some follow-up. Are you interested in doing anything more"? They said, "No." That was the end of it. Again, this person who was putting together some of the immunology data on the three patients was so slow that by that time she got the immunology prepared on the three patients together, I was ready to strangle her. We presented one of the cases at intercity infectious disease rounds in New York, and several people came up to us and said they had similar cases. So, we went around and we collected a dozen cases. These cases were being seen at a variety of different hospitals.

Harden: What year was this?

Masur: This was 1980. Again, the work was slowed down by the fact that I had gone back to Brazil at some time in the middle of this. When I came back from Brazil, we had 12 cases, and we got all the data together. But unbeknownst to us, one of our co-authors at Memorial was also working with [Dr. Frederick] Fred Siegal from Mount Sinai Medical Center on chronic perianal herpes, which, I guess, he did not realize was a similar issue. He was simultaneously working up some men with chronic herpes simplex plus *Pneumocystis*. We did not know about it although he was a co-author on our paper. Also, we did not know about [Dr. Michael] Mike Gottlieb's cases in Los Angeles. So, at this point, it was clear to us that we had seen about a dozen men with *Pneumocystis*, but it not clear whether they were immunosuppressed because they had been infected with a virulent strain of *Pneumocystis*, which had somehow altered their immunity, or whether they had somehow become immunodeficient due to something else. It was not at all clear that this was a major public health problem. It seemed to be an unusual issue, and the major focus was whether or not there was some kind of an environmental exposure. We did not know that they were all gay or intravenous drug users.

The first evidence we actually had that our initial case was gay came when I was in a room about a third the size of this one, and he suddenly leaned over and said, "Give me a kiss." I just looked at him. In retrospect, it was clear that he was gay and he was demented: he had a red bandanna in his back pocket and wore an earring, but being naive like most physicians, I had not put all that together. Not as many people knew about the gay culture then as they do now. At least I had not read that much. We really did not know anything about the sexual orientation of the other patients. A couple of them were drug users, but a lot of the people who go through the infectious diseases rounds come from hospitals that serve that kind of clientele and have them in their ward populations. It was not clear to us until later that these people shared drug abuse and homosexuality, or that there was a connection between the two.

At this point, we knew that there were 12 cases. We submitted that information to *The New England Journal of Medicine* on the assumption that this was probably something involving a very small number of people and that it would turn out to be scientifically very interesting. But, at that time, there was no suggestion that it might reflect a public health problem. I went off to Brazil again. When I came back, we got a call from the CDC indicating that *The New England Journal of Medicine* was, in fact, considering two other similar manuscripts and that the CDC wanted to put something in *Morbidity and Mortality Weekly Report*. It is interesting how things have evolved since then. We were concerned that putting the information in *Morbidity and Mortality Weekly Report* would preclude publication in *The New England Journal of Medicine*. We talked to *The New England Journal*. They were very adamant that "prepublication" [elsewhere] would prohibit publication in *The New England Journal*. Again, history seems to have changed over the subsequent time. We decided that we should not put our material in *Morbidity and Mortality*, which probably was not the right decision in retrospect, but neither was *The New England Journal*'s decision correct, either. In any event, Gottlieb reported his cases in *Morbidity and Mortality Weekly Report*. I do not actually know who had seen the first case, or who had submitted the first manuscript to *The New England Journal*. However, three articles were published in *The New England Journal of Medicine*, which thus became, simultaneously, the first peer reviewed reports of AIDS.

By then it was clear that there were three foci of this infection, but the extent of the foci was not clear. There really was no race for space or resources. It was more of an interesting scientific phenomenon. That was just about the time when [Dr. Joseph] Joe Parrillo, who had been a classmate of mine at Cornell, came here to the NIH to be the Chief of Critical Care Medicine. He was looking for senior investigators. He knew that I was interested in infectious disease and in seriously ill patients. He thought that it would be an interesting recruitment tool to be able to say, "If you want to study this strange phenomenon, there are a lot of opportunities. Why don't you come to NIH, and while you are working in the ICU [intensive care unit], you can have laboratory space. We could work something out." I knew [Dr. Anthony] Tony Fauci from Cornell. Actually, Tony had been the chief resident when Joe and I were fourth-year students. So, we had both known Tony, and Tony had been instrumental in recruiting Joe to come down to NIH. When I came to look at a job, Tony, at that point, was very interested in getting involved in AIDS, but he had not really initiated anything. He was very excited about having somebody who would help bring in some patients so that he could study them. He was very interested in devoting a lot of his laboratory resources to it, and he had [Dr. Clifford] Cliff Lane in his laboratory as a Fellow, who he thought would be a good person to get involved in studying these patients. At that point, it seemed like another unusual disease like Wegener's [granulomatosis], or Sjogren's syndrome, which was scientifically interesting.

