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The San Francisco AIDS Oral History Series

THE AIDS EPIDEMIC IN SAN FRANCISCO: THE RESPONSE OF COMMUNITY PHYSICIANS,  
1981-1984

Volume II

Paul Monahan O'Malley

AIDS AND THE HEPATITIS B VACCINE  
TRIAL IN SAN FRANCISCO

Stephen Follansbee, M.D.

INFECTIOUS DISEASE PRACTITIONER IN  
THE EARLY AIDS EPIDEMIC

With an Introduction by  
Donald I. Abrams, M.D.

Interviews Conducted by  
Sally Smith Hughes, Ph.D.  
in 1996

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Paul Monahan O'Malley, (b. 1946). Public health agent and AIDS researcher; background and "coming out"; work at the San Francisco City Health Clinic; prevalence of Sexually Transmitted Diseases [STDs] in gay men; hepatitis B study, 1977; confidentiality issues, finding participants, first AIDS cases, use of data for AIDS research; educating gay men about AIDS; discusses Selma Dritz, Randy Shilts, Andrew Moss, other early personalities in AIDS medicine; Stephen E. Follansbee, M.D., (b. 1948). Infectious disease physician; background and early career; early discovery and treatment of AIDS and AIDS-related opportunistic infections; seriousness of the AIDS epidemic, linking it to the gay community; Connie Wofsy, Jay Levy and other early AIDS physicians; Physicians for Human Rights groups and County Community Consortium; infection control measures and the AIDS antibody test; comments on San Francisco's medical response to AIDS, later treatments for AIDS, and the future of the epidemic.

Introduction by Donald I. Abrams, M.D., Chair, Community Consortium.

Interviewed 1996 by Sally Smith Hughes, Ph.D., for the San Francisco AIDS Oral History Series. Regional Oral History Office, The Bancroft Library, University of California, Berkeley.



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## SERIES INTRODUCTION--Donald I. Abrams, M.D.

First, transport yourself back in time to the late 1970s, early 1980s, when solo medical practitioners were the norm in the San Francisco Bay Area. Community physicians, practicing alone in their private practices, were the first to encounter patients with the unusual purple lesion or the rapidly progressive pneumonia. Many of these providers had much in common with their patients--their age, their socioeconomic status, and their sexuality. Even before the establishment of the AIDS clinics at the university facilities, the community physicians were on the front lines as the epidemic erupted. They were truly community providers not only in the contrast to the academicians, but also often as members of the community that was about to become decimated by the ravages of the terrifying new disease. Has such a situation ever before been encountered in the history of medicine?

Recall as well the history of the "Gay Liberation" movement in the United States. In the late seventies, homosexual men and women were just becoming comfortable with emergence from their closets, enjoying an openness and sense of empowerment that accompanied the newfound freedom and acceptance. Nascent organizations of lesbian, gay, and bisexual physicians were being established, initially with the founding of the Bay Area Physicians for Human Rights [BAPHR] in 1977, followed by the national American Association of Physicians for Human Rights [AAPHR (now the Gay and Lesbian Medical Association--GLMA)] in 1981. In fact, it was at a BAPHR meeting of gay physicians from around the country being held in San Francisco in June 1981 that it became evident that these unusual cases of Kaposi's sarcoma and Pneumocystis carinii pneumonia were more than freak isolated occurrences. These organizations served as early foci for information dissemination and educational efforts to alert colleagues and government health officials about the new disease. BAPHR and AAPHR meetings became informal support groups in a way, providing community physicians with a safe haven to share the sense of fear, frustration, and loss that accompanied caring for their earliest AIDS patients, even before the disease was named or the cause was discovered.

Despite an attempt to centralize care of AIDS patients at a center of excellence at San Francisco General Hospital [SFGH], community physicians maintained a desire to care for their patients in their own practices. After all, it was a brand new disease. It is not as if there were a fountain of information on how to treat it that only flowed at SFGH. Although most of the earliest clinical trials evaluating immune modulators and later antiretrovirals were occurring at the General, providers chose to maintain their primary caregiver role. They were undaunted by the novelty of the disease. They were unhampered by the lack of specialty training since there was no such thing as an AIDS

fellowship and we were all pioneers, out on the edge of medical history. Plus these men and women were bound to their patients in a unique way. Many of the community doctors had established gay medical practices, focussing their attention on the health needs of gay men. Prior to AIDS, in a young, sexually active population, sexually transmitted disease was the worst of the worries. They expected to establish their general practices and follow their patients through their maturity until old age and death. None of these young practitioners could anticipate the enormous premature loss that they would experience over the ensuing decade, presiding helplessly over the wholesale eradication of their community. Loss of a whole generation of young, intelligent, capable, productive men--like a war without guns. Has such a situation ever before been encountered in the history of medicine?

Read now the stories of some of the generals on the front line in this war. Although not himself a member of the gay community, Jim Groundwater was a favorite dermatologist in private practice for BAPHR physicians to consult. He likely saw the city's first case of Kaposi's sarcoma. Bob Bolan, Jim Campbell, Bill Owen, and Ric Andrews were providers on the front lines, tending to both the medical and psychiatric needs of the community under siege. Stephen Follansbee, completing his infectious disease fellowship just as the initial cases of Pneumocystis carinii pneumonia were diagnosed, became one of the first of the new breed of AIDSologists, his entire early career devoted essentially to the treatment and investigation of the new disease. Another investigator involved in attempting to crack the code from the perspective of the epidemiologist was Paul O'Malley, searching for clues in stored serum specimens and serial follow-up of a cohort of gay men who had been enrolled in a local hepatitis B vaccine trial in the late 1970s. All of these individuals made significant, too often unsung, contributions in the very early days of the epidemic and have for the most part continued on the same course to the present day.

In 1985, Mayor Dianne Feinstein asked Paul Volberding, the director of the AIDS program at San Francisco General Hospital, to establish a line of communication with the community providers caring for patients with AIDS in the Bay Area. The first meeting of the dozen or so providers was held in March at the San Francisco Medical Society. Seeing that many of those in attendance were from the gay community, Paul came to me and suggested that perhaps I should continue the dialogue with these physicians, many of whom he knew to be my friends from BAPHR. Links to my BAPHR colleagues had previously proven very valuable during my oncology fellowship when I established in 1981 a cohort of men with persistent generalized lymphadenopathy to follow prospectively in a natural history cohort. Many of the subjects referred for evaluation were sent by the doctors whose stories follow.

It was my pleasure to preside at the next meeting of the community physicians' group, which was initially formed for a number of reasons.



Information exchange was essential in these early days of emerging therapies. As well, we at the SFGH facility saw this meeting as a way to inform the community providers about ongoing research protocols to which they could refer their patients. As the group was a coming together of community physicians and those from the county hospital, County Community Consortium seemed an appropriate moniker. (In time the acronym CCC could never be correctly decoded by those who tried to use the organization's full name, so it was shortened to Community Consortium.)

Within the first year of meeting, it became clear that County Community Consortium providers were interested in taking a more active role in learning how best to care for their patients with the new disease. If memory serves me right, I believe it was Jim Campbell who raised his hand at a meeting and said, "You know, instead of sending all of our patients to SFGH to participate in clinical trials, there are questions we can answer in our own offices." That observation led to the development of a consensus protocol on how to prevent a second episode of Pneumocystis carinii pneumonia [PCP] in patients who had already experienced a first episode. Each provider had their own favorite regimen. Some offered no prophylaxis. Rather than depend on anecdote, we worked to develop a randomized clinical trial that was launched in July 1986 as perhaps the first community-based clinical trial in HIV disease. Soon after its inauguration, the trial was thwarted by the release of the first antiretroviral agent--AZT--because the first patients to receive the product were cautioned not to take any other non-essential medications by mouth. Since patients with a prior episode of PCP now had access to a potentially life-extending antiviral agent, interest in oral prophylaxis against a treatable pneumonia waned.

Undaunted, Consortium physician/investigators next designed a study to investigate PCP prophylaxis using the inhaled pentamidine therapy which had been developed by a UCSF/SFGH pulmonologist. Working together on the inhaled pentamidine protocol, town and gown investigators collaborated in a manner that would become a model for future productivity and success in conducting clinical trials in the sites where patients received their primary care. Ultimately the Consortium's aerosolized pentamidine trial would lead to FDA approval of the modality as the first prophylaxis for an HIV-related opportunistic infection as well as a lead article in the New England Journal of Medicine. It was clear that significant research could be done outside of the hallowed hallways of academic teaching hospitals. This Consortium achievement became a model for community-based clinical trials programs later established by both the American Foundation for AIDS Research and the National Institute of Allergy and Infectious Diseases.

Much of the success of the Community Consortium and even the larger San Francisco Model of HIV care can be traced to the efforts of

the physicians whose stories follow. No such collaborative coming together of the community was seen in other areas hard hit by the epidemic. New York and Los Angeles did not pull together the way the community did in the Bay Area. It can be attributed as well to the collaborative congeniality fostered by BAPHR, allowing its member physicians to strike out united against the common enemy--the disease--and not against each other.

I myself owe much of my professional as well as personal growth to my colleagues you are about to meet. Serving as brave, openly gay role models for a young junior faculty academic, initially fearful of coming-out to avoid derailing my career, the examples of these noble, proud and successful professionals inspired my ensuing openness. I write this today with pride as the current president of the Gay and Lesbian Medical Association. Through two decades of battle, these brave warriors on the front line of the fight have unique stories to tell of a struggle to save their community from a plague that often brought as much political as medical despair. Although the battle is neither won nor over, the contributions of the community physicians have done much to enrich the lives of their patients, the medical profession and society-at-large. Has such a situation ever before been encountered in the history of medicine?

Donald I. Abrams, M.D.

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Association, 1999-2000

January 2000  
San Francisco, California

SERIES HISTORY--Sally Smith Hughes, Ph.D.

### Project Origin and Organization

This series with community physicians is the third phase of an oral history project documenting the medical response of the medical and nursing professions in the early years of the AIDS epidemic in San Francisco. Please see the earlier volumes for descriptions of the particulars of these two previous interview phases.

Phase one and two with university physicians and nurses has effectively, albeit selectively, documented the role of academics in the epidemic, the "gown" component of the traditional town and gown division of medicine worldwide. What was obviously missing were accounts by representatives of the "town," that is, physicians with private practices in a variety of medical specialties relating to AIDS. In 1995, UCSF Library, represented by Karen Butter, now Acting Director, came to the rescue with a grant to the Regional Oral History Office to conduct interviews with community physicians whose practices included substantial numbers of AIDS patients early in the San Francisco epidemic. The grant was sufficient to cover two- to six-hour interviews with seven individuals--six physicians and one professional in the San Francisco Health Department--selected because of their substantial participation in the early medical response to the epidemic. In 1996, interviews were recorded in the San Francisco offices of the participants. The only exception were the interviews with Dr. Robert Bolan, which took place in Glendale in southern California, his new home after a recent career move.

### Primary and Secondary Sources

The interviews were largely based on the reading I had done to prepare for the first two phases of the project, and even more substantially by the information I had acquired in the course of these interviews. The most significant new source for phase three was documentation concerning Bay Area Physicians for Human Rights [BAPHR], a gay physicians' organization founded in San Francisco in the late 1970s. An extensive series of "The BAPHRON," BAPHR's informative monthly newsletter, and documents in BAPHR's office in the Castro District of San Francisco were rich sources of information on the response of gay physicians and the gay community to the epidemic.

### Selected Themes

BAPHR has a large voice in the present series. Five of the seven interviewees were at one time or another members of BAPHR and spoke at length of the organization's contributions. Only James Groundwater, who

is not gay, and Paul O'Malley, who is not a physician, were never members. Furthermore, BAPHR was one of the focal points of the early medical response to the new disease after it was recognized in San Francisco. Its members came to the crisis with the very intersection of experiences that the epidemic seemed to demand: medical skill in diagnosing and treating diseases prevalent in gay men, and sympathy for preserving the personal and sexual freedoms that the gay community had recently won. The vast difference for physicians confronted with previously healthy young men who were suffering and dying from AIDS was that neither cause nor treatment of the mystifying new disease was known and available. These histories recount over and over, but from diverse perspectives, the ways in which physicians responded professionally and personally to the increasing stream of very ill patients with puzzling symptoms and psychological as well as physical problems. They also trace physicians' gradual awareness of the severity, extent, and complexity of the new epidemic, focused initially so frighteningly on gay men. Some of the interviewees also tell of learning to manage the "worried well" who came to their physicians with fears of acquiring or transmitting the new syndrome.

Aside from providing a portrait of AIDS medicine as practiced in private medical offices in the years before AZT and protease inhibitors were available, these interviews describe from a variety of perspectives, the interviewees' responses to major events and crises of the epidemic in the early 1980s. A pervasive theme is the formulation of safer sex guidelines. Bob Bolan particularly, but others as well, were preoccupied with the formulation of guidelines which would simultaneously reduce disease transmission and honor the community's arduous battle for freedom of sexual expression. The accounts are sometimes explicit about sexual practices and attitudes, showing how those active in the epidemic brought taboo issues out of the closet and onto the public stage. In fact, these oral histories suggest that one lasting effect of the AIDS epidemic may be to have made safer sex practices and healthy sexual expression an open topic of discussion in many sectors of American society. The histories offer an intriguing range of viewpoints on this issue in gay politics.

The interviewees also provide accounts of important events in the years closely preceding and following the recognition of AIDS in San Francisco--the deaths of San Francisco Mayor George Moscone and Supervisor Harvey Milk (the latter the first openly gay elected official in the country), the hepatitis B vaccine trials, the crisis over bathhouse closure in San Francisco, controversy over blood donation policy, fears regarding the AIDS antibody test, and so on. Most of these events highlight the intersection of medicine, sexual and gay politics, and human rights, as well as the strengths and fallibilities of individual human actors. The oral histories in this series are rife with colorful examples in all these regards. Readers may be interested



to compare these accounts with those of the university physicians and nurses interviewed for this project.

These comments only begin to tap the range of topics and insights embedded in all three phases of this project. My hope is that these interviews, over thirty in all, will provide a basis for ongoing documentation of the epidemic. Victoria Harden and colleagues at the National Institute of Health Historical Office have recorded the contributions of researchers at NIH and Ronald Bayer and Gerald Oppenheimer at Brooklyn College have interviewed physicians in various cities across the United States. But there is a great need to expand documentation in time and geography. To my knowledge, there is no systematic and sustained work on the history of AIDS in developing countries in which it is expanding at a terrifying rate. Neither are there indepth historical projects on specific topics, such as efforts to develop AIDS vaccines and the associated ethical and social issues. Perhaps this collection of oral histories will serve as an impetus and inspiration for others to pursue the history which remains to be recorded worldwide.

#### Locations of the Oral Histories

The audiotapes and bound volumes of all oral histories in the AIDS series are available for research at UCSF Library's AIDS History Project Archives. The oral histories are also available at the National Library of Medicine, the Bancroft Library, UCLA, and other research libraries. Some are available on the Internet at: <http://www.lib.berkeley.edu/BANC/ROHO/online>. The remainder are in the process of being placed online.

#### Acknowledgements

We are grateful to Karen Butter, Acting Director of UCSF Library, for arranging project funding. I also wish to thank Dr. Robert Bolan for giving me access to three cartons of his personal records which he then donated to the AIDS History Project at UCSF. I also wish to thank Dr. Ric Andrews for arranging access to documents in the office of Bay Area Physicians for Human Rights.

I thank editorial assistants Grace Robinson, Julia Rechter, and Celeste Newbrough, and production manager Shannon Page for their efforts in finalizing the oral histories. I am grateful as always to Willa Baum, ROHO director, for her oversight and helpful comments.

Sally Smith Hughes, Ph.D.  
Research Historian and Project Director

January 2000  
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- Michael J. Helquist, "Journalist of the Early AIDS Epidemic in San Francisco"
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- Diane Miller, M.P.H., "AIDS Policy and Administration at San Francisco General Hospital"

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James Groundwater, M.D., Dermatologist, "Dermatologist Treating the First Kaposi's Sarcoma Patient Diagnosed in San Francisco"

VOLUME II

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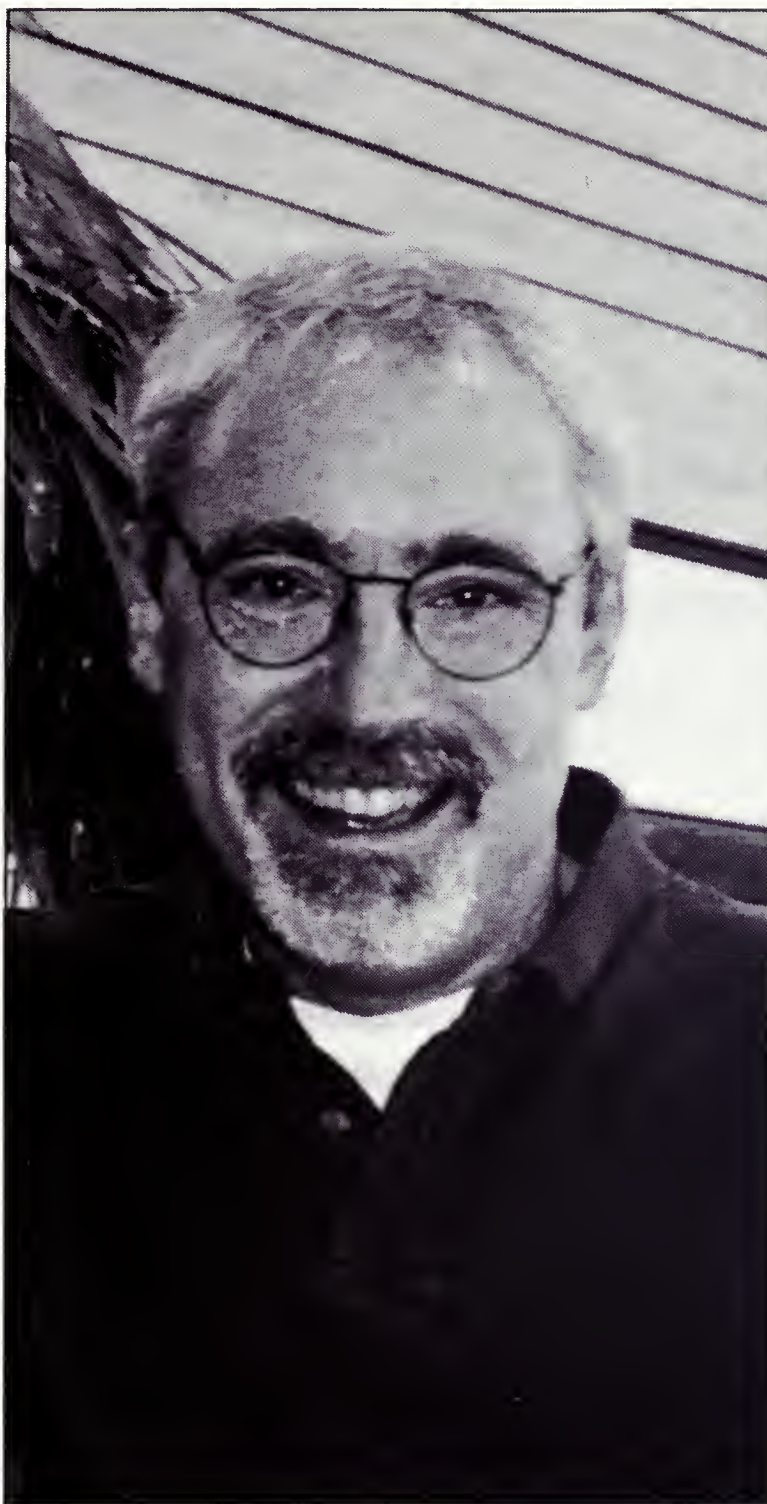
Paul Monahan O'Malley

AIDS AND THE HEPATITIS B VACCINE TRIAL IN SAN FRANCISCO

Interviews Conducted by  
Sally Smith Hughes, Ph.D.  
in 1996







Paul Monahan O'Malley, 1999.



## INTERVIEW WITH PAUL MONAHAN O'MALLEY

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## INTERVIEW HISTORY--Paul Monahan O'Malley

Paul Monahan O'Malley was chosen as an interviewee in the AIDS oral history series because of his key role in mining the hepatitis B vaccine trial of the late 1970s and early 1980s for information about AIDS. As a project manager at the San Francisco Department of Public Health, he coordinated the hepatitis study which provided important data regarding the safety and efficacy of hepatitis B vaccines. More significantly for this oral history project, the serum samples collected for the vaccine trial and the accompanying log books held hidden clues to the genesis and spread of the AIDS epidemic in San Francisco.

Although the hepatitis B/AIDS story is the focus of his oral history, O'Malley, the only person in this series who is not a physician, sets the stage for the arrival of the epidemic by first telling of his work as a communicable disease investigator at the San Francisco City Clinic with its heavily gay clientele. He recounts that even before the AIDS epidemic broke, he was alarmed by the high incidence of sexually transmitted disease in gay men and by their often cavalier attitude towards treatment. Like others in this oral history series, he was concerned that continued reliance on antibiotics was not a reliable prophylaxis and might have long-term health consequences.

One of the six test sites for the hepatitis B vaccines--the conventional and recombinant DNA forms--was the gay community in San Francisco because it suffered a high incidence of hepatitis, which, like AIDS, is a sexually transmitted and blood-borne disease. Hence, the gay community centered in the Castro District was an apt population for testing the efficacy of the vaccines, which were subsequently shown to work. By chance, the hepatitis B study was conducted at the time that AIDS was beginning to infect the same community. What O'Malley and others subsequently recognized was that the frozen and stored serum samples collected in the hepatitis study showed that individuals were infected with HIV before the AIDS epidemic was recognized in 1981. Furthermore, the sera could be used to trace over time the early spread of AIDS in San Francisco and to substantiate its long incubation period. The study served as an alarm to the threat of the new epidemic.

O'Malley explains in the oral history that the serum samples could be linked with donors through log books containing the names of the serum donors. On the basis of the new HIV antibody test and the stored serum samples, it was determined that about 70% of participants in the hepatitis vaccine trials tested positive for HIV.<sup>1</sup> O'Malley describes how in 1983 he

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<sup>1</sup> Christine Russell, "Map of AIDS' Deadly March Evolves From Hepatitis Study," *Washington Post*, February 1, 1987. (See appendix.)

set about the tedious process of tracing donors, some of whom had moved away from San Francisco in the intervening years, in an effort to determine the risk factors for AIDS.

O'Malley was driven by two principal aims: to gather unique information about the origin and spread of the San Francisco epidemic, and to preserve the confidentiality of the study participants. The first goal was achieved in spectacular fashion: the hepatitis B vaccine study has been credited with providing more information about the progression of AIDS in a population than any other single study at the time. Regarding the second goal, preserving the confidentiality of its medical records had always been a prime intent of the health department, but was particularly important in this instance because the donors were gay and a diagnosis of AIDS in the early 1980s carried the threat of stigmatization, alienation, and discrimination. O'Malley also had a personal motive in guarding the log books: he himself was a member of the gay community and had experienced the pain of losing friends and loved ones. He brings his story up-to-date by summarizing his role in the health department's ongoing HIV vaccine preparedness study.

### The Oral History Process

Two interviews were conducted, on July 2 and 9, 1996, at the AIDS Office of the San Francisco Department of Public Health. O'Malley, intently focused and appearing younger than his fifty years, talked expansively, obviously taking his responsibility regarding participant confidentiality extremely seriously. The transcripts of the discursive interviews were heavily edited by interviewer and interviewee in order to clarify the main narrative of the hepatitis B vaccine/AIDS studies.

This oral history describes the professional and very personal dedication of a man who has helped to elucidate the early course of AIDS in a community with which he identifies and strives to protect:

The health department has always been very concerned about confidentiality. We realize a lot of disenfranchised people have reasons to fear that information about their sex lives, or diseases they've had, or drugs they've taken, will be used against them. So I've always felt that we had to bend over backwards to protect them. Confidentiality is paramount, even during so-called "worst case" scenarios.

Sally Smith Hughes, Ph.D.  
Research Historian and Principal Editor

October 1999  
Regional Oral History Office  
The Bancroft Library  
University of California



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BIOGRAPHICAL INFORMATION

(Please write clearly. Use black ink.)

Your full name PAUL MONAHAN O'MALLEY  
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 Father's full name Philip Thomas O'Malley  
 Occupation Public Utility Supervisor Birthplace Clinton Massachusetts  
 Mother's full name MARY Elizabeth MONAHAN  
 Occupation Housewife Birthplace Worcester, Massachusetts  
 Your spouse N/A  
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Where did you grow up? Worcester, Massachusetts  
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MA State College at Worcester

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Areas of expertise HIV/AIDS research  
especially HIV vaccine preparedness  
issues.

Other interests or activities Children's Mental  
health issues, English and  
American history.

Organizations in which you are active HIV VACCINE  
Advocacy Organizations

SIGNATURE Paul M. O'Malley DATE: 10-22-1999





## INTERVIEW WITH PAUL O'MALLEY

## I BACKGROUND, EDUCATION AND MILITARY SERVICE

[Interview 1: July 2, 1996] ##<sup>1</sup>  
[San Francisco, California]

Education

Hughes: Mr. O'Malley, would you start back with where you were born and educated, and bring yourself up to the hepatitis study.

O'Malley: Yes, my name is Paul Monahan O'Malley--I've included my middle name, it's my mother's maiden name. I just turned fifty, so I was born in 1946, the second day of June, 1946. I was born in Clinton, Massachusetts, a small town in north-central Massachusetts, but at a very early age moved to Worcester, Massachusetts, which is the second-largest city in Massachusetts. They always call it "the heart of the Commonwealth."

And that's where I grew up and had my grammar school, high school, and college education. I'm from an Irish Catholic background, and I was educated by the nuns at Blessed Sacrament Grammar School, and then went to a public high school. My college degree [1968] is in biology with a minor in math from Massachusetts State College at Worcester, 1968.

Public Health Instructor in the Air Force

O'Malley: When I graduated from college, it was 1968, the height of the Vietnam War, and this was before the draft lottery. I was

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<sup>1</sup>## This symbol indicates that a tape or tape segment has begun or ended. A guide to the tapes follows the transcript.

drafted immediately after I graduated and had to make a decision, and I wasn't overly enthused about the war.

I joined the Air Force to avoid being drafted into the Army, which was very serendipitous for me in a lot of ways. I was trained as a public health inspector, I was trained in environmental medicine, occupational health and communicable disease. My military training mainly occurred in Texas, at Brooks Air Force Base.

Hughes: Did you have some choice about that field?

O'Malley: Yes. I was sent to a base in Florida after my training where the focus was on tropical medicine. The only downside to this assignment, virtually everyone that had been sent to the base in my job classification were later assigned to Southeast Asia.

Orders came up more than once to send me to Vietnam and then they were canceled, it was pure luck. It was not as if I had someone to "pull strings" for me. I spent a year and a half in Panama City, Florida on the Gulf Coast of Mexico, and I learned a great deal about communicable disease, occupational medicine, and environmental health. Since it was an air force base, most of my training focused on flight lines and services associated with aircraft maintenance. Venereal diseases, STDs [sexually transmitted diseases], were quite prevalent, and I became well experienced in that area as well.

Hughes: Were you doing research?

O'Malley: No, this was real hands-on--going out to flight lines, doing occupational health inspections, going to restaurants, doing environmental health inspections, interviewing and counseling people who had been diagnosed with STDs about source and spread of their infections.

I was reassigned to a base in the Upper Peninsula of Michigan right on the Canadian border. The assignment lasted eighteen months. I dealt with similar public health issues. It was another air force base, so the occupational health, environmental health issues, and communicable diseases were very similar.

"Coming Out" as a Gay Male

O'Malley: During my military service, I started grappling with my sexuality, my homosexuality. And the reason I bring this up is because it did effect my decision as to where I would live when I left the military. I had a relationship with a fellow airman when I was stationed in the Upper Peninsula of Michigan. It was an unfortunate time to deal with, "coming out"; while also forced to be "closeted" in the military.