The NIH could be a good place to study this unusual disease because we could bring patients in from all over the country and study them. There seemed to be no need for a major initiative. This appeared to be another disease that, with the NIH's good virology, immunology, laboratory space, and investigators, could be studied at NIH. So I came with that in mind. There was a lot of interest in this new phenomenon. Before I arrived at NIH, however, I disappeared to the tropics again for another few months. By the time I came, in early 1982, these articles had come out, and there was a lot of interest. There were a lot of people who were interested in collaborating on AIDS.

When we first started studying AIDS, we found—just by word of mouth—that there were a lot of people who wanted to look at various aspects of it. It was not an issue of resources because I was by myself in Critical Care. There were other people who, as individuals, had an interest. Tony and Cliff did the immunology. We had a meeting each week that grew larger over time. [Dr. Edward] Ed Gelmann, who is now at Georgetown, was a Fellow in the Cancer Institute [National Cancer Institute, NCI]. He was interested in Kaposi's sarcoma and searching for a viral etiology. [Dr. Phillip] Phil Smith, who was then in the National Institute of Dental Research, was interested in some other immunologic aspects. [Dr. Gerald] Jerry Quinnan, from the Food and Drug Administration [FDA] here on campus, who ran a herpes virus laboratory, was very interested in looking at CMV [cytomegalovirus], HSV [Herpes simplex virus], and VZV [Varicella-zoster virus]. They would come to the meetings each week, and [Dr. Stephen] Steve Straus, who is with NIAID [National Institute of Allergy and Infectious Diseases], was interested in the herpes virus aspects of this. So very quickly we got a group of people, all of whom were interested in different aspects of the problem.

To me, that was what made NIH an exciting, attractive place to work. You could put together a group of people who did not need an organized program because they all had a common interest. They could all pick off a piece of the problem to work on. Somebody could publish on CMV; somebody else could publish on immunology; somebody else could focus on the formative problems. This collaboration worked out very well. Some of the other institutes also quickly got involved. [Dr. Alan] Al Palestine and [Dr. Robert] Bob Nussenblatt very quickly recognized that eye involvement was a common problem in AIDS, so they got involved very early. We had an expert on every organ system and every major category of laboratory abnormality. [Dr.William] Bill Travis was very interested in the pathology of the disease. We were able to use one critical care therapist ([Dr.] Jack Ames, now a radiation oncologist) part time to deliver specimens to all these laboratories. We had a very small number of patients who were from all over the country because treating physicians did not know what to do with them, but there were not very many at that time. We had the patients come in, and while

taking care of them, we would try to study them. We had one therapist who would draw blood in the morning and then go around to all these laboratories and deliver the specimens. We would meet once a week, evaluate what was going on, and decide what to do next. That was just at the time in 1982 and 1983 when the CDC data began to show that this was more of a national problem. But it was not until after this that AIDS was recognized as having a retroviral etiology. That was really the beginning of the crunch for resources. Up until then, AIDS was more of a curiosity.

So, AIDS began the way a lot of NIH events begin. There was a scientific issue, for which the atmosphere on campus was very attractive as an environment where one could study this kind of problem. We could get a lot of people who were free to choose their own interests. They all had an opportunity to take part in this because there was something in it for everybody academically, scientifically, and intellectually. It worked out very well. Now there are 85 committees trying to dole out resources, and it has become a much different kind of issue. This is an example of what the intramural NIH can do very well as a community of basic scientists and clinicians. It really took a combination of basic science and clinical science to bring the patients in, to recognize the important patient-care-related problems, but also to do, very quickly, a lot of the groundwork in immunology and virology. It required the range of expertise that we have at NIH from basic immunology, basic retroviral studies, basic herpes virus studies, to very good autopsy studies. From the study group that we had, we got autopsies on patients to figure out what the range of the pathology was. The ophthalmologies were interesting. They enucleated all the patients who died, so they very quickly recognized what the retinitis was all about. Because there were people here who were free to choose what they wanted to do, who had the resources to devote to it and the esoteric backgrounds to take advantage of it, it all worked out. There were people who had skills that might never have been publicly recognized because they were not very important until something like this came around. Suddenly there were retrovirologists who had been working on veterinary problems who found a human clinical application. If we had not had that group of people doing basic research at NIH, and if we had not had people doing electron microscopy on retinas who could recognize CMV, we would not have been able to make the progress that was made on campus. Progress was made as part of this integrated team. Other progress was made independent of the group, however. Dr. [Robert] Gallo's group, for example, was an independent entity that did not wish to maintain communication with our group. They got their own specimens and made progress independently of our group. That was the way it began.

Rodrigues: You mentioned that before AIDS there were only a small number of cases of *Pneumocystis*, 60 or 70, per year. Were almost all of those cases due either to malnutrition or to the effects of immunosuppressant drugs or cancer therapeutics?

Masur: Yes. There was an article in the *Annals of Internal Medicine* in 1972, which reviewed 180 cases in three years. In this country, they all occurred in patients with congenital immunodeficiencies or ones who had a previously diagnosed immunodeficiency. When they developed *Pneumocystis*, they were all known to have cancer, for example, or have had a kidney transplant. They did not walk in with *Pneumocystis*. There were a few unusual cases in the literature suggesting that it could appear out of the blue, but they were all questionable. Actually, looking back, it is conceivable that some of those were the first cases of AIDS, although they were not worked up for that. In this country malnutrition was not so much an issue, but in Africa, in Iran, and in post-World War II Eastern Europe, malnutrition was certainly an important issue.

Rodrigues: At that time, how effective were the available therapeutics for *Pneumocystis* infection?

Masur: The first successful treatment of *Pneumocystis* was about 1955 or 1956. Actually, NIH was involved very early on. The first successful treatment was by an Eastern European doctor who developed pentamidine. From the mid-1950s until the early 1970s, pentamidine and sulfadiazine-pyrimethamine were the only recognized drugs that were available. There was a group here that was interested in *Pneumocystis*. It included Dr. [Vincent] DeVita, though, in the middle 1970s. They did some work with pentamidine. There was a group in the Cancer Institute [NCI], led by [Dr.] John Whisnant, that worked with sulfadiazine. They published a monograph in 1976 that summarized the world's literature. At that point, intramuscular pentamidine was very toxic, and sulfadiazine-pyrimethamine was not always very easy to give. But, until the mid-1970s, those were the only two choices. Then, [Dr.] Walter Hughes developed trimethoprim-sulfamethoxazole, which is a good, all-purpose treatment and is not terribly toxic. So, by the time the AIDS epidemic came along in the late 1970s, there were at least two alternatives: intramuscular pentamidine, which was very toxic, and either oral or intravenous sulfa-trimethoprim, which was very effective and not very toxic in cancer patients. However, it turned out to be relatively toxic in AIDS patients. But there were only those two choices in the late 1970s.

Rodrigues: You said, as have many others, that there was the feeling that these cases you were seeing represented an anomaly—something related perhaps to some environmental cause or to amyl nitrites. In your mind, when did you begin to move away from that thinking and to consider that there might be an underlying viral origin?

Masur: By about 1983 or 1984, the presumption was that it was something transmissible.

Just as I was leaving New York at the end of 1981, we started seeing women with the disease, although they were mostly drug users. That was published by my group in about mid-1982, but by the end of 1981, we were beginning to see it in women. Then, in 1982 and 1983, it was clear that there were two main groups infected: homosexuals and IV drug users. The assumption was that it was something transmissible through blood. Although there were people speculating about a virus, there were a lot of different theories as to what kind of thing might be transmissible. For example, it might be lymphocytes that created some kind of graft-versus-host response. There were all sorts of crazy ideas, but I do not know anybody who was focusing on a virus and excluding other things. There were a number of people who felt strongly that it could be a virus, but until Dr. Gallo or the French, depending upon which you want to give the initial credit to, showed the strong correlation, viral etiology was just one of a number of different theories. Everything was so unprecedented that none of them seemed very likely.