Sometimes I think there's more of a witch hunt now. I didn't realize then, but because a war was being waged the military tended to look the other way to some degree if you were homosexual.

Hughes: Because they needed bodies to fight the war?

O'Malley: Yes. There was one incident that I often wondered about. An inspection was done without notice. In my locker I had a card from a gay bar in southern Michigan, and I thought, If someone looked at this, they should have been able to put two and two together. Additionally, I was in a relationship with this airman, I thought other people may have put two and two together, but nothing was ever said.

But in any case, when I got out of the military, I had this background in infectious diseases, and I felt like I really had learned something from my time in the military. I was honorably discharged from the military during the holiday season in 1972--I went back to Massachusetts to spend Christmas with my family.

But there were two things on my mind. I'd wanted to get away from cold weather, but more importantly I wanted to explore my sexuality without being judged. I have a New England, Roman Catholic background so I wanted to do it in an environment somewhere where no one was looking over my shoulder. I hadn't told my family about my homosexuality; I hadn't dealt with that at all at that point. And for all those reasons, I wanted to be somewhere like San Francisco, where I could be open about my sexual orientation.



## II MOVING TO SAN FRANCISCO

### Work at the City Health Clinic Before the AIDS Epidemic

O'Malley: So I moved to San Francisco. I decided I was going to give San Francisco six months. If I couldn't find work, I would return to Massachusetts. My interests were very specific: I wanted to work in the public health field. I took the U.S Public Health Service exam. There were openings in the surrounding counties, and I was applying for jobs all over the Bay Area. Luckily I was able to collect unemployment during this period of job-hunting.

Luckily, a job materialized at six months. I had just missed getting a job in Marin County, and the person I lost out to was leaving a job in San Francisco, at the health department. This was in July, 1973; I had just turned twenty-seven when I applied for the job with the health department. As a communicable disease control counselor. The interview went well and I was offered the job.

Hughes: Now, was Erwin Braff head of the clinic at that point?

O'Malley: Yes, exactly. Dr. Francis Curry was the director of health. Dr. Braff was the director of communicable disease control, and then there was Selma Dritz, who you probably have interviewed.

Hughes: I've talked to her<sup>1</sup>.

O'Malley: Did you get a chance to talk to Braff?

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<sup>1</sup> See the oral history with Selma K. Dritz, MD. San Francisco AIDS Oral History Project: The Medical Response, 1981-1984. An oral history by Sally Smith Hughes, PhD, recorded in 1992 and 1995, Regional Oral History Office, The Bancroft Library, University of California, Berkeley. Hereafter, AIDS physicians oral history series.

Hughes: No, I didn't.

O'Malley: Unfortunately, he died last year. In '83, '84 Dr. Braff and Dr. Dritz had both decided to retire. Their last years with the health department was the same time the AIDS epidemic was exploding. So when they would have under normal circumstances been able to wrap things up and leave quietly, they had this enormous epidemic exploding in their face. Dr. Braff pretty much turned this all over to Selma, as I remember.

Hughes: Yes, that was the impression she gave to me.

O'Malley: In the beginning, it clearly was her baby. There were also CDC epidemiologists that came out from Atlanta, Georgia [from the CDC] to help and advise.

But back to my story: Dr. Braff oversaw the VD clinic. City Clinic was located at 250 Fourth Street, San Francisco. It's now the Ansel Adams Gallery. It's right across from the Moscone Center. The neighborhood was very different in those days. In the early 1970s, long before the Moscone Center was built, there was a flophouse across the street, there was the Salvation Army soup kitchen next door, so it was a very different feel than you get down in that area today.

The clinic was an amazing experience. Especially for someone who was dealing with their own sexuality. I'd heard all these wonderful things about the city, especially about the thriving gay community. It wasn't as well known then as it is today, but at the time San Francisco was known, at least amongst gays, for its long history of tolerance.

I was working at the clinic with a staff that was primarily my own age--baby boomers. The people walking in the door were young people too. Working there was a great way to get your career off and started. I mean, if you can handle a busy STD clinic you can handle anything. It was a three-ring circus. We used to see 350 to 400 people every day, day in, day out. So 1,700, 1,800 people a week.

#### History of the San Francisco City Clinic

O'Malley: The clinic itself had a history, a reputation, going back to the thirties. I know the clinic existed then; we still have the charts and all that. Because of San Francisco's history of being very tolerant, people felt more comfortable going to



the Clinic instead of to their private physician if thought they had a sexually transmitted disease. They knew the staff there was nonjudgmental and experienced and that they'd get treated properly.

Hughes: But it wasn't known as a gay clinic?

O'Malley: It was never publicly known as a gay clinic. It was more a word of mouth thing amongst gays. When I first went in there, I would say the majority of clients were gay males. But see, the clinic first became well known in the Haight-Ashbury era in the sixties when there were an STD problem developed in the hippie communes. As a matter of fact--this was before I moved here--they apparently used to have a shuttle bus that would go up and down Haight Street. By the time they got out to Golden Gate Park they had a full bus, and they would go to the clinic and drop everybody off. So the clinic really developed a reputation during the hippie era.

I have two friends who I refer to as my gay mother and father, whom I met right after I moved to San Francisco. They are actually an important part of this story, especially when the epidemic begins. They had moved here in the late fifties, and they said that when they went to the clinic for an STD check back in those days, they really stood out like a sore thumb. They were "three piece suit" gay men so if you walked into the clinic in a shirt and tie it made you stand out, period, because it was the hippie era. I received the history of the STD clinic from them, long before I worked there.

#### Prevalence of Sexually Transmitted Diseases in Gay Men

O'Malley: When I went to work at the clinic, there was a real high incidence of gonorrhea and syphilis, in particular amongst gay men, and the clinic was pretty much focused on serving those needs. The clinic was very good at getting people seen by a clinician in a timely manner. There was a lot of what they call "epi treats", epidemiologic treatments. If someone came in who had had sex with someone who was recently diagnosed with syphilis or gonorrhea, the clinicians would usually treat them prophylactically. They didn't want to take the chance that the test would produce a false negative result and there was little or no concern about overtreating with antibiotics in those days. That attitude has changed since then. It was like, Give them the medication to prevent any possibility that the infection would be spread to a wider circle of sex partners.

Hughes: Did anybody think twice about that at that time?

O'Malley: No. There was such an innocence. Following World War II with the development of penicillin, my generation grew up with the idea that there was an answer to everything, a treatment for everything. Some of the younger gay men now will ask me, "After you'd had gonorrhea four or five times, wouldn't that have been enough to worry you about overuse of penicillin and motivate you to start using a condom?" And I say, "You have to have been there. You could take a handful of pills, and you were on your way. It wasn't like people were concerned about, Well, what are the longterm implications of taking these massive doses of penicillin, a couple of times a year, year in, year out? Everybody was young, living in the present, and focused on the sexual revolution. And it wasn't just the homosexual community. It was like, no limits. We're going to have a good time, and we're not going to let anything get in the way of it. The health department did not try to counter that attitude. The strategy was to do absolutely nothing to discourage sexually active individuals from seeking testing and treatment. Condom use when it occurred was not due to a health department campaign.

Hughes: So there were two factors: sexual liberation, and the attitude that if you did get a sexually transmitted disease, there was an antidote, so why worry?

O'Malley: Exactly. And the health department did not push other alternatives such as condom usage as I just mentioned.

A question I've been asked a million times is, did I have any premonition of what was to come? No, not really. We did get concerned at times that there was maybe a strain of gonorrhea or whatever that was going to become resistant to drugs and harder to treat. People would also develop an allergy to certain drugs, but there always seemed to be the next drug coming down the line that was not resistant to gonorrhea and provided another option to those with allergies to licensed drugs.

My biggest concern was seeing 350, 400 people a day, everyday at the clinic. I do remember talking with Tom Hoynes, a co-worker, and saying, "Can this really go on forever?" But I was thinking more in terms of all these antibiotics that people were taking. Antibiotics were a relatively new phenomenon, they'd only been around since 1945. There was no historical data on people taking the massive doses of antibiotics that were being distributed at the clinic. There was a public health justification for it, but now in



retrospect, maybe we should have questioned the dispensing of antibiotics more closely for prophylactic purposes.

Hughes: Was this the approach throughout the Public Health Service?

O'Malley: Yes, definitely.

Hughes: It was common practice to treat prophylactically?

O'Malley: Right. Syphilis is a good example. Someone walks through the door and they've had sex with someone who's diagnosed with syphilis the prior week but the incubation period is ninety days. The easy solution is to treat prophylactically. Another choice is to have them come in for a blood test every thirty days for three months, and then if they stay negative for three months, then we know they didn't get infected.

The problem with this solution was expecting someone to be abstinent for 90 days or to use condoms for 90 days. This was the sexual revolution. The potential for further spread of the infection was high. If you defer treatment, and they become infected between tests, what do you do when they walk in at day thirty or day sixty with a positive blood test? If they have had new sex partners during this period the potential for further spread of infection is high.

Hughes: Yes, the infections mushroom.

O'Malley: There really wasn't any data suggesting concern about longterm distribution of these antibiotics. We gave 4.8 million units of aqueous procaine penicillin by injection for gonorrhea. 4.8 million units! That's the highest dose you'd ever get by injection! I used to think about all this penicillin year in, year out. It wipes everything out in your intestines, and you have to sort of start from scratch. The treatment for repeated infections has to be hard on your immune system. I thought, We're all in our twenties now. Let's fast-forward fifteen or twenty or thirty years. Are we going to find out that people are coming down with rare cancers and diseases because of the impact antibiotics may have had on people's immune systems?

Hughes: Could you attribute the increase in STDs to more than just the fact that the population of gay men in San Francisco was increasing in large numbers?

O'Malley: Hmm. If you look at STD statistics from the seventies, they just continue to climb right up until 1980, '81, when everything started changing. When cases were reported in the newspapers about the number of gonorrhea or syphilis cases--and

this was supposedly being very protective and paternalistic towards the gay community--it would be covered up as to how many were attributed to gay men. There was never any information about where it was coming from.

Hughes: For example, they wouldn't call it rectal gonorrhea?

O'Malley: No.

Hughes: Would it be listed as simply gonorrhea?

O'Malley: Yes. Well, that was a harder one to cover up. If people asked the right questions, the statistics were available. [laughs] In retrospect, I see the downside: gay men weren't getting the message about risk, especially about the prevalence of syphilis and gonorrhea, especially rectal gonorrhea, in their community.

##

O'Malley: We encouraged people that were sexually active to get checkups every three months. So part of the reason that clinic was very busy is because we really promoted routine check up and follow-up checks after a diagnosis known as "test of cure". If someone didn't come in for a test of cure, they would get a reminder letter to come back. If someone had been treated for syphilis, we had them come in for follow-ups for awhile, usually every three months or until their blood test results returned to negative.

#### San Francisco City Clinic as a Gay Clinic

O'Malley: It was the sexually active portion of the gay community that was using our clinic. And it did develop a reputation over time within the gay community as a gay clinic. I think probably by the late seventies 70% of the clientele were gay males. We had a slew of gay physicians who were working there by that time. It was a very gay-friendly clinic.

Hughes: So if somebody other than a gay person walked in, they might be likely to choose a different clinic the next time around?

O'Malley: Maybe. We might have seven or eight doctors working at any given shift, but there would be only one physician assigned to see women. And part of that was because there weren't that many women walking in there. It wasn't that staff, or gay male patients, made women or straight male patients feel unwelcome.

I think that they automatically felt like a minority due to the number of gay men attending the clinic.

Hughes: A woman was sort of a fish out of water.

O'Malley: Yes, she'd rather go elsewhere, but the options were limited. That has all changed now for women and gay males. Now there are several health centers where you can go to get STD checks as well as to be evaluated for other things. But in those days, regardless of whether you were a female, straight man or gay man, the clinic on Fourth Street was about it. If you had syphilis or gonorrhea, if you did not have insurance, the clinic on Fourth Street is where you went for a checkup & treatment without judgement. Today there are numerous private gay practices with gay physicians, which started in the late seventies, early eighties. Before then, most gay men even if they had medical insurance, would go to the clinic for STD checkups, because they didn't want to go to their private doctor.

Hughes: Because of the fear of stigma?

O'Malley: Yes, right.

An interesting part of the clinic is that every type was represented. We used to see the three-piece suities from the financial district to the down-and-outers from the Tenderloin. I mean, we had the whole gamut, because the Clinic had that reputation. There were people coming in whom you knew had money, the Pacific Heights types. They came because they knew they would get handled properly and nonjudgmentally.

Hughes: Was there any counseling?

O'Malley: Yes. And especially if you were infected with syphilis and urethral gonorrhea, you were interviewed for contacts as well. As I mentioned earlier, condom use really wasn't promoted strongly at that time. The emphasis was placed on getting tested every three months if you were sexually active, and educating people as to what the symptoms were so that they'd know, recognize, be sure to run to a doctor if they got any of these symptoms again. This educational component was beneficial. The clinic was always a busy place, period, all through the seventies. I didn't sense the dramatic leap in STD cases over those years. The increase was gradual but steady. It crept up over a number of years so the rise was less obvious on a month to month basis.

Hughes: Do you remember any discussion in the 1970s along the lines of, This group of sexually active people is a perfect setup for a new sexually transmitted agent or a virulent form of an old agent?

O'Malley: Yes, because even then we heard that there were certain penicillin-resistant strains of gonorrhea that had shown up in Southeast Asia, and were usually tied to heterosexual G.I.s. There was always the concern, because we're on the West Coast, that sooner or later somehow it could get into the gay community, and oh boy, would we have our hands full if we had [an outbreak]--particularly if we couldn't use penicillin any longer. We'd have to use some of these newer drugs that were developed.

#### Naive Attitude towards STDs in the 1970s

O'Malley: It was such naivete. There was this feeling, Oh, even if penicillin doesn't work, they'll just keep chugging out new drugs, and they will be keeping one step ahead of the antibiotic resistance problem.

But new diseases? No. I don't think people really thought about that. I mean, now we know. It's funny, since HIV was discovered we've had toxic shock syndrome, Legionnaire's disease, and others.

Hughes: Hantavirus.

O'Malley: Yes. But if you think back to that time period, we did not hear about new emerging diseases. And there wasn't a lot of long-range planning regarding the potential emergence of drug-resistant strains of the known infectious diseases. It was just keeping on top of what was happening, making sure you had a staff to handle the volume of people that were coming in, and providing penicillin. It wasn't just a San Francisco Health Department decision; it was a Centers for Disease Control decision as well. There were busy VD clinics, I'm sure, in several cities in the country at that time, because of the sexual revolution.

I can remember talking about immunological disease or rare cancers that might show up years later because of the sheer volume of penicillin that people had received. I don't think I focused on the disease itself. If I did it was for the following reason: I'd state: "If you keep getting urethral

gonorrhoea over and over and over again, you may develop strictures in your urethra." In retrospect I'm sure I scared people half to death with talk about what would have to be done to get the strictures removed. But I don't think we ever educated people on a broader scope in terms of what could happen on a community level later on down the line. We didn't think through this thing of being nonjudgmental in terms of whether you had one, or ten, or 100 sex partners in a given period of time, and the potential health implications when diagnosed and treated numerous times for STD infections.

Hughes: The ethos of the clinic was that you went out of your way not to say anything that might question somebody's sexual activity.

O'Malley: Yes, at the time the strict public health point of view was, you want to do nothing that discourages them from coming to the clinic. If they get symptoms, we want them to feel comfortable about coming to the clinic and getting diagnosed and treated ASAP, and not taking a chance that it's going to be spread to others.

Hughes: So it was more a public health concern than the clinic staff being in sympathy with gay community sentiments?

O'Malley: Well, I think there were some people at the clinic in those days who were concerned and appeared somewhat judgmental of what they were witnessing. Overall, there was a lot of empathy. I mean, there were gay men working there who came from the gay community, and there were also a lot of young heterosexual people that were in the same age group, and they were in the midst of their sexual revolution too.

### The Hepatitis B Study

Hughes: How interested was the CDC [Centers for Disease Control] in sexually transmitted disease, particularly in the gay community?

O'Malley: CDC and the Health Department realized what was going on. Nobody wanted to talk about it publicly. Some of it was paternalism, but mainly due to embarrassment associated with discussing sex, especially gay sex, publicly. To go forward to AIDS, the embarrassment that was associated with how people got this disease was very apparent. CDC was not alone in that regard. It was a society-wide phenomenon.



We were approached in late 1977 at the clinic by Bill Darrow from CDC about conducting the hepatitis B study. He wanted to do a hepatitis B vaccine preparedness study to get data on the prevalence and incidence of hepatitis B in gay men--as well as assess their willingness to participate in Hep B vaccine trials. Clean documentation as to the risk factors associated with transmission of hepatitis B through sexual practices was also desired.

Merck, Sharpe, & Dohme, a vaccine manufacturer, had been doing research on hepatitis B vaccines in the mid-seventies. They were testing their Hep B vaccine in New York City at the New York Blood Center. The vaccine looked promising, but they needed to do this additional multicentered vaccine efficacy trial to get FDA approval of the vaccine for licensing. Clearly, the CDC was well aware of the fact that San Francisco was an ideal city due to the size of our gay community and because there was clear evidence that hepatitis B infection rates were rising in the gay community.

Originally the disease had been called serum hepatitis, because they thought it was primarily transmitted through transfusions and IV drug use. But when it became a problem in gay men by the mid-seventies, it became evident there's more than IV drug use transmission occurring in gay men. It became clear that hepatitis B was being transmitted sexually, and more likely sexually than by intravenous drug use.

In addition to San Francisco, L.A., Denver, Chicago, and St. Louis, all of which have gay community service centers, were approached to participate in this trial. The original game plan was to collect the data on prevalence and incidence in 1978 with the trial starting in '79. But the trial started in '80, so we had actually two full years of '78 and '79 to collect data on hepatitis B infection in gay males and to recruit men for a vaccine trial.

Hughes: It took longer to recruit than initially expected?

O'Malley: The delay had nothing to do with our ability to recruit. Getting the trial protocol in place and abiding by all the rules of the FDA, et cetera, took a little longer than planned. That was the main reason. There were problems with recruiting, but for an entirely different reason which I will discuss in a minute, once I give you some statistics.

Due to the delay, we ended up screening 6,700 people for hepatitis B between 1978 and the end of 1980; it was a three-year period. The trial began in April of 1980. Although we

screened approximately 6,700 people for hepatitis B, only 360 men actually participated in the trial itself.

#### High Rate of Hepatitis B Infection in San Francisco's Gay Population

O'Malley: One of the reasons we were approached, of course, was that we had an enormous gay community. We figured there would be a lot of men potentially at risk for exposure to hepatitis B, so we should have no problem recruiting a large sample. But one thing we discovered when we started recruiting at City Clinic in '78 was that three out of four men showed a history of exposure to hepatitis B. They either had a marker for hepatitis B antibody which indicated a previous case or infection, or they were currently infected--people that had either a new infection, or were healthy carriers.

Hughes: Now, did that come as a surprise?

O'Malley: Yes. We didn't expect the prevalence of hepatitis B infection to be quite that high, that three out of every four men we screened at the clinic would either have a history of exposure to hepatitis B or show a current infection. We were also surprised by the large number who apparently had asymptomatic cases, and reported no known history of hepatitis B. We found that many men were immune to hepatitis B without a real history of being diagnosed hepatitis B.

Hughes: And that was a new idea as well, that the carrier state was not something that people expected?

O'Malley: Actually, that is a different finding. This is people who were not carriers--literally, their body cleared hepatitis B. They were infected and cleared it and never manifested any obvious symptoms, or if they manifested symptoms, they were so short-lived the patients never went to a doctor. Mild cases of hepatitis B often appear like the flu. We now know that about 60 percent of individuals who get exposed and are infected with hepatitis B never manifest serious symptoms. The odds are actually overwhelmingly in your favor that you won't become symptomatic or sick.

If you look at everybody that is infected with hepatitis B, it's actually only one in ten that becomes a carrier, and not all of those are symptomatic. Only one in one hundred

develop chronic, aggressive, fulminant hepatitis B.

Hepatitis B can be a life-threatening, fatal disease. I knew a physician that died of hepatitis B in 1978 or 1979. It was chronic hepatitis, and it was fulminant. He was a fairly well-known gay physician, one of the first gay doctors who set up a practice in the Castro area focusing on the health needs of gay male patients. Here was somebody who was dying from something they picked up sexually, a life-threatening sexually transmitted disease. This was the first time I was personally aware that an sexually transmitted disease could result in death.

Although we were aware of the problem with hepatitis B, we were mainly focused on the high rates of syphilis and gonorrhea in the late seventies. By 1980, before HIV we were also aware of problems with parasitic infections, enteric infections, in the gay community. Regarding the concern I raised earlier about the effect of continuous drug treatment on the immune system, the treatments for enteric infections were much harsher than penicillin. The drugs were very hard to tolerate due to side effects. You had to take them for weeks. This did motivate some individuals to change certain sexual practices.

Hughes: What were you doing in the clinic with cases of hepatitis?

O'Malley: We rarely would uncover anybody that actually was sick with hepatitis, but if we did, a referral system was activated, referring them to the Infectious Disease Clinic at SFGH.

I would also like to share with you the procedures we used at the clinic to enroll gay men in the hepatitis B study. As I mentioned earlier, seventy percent of the guys walking through the door were gay men, and we were recruiting at random from the waiting room. The clinic was busy. We had a captive audience. There was always a time lapse between someone initially registering and being seen by a doctor. It could be half an hour, forty-five minutes. We first received informed consent, then gave a short fifteen-minute questionnaire, and we tested for markers of hepatitis B. We sent them the results in the mail, with the exception of those that were currently infected. We would call those that were infected as soon as possible. For those who were negative, we also would call and request that they participate in the hepatitis B study which involved screening for hepatitis B every four months.

Hughes: I asked you what you did for cases of hepatitis.



O'Malley: Well, if we got a positive test result back on somebody, we would offer to repeat the test and perform a liver function test. The clinic wasn't set up to take care of people with hepatitis B. Occasionally, we'd catch someone as they were coming down with hepatitis B, and make an immediate referral. We were not equipped to evaluate people, other than to do the initial repeat test and the liver function test. Again, for ongoing care they'd be referred to their own private physicians or to San Francisco General Hospital if they didn't have health insurance.

We were uncovering cases of hepatitis B in men who may have been infected for several years, and it looked like they were healthy carriers. They were basically healthy and without obvious symptoms; their liver functions might have been slightly elevated, but not sufficiently elevated to make the patient aware of the infection.

#### Procedure for the Hepatitis B Study

O'Malley: The fifteen-minute questionnaire focused on sexual practices and drug usage, factors that were thought to be strongly associated with transmission of hepatitis B. After the first four months of screening in 1978, we were finding a very high percentage of people that were already exposed to hepatitis B and thus not candidates for the vaccine trial. We started experimenting with different ways of trying to increase our likelihood of screening men that had no markers for hepatitis B.

The first change we implemented was to stop interviewing everybody on their first visit. We had been interviewing everyone the same day as the blood draw or before we had a blood test result.

We started interviewing only the men who were hepatitis B negative. We would place them on a four-month return plan because the incubation period was supposed to be no greater than four months. We would follow them every four months and question them about their sexual practices, so if they did become infected or go from hepatitis B-negative to hepatitis B-positive, we could see what they'd been reporting in the interim as to their sexual and drug use practices.

This is how the study operated in '78 and '79. We continued this massive screening of men that were coming in to

the clinic and asking them if they'd be willing to consent to getting an extra tube of blood drawn for hepatitis B on this visit. Most patients were being tested for syphilis via a blood test. We told them they could also be tested for hepatitis B while having their blood drawn for syphilis. Most gay men consented. They realized that hepatitis B was a problem in the gay community, and it was important for the community to participate in this project.

Hughes: In the late 1970s, you were doing just the hepatitis study? You'd been assigned to it?

O'Malley: Right, I also sought out the position. I had been from '73 to '77, mainly working with STD's, gonorrhea and syphilis. I was ready for a change. The opportunity came along to be the coordinator of this hepatitis B project. I was excited. It sounded like it would be something very new and different. It interested me, it was a multicenter project, it was a national study, and we hopefully were going to end up with a licensed vaccine, too. That part of it was nice. I had witnessed enough people with hepatitis, what they had to go through. A vaccine against hepatitis B would be a major advancement.

Before HIV, hepatitis B was considered the most serious health problem in the gay community. You would hear about people that were carriers, and occasionally people had died from this disease. Again, it was an STD that could kill. It's not like syphilis or gonorrhea. There wasn't a treatment. For chronic carriers, they were experimenting at Stanford University with some interferon as a treatment, but that was for someone with high liver functions. Hepatitis B could do somebody in.

#### Difficulty of Finding Seronegative Participants ##

Hughes: I read that accession of the first 700 participants in the hepatitis B vaccine trials was achieved only by screening something like 10,000 individuals, presumably in all five centers.

O'Malley: Yes.

Hughes: What were the requirements for eligibility in the trial?

O'Malley: As long as you were a sexually active gay male, you could be screened for the hepatitis study and potentially end up being

enrolled. You had to have no markers of hepatitis B, normal liver functions, and also make a commitment of participation for one to two years. One out of four, 25 percent of the 6,700 people we screened, had no markers for hepatitis B. In general, it was the younger guys that had no markers. They hadn't been out as long. We used to joke about the need to set up a screening booth at the Greyhound bus station to screen them as they got off the bus before they had sex with anybody.

There were all these other hurdles too. We discovered was that certain recreational drugs being used in those days would cause abnormal liver functions, usually temporarily. They would subside, but it would wreak havoc with us trying to determine whether they really had a liver problem or not.

Hughes: You automatically disqualified those people?

O'Malley: No, we would retest. It just added an extra level or step to the screening process in many cases. It was hard to get people back in for retesting.

Also, gay men are very mobile, especially when they're young, and to get a two year commitment was asking a lot. Additionally, there were individuals who had fears about taking an experimental, unlicensed vaccine. Some young people did see the need to experiment with this vaccine, but when you're young you think something bad is always going to happen to the other guy, not to you. Maybe one in 100 people that get hepatitis B get sick and die of it, so some people didn't consider it worth taking the risk. These reasons explain why we had to screen so many people.

In order to increase our odds of screening hepatitis B negative males, we refocused on screening younger individuals. We were trying to focus on people who were born after 1950, or on more recent arrivals in the city. We were looking for different ways to increase the number of people that had no markers for hepatitis B. We knew we were going to lose people from that pool of younger gay males, either because they wouldn't make a year and a half commitment, and/or they were worried about taking an experimental vaccine.

Hughes: Muraskin maintains in his book on the International Task Force on Hepatitis B that one of the biggest obstacles to fighting

Hepatitis B was the high price of the vaccine.<sup>1</sup> Was that an issue here?

O'Malley: We didn't deal with that issue until the vaccine was licensed. Participation in the trial was free. The licensed vaccine was expensive. It costs \$100 for the series of injections. Most at-risk populations could not and still can not afford the expense.

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<sup>1</sup> William Muraskin. The War Against Hepatitis B: A History of the International Task Force on Hepatitis B Immunization, Philadelphia: University of Philadelphia Press, 1995.

### III THE AIDS EPIDEMIC

#### First AIDS Cases

O'Malley: In early 1982 I started to deal directly with AIDS cases. Six months prior to that is when the first cases had been reported in the MMWR [Morbidity and Mortality Weekly Report] and the New York Times, et cetera. I was at a meeting in San Diego and I remember specifically talking about the cluster of cases with Harold Jaffe.

Hughes: What was that meeting?

O'Malley: It was just a standard STD [sexually transmitted diseases] annual meeting. I had just been to China, and had heard nothing about this new disease. One evening I was at dinner with some of the people I'd been working with in the hepatitis B vaccine trial. Bill Darrow and Harold Jaffe from CDC were talking about this cluster of cases, a few in New York, a few in Los Angeles, and apparently a couple in San Francisco too.