Rodrigues: You mentioned the cases in women. I looked at that paper you just mentioned. In one case, an individual, patient number three, manifested symptoms 34 months before diagnosis. That suggests that AIDS was around practically in the mid-70s.

Masur: If you assume that the average incubation period for AIDS is 8 to 10 years, i.e., the time between acquiring the virus and developing clinical disease, the first patient that we saw in 1979 was probably infected in the early 1970s. There are some people in whom the disease is manifested as early as two years after infection, so maybe the first patient was infected in 1977 or so. At some point during the 1970s, the virus was widely introduced into this country, but it was not until the late 70s or early 80s that we began to see the clinical manifestations. One of the other interesting things about that first patient that had thrown us astray was that he was a hospital security guard who worked in a busy emergency room. So one of the first things we wondered about was whether he had been exposed to something in the emergency room.

If you look back in the medical literature, you see that at first they talked to practitioners. A lot of people had big AIDS practices, and for several years they had been seeing more lymphadenopathy. If you look back on some unusual cases in the literature, either from abroad or from the United States, you can find some cases that go back as early as 1960 that might have been AIDS. There are even some who say that they have serum that has been tested.

Harden: While we are on this subject, this incubation period is one of the things that [Dr.] Peter Duesberg has attacked in his arguments that HIV is not the cause of AIDS. He also notes the difficulty in detecting antibodies to HIV. As an infectious diseases expert, what is your view of his ideas?

Masur: I think one has to keep an open mind to all possibilities. I think the most compelling evidence is the transfusion cases where you can show that somebody got the virus from a transfusion and then developed the syndrome. You can say that maybe there is something else that is being transfused that we have not recognized. It is becoming clear that there is at least a logical explanation for long incubation periods. You can see a very slow, immunologic decline. It is just a chronic disease that takes a while to wipe out your immune response. The fact that antibodies are not produced is a function of the type of virus that it is. I think that there are logical explanations for what Duesberg considers to be discrepancies in the theory. Whether those logical explanations are accurate is another issue. Everything that we know about retroviruses right now at least makes a logical picture about their being the cause of AIDS. Duesberg has become well known because of his skepticism.

Rodrigues: In going through some of the past records I found a protocol for which you had provided a written description. I believe that it was the first formal protocol at NIH for AIDS patients? Was that so?

Masur: Yes. In 1982. It was a sort of "catch-all" for everything. I am impressed that you could find this in somebody's files.

Rodrigues: One of the things that you mentioned earlier was the considerable coordination taking place among the Dental Institute [NIDA], the Allergy Institute [NIAID], the Cancer Institute [NCI], the CDC, and the FDA. There were people from these agencies working together. Part of the criticism that the NIH has come under has to do with the expectation that first an agency should build an administrative mechanism, which then provides momentum to drive science and provides resources. What you are telling us, however, is that there is an unspoken, underlying logic behind research, and that this logic created this embryonic program simply by the steps that presented themselves in the conduct of research. Later, more formal programs grew out of these efforts rather than the opposite taking place.

Masur: Yes. My perception of scientists is that they are like businessmen in that, although there are some who are purists and will do what interests them regardless of what else is happening in the world, most of them are very practical. If they see a new disease that will help their careers in terms of publications, of getting a more prestigious job, and if they see opportunities, they will be attracted into that field. They are not going to be attracted by a dead issue no matter what the leadership suggests. If someone says there is going to be a war on Sjogren's syndrome, they are all going to look and say, "That is nice, but I don't think I am going to work on it, because I don't care about it." Fortunately, somebody cares about Sjogren's syndrome. We are not going to have a war on it, however.

Sjogren's was a very interesting scientific opportunity, but I think people got involved in AIDS not only because it was interesting scientifically but because it looked like it was important clinically.