Hughes: Now, he was talking about KS and PCP at that point?

O'Malley: Yes. KS, which was being called a type of cancer, was the focus of the conversation. It was what was more unusual, these guys were presenting with getting purple spots or lesions on their skin. It sounded so bizarre compared to a pneumonia, even a pneumonia that people didn't seem to respond to treatment to. But these purple lesions were really new and different. That meeting was the very first week of June, in 1981.

So by the end of '81 you have a licensed hepatitis vaccine, but now no one was interested in hepatitis B. I mean, all of a

sudden, you've got alarm bells going off about this new disease that may be coming through the community.

Hughes: Why was your attention drawn to it?

O'Malley: Initially it had more to do with friends than with my profession. Sid and J.B. were an older gay male couple whom I saw almost as parents. They owned the apartment building in which I lived in the Castro district. They had been very sexually active for years in San Francisco, and they used to always joke that although they enjoyed life to the fullest, they also were prone to picking up STDs, and that's why everyone knew them at the clinic! They used to joke that clinic staff had to get a dolly from the back room to bring out their medical charts.

In August 1981, after I told them about what I had heard at the STD meeting, J.B. said, "You know what? Sounds like another sexually transmitted disease to me." And I remember he said to Sid, "We're not going to the bathhouses until they find out exactly what the hell is going on." J.B. also added, "With our luck we'll get this new illness if it is an STD." The ultimate irony is that they both did get it. They got sick in late 1982, and both died of AIDS in 1984. It's clear that by the time we were having this conversation, in 1981, they were already HIV-infected. We also know from the hepatitis B study's stored serum specimens that gay men were already showing positive [for HIV] in '78 and '79.

The gay physicians I was working with at that time were in denial, and the sad thing is that most of those doctors died of AIDS. If they used their logic they probably would have realized that, Hey, there's a good chance this is a sexually transmitted disease. But to accept that meant that they had to accept that they were all at risk as well. In retrospect that denial is understandable.

I had been diagnosed with cancer the year before, in '80, just before all this happened. I think my recent bout with cancer helped prepare me for this new epidemic and not succumb to fear and irrational explanations as to what was occurring. I thought that the STD theory made sense. I acknowledged that I was at risk as well, but I didn't consider myself at high risk. This was back in the days before we realized there was a long incubation period, and I had been relatively inactive sexually while I had cancer. I had dated two people. When this new disease started unraveling, I felt like maybe my risk was low. There also were implications that drugs played a role



in this new disease. I felt my risk was low in that regard as well.

AIDS Research Interest in Hepatitis B Blood Samples

O'Malley: The focus of AIDS research on the hepatitis study began when it was discovered that one of the very early AIDS cases, diagnosed in late '81, early '82, had been interviewed for the hepatitis B Study. Then Carlos Rendon who worked closely with Selma Dritz was investigating all the early [AIDS] cases for the Health Department. Our patient from the hepatitis B study was interviewed by Selma and he must have mentioned to her that he'd been in our hepatitis study.

Selma and Carlos contacted me. The patient had signed a release of medical information. They were looking for whatever information we might have on him. I mentioned that we had some stored blood specimens and interview data. This was early '82. I remember very clearly saying, "But you know, this is information from '78 and '79. We're talking about serum and interviews that are three and four years old." I thought it was pretty dated information. Now that we know what we do know about HIV, it was actually right in the important time frame in which people were first becoming infected with HIV. We decided to look for more individuals with AIDS that may have been we screened for hepatitis B.

Hughes: Did you have any particular rationale?

O'Malley: It was more of a hunch initially. I thought that the stored serum and interviews covered the three years prior to the AIDS outbreak, and might provide clues to this new disease. I did not make a strong connection at this time. I did not think hepatitis B was connected with AIDS. I just thought both diseases might be blood borne agents, since similar groups were at risk.

Information was starting to trickle in on a few other AIDS cases that were not in gay men--in IV drug users, transfusion cases. Every new risk group they come up with seemed to match risk groups for hepatitis B. So again I was thinking, Maybe we've got another sexually transmitted disease here that's caused by a blood-borne virus, just like hepatitis B. Except we didn't have documentation then that it had such a long incubation period.

Confidentiality Issues with Hepatitis B Study Participants

O'Malley: We had old logbooks of hepatitis B cases in gay men in SF. This was long before anything was computerized. We were working out of shoe boxes in those days. It was a very cumbersome task, but we went looking for hepatitis B study participants who matched cases of AIDS that were being reported.

Hughes: You did that on your own, or were there others involved?

O'Malley: No, I did this on my own. These men had entered a research study, and there are certain rules around the integrity of the research process--who should have access to names. I was the coordinator of the study and also worked for the health department, so they could justify letting me have access to the names of the AIDS cases. But I said, "It can't work the other way. I cannot be turning over these logs to non-research study staff in the health department. The consent forms that were signed are very clear. Not just anyone in the health department can see who's in these studies."

Hughes: Would that have been true of any sort of study you had done through the health department, or were these confidential issues because these were gay men?

O'Malley: Even before HIV there were concerns about discrimination and stigmatization. We had to be very careful about maintaining confidentiality in any research study, whether with gay men or not. If a consent form states limited access, you abide by those rules and regulations. Additionally, the gay community was already distrustful of government, just like other disenfranchised populations in our country.

Hughes: Was it rather a new thing to take a detailed sexual history?

O'Malley: Well, people came in with STDs and had to talk about their sex partners with clinicians and counselors. But yes, to interview people whose names you knew about a variety of sexual practices and illicit drug use and then to record their answers on a questionnaire, that was very new.

One of the reasons CDC came to San Francisco and proposed doing the hepatitis B study was that there was a long history of trust, in San Francisco, between the health department and the gay community. I'll use my own example. I went to get an STD check at the clinic during my first six months in San Francisco. This was before I worked there. I knew about the



clinic because Sid and J.B. told me. They said, "This is the place to go. The doctors are great; they're sensitive." It was a word-of-mouth thing that got around in the gay community to use the clinic.

So people, for the most part, were trusting. But there were always people that had concerns. How do I know where my name might end up sooner or later? People round up gays, whatever. The mid-seventies were post-Watergate and Nixon, followed by the CIA scandal, etc. There was still an enormous amount of distrust of government in the community at large, not just in the gay community.

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O'Malley: The hepatitis B vaccine was a plasma-derived vaccine; the manufacturers used the blood of hepatitis B carriers to make this vaccine. One fear voiced was that the [AIDS] virus may have been around longer than people realized; maybe even in the late seventies. Some people had this fear that the vaccine itself could potentially be contaminated with HIV. You couldn't get people even in the health care professions to take the vaccine once it was licensed. Now there is a genetically engineered vaccine where that just couldn't be the case.

Due to this concern, Merck, Sharpe, & Dohme, the hepatitis B manufacturer, put the blood of someone who was infected with both hepatitis B and HIV through the vaccine preparation process to prove that the virus could not survive the preparation process and contaminate the vaccine. This was an issue that had been addressed even before we knew about HIV, due to other infectious agents like typhoid fever. Part of the vaccine preparation process is to ensure you destroy any disease agent that could potentially be in a person's blood.

#### Gay Community Conspiracy Theories About AIDS

O'Malley: The distrust of government within the gay community lead to the articulations of many fears. Several gay newspapers and individuals started postulating that the hepatitis B vaccine

might have been a government plot to infect the community with HIV. They said, "Isn't this interesting? They did those hepatitis B vaccine trials in '78, '79 and '80 in New York and San Francisco. And then in '81 the AIDS epidemic begins." This rumor was completely shot down, because once we started testing old blood specimens, we could clearly show that people were infected with HIV in '78, '79, before the first gay men had received the hepatitis vaccination, and we clearly showed that the prevalence of HIV was actually less in the men in the vaccine trial than in the larger population. Men that had not been exposed to hepatitis B were more likely to have not been exposed to HIV.

### Speculation About Etiology

[Interview 2: July 9, 1996] ##

- Hughes: Mr. O'Malley, last time you told me that you first heard about what we later recognized as the AIDS epidemic at a meeting in San Diego in May of 1981?
- O'Malley: It was actually end of May, first week of June, yes.
- Hughes: Was there any assumption that this new disease might be sexually transmitted, or were there too few cases at that point to think about that?
- O'Malley: I think no one jumped to the conclusion initially that it was sexually transmitted, although in retrospect, why wouldn't it have been right there high on the list? Many people were afraid to verbalize their thoughts, because of what the broader implications might potentially be if it was an infectious agent.

But here are some of the things that legitimately steered people away from thinking it might be a sexually transmitted disease. We knew there had been this sexual revolution that had gone on throughout the seventies here in San Francisco. There wasn't just a handful of gay men that had been very sexually active; there had been large numbers of gay men that had been sexually active with large numbers of partners. And they were not getting sick. Additionally, the first question people would ask when they heard about a new case of AIDS was; Okay, well, John Doe has this rare cancer, or John Doe has this pneumonia. Well, if he has a steady partner and that partner is totally healthy, how could this be an infectious disease? If this is sexually transmitted then why aren't the partners sick? It was hard to argue that point, it was a good question.

Clearly, there was immune suppression, but maybe it was chemically induced or drug induced or some people are just more susceptible than others.

I think there may have been some researchers who right from the get-go might have been fearful that what we were witnessing was a disease that was sexually transmitted. At that meeting, I was thinking it might be something environmental.

At that time I thought it might be the cumulative effect of having multiple sexually transmitted diseases, and subsequent treatments, and that's why the most sexually active gays who had had the largest numbers of partners seemed to develop the syndrome in the beginning. But of course, now it all makes sense. When you have a new agent introduced into a community, it's going to be the ones who are the most sexually active that are going to cross paths with it first.

Hughes: Did you see cases in the clinic that you thought might be related to the new disease?

O'Malley: That happened six months later in was early '82. As I mentioned last time, an individual that I knew who had been in the hepatitis study had been diagnosed with Kaposi's sarcoma in November of '81. So he was one of that first handful of [AIDS] cases here in San Francisco.

One thing that helped motivate the CDC to do a research study in this hepatitis B group is we started collecting information on AIDS cases diagnosed in '81. I think by the end of '82, early '83, we realized that of the twenty-four cases that were reported with AIDS in 1981 in San Francisco, eleven of them were amongst men we had screened for this hepatitis B cohort study. If almost half the men diagnosed with AIDS in 1981 were screened by us, then clearly the hepatitis B was the place to start looking for a causative agent. It was probably going to be found in the blood of these men. Since the blood we had on these men was from '78, '79, and 1980, and this was now 1982, early '83, the question was whether we had serum close enough to the time that these men were diagnosed with AIDS.

I got a few calls from CDC like, "What do you think is going on?" They valued my opinion as to what might be happening.

Hughes: This conversation occurred amongst this dinner group after the San Diego meeting?

O'Malley: It was during the May-June 1981 San Diego STD meeting with Dr. Harold Jaffe and Dr. Bill Darrow from the Centers for Disease Control. There was a little something in the San Francisco Chronicle shortly thereafter about these cases.

CDC Involvement with the Gay Community

Hughes: Jaffe didn't say anything about whether the CDC had any plans to investigate this outbreak?

O'Malley: Well, yes, CDC had sent a CDC case officer to New York and L.A., but it was just like they would do with any new diseases. There wasn't any discussion about broader national responses. I mean, they had this little outbreak and it appeared to be only occurring in gay men. A lot of data had been collected on gay lifestyle; there was a lot of drug use; there were a lot of sex partners, et cetera. So there were clear ideas about what would probably need to be investigated.

Hughes: Was the CDC used to dealing with the gay community prior to the epidemic?

O'Malley: Yes. Bill Darrow is a good example. Bill Darrow had come to San Francisco, to L.A., and went to every one of the five sites selected for the hepatitis B study. He came to these cities in late '77 and spoke to me directly. He clearly was very familiar with the gay scene and knew people within the gay scene. I didn't get to know him really well until later when the epidemic became full-blown, when he would periodically visit our clinic in San Francisco.

CDC was providing federal grants to STD clinics all over the country. Additionally, there were federal assignees to most of these clinics.

Hughes: Who were the federal assignees?

O'Malley: They were public health advisors. During those years Dr. Braff, a local physician, was the titular head of the clinic, but there was also a man named Don Hawkins who was a federal assignee who really ran the place. He ran the place with an iron fist. The clinic was clearly run by the "feds" that were working there, the federal government assignees, public health advisors. Hawkins was there all through those years.

So CDC definitely had a presence which was felt at the clinic. Hawkins and his assistants knew all these people at CDC as well. He'd been an old-timer with the U.S. Public Health Service. CDC was well aware of what was going on in the gay community.

There was a tendency for CDC to be paternalistic or try to hide what was going on to some degree regarding what populations were generating the high STD rates. They didn't want front-page news stories about STDs in gay men. All this changed with the AIDS epidemic.

Hughes: What was the motive there?

O'Malley: I think there was concern about homophobia and public reaction. Just like people sometimes say today, "Why the hell should we spend all this money on AIDS? They're not innocent victims." I think there was a concern that if it became common knowledge to the average citizen in the United States of America that a lot of their tax dollars were spent running these STD programs around the country which were needed in part due to the high rates of STDs in gay men, there would be a homophobic reaction. So CDC would always try to hide the statistics if they could.

Hughes: Do you have any feeling about the government's motive? You could look at it in at least two ways: The government wants to keep the gay population healthy, because they're American citizens. Whatever rationale they wanted to use. Or, they could say, We want to keep the gay population healthy, because we don't want them infecting the rest of us, the larger population.

O'Malley: Well, I think you could say both. It was also partly that government creates their bureaucratic fiefdoms. Federal officials sometimes do not want anything to jeopardize the infrastructure they have created. People say this about AIDS now too. "What are you going to do if they find a cure tomorrow? That could put you out of work." So I think there was some of that going on as well with some of these federal bureaucrats. They weren't always motivated by great love or interest in taking care of gay men and making sure they stayed healthy or protecting the larger community.

The way the clinic's mission was put to the public was keeping STDs under control so that they don't explode into a larger and larger problem. I think they used to say that about TB and other diseases as well.

Hughes: So homophobia wasn't patently obvious.



O'Malley: No, I don't think it was patently obvious. God knows what many of them said behind closed doors about all this money being spent on gay men. It would not have reached my ears since everyone knew I was gay. I will say that I was always treated with respect by CDC staff and the federal assignees at City Clinic.

### Defining AIDS

Hughes: From what you told me last time, it sounded as though the immune overload theory was an old idea, that it wasn't developed specifically in the context of the AIDS epidemic.

O'Malley: Yes. When the first cases were being diagnosed, it seemed very logical that this was put on the platter as one of the possible answers. It made sense in connection with the diseases that gay men were getting--Kaposi's sarcoma was a known disease in older Jews and Mediterranean males, and all these other opportunistic infections. It all fell back on, Well, something's causing immune suppression, and immune overload obviously was a very logical way to explain it.

### Case Clusters

O'Malley: But then Bill Darrow's cluster study made it look clear that this new disease was probably caused by an infectious agent because of the clustering of cases. It was very serendipitous in a way that Bill Darrow was able to tie things together the way he did. We now realize that there were many gay men that were already infected. They blamed that Canadian airline steward in Randy Shilts' book<sup>1</sup>, Gaetan Dugas, for infecting everyone, but there were a lot of so called "patient zero"s by that time. If he was spreading virus, there were a lot of others doing the same thing at that point. He clearly was not Patient Zero or the first infection.

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<sup>1</sup> Randy Shilts. And the Band Played On: Politics, People, and the AIDS Epidemic. New York: Penguin Books, 1988.

Hughes: Are you saying that Darrow was lucky that he could actually link these cases, because it actually was much more complicated than it at that time appeared to be?

O'Malley: Yes. We now know people progress at such different rates that it was very lucky in those early years to be able to tie them together. Let's use a hypothetical example, twenty gay men go to a party on Fire Island in the summer of 1978 and have an orgy. Let's say they all got HIV-infected at that party. We now know that people progress to AIDS at varying rates after infection. But in the case I just described, only half of those men, 10, would probably become immune suppressed with AIDS ten years after their initial infection at that party.

#### AIDS as a Gay Disease

Hughes: Did you think of it initially as a gay disease?

O'Malley: Well, I guess you couldn't help but think of it initially as a gay disease, because gays were diagnosed with the first cases. At the dinner with Harold Jaffe, when I first heard about it, I heard that it was in gay men. It wasn't like I heard, Well, they've got some hemophiliacs and some transfusion recipients. It was, They've got these gay men in New York and L.A. So yes, it was being called gay cancer and gay pneumonia; you couldn't escape the association with gay men right off the bat.

Hughes: And you thought that was fair enough, to characterize the disease in that way?

O'Malley: Well, I didn't question it initially, because that seemed to be the case. Gay men were the first cases. Again, it wasn't like we were automatically thinking of a blood-borne agent at that point, especially in that first month or so after I heard about it. So as the data started trickling in about transfusion cases, it became much more logical to make the jump that maybe this is a blood-borne agent of some sort that's causing this and that the risk groups may be much broader in scope.

Hughes: But some members of the community objected to the names, particularly GRID [Gay-Related Immune Deficiency]. Because whether or not you thought that it was a disease limited to the gay community, the "in" thinking was that the name of the disease shouldn't indicate that.

O'Malley: I agree. Whether it turned out to be an infectious agent, or even if it was something about gay men's lifestyles, like immune overload from repeated treatments, then it was an environmental thing that technically could have happened to anybody if they'd had similar exposure.

I wasn't overly excited about the fact that we were calling it gay pneumonia. I could be in a social situation where someone said, "That guy over there I heard has the gay pneumonia." It just seemed so ridiculous that even within the community gay men were using the term amongst themselves.

But I all along thought, the main thing we've got to focus on is trying to get to the bottom of this disease and to do everything possible to make gay men feel comfortable about participating in getting to the answer. Distrust of government was something that was going to be a battle right from the get-go. There were fears that were voiced early on like, Could this be some kind of massive plot on the government's part to do in the gay community?

Hughes: Was that a prevalent suggestion?

O'Malley: Not right from the get-go, but as time passed it was. When you think of the seventies and everything that came out about Watergate, everybody was, Oh, my god, who knows might be going on? The Reagan administration's apparent lack of concern also helped to foster these feelings.

Hughes: It was an us-against-them attitude?

O'Malley: Well, in '77 there was the Anita Bryant affair. The gay community's eyes were opened to the potential of real discrimination. This was followed up by the Briggs initiative against gays as school teachers, and then Harvey Milk was assassinated at the end of '78. To think on top of all of that, at the same time this virus was taking hold in our community.

#### CDC Interest in the Hepatitis B Study

O'Malley: As I mentioned last time, in early 1982, I brought to the attention of CDC this AIDS case in one of the participants in the hepatitis B study. I told them that we had stored blood specimens and interview data on this man. Then I started cross-referencing to see if there were other men with AIDS that



were in our hepatitis study, and we found there were others and notified CDC.

In 1982 the only money provided by CDC was for hepatitis B vaccine trial follow up. My position was the only one funded at that point. It was a wrap-up activity of the vaccine trial. Luckily, I was there and I had this established connection with people at CDC--Don Francis, Bill Darrow, Harold Jaffe--they were calling me up, asking me for suggestions, advice, information as to what I thought was causing this disease.

For example, we had an unusually high number of men in the trial become hepatitis B carriers once they became infected with hepatitis B. The data on hepatitis B is about one in ten become carriers. If six months after the date of infection they're still showing positive but don't have hepatitis, then that's an indication they're probably going to be a carrier.

We started noticing that among these men, we had seen a higher number of carriers than usual. The question was, does it have something to do with gay lifestyle? Maybe their immune systems aren't functioning as well as they should, and maybe that's why some of them are having a harder time clearing the hepatitis virus than you would expect from the general population. But then again, is there something new happening in our midst here? These men are all "healthy"; they hadn't been diagnosed with AIDS, but why is it they're becoming carriers at a higher rate than you would expect.

We asked these carriers to come back for further evaluation. Everyone suspected that something was probably going on in this group, and possibly immune suppression. There were suspicions early on that HTLV-I, the leukemia virus, might be the causative agent. While NIH and the Pasteur Institute were doing their research on the causative agent for AIDS, they were actually using some of these specimens. These specimens were blinded. CDC, NIH and Pasteur did not have access to the names. We knew that whatever the disease agent was, it was going to be found in this group of men.

We operated without extra funding from CDC during 1982 and most of 1983 on the hepatitis B study's investigation of AIDS. And this is where the criticism comes, Why didn't the government jump faster? Why didn't they offer us money sooner?

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O'Malley: In all fairness to CDC, a CDC man did come to the clinic independent of the hepatitis B study to conduct a case control

study which is to look for men who were using clinic services who would match the profile of the [AIDS] cases.

Hughes: Did the CDC run the study using the City Clinic?

O'Malley: They worked with the federal assignees at the clinic. We would get involved in it in some cases, but generally by accident. I just got in with the CDC because I was working with the hepatitis group at the time. I didn't go out of my way to get involved. I did not need to--they came to me. If they wanted to conduct research on the hepatitis B study they had to work with me. There were tensions. We used to always feel that the feds had too much power at the clinic. A lot of the tension had to do with Don Hawkins. There were constant personnel problems in those days between local and federal staff. I take a lot of credit for being a good supervisor, and I say the only reason I'm a good supervisor is when conflicts come up, I think about how conflict was handled in those days, and I always do the opposite--180 degrees, and it always seems to work out better!

#### First AIDS Study Using Hepatitis B Samples

O'Malley: During '83, we were offered \$100,000 [by the CDC] to go after approximately a 10 percent weighted random sample of the 6,700 men that we had screened for hepatitis B. I was working very closely with Bill Darrow at this point. I think the CDC first wanted to see if we could attain our objectives. In retrospect, they should have taken chances and thrown even more money at us. We could have tried to reach a larger percentage of the 6,700 men than we did in 1984.

But the reason I say it was a weighted sample is because we interviewed the first 800 men that had been enrolled in the hepatitis B study. They had had blood drawn and were interviewed about risk factors before we even knew whether they'd been exposed to hepatitis B or not. After four months, as I told you last week, we discovered we were finding too many men who had already been exposed to hepatitis B, and they wouldn't have been eligible for a vaccine trial. At that point we changed the rules and then screened everybody first. Those who had a negative test result for hepatitis, we'd call back and then interview and follow them every four months. We changed this protocol in May of '78 and we clearly were moving away from the riskiest people at that point, because we were trying to get only men who were seronegative for hepatitis B.

The thing that was really valuable about those first four months of '78 and those 800 people we interviewed is that we clearly interviewed a lot of very high-risk people who were either exposed or about to get exposed to HIV. We had this weighted sample, and in that first year we were able to locate about 80 percent of these men. In general, people were willing to participate.

So back to this question you asked a little while ago, What was the feeling in the community? Were people nervous about participating? There was some nervousness, but in general, people were much more likely than not to participate. And that had a lot to do with the clinic's reputation in the gay community. I had worked in the clinic since '73, so I was a known commodity as well to a lot of these guys. They knew I was a gay man, there was a lot of gay presence in the clinic, [gay] doctors, nurses, clerks, counselors, so people felt comfortable participating.

Then between '84 and '85, CDC threw a little more money at us and said, "Okay, go after another 10 percent weighted random sample of the 6,700. Additionally, anyone diagnosed with AIDS would be enrolled in the study.

Between year one ['83-'84], and year two ['84-'85], the development of the HIV antibody test occurred with the announcement of isolation of the HIV virus. This was considered great news, We've got a causative agent! Maybe we'll have vaccines, treatments, a few years down the line.

#### Paranoia

O'Malley: Then there was a sea change within just a few months in the attitude of the community regarding this scientific advancement. The implications surrounding the taking of this test, and how it could be stigmatizing and discriminating. Some gay men soon felt that you've got to be out of your mind if you would take the test in anything other than an anonymous setting. There were a lot of fears like, What if this disease really does get out of control and really does start spreading into the general population? There might be roundups. They may want to lock everybody up that's HIV-positive. So there were a lot of reasons why people were hesitant about getting tested for HIV in a non-anonymous setting.

Additionally, nobody really knew what exposure to the virus meant. You can find out you've been exposed to this virus, but then what do you do? Sit around and wait to get sick? Am I going to die tomorrow? There weren't treatment options then.

There was an enormous change in attitude. We had had people more than willing to participate, and all of a sudden most people just said, "No way, I'm sorry. I won't give you permission." They'd say, "You have this stored blood on me?" Some didn't believe that we were actually seeking their permission to test their blood samples for HIV, they assumed we had already tested all those stored samples anyway, and that we were actually calling to see if they were alive, sick, or just to keep track of them.

It was hard, because it was very clear that from a purely scientific standpoint it would have been convenient to be able to test those 6,700 samples. Instead of trying to find 6,700 people, you'd want to try to find the ones that showed up positive and see what was going on in their lives healthwise. I thought we would lose in the long run if we did operate that way. Although a lot of information could have been gained quickly, there would have been a bad taste left in everyone's mouth because of the method's we'd used. Bottom line, the end does not justify the means.

As it was, it was horrible. A lot of people were very upset. They were paranoid about the fact that we even had the stored serum. Like, Oh, isn't this interesting, that the year the virus first starts showing up is the year you started saving these specimens? So a lot of people thought, Oh, this is just a little too much of a coincidence. So there were justifiable reasons for paranoia.

Some people have said that even the hepatitis study was all a big front for something else that was going on on an even larger level than we realized. After all my years of working with the government, I'd never really given much credence to that idea because the government's just not that intelligent and organized. I really do think it was serendipity that we were doing the hepatitis B trials at the same time that the AIDS epidemic broke.

Hughes: As I remember, there were five--or was it six?--centers for the hepatitis study?

O'Malley: Well, there were six, counting the New York Blood Center, which wasn't really part of the CDC group.

Hughes: Why wasn't New York part of the CDC group?

O'Malley: Well, the New York Blood Center did the first hepatitis B vaccine trial. They were ahead of us. I think the FDA wanted two trials conducted, and the New York Blood Center was seen as one trial. They started in '79, and then there were the five multicenters, which started in 1980, a year later. So yes, from the outside looking in, there were six sites.

#### Primacy of the San Francisco Database

Hughes: Why, in terms of the AIDS epidemic, was it San Francisco where the valuable information was gathered from those sera?

O'Malley: That's a good question. Partly because our records were maintained. The L.A. Community Health Center shut down--they didn't have any infrastructure in place to even try to relocate all these guys. I think their logs were destroyed when the center closed. So to this day, there's no way of matching people with those blood specimens in Los Angeles. I think that was the problem with the New York Blood Center as well--destruction of records once the trial was completed.

It could have happened here too. The original master logs are in our safe. I've looked at them; they're just torn and tattered. They're priceless when I think of all the research those logs have given to us over the years.

But the fact that we had these old logs, plus we had clinic records on most of the 6,700 men, I think was why the CDC realized San Francisco had the best shot at reaching a significant portion of these men.

Hughes: Did the other centers have log books?

O'Malley: Yes, Chicago and Denver. I think of what could have been done if more money had been made available for Denver and Chicago in 1983 for contacting former hepatitis B participants. CDC obviously didn't have enough money to provide Chicago, Denver and San Francisco all at the same time, they only had money for us.

Also, they had all these stored specimens at CDC that had not been catalogued. We're talking about 6,700 men who had provided 15,000-something specimens. One of the things we were up against right at the beginning, once things really started



moving along and we started getting people in and asking permission to pull their blood, is it became very clear that we needed to hire people at CDC to go into these freezers and get these samples organized so that the specimens could be easily located. It took them almost a year to get all these specimens catalogued. I think CDC just didn't have the money to invest in all these necessary tasks in the early 1980s to get this study off the ground at both CDC and multiple clinic sites.

Hughes: Where were the serum samples located?

O'Malley: They initially had been sent to Phoenix, Arizona, the location of the CDC hepatitis branch during the 1970s. They moved to Atlanta<sup>1</sup> in the early 1980s. By the time specimens were being pulled for this study, they were all stored in Atlanta, Georgia.

Hughes: When you needed to test them the CDC did that?

O'Malley: Yes. We had a whole mechanism set up through our computer with the code numbers. CDC never had access to the names of the hepatitis B study participants. We were the only ones that had the connection with the name and code number. Once they had all the specimens catalogued it was a weekly activity: we sent off a list of specimens that had to be pulled, and they would send us the test results.

There were 800 individual specimens that they had tested for HIV antibody by 1985. I received all the results of all these tests all at one time in 1985. This is, by the way, where this story becomes much more interesting (and upsetting I might add) and why it became so clear how valuable this study was.

Two things became clear in late '84, early '85, when CDC started providing test results on these specimens. We were able to document when the virus entered the community. We could see that about 4 percent of the specimens from '78 were positive, 12 percent from '79, and then it jumps up to 20, 25 percent by 1980. So there was conclusive proof. It becomes clear that at least three or more years before the first case was diagnosed, the virus was present in San Francisco. Obviously there was a long incubation period.

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<sup>1</sup> For more on this episode, see the oral history with Donald Francis in the AIDS physicians oral history series.

AIDS as a Disease Continuum

O'Malley: Additionally, some of these guys who were HIV antibody positive since '78, '79, '80 were walking around in totally perfect health. People were obviously progressing at different rates. A few years later we made the association between duration of HIV infection and the risk of developing immune suppression. Initially, we thought AIDS might be like some other diseases. AIDS was an endpoint for some people, but lymphadenopathy or another symptom, like hairy leukoplakia or what was then called ARC [AIDS-related complex] was an endpoint for others.

Sadly, no symptom was an endpoint. We now know there is a continuum that runs at different rates for different individuals but that ultimately leads to AIDS for most HIV infected individuals if treatment is not provided. By '85-'86 when we had these results, it was seven years from '78, six years from '79. We knew some of these guys had been infected for five, six, and seven years and were healthy. There was a feeling of hope: Well, if something's going to happen, why hasn't it happened by now?

The government started to spend more money on the study. After those first two years of going after two percent random samples, CDC basically said, "Hire as many people as it takes to locate these 6,700 people." They wanted us to increase the samples of men that were what we called "well-characterized" HIV positive. These were study participants whom we could pinpoint within one year as to when they became HIV infected.

Confidentiality Issues**The Hepatitis B Study**

- Hughes: The AIDS study followed very closely on the hepatitis study. In fact, there's an overlap.
- O'Malley: Yes.
- Hughes: Say there had been ten years in between, would the hepatitis serum samples still have been available?



O'Malley: Well, the samples would have been available. The question would have been whether the logbooks would have been there. For example, if I had decided to leave I would have been a little bit uncomfortable about those logbooks being left behind. I might have recommended they be destroyed. There's only a certain number of years you are required to hold onto these documents.

Hughes: Why would you have destroyed the hepatitis B logbooks?

O'Malley: Well, confidentiality concerns. I just wanted to make sure that these individuals' confidentiality was respected. Their names, addresses, and phone numbers were in this logbook and the code number which linked them with their serum specimens, test results and sexual and drug histories.

Hughes: That's what I thought you meant, but then you turned it to, Well, scientifically, this information isn't of much interest any more, why hang onto it?

O'Malley: Well, only partly. It was clear that you wanted to hold onto information from the group in the vaccine trial. I had been told right from day one by vaccinologists, "You never know, five years, ten years from now, we may need to get hold of these people to find out whether they're still immune to hepatitis or if there's any longterm side effect." So I knew the information on those individuals was important, but only 359 out of 6,700 were in the vaccine trial. I could have destroyed the other logs. Of course, now I think no one will ever throw anything away ever again.

#### Selma Dritz and Randy Shilts

Hughes: Selma Dritz<sup>1</sup> told me that some of her notebooks were destroyed when she retired. She didn't know who had done it. Do you have any more information?

O'Malley: This is purely speculation, but I think there may have been some concerns that some of the stuff she collected had served their purpose and was extremely personal. The health department believed it was no longer important to hold on to this information.

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<sup>1</sup> See the oral history with Selma Dritz, M.D., in the AIDS physicians oral history series.

There was some concern as to how Randy Shilts knew some of the information he wrote in his book. He was someone who lived here in the community, and he wasn't like an outsider coming in here; he was very savvy at what he did. I don't think Selma may have realized that he was able to ask questions and put two and two together sometimes, because he was so familiar with what was going on in the community. I don't really know who destroyed her books. She might be really irritated to hear this, but I would rather see health information like this destroyed if it is no longer relevant to investigating this epidemic.

Hughes: What about the use of specific names, which of course Shilts does all the way through his book? It's a very personal story.

O'Malley: That's because he interviewed a lot of those people himself. The concern always was, How did he get confirmation on some of this information? Usually if you're a good reporter, you try to get a second and third confirmation of what somebody is saying.

#### Health Department Policy

Hughes: Has confidentiality and perhaps the resulting methodology that grows up around those concerns escalated because of the epidemic, or has the health department always been very concerned about it?

O'Malley: The health department has always been very concerned about confidentiality. We realize a lot of disenfranchised people have reasons to fear that information about their sex lives, or diseases they've had, or drugs they've taken, will be used against them. So I always felt that we had to bend over backwards to protect them. Confidentiality is paramount, even during so-called "worst-case" scenarios. For example, you get a phone call from the police department, and they've arrested someone because they've molested a three-year-old. The police have found their clinic card that says they're in our hepatitis study, and they want to know whether somebody's infected with HIV or not. I'd ask, "How are you going to deal with something like that?" It's an interesting moral dilemma but we the health department would always choose confidentiality.

### Effect of the LaRouche Initiative

Hughes: So you have more or less a flat policy not to release personal data?

O'Malley: Yes. Especially since the the LaRouche initiative and Proposition 103.

There were times where we were really concerned that we were going to be stuck with this law saying, "You've got to report HIV-positive status on your study participants." We thought out how we would fight it out in the courts. I used to think about these logbooks. What am I going to do if the elevator door opens up some day and the police say, "We're going to confiscate your records." Am I going to burn them on the spot? I used to think about how we would hide them; I didn't want them to get into the wrong hands. I was also aware what a terrible loss to science it would have been if we had destroyed this information.

Hughes: Had you always realized what a scientific gold mine they were?

O'Malley: Oh, yes. It was very obvious because I realized L.A., which was another early center of the epidemic, had all these blood specimens and they had no way of linking them to the study participants because their logs had been destroyed when the clinic closed.

Hughes: You had the serum samples on one hand, and you had a logbook with the names on the other.

O'Malley: Yes, and the interview data.

### Finding the Participants in the Hepatitis B Studies

Hughes: Obviously some of these people were still here in San Francisco, and you could find them. How did you find people that had moved away?

O'Malley: We would start with the clinic registry, which we had access to. The first thing we'd do is see how many of them had visited the clinic recently, to see if we could get and updated address and phone number on them. So those were the easy ones. We'd just call the number, and there were a certain percentage of people that we found that way in the city.

Then we used the voter registry. Everybody has access to the voter registry. And we had birth dates on a significant number. One of the flaws we discovered was that we didn't have everybody's birthday. But in any case, we were able to find an awful lot of people through the voter registry.

We'd send address requests to the DMV [Department of Motor Vehicles]. The DMV occasionally had information besides address updates. For example, families will sometimes let the DMV know when a family member dies, so we did find occasionally that some men were deceased. Additionally, if someone goes to another state and registers with their DMV, the California DMV was often notified by the state. We'd get a clue to where they went. For example, the form would say, "Moved to Florida." The DMV no longer provides this information to the Health Department.

Also, being part of this community, I knew where gay men were moving. I knew many gay men were moving to Los Angeles, San Diego, New York and Miami. So we would look for names in these cities. If your name was John Smith, that wouldn't help, but if your name was somewhat unusual then you might be the only one there with that name.

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O'Malley: Technically, you probably should notify a local jurisdiction health department when you're calling somebody about something like this. But we thought, There's no way we could make such a call and maintain confidentiality, especially if someone lives in a small town. We feared that if the word got around town that we were investigating someone regarding an HIV study stigmatization could occur. So a lot of times, I would just call Information. I had all these skills from working at the STD clinic for trying to locate people without breaching confidentiality, so I knew how to call people out of the blue and ask questions. You try to find out if you've got the right person without breaching confidentiality.

Hughes: Can you give me an example of how a conversation might go?

O'Malley: Well, luckily, the medical charts sometimes would have just enough information so I could identify a person. We would say we were a friend, or trying to find the person for a reunion or whatever. I took classes with Joe So-and-so that used to live in San Francisco, and I'm trying to find him. If you got the right person on the line he'd say, "Oh, yes, that's my brother," or, "That's right. He used to live in San Francisco."

Hughes: When you did find the right person, what in general was the reaction?

O'Malley: In general, it was amazing. People who had lived in San Francisco saw it as an island of safety. So gay men never reacted like, "How the hell did you find me," or, "My god, how did you track me down?" to a call from San Francisco. Once I described who I was and what I was doing they generally wanted to help.

People here in the city would get more paranoid. Locally you had activists and everybody screaming from the rooftops to watch out for this, watch out for that, they're gonna round us all up. It was the reverse of what you'd think it would be; there was more paranoia locally than there was out there in the boondocks about the San Francisco Health Department and governments sponsored HIV studies.

Hughes: How did you present yourself?

O'Malley: Well, I learned how to do this: it was to not come across like the bill collector or whatever, and to be as relaxed as possible. I used to say, "Now, Paul, think of it as trying to find somebody you went to college with twenty years ago. Think of it that way. Don't set off alarm bells in people."

Hughes: Did you make it clear that you were gay?

O'Malley: I wouldn't automatically preface everything by saying, "I'm gay," but I think in the course of the conversation, it probably was apparent. People would say, "Oh, god, this horrible disease; I lost somebody." I would let them know that yes, I understand what you're going through; I've been there myself. Sometimes they would remember me from the clinic and seeing me out and about in the gay community. I lived in the Castro district.

Hughes: I read that, despite all this, there was about 29 percent of those 6,000 whom you could not locate. Does that sound about right?

O'Malley: Yes. We've actually located and tested about two-thirds of the men. There were also flat-out refusals in addition to the untraceable. There were people that we just reached a total dead end on. Not surprising, when you think of the number of years that went by before we started this search. It wasn't until '89 that we completed our attempt to reach every one of those men. We tried to locate 10 percent of the sample in '84, another 10 percent in '85, and the remaining 80 percent between 1986 and 1989.

Hughes: Why the 10 percent limitation?



O'Malley: Well, I think there were two things. There were the budgetary restraints and CDC also felt that we only needed to locate a ten percent sample. If we could get a representative ten percent sample, they figured, then we make some generalized statements about the entire sample.

#### Usefulness of the Hepatitis B Study Data

O'Malley: But that changed in '85. In '85, CDC finally realized that they were sitting on a gold mine here with these specimens and the data that we were uncovering, so they sent Dr. George Rutherford, who was an M.D. epidemiologist. He was followed by Dr. Alan Lifson from CDC, and we then had the expertise we needed. They were very competent and knowledgeable and sensitive to the needs of the community. But from '81 to '85, for four years, we lacked the local support we required.

It took a while to realize fully the significance of this data. The reason CDC now wanted to locate everybody was to develop a significant sample of men who were "well characterized" positives. The public health implications were, Okay, we have 500, 600, 700 men about whom we can say, "This is when they got infected, and this percentage progressed after X number of years to AIDS." They started doing similar studies in hemophiliacs. You could get some sense of what was going to happen as this epidemic progressed. The demands on the public health would be considerable if all HIV infected individuals were eventually going to progress to AIDS.

So in '86-'87 the first projections were made about the spread of AIDS, based in part on our study. There was an enormous amount of publicity around the Kaplan-Meier progression curve, which estimates the progression rates to AIDS. It was our study that first came out with the projections that roughly 50 percent of the HIV infected would progress to AIDS in ten years.

Hughes: Before Dr. Rutherford and Dr. Lifson were assigned here, how close were your ties with the CDC, and under what circumstances were you contacting them or vice versa?

O'Malley: Oh, there was a lot of contact. Bill Darrow visited periodically, and I was talking to Don Francis all the time. Dr. Braff and Dr. Dritz were focused on other issues. Keeping track of cases was a full-time job. They did not have time to focus on this study. Most of the attention I received was from



CDC. I did not have local expertise available. They also knew I was self-motivated, but I think they were just overwhelmed with everything that was going on here in the community, setting up all the service organizations that were required.

Hughes: That wasn't specifically your responsibility?

O'Malley: No, it wasn't. That's another whole part of the response of the health department to the epidemic.

Hughes: Did you see it as part of your job to educate the gay community?

O'Malley: Yes, but that's another whole story. There were people that were assigned to deal with education. We would pass information around to them. I remember feeling like I was working morning, noon, and night just dealing with the research end and also dealing with the disease in my personal life. In retrospect, though, I wish I had screamed from the rooftops a little more about all the implications for the gay community regarding this burgeoning epidemic.

#### Reluctance to Talk About Gay Sex Practices

O'Malley: There was a lot of hesitation to get up and publicly say, "We think receptive anal sex is the primary risk factor for HIV transmission." There was such concern that everyone was going to say, "Well, of course the health officials are going to say that, because they're being judgmental, moralistic, anti-gay and all that, and of course they're going to automatically assume that's the major risk factor. And here we are as a community finally starting to feel comfortable about our sexuality and about engaging in these practices, and all of a sudden someone's saying, 'No. Don't. Sex has to be protected, or don't do it at all.'"

So there was a lot of hesitation, even though some of the early signs were that receptive anal sex was probably a major risk factor. We didn't really get up there and scream and holler about it, because of the feeling that there was going to be negative feedback and if we were wrong we would lose all credibility and leave ourselves open to charges of homophobia and raising unnecessary fears.

Hughes: It seems to me that those very concerns of yours are epitomized in the bathhouse episode, which polarized the gay community.

O'Malley: Yes, and it's funny, because you talk to some people today who were adamantly against closing the baths at the time but will now say, "Well, I guess it was the right thing to do at that time."

Hughes: Did you have any sentiment one way or another about the bathhouses?

O'Malley: I didn't get drawn into it. I remember feeling, because I was dealing with research, I am not going to take a public stance and become controversial. I thought it might jeopardize study participation. There were men on both sides of this issue participating in the study. I felt that I had to keep my sights on the fact that there was so much to be gained from this study and getting the cooperation of the gay men. I would also like to say more about the struggle it was for gay men when trying to decide to participate in a study which required testing without anonymity. They worried about losing their civil liberties in the long run if they chose to participate, and about losing their lives if they chose not to participate in these studies. I could sympathize completely with gay men who said, "I'm sorry, I'm just not going to take the risk that information I provide you could come back to haunt me later." Luckily there were many men who finally said, "Maybe that's a possibility, but I also feel that this disease may do us all in, so I am going to take that risk." I wanted to try to provide the best environment possible so people felt comfortable making that decision. I felt that there were a lot of people that were not going to participate. People were saying, "Don't get tested, and even if you get tested as part of research, don't get your result, because who knows what it means."

But I also thought, The only way that this community is really going to deal with these issues and push treatment research was by having a groundswell of participation in research. Men were ultimately motivated by the fact that their friends and lovers were getting sick, and the possibility that they were positive. Once gay men dealt with their fears and came forward, there would be a groundswell of activity on numerous fronts--political, scientific, education and healthcare. And that's indeed what finally played out.

Andrew Moss's Epidemiological Studies

Hughes: Well, a name that we haven't mentioned yet is that of Andrew Moss,<sup>1</sup> who as you well know was doing epidemiological studies, and ran into turf problems in the early part of the epidemic. I found a letter, which I'll show you.<sup>2</sup>

O'Malley: [reading letter] This is really mind-boggling. I can't believe that I never knew that Andrew was supposed to have access to this hepatitis screening cohort. I can't believe he never told me this either. I knew he was interested in the cohort and would have liked to have access, but I never knew that it was formalized in writing. I mean, I worked with him in '82.

In '82 Andrew started up a study at General in which was interviewing cases and partners of cases, and there were people that they found coming through Ward 86 the AIDS Clinic at San Francisco General Hospital. Martha Rodriguez, who I worked with at the time in the hepatitis study, and I, volunteered, and we went up and started interviewing cases, because Andrew needed help and we had interviewing skills.

But this letter is really interesting. [reads] I know one thing: the confidentiality of the hepatitis study data would have been a roadblock. The question of who should have access to these participants' names was a question within the health department, and Andrew was not a health department employee. I told them, "I think there's justification for me having access to the names of the cases because I work for the health department. But I don't think the people doing case reporting should have access to our lists of names. They're not employed by the research study, and the consent form is very clear that only people on the research staff have permission to have access to their names."

The Health Department AIDS Surveillance Unit was annoyed because I wouldn't even tell them who was in the cohort and who wasn't. They asked me, "At least when you do get a match, let us know that they're in the cohort." And I said, "Well, the

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<sup>1</sup> See the oral history with Andrew Moss in the AIDS physicians series.

<sup>2</sup> Andrew R. Moss, Ph.D. to Mervyn Silverman, M.D., April 25, 1984 (Ward 86, Carton 5, folder: research studies in or related to San Francisco 1984 AIDS History Project, UCSF Library). This letter is reproduced in the appendix.

only way that will happen is if I can get in touch with these people and get permission to let them know that this is information that we would like for research purposes."

I know there were personality problems between Andrew and people at CDC, and I know there were some tensions between Bill Darrow and Andrew at one point. I can tell you right now that I would have been a roadblock, although I wasn't aware of Moss's request for access. It had nothing to do with my opinion of Andrew. He was well respected, competent and ethical, but Andrew, again, was not an employee of the City and County of San Francisco.

I'm surprised Andrew didn't try to head this off by coming to me directly. This letter also helps me understand a little better why he was irritated with Harold Jaffe and Bill Darrow.

Hughes: How was Moss's request for access resolved?

O'Malley: Well, he never had access to the hepatitis B cohort. It never occurred. He was working on the General Hospital cohort. There was also the San Francisco Men's Health Study directed by Warren Winkelstein. There were three HIV-related studies in gay men at that time. I do not think AIDS research suffered either locally or nationally due to the restrictions that were placed on accessibility to data by the health department.

##

Hughes: As you see in that letter, Moss protests the CDC's plan to start a study of sexual partners of AIDS cases.

O'Malley: Well, the first year of the study we did the 10 percent random sample. The second year, there was talk about recruiting a new, younger cohort, looking at sex partners of study participants, another 10 percent sample, following 50 percent of the guys that we had enrolled in the first year of the study. But the sex partner study was thrown in there with everything else, and it never really got off the ground. Partly because we knew that Andrew Moss was doing something similar, I think a lot of us felt that there were other things that made more sense using this cohort. This cohort had very specific value. You could do interviews of sex partners of AIDS cases with any group of AIDS cases. What we had, of course, was the history of the stored samples. I understand Andrew's position on this matter.

[continuing to read letter] Some of these personalities aren't around any longer. Dean Echenberg was hired by the health

department to replace Selma Dritz in the Bureau of Communicable Disease Control. He did not fit well in this position.

Hughes: Why?

O'Malley: The position required working with numerous other AIDS researchers on the the local and national level. Tensions developed with several of these researchers and he finally decided to vacate his position.

For example, CDC's Dr. Bill Darrow and Echenberg locked horns. Dean wanted to get much more involved in all that was going on, where Dr. Braff hadn't really paid any attention on a day-to-day basis. CDC had developed a relationship with me over four years, and Dean was seen as interfering. In retrospect, this was somewhat unfair to Dean because he really was my boss and had a right to be involved.

#### Shift of AIDS Services from UCSF to SFGH

Hughes: In the very earliest years of the epidemic, it seems as though the center of power was at the university, first at UCSF because of the KS Clinic--

O'Malley: Yes, Dr. Conant and Paul Volberding.

Hughes: And then it began to shift. For complicated reasons, the major effort of the university moved over to the General. Where do you see the center of power in AIDS medicine lying from about 1983 on?

O'Malley: I know very little on this subject other than that Jay Levy, Paul Volberding, Marcus Conant, were the most prominent M.D. researchers. They were experimenting with the treatments, and that was an enormous focus of what was happening with AIDS at that point in time. At the end of '86, of course, there was AZT, but even long before AZT there was progress occurring with treatment of opportunistic infections. UCSF was the center of power, and you're right, Andrew Moss worked right there alongside Donald Abrams and Paul Volberding.

Hughes: How did the health department feel about that UC's prominence in AIDS medicine?

O'Malley: There was definitely some competition there. Our roles were very different. I was not involved and not concerned. I



thought the health department role and the university's role were distinctly different and should be non-competitive. The AIDS Activity Office at the health department was run by Jeff Amory and Michael Bala. They oversaw the provision of health services and HIV prevention education. Mike Bala is the only one around now who can speak from the perspective of the health department's initial response. He took care of all the contracts that had to be set up for service organizations here in the city to take care of the needs of people that had AIDS.

Our study was housed at the City Clinic from '81 to '85, and then, due to growth, there was no longer enough space at the clinic and we rented offices on Market Street in '85. We moved into this building here at 25 Van Ness in '89.

Hughes: This building is?

O'Malley: Well, the AIDS Office. All the health department AIDS related operations are now conducted in these offices.

#### Multiple Uses of the Hepatitis B Cohort Data

O'Malley: In retrospect I think there was definitely competition between CDC and the local researchers concerning the hepatitis B study. I think CDC saw this study as their baby in a lot of ways; they have reaped a lot of favorable publicity due to this study over the years. It's continued to be a source of pride for them. Susan Buchbinder is at the International AIDS Conference in Japan right now giving an update on our study participants, and people are always interested in the latest on this cohort.

Fast-forwarding a little bit, in '89-'90 the focus changed again here. We were still interested in progression rates, but then what became interesting was a subset of men that apparently weren't progressing to AIDS. This has been the main focus of this study for the past ten years and that's where most of the recent media attention is focused.

Hughes: The AZT study used the same group of men, right?



O'Malley: Well only the AZT-acyclovir study in 1987. It was a very small phase I study. It involved twenty study participants. They had to be asymptomatic. It was a Burroughs-Wellcome sponsored study. They had to be asymptomatic and have T cells above 500. The idea of testing this drug in people that were healthy positives was new. It also had broad implications for Burroughs-Wellcome. If all of a sudden there was this much larger population which might be accessing drugs. But that's another story.

The Health Department's HIV Vaccine Preparedness Study, 1993-1996

Hughes: Do you want to briefly say something about the vaccine study that you're working with now?

O'Malley: Yes. Just prior to the vaccine research study, my lover, Doug Franks, died. Additionally, the office, in '89, '90, '91 had also lost several staff members to HIV. We lost several of the interview staff; we lost doctors, nurses. It was just an awful time and we needed a boost.

Luckily, the boost was when CDC approached us about doing an HIV vaccine preparedness study, and they approached Denver and Chicago as well. Denver and Chicago, of course, were also sites where the hepatitis B study had been conducted, and they did do some HIV studies in those cities as well during the eighties. Everybody always asks, "Well, what do you mean by an HIV vaccine preparedness study?" Actually, it was very similar to why we did the hepatitis B vaccine preparedness study: we needed to get some information on prevalence and incidence of HIV in the community, updated, well-documented information on HIV-seronegative men and what the risk factors for HIV transmission were. Then we could develop a good screen for people that would be eligible for a vaccine trial and obtain a population of men who potentially could be enrolled in a vaccine trial if a vaccine product came along that looked promising. So the idea was to set up an infrastructure now, so that if a vaccine comes down the pike, you've got an infrastructure in place to go forward. So that was part of it.

Another factor, which is very different from the hepatitis B vaccine trial, was the complications around conducting HIV vaccine trials. The concerns people have about vaccine-induced seropositivity, was a new concern and the stigmatization around that term, HIV-positive, whether due to infection or vaccine.

Then people were concerned about the vaccine itself. Would it cause HIV infection. Or if it didn't cause HIV infection, would it cause immune irregularities? There were issues of enhancement, that is the concern that if you get vaccinated and you have a breakthrough infection, are you then susceptible to progressing more rapidly to AIDS than if you hadn't been vaccinated at all? There are a lot of serious scientific questions.

Additionally, we don't know the correlates of immunity. We're not sure exactly what a vaccine is supposed to do, immunologically, to provide protection. With hepatitis B, you could always look at the immune profile of someone who cleared hepatitis B infection and say, "Okay, this is what we want the vaccine to do. We want them to produce these markers in their blood. Then we'll know we've got a vaccine that will be efficacious." We don't have that with HIV, because we don't have a cured individual. We have healthy longterm positives, and we do do immune profiles of those guys, because that's part of solving the mystery for creating an effective vaccine and therapies for that matter. So I think this is a wonderful area to be working in now, because it's an area of hope.

Just this week with all these news stories coming out about the International Conference on AIDS, they talk about advances in therapies and how things have moved along with education and that even condom usage is up in some of the developed world. But it always ends, "Ultimately, we're going to need a vaccine." They're talking about technology now where you might even be able to put a vaccine in a banana.

And that's the other thing: even with a vaccine, which is a lot cheaper than therapies, how are developing countries going to afford them? The distribution of the vaccine is a problem, even if you could reduce the cost.

This is why we need to conduct this HIV vaccine preparedness study. We still find people getting infected, and we're trying to nail down, with education and all the information that's out there, why people are still putting themselves at risk. It's very complicated. It's self-esteem and drug use and a variety of things. For all these reasons we need a vaccine and continued HIV prevention education.

Current Collaborative AIDS Studies

Hughes: I was wondering, Paul, about the tie-in with the longterm survivors of HIV infection and Jay Levy's virological work on the same subject.

O'Malley: Jay Levy is one of many of our research collaborators. These collaborations have developed over the last five or six years, as it became more and more apparent that we had these unusual individuals that weren't progressing. Jay in particular is very interested in the CD8 cells, the suppressor cells, and the fact that healthy longterm positives tend to maintain very high levels of CD8's. We send blood samples to him on these select participants. We also refer individuals to UCSF directly and they are involved in studies there independent of what we're doing.

We're also doing collaborations with the National Institutes of Health. We've looked at genetic factors that may play a role in non-progression. We're also sending blood specimens to Massachusetts General Hospital. Those researchers are looking at white blood cells known as cytotoxic lymphocytes, which may be controlling viral replication. We have a collaboration with the Gladstone Institute for Virological Research at San Francisco General Hospital. Mark Fineberg is looking at the virus itself as a factor in explaining non-progression.

We have probably about twelve collaborators at this point. A big staff responsibility here is the coordination of blood specimen shipment around the country to these various research institutions. It's a major undertaking, believe me, in dealing with FedEx and the hazardous material regulations. We're lucky that we have prominent researchers like Jay Levy and Mark Fineburg in our own community.

Hughes: Well, thank you.

Regional Oral History Office  
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Berkeley, California

The San Francisco AIDS Oral History Series

THE AIDS EPIDEMIC IN SAN FRANCISCO: THE RESPONSE OF COMMUNITY PHYSICIANS,  
1981-1984  
Volume II

Stephen Follansbee, M.D.  
INFECTIOUS DISEASE PRACTITIONER IN THE EARLY AIDS EPIDEMIC

Interviews Conducted by  
Sally Smith Hughes, Ph.D.  
in 1996







Stephen Follansbee, M.D. and daughter Stephanie Rose, December 25, 1995.





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## INTERVIEW HISTORY--Stephen E. Follansbee, M.D.

Stephen E. Follansbee was interviewed for the AIDS oral history series because of his role as infectious disease physician in the AIDS epidemic, from its earliest recognition up through the present day. Because of his ties with the University of California, first as a trainee in infectious disease and later in his association with the AIDS Clinic at San Francisco General Hospital, he served when he set up a private medical practice as a link between academic medicine and the private practice community. Contrary to what one might think, the flow of information, as he explains in the interviews, was not one way--from the university to the community. Rather, it flowed in both directions, with private practitioners providing crucial information about treatment in a real-world setting.