One of the things that to me reflects a real tragedy, in terms of the direction that science and NIH are going, is that there is not as much emphasis any more on clinical investigation on this campus. It means that the NIH is shifting more and more to very basic research. Nationally, research is being split into two camps. More and more of the basic science branches are going to Ph.D.s, and the physicians are doing the clinical studies. This pulls people out of opportunities to respond to the kind of situation that AIDS presented. Here there were clinically trained people who were involved in basic science, and it was these people who initially saw that this was something very interesting and that there was a social problem out there. They knew that there were patients coming in. When a patient comes in and has a problem, it stimulates a lot of people to go back to the laboratory and say, "We should look at that." It is a lot different when you are a Ph.D. There is not that same stimulation. I realize that not everybody sees things in the same way. I think this is a good example of how training physician-investigators pays a dividend, however, because physicians, microbiologists, and other people who had both clinical and research skills were able to take on a problem that piqued their interests scientifically. It looked like it was going to be a problem for them to take care of patients clinically, so they went to the laboratory and came up with some of the initial answers. Admittedly, it took somebody like Dr. Gallo, who does only bench research, to come up with the important answer about retroviruses. But I do not think that he ever would have recognized that there was a problem unless there had been a group of people who brought things along to a certain stage where he could jump in. That is not to take any credit away from him, but I think that there is not a lot of recognition that physician-investigators are the bridge between two worlds: people who have to deal with public health problems and the people who come up with the answers.

Harden: As a physician-investigator working with others and attempting to cope with AIDS as a new disease, could you describe the strategy the group used? Were people attacking the problem, bit by bit—dealing with discrete opportunistic infections such as CMV or *Pneumocystis*—or did you rapidly shift to efforts to reverse the underlying immune deficiency? Or did you try all these things at once?

Masur: I think our efforts were a function of our interpretation of what the problem was and what resources we had available. Again, one of the real virtues of the NIH community is that there is an expert on almost everything here. When we saw that the herpes virus was a problem, we went to Steve Straus and said, "Why don't you come and do the cultures?" And he said, "Fine." If we had needed to go to

Baltimore or Philadelphia, that would not have happened, or would not have happened very easily. So the diversity here was an important issue. It thus depended on the personality and the imagination of the people who were here. For instance, nobody had any idea how to go about figuring out what a good antiviral drug was. That was when [Dr. Samuel] Sam Broder made his important contribution. I would doubt that he knew anything about retroviruses, but with intelligence and hard work, he figured out where to start, and he got some people working on it. The therapeutic attacks went along the lines of the people who were involved and what their expertise was. There was a lot of interest, for instance, in herpes virus, but no herpes virus drug, so we did not really do anything about that. There was a lot of expertise in immunology and there are lots of things you can do about immunologic deficiencies, even though most of them had never worked. But there were many things to try and a lot of ideas. Some were crazy; some not so crazy. It was really Tony who did one of the first remarkable things. Fortuitously, we had a patient who had an identical twin brother. We said, "This sounds like something for which we ought to be able to do a bone marrow transplant and get a cure." That was one of the exciting first initiatives. The problem was that it did not work. There was somebody else who was interested in the interferons. So using alpha interferon was one of the first big initiatives, just because there somebody here who measured alpha interferon levels. We were able to figure out the dynamics of gamma interferon and alpha interferon. The initiatives were the function of the expertise and methods that were available for attacking AIDS. Some things you could attack; some things you could not. Again, we did not know it was a retrovirus, and, besides, there were not any antiretroviral drugs. Drug therapy was not a possibility until Sam Broder helped develop AZT [3'-Azido-2', 3'-dideoxythymidine], and those trials started in 1985 or 1986. The researchers started unsuccessfully with some drugs that did not work, and then eventually came to AZT.

Harden: I would like to ask one more question before we end the interview. From the patient's perspective, what did he see during treatment—a whole host of doctors crowding around him, or one primary care physician with consultants?

Masur: Most of the NIH people did not see the patients. Cliff Lane, Ed Gelmann and one of his Fellows, [Dr. Ronald] Ron Steis, and I saw all the patients and took care of them. If they needed an ophthalmology consult, Alan Palestine was particularly interested, so he would come and see them. If they needed a gastrointestinal work-up, Phil Smith would come and see them. So, we had our own AIDS service, which would act like any other service around here. The patients would see a few people as their primary people and then we would call in a consultant as needed. We quickly had an informal AIDS team rather than the traditional clinical services. Some of the patients were on Cancer Institute [NCI] protocols, some were on Allergy and Infectious Diseases [NIAID] protocols, and some were

in critical care, so they were spread around, depending on where we could find a bed.

Harden: Thank you very much, Dr. Masur.

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