Follansbee's story, largely that of a physician in the trenches attempting to deal with AIDS patients with unexplained infections, provides fresh insight into early treatment dilemmas in respect to the new syndrome. He comments extensively on diagnosing and managing AIDS-related infections, a primary focus of his private medical practice at Davies Medical Center in San Francisco. As an infectious disease practitioner, he was fascinated--and horrified--by the infections his patients manifested:

...I think discovering new infections is to some extent what keeps AIDS doctors humble but also excited about keeping our eyes open--every problem is a potentially new problem.

Like most of the private practitioners interviewed for this series, Follansbee is a member of Bay Area Physicians for Human Rights, and he provides yet another view of BAPHR functions in the early years of the epidemic. He also talks about his involvement at the university with the formulation of early infection control protocol, adding to the perspectives provided by Merle Sande, Grace Lusby, and others interviewed for this series.

A major contribution of Follansbee's oral history is the picture it paints of the problems and successes of the relationship between AIDS physicians at the university and in the community. Complementing Donald Abrams' account in his oral history, Follansbee tells of the formation and functions of the County Community Consortium, a group established and continuing to flourish in San Francisco, which, among other things, provides a forum for the interaction of university and community physicians.



The Oral History Process

Two interviews were conducted on August 23, 1996 and September 6, 1996, in Follansbee's office at Davies Medical Center. He reviewed the transcripts and hand delivered them to the Bancroft Library. Although he told his story in straightforward, unadorned manner, he was eloquent on the subject of his own contributions:

I think my greatest contribution was the fact that I took care of people with HIV, that I was one of the people who had the privilege, honor, fate to be in the right place at the right time, and to take care of people who really needed health care and who still need health care. It's that one-to-one contribution, with those patients, their partners, their family members, that is my greatest contribution.

Sally Smith Hughes, Ph.D.  
Research Historian and Principal Editor

February 2000  
Regional Oral History Office  
The Bancroft Library  
University of California, Berkeley

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## INTERVIEW WITH STEPHEN FOLLANSBEE, M.D.

## I BACKGROUND, EDUCATION, AND EARLY CAREER

Background and Education

[Interview 1: August 23, 1996] ##

Hughes: Dr. Follansbee, start back, if you please, with where you were born and educated.

Follansbee: I was born in Pasadena, California [1948]. My father was a Presbyterian minister so we moved around, but I was mostly educated in Sacramento through high school, Pomona College in Claremont, California [1966-1970], for four years. I went to graduate school in molecular biology and biochemistry in a Ph.D. program, and after a year at Harvard for a number of reasons decided that that was not what I wanted to do. So I took a master's degree from Harvard [1972] and moved to Boulder, Colorado, and did research for about a year and a half.

Hughes: In molecular biology?

Follansbee: In molecular biology, trying to decide what I wanted to do with my life, and decided to go to medical school. So finished medical school in 1977 in Colorado, in Denver, University of Colorado, and came up to San Francisco. Did my internship in what was the last year of the straight medical internship program at San Francisco General Hospital, which was part of UC [University of California at San Francisco], and finished my residency in medicine at UC in 1980, and then did two additional years in infectious diseases at UC between '80 and '82.

Early Career

## Choice of Medical Career, 1970-1977

Hughes: Now, why did you choose first medicine and then infectious disease?

Follansbee: I think I had always liked medicine as a child. I've never been sick, and that was a question that was asked when I applied to medical school.

For a lot of reasons that are probably not of interest to this epidemic, I originally decided not to go into medicine and to go into more basic sciences. But I realized, actually before I ever went to graduate school, that that's not what I wanted to do. I had this sort of elaborate plan where I was going to actually defer my graduate school education for a couple of years because I was low in the lottery to be drafted and was applying for a C.O. [Conscientious Objector Status]. I had lined up a job if I got a C.O. to do research for a couple of years at Harvard, and I really wanted to do this. It was pretty obvious that, as a career, I didn't like the academic side of molecular biology/biochemistry, and so I stepped back from that path.

It turned out that my boss, Dr. Charles Yegian, in Boulder where I was doing research, had leukemia, unbeknownst to those of us in the lab. I might still be a lab technician if he hadn't relapsed his leukemia and called us all in one at a time to plan our futures, literally from his hospital bed at the University of Colorado Medical Center in Denver. He had decided that I should go back to graduate school in biology at MIT and had written letters for me. I was a little taken aback and said, "No, I don't want to do this." "Well, what do you want to do?" And I had been toying with the idea of medicine, because I like contact with people, so I said, "Medical school." He looked at me and said, "You're not going to like it." I said, "I know myself I may not like it, but that's what I want to do." So he said, "Well, fine," so he wrote me letters and I applied to medical school.

So I was kind of prompted a little bit, because I like contact with people--the human side or personal side of medicine. I still like science a lot, but I also like the other, human, side of it, and some of the mystery of medicine, I think. Over time, my early prejudice that medicine was

"glorified auto mechanics" dissipated, so I was more willing to look at it as a profession.

**Reasons for Specializing in Infectious Disease (UCSF  
1980-1982)**

Hughes: And then why did you decide on infectious disease?

Follansbee: Well, that is related to this epidemic, because it became quite obvious to me in internal medicine that internists don't cure people, and at least weren't curing people in the late seventies. And infectious diseases was the one branch of internal medicine that I saw where I had a chance to actually cure people. So it was attractive to me. And because of my background in molecular biology and virology, I also felt that I had some of the necessary scientific background. I knew I didn't want to stay in academic medicine, which was unusual in that era for someone going into infectious diseases. Most people who were in infectious diseases did a third year of research in infectious diseases and then stayed in an academic manner. I already knew I didn't want to do that.

Hughes: Why didn't you want to do it?

Follansbee: Because I already knew what academics was like. I mean, that's one of the reasons I had gotten out of molecular biology. I didn't want the constant pressures of research grants and publishing, with teaching squeezed in. I really wanted to engage in more patient care, and saw myself in that light. So I applied for a position that was vacant in infectious diseases rather late in my internal medicine training at UC.

I didn't want to leave San Francisco. I had moved out here with the idea that I'd go back to Colorado, and, in fact, had sort of arranged before I even left medical school with some of the people at the University of Colorado to talk about future job possibilities in a clinical realm, and thought that I had options back there. But at the end of three years, I wasn't really quite ready to leave San Francisco, so it gave me an opportunity to stay for a couple more years before I left. And I haven't left.





## II THE AIDS EPIDEMIC

### Early Warning Signs of the Epidemic at San Francisco General Hospital, 1978

Hughes: Well, with the advantage of hindsight, we can now see that there were certain things going on in infectious disease that set the stage, so to speak, for the arrival of the epidemic. Were you seeing things that you now realize were warning signs?

### First Probable AIDS Patient, San Francisco General Hospital, 1978

Follansbee: Absolutely. There's one patient who sticks in my mind. The last week of my internship at San Francisco General Hospital [SFGH]--well, it was not actually the last week. It was several weeks before that. My internship finished in June of 1978, and I remember assuming care of a patient that last month, I think, at San Francisco General Hospital. He was a young Hispanic man who presented with high spiking fevers and diffuse lymphadenopathy. At that point his lymph nodes had been biopsied and just shown hyperplasia, and this was again June of '78. Because he was Hispanic and had no real risk factors for anything else at that point, we treated him presumptively for tuberculosis. I then started to follow him in my general medicine clinic at San Francisco General Hospital.

So I followed him in my clinic, and about three months later, he came down with hepatitis. I thought, "Oh, my gosh, hepatitis from the medication." We stopped the tuberculosis medications, but it proved to be hepatitis B. And hepatitis B, late 1970s, was a sexually transmitted disease in the gay

community. He denied that he was gay; he told us that he had a girlfriend and didn't inject drugs, so I didn't know how he got hepatitis B, but that was fine, we just do the best we can, treat it.

Then he came back into my clinic, and all the TB [tuberculosis] cultures were negative. He came in a couple of months later having been assaulted while visiting friends in Denver. I finally got him to tell me why he was assaulted. It was because the brothers of his girlfriend or somebody had accused him of being gay and beat him up. He had minor lacerations and bruises. I asked him if it was true that he was gay; he said no. I said, "Well, fine, we'll just go ahead and continue to take care of you." And then I sort of lost him during follow-up, but I'm sure in retrospect that he was an acute HIV seroconversion, that he was an HIV seroconversion as early as 1978.

#### More Early Probable AIDS Cases, Late 1970s

Follansbee: We saw periodically people with similar symptoms. I remember another guy who was a male prostitute but said his clients were all female, who had a febrile illness when I was a resident in the late 1970s. And again, we just saw these things, and we chalked it up to acute CMV [cytomegalovirus]; this was the most common "presumptive" diagnosis. This CMV was an unusual sort of diagnosis of exclusion, and of course not confirmed.

So yes, we were already seeing unusual symptoms in patients, and not being able to sort them out, because there really weren't any distinctive findings. Just fevers, swollen lymph nodes, maybe pharyngitis and a rash. These patients got better.

Hughes: Did it seem to be occurring in gay men, or did you make that connection?

Follansbee: Well, we did make the assumption, because the first case I mentioned I assumed was gay, but I didn't know for certain. I mean, it was one case, and it just didn't seem like a pattern.

And we saw at San Francisco General, in particular, a lot of IV [intravenous] drug users with fevers. Usually I'd get three or four of these cases a night when I was on call, and I would rule out endocarditis and send them home in two days if

the blood cultures were negative. And so these exceptional cases with unexplained fever which got better spontaneously just didn't stick out. However, because these cases weren't satisfactorily explained, I remember them.

So, I didn't make the association at the time that this was the beginning of something, because the patients all got better, and then we never saw them again. The health care and social situation was that we sort of lost them to follow-up.

#### Reasons for Not Initially Recognizing the Significance of Early Cases

Hughes: Were these cases any particular source of conversation with your medical colleagues, or was this just sort of a run-of-the-mill problem?

Follansbee: We did not connect the dots. I don't think that there was a recognition, at least in my circle of friends, that these cases were odd, because again, I think the CMV serology they exhibited really fooled a lot of people. Larry Drew had already recognized that CMV was sexually associated and sexually transmitted. And part of the problem is that the antibody tests that we do for CMV, the IgM antibody and the IgG antibody, were confusing. The IgM, which is classically an early antibody that goes away after six months to a year, would sometimes reappear in a patient for unexplained reasons. So we were often associating this rise in CMV IgM with CMV, because Larry Drew had already recognized that phenomenon. These cases could have all been CMV, and we had no way to really confirm or deny it at that point. So I don't think that there was speculation that we were seeing something new other than CMV or another self-limited virus.

Donald Abrams' Similar Experience, Kaiser Hospital, Late 1970s

- Hughes: I remember Don Abrams telling me a similar story.<sup>1</sup> He was a medical resident at Kaiser and was seeing cases of lymphadenopathy. He eventually stopped having the lymph nodes of these patients biopsied, because the results revealed nothing definitive, just reactive hyperplasia or enlargement.
- Follansbee: Right, exactly, and that was exactly the case. And by the way, call him Donald. He does not like Don. It should never appear in any transcript! [laughter]
- Hughes: All right, I'll remember that: Donald.

Early Cases of *Pneumocystis Carinii* Pneumonia [PCP] in San Francisco

- Hughes: When did you begin to recognize that there was something significant going on?
- Follansbee: Again, the exact dates all escape me, but I remember very distinctly when I was an infectious disease fellow at UCSF, 1980-1982, and we had our first case of unexplained *Pneumocystis carinii* pneumonia [PCP].

I remember the patient very clearly, and I remember that the Infectious Disease Service didn't get called in on the case until the patient had already been diagnosed. By that time, he had already been in the intensive care unit and the ICU staff had performed all the lung biopsies and they had found *Pneumocystis*. The pulmonary fellow at the time said, "Steve, you ought to see this case. We don't know why he's got this." I did a literature search to find out how often *Pneumocystis* occurs, and found out that except for some report out of Seattle in a homeless person who didn't sound very healthy to begin with, it really hadn't been reported in adults without a well-defined, pre-existing immune system deficiency.

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<sup>1</sup> See the oral history with Donald Abrams, M.D. San Francisco AIDS Oral History Project: The Medical Response, 1981-1984. Regional Oral History Office, The Bancroft Library, University of California, Berkeley. Hereafter, the AIDS physicians series.

It was either that pulmonary fellow or another pulmonary fellow who had connections with people in New York City-- either he'd done his medical school training there or whatever. He told me: "I've got a friend who's doing a pulmonary fellowship in New York; you ought to call him, because they've seen some Pneumocystis." So I actually called--I don't remember who that was whom I called.

Hughes: So what was unusual about this case was that it was an adult?

Follansbee: It was an adult with Pneumocystis pneumonia, and we'd never seen it in an otherwise healthy adult. We would certainly see Pneumocystis on occasion at UCSF because of their kidney transplant program, but here was a person who came in off the street presenting with Pneumocystis.

I remember distinctly interviewing him, and the only reason he got admitted to the hospital in the first place was that he had terrible diarrhea. He was being followed in the GI [gastrointestinal] clinic, and they couldn't find out what was going on. The GI staff wanted to send him home from the clinic, and he actually lay down on the floor on the hallway of the GI clinic and threw a temper tantrum and pounded the floor and said, "I'm sick, you've got to do something about it."

So they admitted him for this diarrhea, and the staff didn't know what to do with him. They got a chest x-ray as part of the routine admission orders, and there were infiltrates. The medicine team worked up these x-ray findings, and so they ended up ultimately finding the Pneumocystis. But the patient had to throw a temper tantrum to get admitted, and wasn't recognized as having pneumonia until the chest X-ray.

Hughes: The GI admitting staff didn't realize that he also had pneumonia.

Follansbee: Right.

Hughes: When was this?

Follansbee: This must have been--I'm sure I've got records of it--some time in 1981.

Hughes: I have your paper that was published in 1982.



- Follansbee: The Annals of Internal Medicine paper.<sup>1</sup>
- Hughes: The Annals paper, yes. There were cases being seen as early as November of 1980.
- Follansbee: Yes. I think the patient who was admitted first with diarrhea was not the first case in San Francisco. I think the first case in San Francisco may have been David Busch's case at Davies Medical Center. This case in the Annals paper was also one of the first reported. It was co-authored with Connie Wofsy, who was at San Francisco General at the time, and David Busch, who's now one of my associates in private practice. I can't remember which was the actual first case in San Francisco, but there was a case from each institution represented by authors on this paper.
- Hughes: Where was Busch then?
- Follansbee: Busch was at Children's Hospital, but was in practice--and still is in practice--doing infectious disease at several private hospitals in San Francisco.
- Hughes: There are a couple of cases that were first observed in November, 1980.
- Follansbee: Yes. Busch actually saw the first, it was his case, here at Davies Medical Center. I think that the first case that I saw at UCSF was not hospitalized until April of '81. In other words, he had been sick in '80 but had diarrhea and had Entamoeba histolytica. So his history had gone back several months. I think that the case I saw at UCSF wasn't hospitalized until '81. Yes, some of these cases do go back to late 1980. In retrospect, reviewing the paper, maybe the case of Dr. Wofsy, the San Francisco General case, was the first one.
- Hughes: Was this the first case of Pneumocystis that you'd ever seen?
- Follansbee: This was the first case of Pneumocystis that I'd ever seen in someone who didn't have another risk factor. We'd had a couple of cases at UCSF, I think, in the renal transplant population. But this was the first case we'd ever seen outside of a normal risk group. So we did a literature search on Pneumocystis in healthy adults.

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<sup>1</sup> Follansbee, SE, Busch, DF, Wofsy, C, et al. An Outbreak of Pneumocystis carinii pneumonia in homosexual men. Ann. Intern. Med. 1982, 96:705-713.

Hughes: And what did you think?

Follansbee: Well, again, it came out at the same time--I think this was April of '81--that someone told me, "Hey, cases of PCP are appearing in New York City."

Hughes: That's interesting that you were referred to the cases in New York, because what I think of in terms of early *Pneumocystis* cases are Gottlieb's cases in L.A.

Follansbee: Right, in Los Angeles. It just so happened that the way the communication was going on, that yes, I heard about New York before the CDC [Centers for Disease Control] report from Los Angeles. Very quickly, I, like others, recognized that something unusual was happening. I think that was pretty obvious. I don't know how quickly it took those of us who saw these cases to get together, but San Francisco's a very small town, and so I think it was fairly soon that we all recognized that we had all had this unusual experience.

#### Testing for Immunodeficiency in PCP Patients

Hughes: What about the immunodeficiency aspect of these unusual PCP cases?

Follansbee: Well, you know, it was interesting. Certainly what we're used to now in terms of ordering T-cell subsets as if we were getting a routine urinalysis was very unusual then. The equipment to do that T-cell subset count was certainly available in research labs, and there was the equipment available at UCSF to perform this kind of testing. So it was suggested that we begin to investigate the immune systems of these patients and find out if an immunodeficiency existed.

Hughes: Did you explore this angle with that very first case?

Follansbee: Again, I'd have to go back and look at the records, but I think that in the report on the first case, it did say that after the patient had survived the first episode of Pneumocystis, that, yes, Art Ammann<sup>1</sup> did the T-cell subsets.

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<sup>1</sup> See the oral history in the AIDS physicians series with Art Ammann, M.D.

Hughes: Ammann did the T-cell counts because he had the capability to do them?

Follansbee: Right. Again, I don't remember the time course exactly, but we relied very heavily on the pediatric immunologists, like Art Ammann, because they had experience with SCID, Severe Combined Immunodeficiency Syndrome. At that point, Art Amman headed the program at UCSF that looked at those infants and children with SCID. So UCSF was an important resource for testing the immunodeficiency angle. Quickly, we began to extrapolate from the pediatric immunology experience with SCID to our adult patients with PCP.

### Immune Overload Theory

Hughes: I see. Did you initially suspect that the immune systems of your PCP cases were compromised because your experience thus far with PCP had been with the kidney transplant patients? Did that immediately say to you: "immunodeficiency"?

Follansbee: Right, something was wrong. It was known that the epidemiology of Pneumocystis was already very clearly established: that Pneumocystis is a very common organism in the environment; that by the age of five, 80 percent of healthy northern European children have antibodies to Pneumocystis; that PCP is a disease of some altered immunity or some sort of concurrence of events.

Thus, PCP in this setting immediate prompted a lot of concern about what was happening. We wondered what was the role of the antibiotic tetracycline in particular? Because a lot of these guys were getting tetracycline for atypical pneumonia, and we wondered whether tetracycline was involved somehow. In that same context, we considered the use of poppers [amyl nitrate]. Was this somehow causing pneumonia? This was the immune overload concept. These guys were getting a lot of sexually transmitted diseases in the 1970's; maybe their immune systems were "exhausted" by all the internal parasites, gonorrhea, CMV, etc.

I remember Dave Busch and Bob Bolan, who was in practice in San Francisco at that point, getting hold of amyl nitrite bottles that were being passed out in the clubs and the bathhouses. They were having these amyl nitrate samples concentrated down and looking for Pneumocystis in them,

thinking, Well, maybe there was a point source of Pneumocystis somehow contaminating the popper that was commonly shared.

Hughes: Does this immune overload theory have a precedent before the epidemic breaks?

Follansbee: Not technically. Pneumocystis itself was recognized first in malnourished children in foundling homes in northern Europe after the Second World War. And so there was concern that maybe there was some association with malnutrition, which is entirely reversible. These guys were having so many infections, particularly with a high inoculum or high rates of exposure, that somehow these men were just more vulnerable to something that they should have been able to fight off under normal circumstances.

At the same time, Kaposi's sarcoma was being seen. The epidemiology of Kaposi's, as you know, was already very well worked out as well, in terms of elderly Ashkenazi Jewish men and, again as with PCP, in patients with kidney transplants and other immunocompromising situations. I remember it wasn't very soon afterwards, after seeing the adult PCP patients, at one of the early KS [Kaposi's Sarcoma] Clinics, that Paul Volberding talked about the fact that you could cure KS in the kidney transplant population by taking their donated kidney out and stopping their immunosuppressant medications. Again, you could conceive of this disease as sort of a temporary situation.

But other than these specific immunosuppressant conditions, no, there really was no precedent in terms of life-threatening illnesses and immune overload. But I think when people recognized that this new syndrome was something serious and that something new was going on, they began searching for answers.

Recognizing the Epidemic, April-June, 1981: Associating PCP and KS as Part of a Syndrome

Hughes: Please talk about the process of building up from individual, specific conditions to what we now recognize as a syndrome.

Follansbee: Again, I don't remember the weekly sequence of events. When was the first CDC report of Pneumocystis?

Hughes: That was in June, 1981.<sup>1</sup>

Follansbee: June. I don't think I was aware of [the CDC report] before the hospitalization for case one in the Annals article for Pneumocystis, which was in April of '81. I think that this epidemic was recognized within those two months [April-May, 1981]. By the time the CDC report came out, it was no longer news. Something was going on.

Hughes: It was not news within rather restricted circles.

Follansbee: Exactly, not news to us medical professionals in San Francisco, to me. So the process of recognizing that there was an epidemic went on very quickly. As soon as someone said, "I think this is happening in New York," then I began to think, Oh, gosh, there is a problem here.

#### Connecting the Epidemic to the Gay Community

Hughes: When you said, "There is a problem," did you add, "and it's in the gay community"?

Follansbee: Yes.

Hughes: Was there that link?

Follansbee: Yes, it was already noted in homosexual men in New York, Los Angeles, and now in San Francisco.

Hughes: That was clear?

Follansbee: That was clear. That became clear very quickly. Although the cases that I had seen during my internal medicine training [SFGH, 1977-1978]--the ones which I mentioned, the young Hispanic man and the male prostitute--both denied having sexual contact with men, the first Pneumocystis patient had no problem identifying that homosexuality was his sexual preference. It wasn't until much later that I began to think about the old patients I used to see who had these seroconversion reactions.

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<sup>1</sup> M.S. Gottlieb, H.M. Schanker, et al. Pneumocystis pneumonia--Los Angeles. Morbidity and Mortality Weekly Report 1981, 30:21, 250-251. (June 5, 1981)



So the connection to gay men was quite clear very quickly. Then the KS was recognized by Jim Groundwater and Marc Conant very quickly.

Hughes: How you would link those two observations?

Follansbee: To gay men? It was basically through gay men. Again, I don't know the specific dates; I'll have to read Randy Shilts's book, And the Band Played On. I don't remember how or precisely when Groundwater and Conant thought of and started doing some of these immune studies in gay men with Kaposi's.

Hughes: You mean measuring T-cell subsets of these patients?

Follansbee: Yes.

Hughes: Through the KS Clinic?

Follansbee: Yes, but I'm not sure whether that was happening when Conant and Groundwater were doing their immune studies, before the KS Clinic formed. The KS Clinic was a response to the recognition of the epidemic.

Hughes: Oh, so you suspect that they might have started these immune studies before setting up the KS Clinic?

Follansbee: They may have already been doing it, or may have been thinking about doing it, because it was very clear very quickly that this was an epidemic of an immune deficiency problem in the gay community.

#### Linking PCP and KS to a Common Syndrome

Hughes: I can tell you that the first KS case that was recognized in San Francisco was in April of 1981, so about the time you were seeing the cases of Pneumocystis.

Follansbee: Same time as the Pneumocystis. And wasn't that first KS case Jim Groundwater's?

Hughes: Yes. I don't believe he did T-cell subsets, certainly not right away.

Follansbee: No, he would not have, but I think that the KS epidemic was pretty quickly recognized to outstrip the Pneumocystis epidemic. Probably because some of the Pneumocystis cases,



although this first PCP case I saw did not have KS, also developed or presented simultaneously with KS. But the patients who survived their PCP were at risk for developing KS and vice versa. So it was quite clear that there was an overlap of the same diseases in these men.

- Hughes: In a situation like this in which you had a patient manifesting several different infections at once, is the physician's natural response to think that these two or more conditions must be related?
- Follansbee: I think because it happened in gay men, the answer to that is yes, that these conditions must be related. Because the recognition of KS was so rapid that I think that through the sexual practices connection, in groups of men with health care coverage and a good "network," the recognition of overlap was swift. If this had been in IV drug users or some other population where there are all the issues of access to health care, I think recognition of the connection between these conditions would have possibly taken longer. Also, recognition of the syndrome would have taken longer if there had been fewer cases of several different manifestations of immune deficiency. But I think that because it was a disease in the United States in gay men, that the association was made fairly rapidly, at least once life-threatening manifestations occurred. In retrospect one wonders how many cases of oral candidiasis [thrush] or shingles had been occurring in the same population without "a light going on."
- Hughes: Is it as simple as linking, in this case, PCP and KS, ergo, you have a syndrome?
- Follansbee: Yes.
- Hughes: As soon as you understand the linkage, it's a syndrome?
- Follansbee: Yes, I think that it was determined to be a syndrome because both of those diseases were recognized as diseases of a poor immune system. Society made an error early in linking disability to the diagnosis of AIDS. Both KS and Pneumocystis pneumonia were AIDS diagnoses, but KS patients often had a much earlier disease and a much different short-term future. If you came in with KS, you had AIDS and were disabled. In other words, people with GRID [Gay Related Immune Deficiency] and then AIDS were disabled whether they had KS or PCP, despite the fact that the life expectancy and the natural history of disease progression in a person with KS was much less severe than in the person presenting with Pneumocystis pneumonia.

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Follansbee: The profile of patients with KS is and was much different than of someone that came in with Pneumocystis, and yet they were lumped together even before the epidemiology, before the natural history was worked out. The various conditions associated with the syndrome were all lumped together, and they were equated. It was only after more time that we recognized the relation of these various conditions as manifestations of progression in immune deficiency, occurring at different time points in the progression.

Hughes: What did you, with your infectious disease background, see first in this syndrome? What stood out to you?

Follansbee: There are so many levels to answer that question.

Hughes: Some of the dermatologists and oncologists involved with the AIDS epidemic to whom I've talked say that the most salient aspect of this syndrome in San Francisco in the very early days was KS. Because KS produces a visible lesion, it's something that a patient would almost immediately recognize and be worried about, and probably get some consultation. A person with PCP, which was the case with your first patient, would have to get pretty sick before he would say, "Hey, there's something going on," or even suspect that it was something unusual.

Follansbee: Yes.

Hughes: Were you and other infectious disease specialists a few cases behind the physicians seeing patients with KS?

Follansbee: At the beginning, probably not, because many of the early KS lesions were not identified, or just watched for several weeks, thinking they were bruises. There wasn't a lot of alarm initially, until KS became identified and pieced together with a life-threatening, more rapidly progressing problem, i.e. Pneumocystis pneumonia. There also wasn't a lot known about what to do for KS.

#### Early Treatment and Prognoses for KS and PCP

Hughes: Physicians tried, did they not?

Follansbee: Oh, lots of things were tried, but no one even thought that treating KS may intensify risks for other problems, such as PCP. I assume you're still referring to the early days.

Hughes: I am, the very early days.

Follansbee: About the only thing that gay men could do to protect themselves was shun the people who had KS. So the response within the gay community was to think that as long as a person's skin looked okay and he didn't have swollen lymph nodes, that that person was safe as a sexual partner.

As care providers, our early reaction was to offer good nutrition and general health advice. But we didn't know what was really going on; we didn't know this syndrome was even infectious. I mean, we assumed it was, but we were still looking at drugs as possible causative agents and at other noninfectious vectors for this disease. Later we recognized that this disease was transmitted like hepatitis B. But early on the whole Mervyn Silverman [Director of San Francisco Department of Health] argument and "can't we close the bathhouses until we have an infectious agent?" argument still related a little bit to the advice that was going on in the doctors' offices.<sup>1</sup>

But you're right that it didn't take very long to recognize that people with Pneumocystis as their first diagnosis didn't do as well, that they had a shorter life expectancy than someone with Kaposi's sarcoma. The interesting thing was that in the first few years of the epidemic, if you look at what happened to people with Pneumocystis as their first manifestation of AIDS, their life expectancy stabilized or improved a little bit over the first few years of the epidemic. With KS, exactly the opposite was the case. Early in the epidemic, people with KS had a better prognosis than later, after the first couple, three years of the epidemic. This was because the oncologists were just blasting the KS patients with chemotherapy and further depressing their immune system, and so their life expectancy went down.

So there was this dip in the life expectancy of KS patients. In fact, the early response, despite what the dermatologists and oncologists may say, was to overtreat these

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<sup>1</sup> For more about Mervyn Silverman's reluctance to close down the gay bathhouses and sex clubs, see Mervyn Silverman's oral history in the AIDS physicians series.

patients with chemotherapy. This hurt patients in terms of their life expectancy, because they were treated aggressively for their visible KS lesions. At least with the infectious disease practitioners, we didn't have this dip in life expectancy as a result of treatment, since we didn't have anything to treat with.

Hughes: Did you go to the KS Study Group at UCSF?

Follansbee: Yes, I would go to the KS Clinic.

Hughes: Do you remember any discussion of KS treatment?

Follansbee: I do remember the discussion where there was a recognition that, "Hey, maybe we're treating KS too aggressively."

Hughes: Can you put any date to that discussion?

Follansbee: No, I honestly can't. I think that it was the mid-eighties.

Hughes: That late?

Follansbee: Yes, I think it was recognized by '84, '85, as opposed to later than that. But I honestly can't tell you.

Hughes: Were those aggressive therapies being used right from the very start, when the clinic first got up and running?

Follansbee: Yes, I think when the clinic got going, there was a tendency to use systemic therapy early. Injecting chemotherapeutics into the lesions wasn't the initial approach to treating KS. Because KS was visible, and because we're all a little narcissistic, and certainly gay men aren't spared that, there was a lot of pressure to treat the KS aggressively.

Hughes: So the injection into the lesion didn't work?

Follansbee: No, use of this modality didn't come at the beginning of the epidemic. It does work, but it's use appeared a few years later.

Hughes: I see.

Follansbee: The initial reaction was to treat KS systemically with what they had used to treat the aggressive forms of Kaposi's prior to HIV, prior to the epidemic.

Alvin Friedman-Kien at BAPHR Meeting, June 1981

- Hughes: Did you go to a June, 1981, BAPHR [Bay Area Physician's for Human Rights] annual meeting at which Alvin Friedman-Kien spoke?
- Follansbee: Where was it?
- Hughes: Here in San Francisco.
- Follansbee: I don't remember. I was one of the founding members of BAPHR in about '77.
- Hughes: So you probably were at the meeting?
- Follansbee: I certainly was in town. It was often held in association with the Gay Pride Parade--the weekend just before, or the weekend of. So I may have gone.
- Hughes: Well, if it doesn't stand out in your mind--
- Follansbee: It doesn't stand out.
- Hughes: For some people, that's the first time that they recognized that there was something going on, because Friedman-Kien reported on KS cases in New York.

Follansbee's Journal

- Follansbee: Yes. I can't remember when I first heard about the KS in New York. I'll have to look. I keep a journal--it's a very personal journal. I don't write in it on a daily basis or a regular basis, but I have kept it since I dropped out of graduate school, so I've kept it since '71. So I can check and see if that impressed me, but again, it's such a personal journal that I don't necessarily include what I'm seeing professionally. What was going on personally usually gets into the journal before professional issues.
- Hughes: Of course, I recognize your privacy, but if there are insights that we can add to our discussion, that would be wonderful.
- Follansbee: Sure.



- Hughes: The CDC in late May of 1981 had a meeting of I.D. [infectious disease] specialists in San Diego.
- Follansbee: I did not go to that.
- Hughes: That again was a meeting where the word got out that a serious epidemic was beginning. You learned among your own circle how things were going?
- Follansbee: Yes.

The KS Clinic at UCSF, 1981-ca. 1984

- Hughes: Let's talk about the KS Clinic. How did you become associated with it?
- Follansbee: Well, the clinic was up at UC[SF], and I was in the infectious disease program there. And because I had been involved with the first case of PCP at UCSF, I became sort of the point person in the infectious disease program for this epidemic. The faculty were very supportive, but not particularly involved in seeing patients at risk for AIDS. I became sort of a resource for this problem, at least the infectious diseases associated with AIDS.
- Hughes: Now, the faculty weren't involved because they saw that you were already involved.
- Follansbee: Yes, but it was mainly because they had other faculty responsibilities and were involved with lots of other issues. There was no push to have an infectious disease clinic for this population, since initially the epidemic seemed small and there already was this regular meeting in oncology/dermatology. So I.D. doctors would attend this regular meeting that already existed, the KS Clinic. I forget what day a week it was, Thursday?
- Hughes: I think so.
- Follansbee: The infection control practitioners who I worked with and I went.
- Hughes: Attendance served to keep you informed about the patients with this disease?



Follansbee: I didn't see patients in the KS Clinic. I only went to the meeting which followed the clinic. There was a meeting that would go on for an hour. So I didn't generally see patients in the KS clinic unless there was a patient there who had a problem that was infectious disease-related. Then they would call me.

Hughes: Wasn't that quite often?

Follansbee: Not really, because again, it relates to your earlier question: KS was more visible than the sporadic infectious disease complications. Also, KS occurs in people with a higher CD-4 cell count, and so the initial population weren't particularly sick with Pneumocystis and the other serious opportunistic infections.

Hughes: But what about in your non-KS-Clinic-associated practice? Were you continuing to see patients?

Follansbee: Yes, but again, it was fairly sporadic. There is one patient who stands out, but other than that, I can't think of patients who stand out in the haziness of that UCSF infectious disease clinic several years ago.

#### The Response of the UCSF Parnassus Campus to the Epidemic

Follansbee: There was a regular infectious disease clinics at UCSF, and I'm sure [AIDS] patients were being seen. But you know, I don't remember the epidemic sort of just exploding, at least at UCSF. Part of it was, I finished my infectious disease training at UC in June of '82 and then went into private practice. So, although I was at UC for another year as a hospital epidemiologist (1982-83), I was not involved with the infectious disease clinics per se. Also, UC has always had, outside of San Francisco General, a very peripheral response to the epidemic in terms of the numbers of people with AIDS seen regularly on the Parnassus campus. If you look at where AIDS patients are seen, UC ranks usually fourth or fifth in the numbers of patients. San Francisco General is clearly number one, but if you separate the campuses, the university campus was always trailing other San Francisco practices.

Hughes: That's because San Francisco General is also a county hospital, and so, for many people, it's the place of last resort?

Follansbee: Yes, right. UC, historically, in terms of being responsive to local community needs also deferred to other institutions. The program at San Francisco General Hospital is a UC program, as you know. So I don't necessarily mean to say that the University of California hasn't responded to the epidemic, but the Parnassus campus<sup>1</sup> has never been very prominent in this context. I think in part because the Parnassus campus has never been a community campus, it's more remote and more focused on tertiary care. Yes, some people get their primary health care there, but it has historically not been a place which has really responded to the problems that have come through its neighborhood. It's close to the Haight-Ashbury, et cetera. And so I think that there were probably a lot of reasons for that, mostly related to reimbursement for health care, and that for many GI admissions, the neighborhood problems--sexually transmitted diseases, drug/alcohol use, psychiatric problems--just didn't appear intellectually challenging.

But the epidemic didn't explode at the Parnassus campus as much as it did at San Francisco General, or as it did in doctors' practices here at Davies [Medical Center] and at Kaiser, where patients are being seen in large numbers.

Hughes: Yes, and even as far as UCSF did experience the epidemic, it was not as early as what we were just talking about at SFGH.

Follansbee: Exactly.

Hughes: I believe that the KS Clinic, by the end of 1981, had only seen something around a dozen patients.

Follansbee: Right.

#### Follansbee's Second Case of PCP, 1981

##### Cotton-Wool Spot and PCP

Follansbee: For the first patient that I saw at UCSF, the diagnosis of PCP was handed to me on a platter, because he had been worked up and had had an open lung biopsy, and I saw him after the

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<sup>1</sup> The UCSF main campus is located on Parnassus Avenue.

diagnosis was established. The second patient, curiously enough, was referred to me at UC from David Busch, in 1981. David Busch was to be my associate.

David had started to see this patient as an outpatient at Children's Hospital. But the patient had no insurance. David couldn't go any further with this patient without incurring a great deal of expenses. The young man had fevers, and David saw a cotton wool spot in his retina. So he sent him to the infectious disease clinic at UC, because the clinic had a sliding scale of payment, and so they could see him without insurance. I worked him up at the I.D. clinic. I walked him over to ophthalmology because of this cotton wool spot, and they said, "You have to rule out endocarditis because of this spot." So we got an echocardiogram, and sure enough, he had a congenital heart valve problem that was very minor. I was in the process of working him up for fungal endocarditis or something remote since blood cultures had been negative for routine organisms.

The reason I tell you this is because the case sticks out, and because San Francisco's a small town. During my I.D. internship, I did moonlighting as a house officer at St. Luke's Hospital out on what's now Cesar Chavez but was then Army Street. I would sit there on Friday nights responding to emergencies in the hospitalized patients. So I was reading the *New England Journal of Medicine* at like twelve-thirty in the morning, sometime after midnight, and there was an article out of Boston on the association of cotton wool spots and Pneumocystis pneumonia. Here I am sitting at St. Luke's Hospital, working this patient at UCSF up for fevers, and I see this article. I think the article came out in January of '82 or December of '81, it was around then. I thought, Oh, my god, I know what this guy's got.

So as soon as my shift was over, which was eight a.m., I rushed over to UC, went to medical records--they must have thought I was nuts--to have them pull an outpatient chart. I called up the patient, whose name was Pat, and I said, "Pat, I know what you've got." He goes, "What do I have?" I said, "You've got pneumonia. Are you short of breath?" He said, "No, I'm fine." I said, "You're coughing." Because the cough was always a minor part of this. Here he was with fevers and an eye problem. I said, "Well, I think you've got Pneumocystis. If you're not short of breath, I'll see you Monday, we'll get you worked up." And sure enough, he had Pneumocystis pneumonia.

He sticks in my mind because I learned a lot from this person. One, is that you keep your eyes open. Here I was not even thinking about his lungs in the process of working him up. And I also learned a lot from him in part because I followed him until his death. He just taught me a lot, about myself, about this illness, because he was just a wonderful person. And also, he sensed that this problem was going to be big.

Having seen a second case, I think it kind of confirmed my involvement in this epidemic. I can remember--and I wish I could remember when, maybe other people can remember--but there was a period in the KS Clinic--I think it must have been probably early, like '82--where we thought the epidemic was going away. We'd seen a few cases, we had this kind of fluttering of cases, but then several months went by and we didn't see anything new. We thought, well, maybe this was some sort of a little blip. Yes, there was something going through the gay community with a short incubation period, and it's gone, or it's going away, so this is all over.

#### Professional Stakes in the Epidemic

Hughes: Did the thought that this epidemic would be short-lived make you less eager to put a real stake in this field?

Follansbee: That's a complex question, because it's a very personal question at one level. I don't think at first anyone saw a career in this epidemic. Nobody closed the KS Clinic; they were still taking care of patients. At that point, and in my own mind, nobody was putting a stake professionally into this disease. We health care professionals were taking care of patients with the syndrome, but Marc Conant was still seeing all his dermatology patients, and Donald Abrams was in his oncology fellowship learning about other malignancies as well. Yes, he was about ready to establish his lymphadenopathy clinic; that didn't get established until 1982. It was first established at UC, so it must have been established in 1982, before it moved to SFGH. But I don't think even Donald at that point saw himself as putting a stake of his whole professional career into this.

Hughes: How could any of you know how long and serious the epidemic would become?

Follansbee: Exactly, because we didn't know. So certainly by 1982, Paul Volberding had moved to San Francisco General. He had moved, quote unquote, because there was this interesting problem going on, but he was moved there officially as the chief of oncology. As Merle Sande<sup>1</sup> [Chief of Medicine at SFGH] loves to say over and over again, "And I'm still waiting for him to establish the oncology program out here." [laughter] Well, he's actually not waiting now, since Merle's gone to Utah. But again, I don't think anyone was putting a stake on their career based on this epidemic in those early years.

The reason that I kind of hesitated was because I certainly didn't predict that this would be a problem that would probably last my entire professional career. It would be wonderful in my own mind if, by the time my professional career is over, this problem was licked and gone. I would consider it a privilege to have seen a disease come as my training was finishing and be gone by the time my career was over. And in part because of my desire to see AIDS disappear, I don't call myself an AIDSologist or an AIDS expert. I'm an infectious disease specialist, and I don't want the ownership of AIDS in that sense. I mean, yes, I recognize that I run a research program and I have expertise, but it's a funny professional distinction to make between AIDS expert and an I.D. specialist. I have not made a career change to be simply the AIDS specialist or the HIV specialist. I'm still an infectious disease specialist.

Hughes: And not just for practical reasons, but for personal and psychological reasons as well?

Follansbee: Right, absolutely.

#### Realizing the Seriousness of the AIDS Epidemic

Hughes: When do you think you and others began to realize that this was not an ephemeral disease?

Follansbee: Well, I think there was this initial period of hopefulness that the epidemic was going away. Again, I wish I could tell you exactly when that was. It probably was about the time the syndrome was being called GRID, because it was gay-related.

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<sup>1</sup> See the oral history with Merle Sande in the AIDS physicians series.



But, it wasn't very long before we recognized that the epidemic wasn't going to go away.

In terms of just how profound this infection is, I don't think the full recognition of that came until we really had an etiology and began to work out some of the complex life cycles of the virus.

Hughes: You mean as late as the discovery of the virus [1984]?

Follansbee: Right. Up to then, I think that there was still hopeful thinking--certainly within the gay community, but within even probably the professional community--that this was somehow some sort of transient problem. We certainly had no indication of how long the incubation period was before the disease manifested itself, and some of these other issues. So I think there was always the hope that it would just sort of go away. We'd take care of the people who got it, but we could stop the transmission and it would just go away.

Although I have to admit, I do remember an early meeting--boy, I wonder what year it was? It may have been one of these BAPHR [Bay Area Physicians for Human Rights] meetings where Jim Curran from CDC came out. No, it was during grand rounds at UCSF. Jim Curran came out and reminded us--and this has to be '83, '84, maybe '85, but probably was '83-'84--when he said, "This is a sexually transmitted disease. Look at our society's response to syphilis. We have the cure. We could eradicate syphilis. This society doesn't deal well with sexually transmitted diseases. Don't think this is going to go away." I thought it was very profound. I remember him saying that, and I remember being struck by this, because it just impacted me that this disease is not a medical problem; this is a social problem. This is not simply about medicine. Medicine isn't in a vacuum.

And look what happened with syphilis under Ronald Reagan's tenure. We had an epidemic of syphilis in the midst of AIDS; an epidemic of syphilis because of lack of funding for public health programs. I just kept thinking about Jim Curran's comment, very prophetic, I think.

Hughes: And particularly relevant coming from Jim Curran, because the CDC was one of the places that was hurt by the Reagan cutbacks.

Follansbee: Absolutely, absolutely.



Hughes: Well, you mentioned that you served as a consult to the KS clinic?

Follansbee: Yes, it was mostly consulting--

Constance B. Wofsy, Infectious Disease Specialist and AIDS Physician

Hughes: When does Connie Wofsy<sup>1</sup> come into the picture at UCSF?

Follansbee: Connie and I had a really wonderful history. Connie was on the admissions committee to the medicine program at San Francisco General. Years later, she told me that she remembered my application and remembered accepting me because anyone with curly hair couldn't be all bad. If I don't comb my hair out every morning, it's very curly. And that's the way I wore it in medical school. Connie was on the faculty of UCSF, in the emergency room in the Department of Medicine at the time. Then later Connie and I went through our infectious disease training in parallel. We were I.D. fellows exactly the same time [1980-1982]. Connie is on the paper because she was involved with that first patient as well at San Francisco General Hospital.<sup>2</sup>

I'm positive that Connie came out to the KS Clinic meetings. It was easier for me to get there regularly because I didn't have to come from San Francisco General, but I know she came.

The KS Study Group Attendance

Hughes: Connie did not see patients there? Like you, she came to just the study group meeting?

Follansbee: I don't think she saw patients in the clinic either. I think she just came to the meeting. I think most of us at that

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<sup>1</sup> See Constance B. Wofsy's oral history in the AIDS physicians series.

<sup>2</sup> This document, entitled "An Outbreak of Pneumocystis carinii Pneumonia in Homosexual Men", is reproduced and cited in the appendix of this volume.

meeting came just to the meeting and not to the clinic before it. I don't know who saw patients other than Marc Conant and Paul Volberding and maybe residents and other staff. Art Ammann would come, and Selma Dritz would come to the meeting after the clinic. Everyone would come to the meeting.

Marc or Paul would present interesting cases from the clinic, but most of the time, that meeting of the study group was really a discussion regarding other reports. Sometimes there would be guests who would want to talk about, "We had a case of this, and let's talk about how to treat cryptococcus," or some other issue. So a lot of the cases that were presented didn't come from the clinic per se. Once in a while, they'd bring a patient in from the KS clinic, but most of the time, there weren't even patients.

Hughes: How many people more or less were there in the early days?

Follansbee: At that meeting?

Hughes: At the study group.

Follansbee: I remember twenty to forty. Probably the mailing list was larger than that, but the room was often quite packed. Because there would also be doctors from the community. I remember Jim Groundwater and Jim Campbell and Steve Mehalko from Davies would be there as well. So a lot of times, there were thirty, thirty-five, forty people. A lot of people from the immunology lab at UC. So a pretty large group.

Hughes: Was there anything unusual about the study group in terms of its multidisciplinaryity?

#### Theories of the Etiology of AIDS

Follansbee: No. I mean, that was unusual in itself. It was a very fertile discussion group.

Jay Levy would go. I remember Jay's handout one day, which I wish I'd kept a copy of, about why this disease couldn't be caused by a retrovirus.

Hughes: Oh, really? [laughs]

Follansbee: Yes. All the reasons.

Hughes: Do you remember why Levy believed it couldn't be a retrovirus?

Follansbee: Well, it had to do with some of the things known about retroviruses. At that point, none of the retroviruses were known to be cell-cidal. I believe the identified retroviruses were known to transform cells. How a retrovirus could accomplish this state of immune deficiency without transformation into a malignancy was just not yet known.

Hughes: So what you're saying is that retroviruses up until then had been associated with cancers?

Follansbee: With cancers, with transforming cells into a malignancy, but not with cell death. And there were several other points Levy made, I just can't remember the details. I wonder if somewhere I have that piece of paper.

Hughes: Do you remember that Levy was definitely arguing against retroviruses as the cause?

Follansbee: He presented pros and cons. There was basically a balance sheet about whether the cause of immune deficiency could be a retrovirus.

Hughes: But that implies that Levy at least was thinking that a virus might cause AIDS.

Follansbee: Yes. I think people were thinking that there was a causative agent, that it was not caused by immune overload. The previous lists of possible causes had been pared down to probably a virus.

Hughes: People believed there was some specific causative agent right from the start?

Follansbee: No. I think people felt that other causes had been ruled out, in terms of drug use and things like that. Although Peter Duesberg is still floating that idea that AIDS is not caused by HIV, but by drug use and immune "overload."<sup>1</sup>

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Follansbee: There were a lot of discussions regarding "candidate" infectious agents that may be the cause of this new disease, AIDS. Larry Drew would go periodically to the study group and

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<sup>1</sup> For more of Duesberg's viewpoint, see the oral history in the AIDS physicians series with Warren Winkelstein.

would argue that CMV [cytomeglavirus] was the cause of AIDS, or a major contributor to it. I had forgotten that, the discussions and speculations that CMV was involved.

Hughes: Yes. Was there discussion of a new virus?

Follansbee: Yes. I still have the slide that I occasionally show about all the causes that have been discussed, everything from the wrath of God, to something about Haiti, to new viruses, old viruses, several viruses. It was suggested that maybe AIDS was really just the result of sort of a meeting in an unfortunate person of several viruses that by themselves wouldn't lead to that kind of disease. So that was being touted. There was concern that it was somehow related to intestinal parasites, and that there was some factor or toxin of intestinal parasites that was being absorbed into the body. It wasn't a parasite necessarily in the body, but in the intestinal tract that produced some toxin which was being absorbed and then destroyed the immune system. This idea arose because we'd been following a lot of these guys for recurrent intestinal parasites, some of which we couldn't treat. So there were all sorts of theories being put forward.

#### Jay Levy's Research

Hughes: Was Levy himself engaged in research on the disease?

Follansbee: He was researching retroviruses.

Hughes: But not necessarily associated with AIDS?

Follansbee: You know, I honestly can't remember. I think he was trying to find and isolate this cause as well. I think he was in a race to find the viral agent. He'd been introduced to retroviruses before AIDS. Jay was working with Paul, actually not with Paul so much as with Donald, who was following the lymphadenopathy patients. He was working with gay men with lymphadenopathy and examining lymph node biopsies. I think I remember him asking us for specimens.

UC and Community Physicians

Hughes: Because you have been affiliated with the university and with the community, could you talk a little about the relationship between this university group, which in the beginning was mainly centered at the KS Clinic, and with the community physicians?

Follansbee: Yes. I have to admit that a lot of things passed me by. [laughs] I think I was in another world.

## Private Practitioners Opt Not to Refer AIDS Patients

Follansbee: I think the first case of Pneumocystis in San Francisco that was recognized was actually seen at Davies, which at that time was Ralph K. Davies Medical Center. My associate, David Busch, who's still my associate, argued with the pulmonary specialist at that institution, Abe Aronow. My understanding is that the pulmonary specialist wanted to transfer this person up to UC, and David Busch, who was very well trained in infectious diseases, said, "This is a disease we can take care of," and argued to keep him here at Davies Medical Center.

Hughes: So this first recognized case was Busch's case?

Follansbee: I forget who the primary care doctor was. I'm sure it was neither David's nor the pulmonary specialist's, because David didn't do primary care. He was a consultant at that point, and the pulmonary doc never did primary care, so he was a consultant as well. So they argued over this person, and I don't know whose practice he originally came from. I wasn't involved. But I remember when David told me about this case, and we talked about this.

Hughes: When was this?

Follansbee: This would have been in late 1980. And the consensus evolved in the staff at this small hospital that, "No, we can take care of him here; we'll take care of him here. We don't need to transfer him to the university; we have the expertise in the private practice community." So I think that very early on, the precedent was set to take care of patients and not use the university in this relatively small town as sort of a dumping ground for people who are very sick. We, the private practice community, can take care of sick people. And UC



didn't do anything to alter that. Certainly by essentially moving Paul Volberding and Donald to San Francisco General, UC on Parnassus sort of acknowledged that they didn't really want a prominent role in AIDS care at that main campus.

### Refusing the Directorship of the UCSF Adult Immunodeficiency Clinic

Follansbee: At the end of my fellowship at UC in 1982 I had to make a decision what to do, so I went into practice with David Busch. It was the two of us. Recognizing, by 1982, that this epidemic was going on now, there was discussion about opening up an AIDS clinic at UC. I was sort of informally asked if I wanted to head that, and had to make a decision about whether I wanted to go back to the university. I had never really cut my ties; I was still employed there part-time. But I had to consider whether I really wanted to make the full-time commitment to UC. I said no, that I really didn't want to do that.

The name of the clinic became the Adult Immunodeficiency Clinic. It was never the AIDS clinic; it was never named to be explicitly associated with HIV. The university administrators didn't want the name on their door. I think that it was just kind of part and parcel of what went on at the Parnassus campus. Because of that, and in part because I think that approximately two-thirds of the patients in this epidemic have come from practices outside San Francisco General Hospital, AIDS care was thought of as a community-based responsibility.

### Communication Between Community and University Physicians

Hughes: Was some of your reluctance to refer patients to UC because, sure, you could take care of these patients, but also because you might have lost your patients if you referred them to UC?

Follansbee: Yes. I think that came later. I went to the first meetings of what became the consortium of San Francisco AIDS Providers, when Dianne Feinstein, who was mayor at the time, mandated that there be a community-San Francisco General-UCSF forum for discussion because of the lack of communication between the community providers and those at SFGH. Soon thereafter there



was a move here on the part of the administration of Davies Medical Center to establish an AIDS research program, and I initially resisted participating in that. I was the logical person to be involved from the medical staff because of my infectious disease background, but I resisted that because there was already a research program at San Francisco General Hospital to which our patients had access. The consortium mandated by Dianne Feinstein was in part founded to help expedite research and access.

But what would happen is patients would go over to SFGH for research. The private providers would never see those patients until they got very sick, and then they'd all of a sudden appear back on our doorstep. There was no communication going on about the sort of day-to-day research visits and lab results from SFGH to the providers. And so yes, in part in order to keep the patients within the system and get information in a timely manner, we did start a research program at Davies. That happened a little bit later.

Hughes: Is that a typical response of an academic institution? Or is it characteristic of San Francisco General itself, the fact that it doesn't seem to have a very good communication system?

Follansbee: I think it's typical. I think what's atypical is that San Francisco is such a small town.

Hughes: So the word spreads--

Follansbee: And so the word spreads fairly quickly. But I think that the universities in general, and I don't have a lot of experience with other universities, the UCSF is historically fairly insular. But I think San Francisco's response was also somewhat atypical. In San Francisco, this medicine was being practiced by general practitioners. In the rest of the country my infectious disease colleagues were looked to as primary HIV providers, both in the community, but a lot more of them were university-based. Infectious disease physicians tended to go to the universities or to the public health system.

The response of the private community in San Francisco was, I think, more rapid, in part because there were a lot of gay practices in San Francisco that were already up and established. A lot of those physicians were already involved, had academic appointments, were very well trained, and so they wanted to continue to practice in this community, to meet the needs of their patients. They had relationships with these

patients who were getting this new illness. They didn't want to just sort of send them off to the university or SFGH.

Bay Area Physicians for Human Rights [BAPHR]

Hughes: Let's talk about BAPHR because BAPHR played a strong role in the AIDS epidemic. You said you were a founding father?  
[laughs]

Follansbee: Yes, one of the founding fathers. Bill Owen is the founding father; the first meeting was at his apartment. But yes, I was one of the founding members.

Hughes: How did you think of it? Was it a medical organization, or what was it?

Follansbee: I considered it sort of a support group. My history at BAPHR happened because during medical school in Denver, as a gay man, I had a friend who was on the faculty, and we've remained actually quite good friends over the years. We founded in 1976 sort of a gay medical group in Denver, because one of the community physicians came out publicly and went on TV. We were watching this program thinking, My god, here's this doc on TV talking about being a gay man. And so we started talking about the people we all knew who were physicians and gay, and both of us knew quite a number of people, and so we organized a group. For about a year before I left Denver in June of '77, we met periodically and had social programs and would talk about what it was like to be gay and lesbian in the medical profession. It was sort of a support group.

At the last meeting I attended before I moved out here-- there were a couple of physicians from San Francisco. I said, "Do you have an organization in San Francisco?" And they said, "No, we don't need one." I said, "You don't need one?" They said, "No, everyone's so out that it's just a wonderful place. We don't really need an organization like that in San Francisco." Well, that's great, that's fine, I thought.

When I got out here in 1977, the community clearly didn't have one and clearly they needed one. [laughter] I mean, it was not nirvana in the medical community. I saw an article in what was called AMSA, the American Medical Students Association, newsletter that there was a gay physicians' support group forming. So I called them and then they gave me Bill Owen's name.

I remember the first meeting at Bill's. It was quite an eye-opener about just how badly this group was needed, because people were afraid to give their last names. And everyone around the room said, "It is okay for you to be here, but I shouldn't be here, because I'm a resident, and if my resident director ever found out, I would be kicked out." or, "Okay, you're a resident, you're safe, but I'm a fellow." And, "I'm in private practice; I'll lose my referral base." It was just bizarre, it was just totally bizarre to see everyone there talk about their own risk in participating.

So it was really a support group, mostly for men initially, but we always made attempts to bring women into the organization. Lisa Capaldini was involved quite early on as a medical student. So BAPHR was not really a medical group, although clearly, there was a medical side of it. But initially, it was just sort of a support group, to say, "Hey, listen, we're gay, and we're in this profession."

Hughes: What changed BAPHR from mostly a support group to a group actively involved with medical issues?

Follansbee: Well, I mean, there were always discussions of medical issues clearly relating to sexually transmitted diseases and homophobia and all gay-related medical issues. Again, I don't know how we got into the business of writing safe sex guidelines, but I actually chaired--I don't know the years--the scientific advisory committee. BAPHR had all these little committees, and so I chaired the scientific advisory committee. We came up with safe sex guidelines when we realized that AIDS sort of followed the pattern of hepatitis B.

We had these committee meetings. And it just seemed like there was some need to talk about how to take care of people with AIDS. So we had created algorithms: how to work up fever, which was the one I did, and how to work up diarrhea and other severe symptoms. These were very early attempts at trying to provide information to the medical community about AIDS. I'm certainly not the one who stimulated the need for this. I think requests for information probably came from the community physicians who were seeing people with AIDS all the time and wanted more information. And again, I don't know how

we got asked to come up with a pamphlet, or whose idea it was for a pamphlet on safe sex guidelines.<sup>1</sup>

#### BAPHR's Safe Sex Guidelines

Hughes: Were you involved in the debates over how the safe sex guidelines should be formulated?

Follansbee: Oh, definitely.

Hughes: Can you re-create some of the debate?

Follansbee: Well, again--[sighs] I remember a lot of the committee discussions. I seem to remember our holding meetings in the doctors' dining area at California Pacific Medical Center-- what was Presbyterian Hospital at the time. We had a room there where we could meet regularly in the evening. I don't remember where else the meetings occurred. This was probably 1983-1984.

It was very interesting, because again, we still didn't have an agent as a cause of AIDS, and we didn't have a test. We were talking about the way we seemed to think that this disease was being spread. My memory is that there was a lot of speculation. It was very difficult for some people to distinguish their own personal sexual preferences in terms of certain activities from what really we had information on. And we didn't have much information, so it was a lot of supposition. Therefore there was a lot of room for people to put what they did sexually into the safe category.

So it was a very odd discussion. We would talk about what we knew about hepatitis B and sort of try to apply it to our formulation of these guidelines. Then, of course, there was a lot of discussion about, well, could we say anything was safe? And what was the impact of these guidelines in terms of male sexuality and sexual expression? How could we do this?

I think that in a sense, the Sisters of Perpetual Indulgence did a better job in terms of coming up with a user-friendly pamphlet at the time. But their recommendations were

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<sup>1</sup> For more information on AIDS medical care and BAPHR safe sex guidelines, see the oral histories in this series with, respectively, Jim Campbell and Bob Bolan.

very much similar to the guidelines we came up with. I am sure that the archivist of BAPHR has the initial pamphlets and the dates they were written.<sup>1</sup>

- Hughes: By late 1982, BAPHR and the KS Foundation, which was the forerunner of the San Francisco AIDS Foundation, collaborated on a brochure on guidelines. Do you remember that?
- Follansbee: I remember that. Again, I don't remember the details or the specific logistics of that collaboration, because I don't think I was involved in the logistics of how the association between BAPHR and the KS Foundation worked. I'm sure that Steve Mehalko and Marc Conant were involved in the logistical side of it.
- Hughes: Were you in the habit of going to the National Lesbian and Gay Health Conference?
- Follansbee: No.
- Hughes: Then there was another conference in 1983 in Seattle. It was a symposium called Current Aspects of Sexually Transmitted Diseases. There had been two previous meetings, presumably a year apart.
- Follansbee: I don't think I attended any of them.
- Hughes: Again, risk reduction guidelines were discussed.

#### Community Response to Safe Sex Guidelines

- Hughes: Do you remember any specifics about how the community responded to these pamphlets?
- Follansbee: I remember pretty early on--and again, I just can't remember the chronology--trying to educate the community that you can't look at somebody and tell whether they have AIDS. There was this concern about informing people that these safe sex practices were something that really should be applied universally, and that it was dangerous to try to second-guess whether a partner was infected. There was an attempt to try

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<sup>1</sup> Over the years, BAPHR produced a number of pamphlets on safe sex guidelines. An example is reproduced and cited in the appendix, unfortunately undated.



to tell people that there weren't "safe" bars you could go to to meet people who weren't infected or types of men who weren't infected. It was to some extent entirely analogous to the bathhouse controversy, and the feeling that there should be information available to people which discussed frankly how this is acquired.

As far as the response was concerned, the majority of people, I think, were quiet and took the information at face value. It may or may not have been helpful in terms of an educational effort. There was no way to assess that. There was obviously controversy over the message, which was a political controversy over male sexuality and disease and homosexuality and the whole bigger picture. Was our safe sex message a positive statement or a negative statement, and were we telling gay men not to have sex? Which we tried not to do.

Hughes: Was that the main issue in people's minds? Did people ask: what right do you or anybody else have to say how I conduct my life?

Follansbee: Yes. I don't think there was a civil rights issue that I remember being discussed, because obviously they were only guidelines, and people could do what they wanted. In terms of the closing of the bathhouses, that became more of a civil rights issue. But in terms of the guidelines themselves, no.

I think that everyone felt that there should be guidelines, and the sooner the better. And the more we could distribute them, the better. And as you say, there were several brochures.

#### Bathhouse Issue

Hughes: Did you have a role in the bathhouse issue?

Follansbee: I had an opinion but not a role.

Hughes: What was your opinion?

Follansbee: I don't go to bathhouses; I never did go to bathhouses, so in a sense, it was no skin off my nose. Whether the city closed them or not. My concern was--and I don't think that it was a simple issue then or now--that we had to make sure that a public health response to the epidemic was appropriate and not just window-dressing.



The bathhouse issue became a very political issue. Bathhouses were offered a tremendous opportunity for education, and monitoring, and policing. And the bathhouse owners for the most part were pretty irresponsible and not in the forefront of AIDS activism. The success of their establishments really hinged on providing unsafe places for sexual activity.

Hughes: Because that's what drew people to the bathhouses.

Follansbee: Because that's what drew them. Bathhouse owners and patrons believed that if you turned the lights up and provided soap everywhere, the romance of this experience was somehow diminished. So it was not a simple issue.

I felt that the bathhouses should not be closed, that they offered a great place for education. You could pass out safe sex pamphlets there. That despite the fact that, yes, one could go to the bathhouse and have a lot more partners than one could by meeting someone in a bar and going home with him. And clearly, this risk of disease transmission or acquisition is directly related to the number of exposures. Nevertheless, even if the city closed the bathhouses, men would still have a lot of exposures in other ways, either in parks or highway rest stops or bathrooms or somewhere. And you can't put pamphlets in parks and these other locales where men would go as an alternative. We needed to educate them; this may be the only way we could educate some people, the only opportunity to affect behavior and lower risk. Many men using bathhouses weren't necessarily "out", and being educated or reached by the safer sex guidelines in other ways.

Hughes: On the other hand, by doing something such as putting educational material in the bathhouses, you could be seen as ruining the experience.

Follansbee: Right. There wasn't ever data that the bathhouses would ever be an effective place for education.

Hughes: I mean, you could argue simplistically that people don't want to go to bathhouses to be educated about sex, that it's not a place where they're going to be receptive to that message.

Follansbee: In no place are those people going to be receptive to education about sex, but at least in the bathhouses it could be mandated by the public health department. We could at least get the bathhouse operators to provide safe sex pamphlets. I guess it's probably the same issue as legalization of prostitution and other social public health

issues; the way to monitor public health may be to legalize it and then regulate it.

Follansbee: So I understand Merv Silverman's concerns: on the one hand, there was the public health and needs for education and on the other hand, how strong was the evidence there was a single infectious agent being transmitted in bathhouses? We knew it was sexually transmitted, but was it through the use of drugs that were being shared or by some other vector? Could Dr. Silverman legally close the bathhouses on the evidence to date? And would it really decrease transmission?

Hughes: You, as an I.D. specialist, had probably already seen a number of infectious diseases in the gay community before the epidemic came along.

Follansbee: Right.

Hughes: From hindsight, it seems such a given that an infectious agent would be involved in causing AIDS.

Follansbee: Right. It does seem like a given. It's hard to reconstruct the concern of the time. The question is, was the epidemiology of the disease strong enough legally to withstand court cases brought against the city for a public health mandate closing the bathhouses? We were obviously operating under the assumption this was a transmissible, infectious agent. Blood banks have lost money over this issue, i.e. with the assumption that they thought that there was an infectious agent. Did they do enough to screen blood products to make them safe for recipients? Hindsight is always a little easier for all of us, I think.

Hughes: You've already done a good job at explaining how complex it seemed at the time, with all the different etiologies that were possible.

#### More on BAPHR Committees

Hughes: You were also on the BAHPR AIDS Task Force, which I believe was first named the BAPHR KS Task Force. Do you remember being on that committee, and how defined are these committees within BAPHR?

Follansbee: You know, I probably remember being on it. I don't remember who else was on it; I don't remember what we did. It was all

volunteer. Everything was sort of "ad hoc" without a lot of infrastructure support.

Hughes: And so either you turned up at a meeting or you didn't.

Follansbee: I remember going to meetings and pretty much trying to turn up as much as I could. I don't even know if we had minutes, and who would have taken them. We didn't have any real clerical support.

Hughes: There were minutes; I've looked through the BAPHRONS--the newsletter for BAPHR--and there are often reports from the committees.

Follansbee: Reports, yes, written by various committee members as recommendations were finalized. I just don't remember all the issues we dealt with, what our mission was, how we usually prioritized our issues--except to say that we had committee members spearhead writing drafts on issues in cases where they had particular knowledge or expertise.

#### BAPHR Physicians' Experiences with AIDS Survey, 1983

Hughes: In 1983, BAPHR started an AIDS survey in which physicians contributed their experiences with patients with what was now being called AIDS. I think this form which I got from Dr. Bolan's papers must have come from that. [shows form] Does that look familiar to you?<sup>1</sup>

Follansbee: Yes, it definitely looks familiar.

Hughes: And is it for surveying doctors' experiences with AIDS?

Follansbee: Yes, I think we tried to come up with a registry, or some sort of an observational database. Yes, I definitely remember this.

Hughes: Did you participate?

Follansbee: Yes, I did participate in this. I don't remember where these questionnaires went or what we did with them. I think again it was one of these ideas that just seemed like a good idea.

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<sup>1</sup> This document is reproduced and cited in the appendix of this volume.

Obviously, we designed it to be computerized because of the numbering system, but I honestly don't remember what happened to this, where this went.

Follansbee: This was '83.

Hughes: Does the form indicate a more specific date?

Follansbee: No.

Hughes: The information that I have came from the February 1984 BAPHRON, and that article referred back to the study beginning in 1983, so I can't be any more specific than that either.

Follansbee: Yes. I honestly don't remember much.

Hughes: The application for the first large AIDS grant for research at UC was being talked about in late 1982, early 1983. The one for which Paul Volberding eventually became P.I. [Principal Investigator]. Because the money came in and a group of academics now had the capability to do some concerted research on this disease, did it become less important for the community physicians to have their own project? Would that have affected your thinking?

Follansbee: I honestly don't remember that at all. Again, the formation of the Community Consortium was in part because there was not communication about a lot of this. The people in the community didn't always know what was going on in the university. If the AIDS survey failed it was probably more a function of we just weren't quite sure what we were doing with the information: who was supposed to be culling this, who was going to use this, where was it going to go, and how it was going to be summarized or updated.

BAPHR and the San Francisco AIDS Community Consortium ##

Hughes: The picture I'm getting from you is that because of the epidemic, BAPHR takes on a more medical-problem-oriented role. Here was a group of people who not only were physicians, but they were gay men. How could you ignore what was going on?

Follansbee: Yes, you're absolutely right. BAPHR did assume a role, and it did attempt to respond to the epidemic. I think that you're right also that whatever role it had did eventually get replaced. If I had to say what replaced BAPHR's role, I would

have to say it was the Consortium. The Consortium really offered the forum for private docs to meet and talk and think about what was going on in a way that BAPHR didn't, couldn't. BAPHR had regular monthly meetings, and we would have rounds and talk about issues or medical problems. That function really did get replaced by the Consortium, and not the KS Clinic.

The AIDS Clinic at UCSF and San Francisco General Hospital [SFGH]

- Follansbee: I'm not sure when the KS Clinic folded. What year was that?
- Hughes: I actually haven't been able to pin that down. There were still meeting notices going out in 1984. Would the opening of the Adult Immunodeficiency Clinic indicate the closure of the KS Clinic?
- Follansbee: I don't think that would work, because I think there was a gap. The Adult Immunodeficiency Clinic was a program within the Department of Infectious Diseases that started at UCSF a year or two after I joined Ward 86, the AIDS Clinic at SFGH, which was July 1983.
- Hughes: So the KS and the Adult Immunodeficiency clinics wouldn't necessarily be in conflict.
- Follansbee: Right. I think that the closing of the KS Clinic probably was more of a personal issue between Marc Conant and Paul Volberding. As Paul got busier at SFGH and that program expanded, Paul and Marc went their separate ways and the program at SFGH became much more the focus.
- Hughes: Do you know any more about that?
- Follansbee: I don't have any particular insights into this process. However, Donald Abrams and Connie Wofsy joined Paul in 1983. As his staff built up, it was natural to see the focus shift to SFGH.
- Hughes: Before the opening of the Adult Immunodeficiency Clinic, Conant became head of the ACRC, the AIDS Clinical Research Center. Funded by the UC State Task Force on AIDS. There was also an ACRC opened at the same time at UCLA. The State Task Force on AIDS was one of the agencies funneling state funds into university AIDS research.



Follansbee: This AIDS state task force is the program that Merle Sande takes credit for.<sup>1</sup> He lobbied for it, I think.

Hughes: Right, and Sande became head of the first committee of the AIDS state task force.

Hughes: In a sense, this doesn't have very much to do with you.

Follansbee: Right.

American Association of Physicians for Human Rights [AAPHR]

Hughes: Did you belong to AAPHR [American Association of Physicians for Human Rights]?

Follansbee: Off and on, I belonged to AAPHR. Some years I do, some years I don't. I guess for my own personal needs, a national organization doesn't have as much impact. Because of my infectious disease associations and contacts, I have not had a lot of need to belong to AAPHR. I don't get any fulfillment or sense of identity from that association.

As you know, BAPHR has gone through many transitions and reincarnations. Lenny Simpson clearly had the vision of BAPHR really dying out or becoming a shell, with AAPHR really taking on a major national, political role in its place. I think he was very successful in that process, transitioning BAPHR into a national association, AAPHR--I believe in the early 1990's. I think it was appropriate in many ways in terms of the politics around not only HIV medicine but gay-lesbian medical issues.

But I guess that I just can't have my fork in every bowl, and so I just have not really been active ever in AAPHR. I've gone to some of the symposiums as a speaker, and AAPHR will have a reception at the infectious disease meetings, and I'll go that, but no, I'm not really involved with AAPHR.

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<sup>1</sup> See the oral history with Merle Sande, M.D. in the AIDS physicians series.



Infection Control Practitioners at UCSF and SFGH

Hughes: Well, let's turn to Conte's infection control committee. Could you talk about how that got off the ground?

Follansbee: John Conte has for a long time been in charge of infection control at UC. He was my mentor as a fellow at UC [1980-1982]. I think he was very instrumental in responding to the infection control issues. There were a couple of very dynamic infection control practitioners. One, who's a friend of mine still, named Linda Rosendorf, is just worth her weight in gold in terms of her contacts and the role she took on to develop a national infection control response to this epidemic in hospitals and medical care systems. Her colleague at San Francisco General was also worth her weight in gold in terms of the issues around infection control.

Hughes: Was that Grace Lusby?

Follansbee: Grace Lusby.<sup>1</sup> These infection control practitioners formed a very active nucleus. I think they were in some sense the driving forces to the programmatic development of appropriate local infection control procedures, that I think became really state-of-the-art, became national guidelines, and really have not changed over the course of this epidemic.

I remember having the first patient with Pneumocystis pneumonia in a four-patient room in 1982. We, the medical staff at UCSF, did not recognize that Pneumocystis in this patient might be causing his immune problem, that there might be an underlying transmissible agent. We just knew he had pneumonia. We weren't worried about having other people in the room, so he was not placed in isolation, either for his own benefit or the benefit of other patients or staff. Until there were more cases, we just didn't consider those issues.

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<sup>1</sup> See the oral history with Grace Lusby, R.N. San Francisco AIDS Oral History Project: The Response of the Nursing Profession, 1981-1984. Regional Oral History Office, The Bancroft Library, University of California, Berkeley. Hereafter, the AIDS nurses series.

Merle Sande and the UCSF AIDS Task Force

Follansbee: Then Merle Sande headed the UC [AIDS] task force that came up with the UC infection control recommendations, which were published.<sup>1</sup> I was already in private practice by then, 1982-1983, but I went to those infection control task force meetings, mostly held at SFGH. I remember some of those meetings very clearly.

Hughes: I understand at times the discussions about infection control guidelines got quite heated.

Follansbee: Yes, the discussions were heated. Merle Sande really played an important role as a very effective chair for that committee. I think, from my own impressions, Sande learned a lot as well.

Infection Control and SFGH's Ward 5A, 1983

Follansbee: I'm trying to think when [Ward] 5A<sup>2</sup> was coming into existence.

Hughes: The formation of 5A was being discussed in early 1983. It didn't actually open until the summer of 1983.

Follansbee: Right. Merle Sande's vision for 5A was of an isolation ward. This would be a ward where there was a big sign in the hall, and there would be sinks and gown-changing areas before you ever walked into 5A. I don't know if you've walked the halls of San Francisco General Hospital, but you can't see the entry doors from the long halls. You have to walk down the hall until the little side turn before you get to the doors. But he was going to set up this isolation area, where you would be warned of its status by a big sign as you walked down the hall and have to enter through a changing area.

I don't know whether Sande was playing sort of the devil's advocate just to make sure that the committee really

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<sup>1</sup> John E. Conte, W. Keith Hadley, Merle Sande, et al., Infection-control guidelines for patients with the Acquired Immune Deficiency Syndrome [AIDS], New England Journal of Medicine 1983, 309: 740-744 (Sept. 22).

<sup>2</sup> 5A, the in-patient AIDS Ward at SFGH, was originally named 5B.

did look at every issue or whether he really believed what he was proposing, but Sande's ideas or proposals became a focus or launching pad for discussion. He prompted the committee to think and to explain all options. We had to examine what we really knew about this disease, having taken care of patients now for a while? So, he was a very good chair, because he really did make sure that every issue was examined in detail. The design of that unit [5B], which was really quite different ultimately than his proposal as an isolation ward, really came out of very hard discussions.

Some of those discussions involved an orthopedic surgeon by the name of Lorraine Day. At some stage, she would come in to those infection control task force meetings. I remember some of the issues, because they involved how we should be protecting ourselves as health care personnel. Lorraine Day was the spark for a lot of discussion regarding HIV. This was way before there was an identified virus, but we were thinking along the lines of hepatitis B, but she also was concerned for CMV[cytomegalovirus]. I think she was worried about CMV infection in health care personnel because she recognized, as we all did, that these patients were all infected with CMV.

Follansbee: So they were very lively discussions, and there was representation from the VA Hospital, as well as from John Conte at UC and from San Francisco General. The task force meetings all occurred at San Francisco General Hospital.

Hughes: But Conte himself is a UC person.

Follansbee: Conte himself is in infectious diseases at UC.

Hughes: Did the participation of UC personnel in the AIDS infection control task force seem legitimate to you, since, by that point, the AIDS care scene was beginning to shift to San Francisco General Hospital? In January of 1983, the AIDS Clinic opened at San Francisco General.

Was it still not clear where AIDS activities were going to be centered at the university?

Follansbee: No, I think it was clear that there were still going to be AIDS patients at all the campuses, and so that's the reason why all the infection control programs were involved: the VA, UC, and SFGH. There needed to be consistency in approach at all the UCSF campuses.

Physicians Involved in Infection Control at UC Hospitals

Hughes: I'm getting the committees mixed up. Sande was head of what's called the UCSF AIDS Task Force. What was Conte head of?

Follansbee: Conte was the head of infection control at the UCSF hospitals on Parnassus. Moffitt Hospital and UC Hospital, much like Dr. Peter Jensen was the head of the VA program. So Peter would come over to San Francisco General for these meetings of Sande's task force, along with their infection control practitioner.

Hughes: And yet when the paper is published in September, 1983--in the New England Journal, Conte was first author.

Follansbee: I think that is because he wrote the drafts for review, and so therefore he would be the first author because he wrote it. Merle Sande was not in fact the head of the infection control program; he was chief of medicine. Now, Sande's an infectious disease physician and very well respected, but he was not the actual head of the infection control program at SFGH. The head of the infection control program at SFGH may have been Dr. Keith Hadley, a microbiologist and head of the SFGH microbiology lab.

Hughes: Keith Hadley I know came to those meetings. He was on the task force.

Follansbee: Right, because he was head of the micro lab at SFGH. He may have been head of the infection control committee at SFGH as well. I don't think John Mills went to those meetings on a regular basis. I'm trying to remember who was there. Phil Hopewell I think was there as a pulmonologist. Connie Wofsy I think went as well.

John Conte's Ad Hoc Advisory Committee on AIDS

Follansbee: There are several things going on at the same time in UC Infection Control. The Ad-Hoc Advisory Committee on Acquired Immunodeficiency Syndrome produced this paper of March 8,

1983.<sup>1</sup> From June 1982 to June 1983, I had a part-time job as an assistant in the epidemiology program or infection control program at UCSF. This position was I think 10 or 20 percent time, I don't remember exactly. John Conte, as chair of the infection control program, convened this ad-hoc committee to look at infection control guidelines for UC. In this paper he reports that there's a task force chaired by Dr. Sande at San Francisco General Hospital to develop guidelines as well. Also, he reports that they're [SFGH] thinking about developing an AIDS ward, and because a lot of these patients were getting bronchoscopies, they needed to write standards for infection control in this setting as well--for sterilizing equipment, handling specimens, etc.

So I think that Conte's committee got subsumed in some way by Merle Sande's committee, which became an overall UC task force. And, yes, Sande's task force did include Peter Jensen from the VA; myself representing UC; Keith Hadley, Connie, Phil Hopewell, who was a pulmonologist at San Francisco General; Paul Volberding, and then Mr. Rankin, who I think was a hospital administrator at SFGH. I think he's still there.

#### UC Infection Control Practitioners

Hughes: Now, the advisory committee led by John Conte that got subsumed by Merle Sande's UC AIDS Task Force is yet distinct from another committee, the one on which I believe Linda Rosendorf but certainly Grace Lusby served.

Follansbee: Right.

Hughes: Some of the principles for AIDS infection control were actually formulated first by Linda Rosendorf and Grace Lusby and the other members of the infection control committee at SFGH.<sup>2</sup>

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<sup>1</sup> Ad Hoc Advisory Committee on Acquired Immune Deficiency Syndrome, [Minutes], March 8, 1983 (Marcus Conant Kaposi's sarcoma notebook, 1983, AIDS History Project, UCSF Library).

<sup>2</sup> For more on formulation of the first AIDS infection control guidelines, see the oral history with Grace Lusby in the AIDS nurses series.



Follansbee: I think the infection control practitioners from the UC hospitals, the VA, and from SFGH met independently. They knew each other; they're still friends. Jackie Octavia from UCSF and Linda Rosendorf remain great friends to this day, and Helen Schietinger, who was actually the nurse for the UCSF KS Clinic, remains good friends with Linda to this day. Linda and Helen live in Washington, D.C. And Carol Viele I think is still at UC. She was involved as an oncology nurse. Many of the nurses and infection control practitioners met on their own and formulated the foundation for policy and clinical care.

Now, there is a Bay Area infection control committee that infection control practitioners go to. Linda Rosendorf is not a nurse; not all infection control practitioners are nurses; they're infection control practitioners. They met regularly and on an ad hoc basis and were instrumental at UCSF in policy development.

Grace Lusby and I went to a meeting in Denver in '83. I think it was the Gay-Lesbian task force meeting, where we chaired a panel discussion workshop on infection control. Talk about controversy! I mean, we came in with a fairly well formulated set of guidelines, all of which were along the model of hepatitis B infection control. And some people, particularly people from New York City, were just adamant that they had a better approach. They came up with really much more aggressive, sort of complete isolation recommendations. So in this huge room with a lot of people, it was very controversial. I remember the anxiety and intensity of discussions around that process, trying to forge a consensus in that national setting.

So these infection control initiatives were all going on somewhat simultaneously. I personally at least--I can't speak for some of the other physicians--really relied on Linda Rosendorf, and to some extent Grace and Jackie Octavia, to help us, because infection control was their full-time job, and we were just docs. [laughs] So I think that a lot of the response to these issues and the alliances were developed privately in order to support the evolution of infection control guidelines and guidelines for AIDS care.



Priority in Formulating AIDS Infection Control Guidelines

Hughes: Grace Lusby, and probably some of her peers feel that the effect of the Sande committee was to overshadow some principles that had actually predated the Sande committee's work. I mean principles specifically in regard to the care of AIDS patients.

Follansbee: At the time of the Sande task force, there were infection control programs in place based on hepatitis B. There were proposals from Centers for Disease Control. There was a lot of controversy about things like, Should pregnant nurses be caring for people with HIV? and, What kind of isolation techniques should be used? and how to handle equipment, and all that.

Hughes: The CDC published guidelines dated November 5, 1982.

Follansbee: Yes. Again, these CDC guidelines follow the hepatitis B recommendations. I think the discussion in the Sande committee was really, Is this enough, or should there be more infection control? I recall that the initial proposal by Sande's task force was that this was not enough, that there needed to be more specific guidelines to protect against AIDS transmission. But these CDC recommendations were what was followed, and these were in effect.

I don't know if Grace or Linda Rosendorf have some publication that came from a group other than CDC, and that did go into the detail of the New England Journal article, that they could say really became the standard first. Obviously, a physician-heavy committee with Merle Sande as the chair is going to overshadow the groundwork of other organizations. But, I think it was simply groundwork, and there was not a consistent UC policy that covered all campuses equally before the Sande committee came up with their infection control guidelines. This was the committee that did that in a formalized way to make sure that there was a unified and uniform response.

Hughes: So in one sense, what the New England Journal<sup>1</sup> paper did was to refine what the MMWR had come out with, which was basically

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<sup>1</sup> "Acquired Immune Deficiency Syndrome (AIDS): Precautions for Clinical and Laboratory Staffs," CDC Morbidity and Mortality Weekly Report, 31:577-580 (November 5, 1982).

the hepatitis B model, and to make the infection control procedures more closely attuned to the needs of AIDS.

Follansbee: Right. I haven't looked at this CDC November '82 directive. I haven't reviewed our article in the New England Journal, but I remember principles like needle boxes and gloves in all the rooms. All these kinds of precautions were the nitty-gritty of AIDS infection control that had to be worked out. How can patients be assigned to an AIDS unit without these details established? Some of this concern about infection control procedure was anticipation, of course, of the AIDS ward at San Francisco General, as well as anticipating there would be larger numbers of patients.

Hughes: Right. That was definitely an issue.

Follansbee: But also, AIDS infection control just became an issue for everybody.

#### Fear of AIDS Transmission to and by Health-Care Workers

Hughes: And by then, too, you were dealing with a certain amount of fear within the hospital.

Follansbee: Oh, a tremendous amount of fear. Yes, there was a tremendous amount of fear that we all had intermittently, and not really knowing who was infected, if we covered all modes of transmission, and even whether we had already been exposed and infected.

I can remember Connie Wofsy at one point talking to me-- we were at the Ward 86, HIV infectious disease clinics. She had been asked by Paul Volberding in June of '83 to put together the first AIDS infectious disease clinic. I think Thursday afternoon was the infectious disease clinic at Ward 86 that Connie and I co-attended, and so we were both there. For a while, we essentially knew everyone who had AIDS and serious infections at San Francisco General Hospital, so Connie and I had a lot of contact with each other. That changed, obviously, over the years.

I remember at one point Connie asking me if she thought that she should ever donate blood. I said, "Why?" And she said, "Well, because here I am seeing people with AIDS all the time. How do I know?" And by then, the guidelines [for blood donation] had come out, and I wasn't donating blood because I

was a gay man. I already fit the criteria for not donating blood. She was asking me if I thought she should, as a health care worker exposed to AIDS patients, also not donate. I said, "I think you should. I think that our guidelines work, and unless you have some reason to think otherwise, that you need to buy into it and trust yourself and our guidelines." I recommended that if she didn't want to donate, she shouldn't, but she should not exclude herself because she was an AIDS physician since we didn't exclude other health care workers as blood donors.

So we all had that fear periodically.

Hughes: You yourself?

Follansbee: Yes. I never knowingly stuck myself or unknowingly was exposed. I never recognized an opportunity as a health-care professional where I could have been exposed. I've not had a needlestick, even though I do spinal taps all the time, and I'm frequently drawing blood.

Hughes: You're implying that it was pretty well assumed that this was not an airborne infection.

Follansbee: Right.

Hughes: And that was just from experience?

Follansbee: That was just from experience?

Hughes: Nobody had gotten it.

Follansbee: Yes, it was pretty clear. My line when I was lecturing about this was, "If this was a disease that was passed through kissing or airborne, then it would be a disease of gay men and their mothers." [laughter] I mean, we had had enough experience; we already knew this. There would have to be an extraordinary change in the epidemiology that would have moved this disease in a different direction.

Hughes: Apparently, one hot discussion point was, What do you do with health-care workers who are infected?

Follansbee: Yes, I remember those discussions. Again, as with the bathhouses and all that, there was a lot of discussion about civil rights. And people asked what were the kind of scenarios where there would be transmission from an infected health-care worker to a patient. I think that our concern became very clear as the discussion evolved, that there was no

risk to patients from health-care workers. AIDS was not casually transmitted, and the kinds of scenarios that would have had to happen would have been extraordinarily unusual.

That was controversial because of hepatitis B. There are obviously epidemics of hepatitis B that have been related to health-care workers. So there was a lot of controversy over what we could do. We were concerned that HIV infected health care workers may be exposed to the infections of AIDS patients and we were concerned about the kinds of problems the health care workers could acquire in this way. That was relatively easy to deal with. But the other question, about taking HIV-infected health care workers out of direct contact with patients, was harder. I think that the conclusion, as I remember, was pretty well thought out. I don't think we took a stand--or, at least, a strong stand. I think we said it was a very individual decision.

Hughes: Yes, you said in the New England Journal article that it was a subject of hot debate, and you couldn't reach a decision.

Follansbee: Right, we couldn't reach a decision, and that the decision had to be individualized. I think it left people open to dealing with it on an individual basis, which was the bottom line, when I actually think about it in terms of the information that we had at the time.

The AIDS Outpatient Clinic (Ward 86) at San Francisco General

[Interview 2: September 6, 1996] ##

Clinics in Which AIDS Patients Were Seen

Hughes: You mentioned last time that in June of 1983, you and Connie Wofsy were asked to form an infectious disease clinic. Was it Volberding who asked you?

Follansbee: Yes. Paul Volberding and his nurse--She's at San Francisco General.

Hughes: Gayling Gee?

Follansbee: Gayling Gee, yes. Volberding and Gee had already started an AIDS Clinic at one end of Ward 86, a unit in an old building which looked like it had been bombed out. Then, in June of

'83--I believe that's the right date--Donald Abrams moved his lymphadenopathy clinic from UC to General. And, recognizing that there were people with the syndrome who had infectious complications, Connie Wofsy joined the AIDS program at SFGH.

I joined the AIDS program at SFGH as a part-time employee. I think I had a 20 percent salary as what they call an assistant physician. It turned out that in one of the two clinics that I staffed beginning in July of '83, Connie co-staffed with me. I don't know, but I suspect that the creation of the infectious disease clinic was by design to some extent and de facto in other ways. Because we, Connie and I, were both trained in infectious diseases, the clinic became sort of the infectious disease clinic, where the AIDS patients with the complicated opportunistic infections were referred from the oncologists.

Hughes: You are calling it an infectious disease clinic, but it was an AIDS infectious disease clinic?

Follansbee: It was an AIDS clinic. It was really the AIDS Clinic at Ward 86. It just so happened that the focus tended to be on the infectious disease aspects of AIDS. It was not called the infectious disease clinic. It was not even in the Division of Infectious Diseases.

Hughes: At the hospital, it was classified under AIDS Activities.

Follansbee: It was under AIDS Activities, under Paul.

Hughes: What was the second clinic?

Follansbee: Well, I think it was just a general AIDS clinic.

Hughes: Where you saw patients with every kind of manifestation of the disease?

Follansbee: Where I saw all the AIDS patients. But I don't think it had the same narrower focus as the Thursday afternoon clinic. I think it was a Thursday afternoon clinic that Connie and I staffed, and became most focused on infectious disease complications.

Hughes: Were others such as Volberding involved in that general clinic?

Follansbee: Yes, although I don't know if Paul ever worked the same half days as I. Certainly my role evolved over the years, but initially I tended to act by doing a fair amount of primary



care in that general AIDS clinic. At the same time, I was trying to act as an attending physician, because there were always nurse practitioners and resident physicians there. J. B. Molaghan was there when I started, or just after, and Gary Carr<sup>1</sup> started soon thereafter. So I tended to supervise the nurse practitioners and house staff that would come through the clinic.

Hughes: There were several clinics that seem generally to have fallen under the rubric of AIDS clinics, like the I.D. clinic. Were the distinctions eventually abolished, and did Ward 86 become an AIDS clinic that didn't make distinctions between the several sub-clinics?

Follansbee: I don't remember if there was ever a formal distinction. There is an official Infectious Disease Clinic that still is very active, but it's in the main building, not in Ward 86 where the AIDS clinic is, and administered by SFGH and not UCSF. They do see some HIV-related problems. Historically, they didn't, and so I just think that it was a natural distinction that the HIV-related infectious complications were seen in the AIDS program.

Hughes: Why were AIDS patients seen in the Infectious Disease Clinic?

Follansbee: I think there are probably a lot of reasons, part of which was that there was another way to do it, which was to send people to the AIDS Clinic at Ward 86. Probably there was some feeling at San Francisco General that [AIDS] was not the focus of infectious diseases per se. Connie was initially somewhat unique, I think, in the Division of Infectious Diseases, recognizing that this taking care of AIDS patients with infectious diseases was something that needed to be done by infectious disease practitioners as well.

### Stigma

Hughes: Was there stigma involved in some of the hospital's decision-making?

Follansbee: Now, you're getting into the area of hearsay to some extent in my mind, because I felt that there was some stigma. There was a rumor that somebody who applied to the infectious disease

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<sup>1</sup> See Gary Carr's oral history in the AIDS nurses series.



program was told by the new chief of medicine at the time when he identified himself as being interested in AIDS as an infectious disease, that this was not really a worthwhile activity. Studying AIDS was something that that particular faculty member felt was not academic enough or not within the appropriate realm of infectious diseases. He, the applicant ended up going to another program to do his infectious disease training because he didn't feel that SFGH was a fertile ground for his particular focus.

I think there was some stigma involved towards this disease in the more classical areas of medicine, for whatever reason. I'm not sure what the reasons were.

#### Lack of Creativity with the Inpatient Unit

Hughes: Were you also seeing patients on Ward 5B, the inpatient unit?

Follansbee: No, I did not have inpatient responsibilities. Part of my concern was, early on in particular, that there was really no liaison between the outpatient and inpatient services. I tried to advocate for a liaison service to try to bridge that gap. But directly, no. I would go over to Ward 5B to see patients, to see how they were doing, to follow up, but in terms of having any responsibility, no. I would occasionally put a note in the chart of a patient in Ward 5B to say, "This is what I know; this is what I think is going on; this is where we should be going." But in a formal way, I did not have responsibility for the patients in Ward 5B, especially since I was on campus only two half-days a week. It wasn't a very satisfying role in terms of continuity.

Hughes: Why wasn't there continuity between the clinics?

Follansbee: I don't know. I think probably it just had to do with how busy people were. I think that the model of care at San Francisco General historically was not one of continuity in the various services. As a physician one rotated through clinics and emergency room and wards, and one's focus changed every time. There was not the opportunity built into schedules to follow patients through levels of care easily. In addition, the faculty at SFGH saw themselves more as teachers or researchers, with patient care playing a somewhat subservient role. I think that's changed. I think it's had to change, in part because our focus on hospitalization has changed over the past two decades. Now the faculty see

themselves much more as "community providers," at least at SFGH.

Hughes: Would a factor in the lack of continuity of care between the units be that the AIDS Clinic was a medical unit, a UCSF unit, whereas Ward 5B was a nursing unit and, hence, a county unit?

Follansbee: I don't think so, not at first. I think maybe if there was any factor, it was just the geography. The AIDS program, Ward 86, was in a building a block and a half away from the inpatient unit. So I think that the distance was probably more of a factor than the distinction between the UC program and the county program. All the faculty at San Francisco General are UC faculty.

I remember when all the phones changed. I don't remember what year it was, but we used to have San Francisco General prefixes, the first three digits of the phone numbers and, all of a sudden, we got UC phones. I remember what a big deal that was, because you could never dial direct anymore to an extension at SFGH. You had to get an outside line and dial through a 7-digit number.

#### Growth in Number of AIDS Patients

Hughes: Is there anything particular to say about the actual day-to-day operation of the I.D. clinic that you and Connie were involved with?

Follansbee: I think the major issue was one of growth. Connie and I at one point knew all of the AIDS patients with infectious disease problems. We talked about them. And, I can say that I knew personally everyone in the AIDS program when it was all at one end of Ward 86. So as the program grew, number one, I knew fewer and fewer of the patients. And, secondly, now I know fewer and fewer of the people who actually work in the program. The AIDS program at SFGH is so big and split in different locations.

But in terms of the day-to-day operations, it was certainly exciting and challenging as we wrestled with clinical problems. Connie started the Pneumocystis for Lunch Bunch that I tried to go to, but I just really couldn't get to. Because by then, I was busy with my own practice, and so it was hard to go over to SFGH for that special meeting.

## Treating AIDS-Related Infections

### Pneumocystis Pneumonia

Hughes: PCP was a problem, was it not, in terms of treatment?

Follansbee: Pneumocystis pneumonia, I think, always had sort of a unique role, even at the very beginning when we had very few treatments for most of the other problems associated with AIDS. We had more treatment options for Pneumocystis than we had for some of the other infections. No one really knew how to treat disseminated Mycobacterium avium complex infection. Disseminated disease was quite rare, and some physicians would argue whether it was treatable at all.

So Pneumocystis always had that role that, yes, we knew it could be treated. But then we had problems with access to medications, as pentamidine was more or less available over the years. And then, we had problems with the toxicity of those medications. Obviously, Pneumocystis was easy to identify from the very beginning, because it was already well established in the medical community as a diagnosis. So, there was a lot of focus on Pneumocystis early on. PCP seems to be less of a problem now, because we prevent it better.

Hughes: Would you say that PCP was the dominant opportunistic infection that you were seeing in the early days?

Follansbee: I would say that as an infectious disease physician, it was the dominant one. There were certainly more minor infections that you could encounter, such as candidiasis, fungal infections, which were probably more prevalent but clearly didn't have the serious consequences of PCP. So Pneumocystis was the dominant one.

### **New Opportunistic Infections**

Hughes: Were you continuing to see new infections--I mean new to you--or had you seen most of these opportunistic infections in some form prior to the epidemic?

Follansbee: Oh, no, that's the excitement of it: to be part of this process of identifying not only new pathogens but new

manifestations of old pathogens that we just hadn't seen functioning or presenting in that way. We now think nothing about doing blood cultures for Mycobacterium, to find out whether it is tuberculosis or MAC [Mycobacterium Avium Complex]. Well, that was unheard of before the epidemic. Occasionally people documented bacteremia during acute infection with tuberculosis, but otherwise, it was unheard of. And the cryptosporidium, microsporidium--all the intestinal pathogens--and the explosion of fungal diseases that we see in various forms, represent new human pathogens in the case of microsporidium, or new diseases from older pathogens.

So I would say it's just really been an explosion of new manifestations. Obviously, the editors of the infectious disease journals still find HIV-associated opportunistic infections fascinating, because the journals are still filled with case reports of AIDS patients with new manifestations of old pathogens or new pathogens.

Hughes: Case reports are sort of a barometer of the state of knowledge in the field?

Follansbee: Yes, right.

Hughes: You wouldn't publish a case description if it was a well-established observation.

Follansbee: Right, absolutely. Also I think discovering new infections is to some extent what keeps AIDS doctors humble but also excited about keeping our eyes open--every problem is a potentially new problem.

#### Searching for Treatment Protocols with Cooperative Patients

Hughes: How do you approach a new medical problem? Was there a standard course that you took?

Follansbee: I can remember distinctly, especially when you go back to the first cases of Pneumocystis--as I think we've mentioned before. Those first patients all got tetracycline, because we all thought, "Well, a young man, middle-aged man, thirties, forties, pneumonia, infiltrates. Must be mycoplasma or something that we can't culture. Give them tetracycline and they will all get better." These people didn't get better. Of course, there was a little delay in our recognition that we were dealing with some new disease, because we thought maybe

tetracycline was causing some problem, and of course it wasn't.

I think we recognized early on that we needed to work harder to get to the bottom of problems faster. It's the patients who have been heroic. They have put up with more biopsies and invasive procedures; they have been willing to undergo brain biopsies, bone marrows, liver biopsies. Patients underwent just procedure after procedure in attempts to get to the bottom of what was going on. Patients recognized that the list of possible diagnoses for problems was so long and that we didn't even know how long the list was. We had to try to keep searching, and patients knew they were part of this search and agreed to participate by undergoing these procedures. So if there was one overriding approach to these new infections, it was to keep looking. It was also important to not get trapped into thinking, Well, I saw a case of "\_\_\_\_", this must be "\_\_\_\_", and treat presumptively. It was important to not just keep treating these new diseases based on past experience, but to keep an open mind to the possibility of new diagnoses.

We, in the BAPHR, would come up with algorithms for how to work up fevers, how to work up swollen lymph nodes, how to work up pneumonia, work up diarrhea. But when you look at these algorithms, they were such scanty outlines. Our algorithms acknowledged that we didn't know what we were looking for, but we often put people through tests.

### An Early Treatment Protocol

Hughes: I want to show you a document, which is undated, titled "Guidelines for the Evaluation, Therapy, and Follow-up of Patients with the Epidemic Immunodeficiency Syndrome, Especially PCP".<sup>1</sup> I am interested in the title itself: the fact that you didn't actually use the term AIDS. This may be simply due to the date that this was written. Let me show you the document. [tape interruption] Does that bring back any memories?

Follansbee: It does.

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<sup>1</sup> This document is reproduced and cited in the appendix of this volume.



Hughes: Just to refresh your memory, the term AIDS was coined sometime in the spring of 1982. It was being used at BAPHR meetings, usually with periods after each initial, at this time. Which may date this document, but there may be another reason why you avoided using that term.

Follansbee: It looks like it is something that came out of San Francisco General. The only reason I say that is that there are beeper numbers for people at San Francisco General, and Keith Hadley's number in the microbiology lab which we wouldn't have offered in the private community, especially as extensions rather than numbers.

Hughes: The comments look like your writing.

Follansbee: Yes, it is clearly my writing, and so I'm clearly responding to what was typewritten here. Initially I thought that I recognized the type, but I don't. I've obviously commented on it. So this paper titled "Guidelines" is something that Connie must have worked out.

It has to be fairly early, for a couple of other reasons. One is that the dose of trimethoprim sulfa is too high. It was the dose that we were taught to give, but we recognized fairly early, probably by '83 at least, that this was too high a dose.

Hughes: Because it was toxic?

Follansbee: Because it was toxic, yes. People got too sick on this high dose, and so we really would give 15 milligrams per kilogram of the trimethoprim component each 24 hours.

Hughes: The dosage which appears on this document had come from previous experiences?

Follansbee: From previous experiences, right, mostly with leukemic children. Likewise with the pentamidine: when we would get pentamidine in the first patients we treated, again in '81, we were giving them all intramuscular injections. That was because the Centers for Disease Control guidelines said that pentamidine intramuscularly was less toxic. AIDS researchers had actually compared IV [intravenous] with IM [intramuscular], the formulation, and realized that IM was more toxic. I don't know if Connie was involved; I wasn't involved in that study, but I think there was one. We recognized real early that you don't give it intramuscularly and we stopped that. I think we stopped that--again, my



memory is poor--by '82. I think this "Guidelines" document came out pretty early.

Another indication that this was early is the section about pulmonary function testing weekly. Well, there was a lot of interest in whether Pneumocystis caused pulmonary scarring. Dr. Diana Coleman was doing a pulmonary fellowship and looking at this phenomenon. Now, we don't do pulmonary function test routinely. That went out of practice; there was no point in doing it. But again, people were looking to follow people as closely as possible for complications that hadn't been previously recognized.

I wrote here, "Talk to K.H. regarding Pneumocystis carinii." I guess K.H. must be Keith Hadley.

Hughes: That's what I thought.

Follansbee: I'm just trying to remember how this document came to me.

Hughes: Maybe Keith referred the document to you?

Follansbee: He certainly could have, although Keith generally didn't forward these outside of SFGH. Boy, this looks early, because look at here, even on discharge: "You should have a primary clinic identified. Infectious disease, chest, or oncology clinic are suggested, depending upon the patient's major problems." Well, it doesn't even mention the AIDS Clinic.

Hughes: So that makes it pre-1983?

Follansbee: So that makes it early as well.

Hughes: The AIDS Clinic was founded in January, 1983.

Follansbee: Yes.

Hughes: Well, look at the last page. Do the tests ordered give any clues to dating this document?

Follansbee: These are things that you would follow for somebody, particularly with Pneumocystis, because the blood gases right here, oxygen levels here. So it doesn't really give much of a clue at all.

Hughes: Now, let me read you the concluding paragraph: "It should be kept in mind that this syndrome is poorly understood, and that further complications of depressed cellular immunity are

likely to occur. Listeria infections, for example, occur in patients with abnormal CMI..."

Follansbee: Cell-mediated immunity.

Hughes: "...but have not yet been reported in this group. While the preceding guidelines are offered to attempt to provide some consistency in the evaluation and therapy of these patients, close observation and thoughtful creativity are important in making further observations on the causes and the complications of this complex of diseases."

Follansbee: It sounds like Connie Wofsy. [laughter]

Hughes: Does it?

Follansbee: "Thoughtful creativity" sounds like Connie. And so I would guess that this is her wording, but I could be mistaken.

Hughes: This medical document is saying between the lines, "We really don't know what this is entirely"?

Follansbee: Right.

Hughes: These guidelines are saying: "This is our best guess at how you might proceed, but don't limit yourself to what we're saying here."

Follansbee: Exactly.

Hughes: Is that non-dogmatic approach typical of how you approached AIDS, or is that, if it is indeed Connie Wofsy, typical of an individual and not necessarily how other people might approach what presumably by this stage was still a relative medical unknown?

Follansbee: I would like to think that this was not an individual but this was the way that all of us approached AIDS, and I think that's true. Part of our non-dogmatic, open approach was because a lot of us AIDS health care providers were humbled by the fact that it was the patients who told us first that this epidemic was going on. Gay men knew that there was something going on, and told doctors that they had friends who were getting sick in some communities. I think I told you about the first AIDS patient at UCSF. To get admitted, he had to throw a temper tantrum and insist on being admitted to convince people he was sick. So we physicians started off by recognizing that we didn't know what was going on. So I think that this open approach was in fact pretty much the standard sort of

thoughtful approach that a lot of people used early in the epidemic.

Hughes: What you're saying, in essence, is that it was so obvious that you couldn't explain what was going on that you could not take a rigid approach?

Follansbee: Absolutely.

Hughes: So medical arrogance was almost an impossibility.

Follansbee: Right.

### A Collegial Approach

Follansbee: When you look at the generation of people who got started in this AIDS business, they were the people of my generation. Here we are, fifteen years, sixteen years later, and we're still baby boomers. We're still fifty years old or less. There are a few exceptions, but the San Francisco General Hospital AIDS faculty were a bunch of people coming right out of their training, at least in this community, and in a lot of communities. People who go into AIDS medicine often still are right out of training. So I think that it was also a group that was maybe trained in a different way than generations before, in terms of the humility and thoughtfulness around what we don't know, following a wave of "medicine can conquer all."

Hughes: Well, elaborate a little on that generational difference. Are you meaning by that that perhaps your eyes were wider open, or that you were less set in patterns? Or that your training was indeed different than the previous generation?

Follansbee: Yes, I think it may be a combination. Part of this generational difference may have been because this disease early on didn't fall into a given academic camp. It was a multispecialty disease. So there needed to be a collegial approach to treating it right away because no specialist could lay claim to AIDS. In fact, at least academically, nobody wanted to. The pulmonologists didn't want to claim AIDS as their disease. And certainly the infectious disease community did not. There was no group who wanted to lay claim to this and squeeze everyone else out. So, almost by default, AIDS took on a collegial approach from the get-go.

Later on, I think there probably have been more turf battles, but at the beginning there weren't. In part, no one fought to be exclusive AIDS specialists because the patient population was not a patient population that most health care providers really wanted to serve. That was manifest for quite a while. It still is at some level.

#### Initial Optimism about Duration of Epidemic

Hughes: Was there any feeling that this was an ephemeral epidemic, that it might be over very quickly? Was there then a hesitation to put a lot of resources into it?

Follansbee: Oh, yes. I think absolutely. Again, I can't remember exact dates, but I do remember thinking at one point as part of the KS Clinic that the epidemic was going away. We hadn't seen as many cases over two or three months, and those of us at the KS Clinic were thinking that maybe this was indeed a "blip." There was talk about the behavior of people in epidemics: that the most vulnerable people get sick and it manifests itself quickly. Then, everybody else probably gets over it. Maybe this was a disease, if it was infectious, that was going to hit symptomatically only 3 or 5 percent of the people. We had arguments about what percent of people with HIV would go on to get clinically ill.

Then we saw, month by month, what we thought was the percentage of people getting clinically ill going up, after that initial period of optimism.

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Follansbee: Early on it was the epidemiologist who should get credit for really leading the way in this epidemic in terms of going back to our infection control guidelines and to the way we approached people in our offices, and to understanding what was happening with respect to disease trends and future impact of this epidemic.

## A Lecture on Infection Control, 1984

- Hughes: Let me show you one more document, which is dated: February 10, 1984.<sup>1</sup> So probably this one was produced after the one you've just seen.
- Follansbee: Yes, it definitely would have been.
- Hughes: Is that yours?
- Follansbee: "Acquired Immunodeficiency Syndrome Part I: An Overview on Infection Control." Yes, this is definitely mine. It looks like it was a talk that I was going to give, and then there are questions as part of a CME [Continuing Medical Education] credit program.
- Hughes: It comes from your folder labeled "Lectures."
- Follansbee: Yes, it was clearly a lecture I was giving, and it clearly is my type and the way I do references. So, yes, this is mine.
- Hughes: Do you have any general comment?
- Follansbee: Well, the only general comment I would make is that early on, it was easy for all of us to know all there was to know about this epidemic, and so I could give a talk on the immunology, the theories of causation, the epidemiology, and treatment of Kaposi's sarcoma. [laughter] All areas that I wouldn't presume to lecture on today--except to maybe a lay audience, giving people an overview.

## Infectious Disease Subspecialists and the Treatment of AIDS

- Hughes: Did dealing with AIDS prompt you and your colleagues to move outside of your narrow specialties in dealing with patients?
- Follansbee: Right.
- Hughes: How different was this syndrome for you as an infectious disease specialist? Normally, an infectious disease

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<sup>1</sup> This document is reproduced and cited in the appendix of this volume.

specialist deals with infectious disease, but here you were dealing with patients that perhaps had wasting syndrome; they had cancers; they had things that were not obviously infectious diseases. Could you blot those out and say, "I am here to deal with the PCP," or other infectious disease, and not pay attention to the other problems?

Follansbee: Yes. Well, one of the good things about infectious diseases was that, since it's not organ-specific per se, I think that the infectious disease community in general in its training and approach never could block out the other medical problems of the patients. They all are interrelated. So I don't think that this disease in that sense is unique. Syphilis is another example. At one point, the dictum went that if you knew syphilis, you knew all of medicine. And I think it's true for HIV. I think HIV is clearly in a sense the syphilis for educators of this century. So from that standpoint, that's not a change, at least for infectious disease practitioners.

Hughes: So infectious disease practitioners, to put it simplistically, are used to thinking holistically?

Follansbee: I think so, much more so than maybe other subspecialists.

Hughes: I've heard that the I.D. community as a whole was not particularly anxious to accept this disease. Is that your perception of the early days?

Follansbee: The infectious disease community in medicine has been very heavily academic-based, university medical center-based. And that's not been my community, although I was trained, obviously, in that base. I knew before I went into my training program that academia's not where I would stay, and I'd made that decision for myself. So I don't think I have any particular insights, other than as sort of an observer. I would say that that [the academic ID community's reluctance to accept AIDS] is true. There are obviously a number of exceptions, and stellar exceptions to that, in various medical centers and communities, but generally, that comment would be, as an overview, correct.

#### Federal Funding for AIDS

Hughes: And again, I'm leaping probably to a simplistic parallel, but I wonder if there isn't some symbolism at a national level to



the switch from NCI to NIAID [SSH question acronyms]? Did this at least represent a struggle for a time about who indeed was going to be the federal authority on this disease. I guess you have to get the CDC in there as well.

Follansbee: Yes.

Hughes: There were tensions between the National Cancer Institute and the National Institute for Allergy and Infectious Diseases.

Follansbee: Yes. I think that clearly, as funding for AIDS research became available, the tensions increased. Initially, it was more like, "This is not my job; this is yours," and then it became more, "Gee, here are monies." So now everything became AIDS, HIV-related. Anyone could write a grant to do anything and tie in HIV, hoping to move it up the priority list.

#### AIDS Diagnosis and T-cell Testing

Hughes: You discouraged people from using only immunodeficiency as a means to diagnose AIDS.

Follansbee: Right, and that was, I think, in response to the people using T-cells for diagnosis of HIV disease or AIDS, because the equipment to do T-cell subsets was available, and it clearly was recognized early on that helper cells were some sort of a marker of the disease. The issue became one of trying to encourage people not to use helper cells by themselves to make the diagnosis. At that time we had no HIV, we had no serologic tests to reveal the underlying cause and to make the diagnosis. The technology was less sophisticated than it is now, and there was so much variability in the T-cells numbers.

Hughes: So T-cell monitoring wasn't an accurate diagnostic test at that point?

Follansbee: It was not necessarily an accurate diagnostic test. You'd do one set of T-cells and say to someone, "Well, you must be part of this epidemic." It seemed premature. Insurance companies were beginning to do T-cells on people for insurability, and there was a lot of confusion. If you fell out of the normal range, then doctors were telling patients that they must have this disease. Yet these patients were well. But the normal ranges were normal ranges for 95 percent of healthy people, and intercurrent illnesses affected T-cells transiently. So there was just a lot of misunderstanding. And obviously there

was a lot of anxiety in the gay community to have T-cell counts done to try to figure out if they were part of this epidemic.

Hughes: So you were saying that T-cell counts were only one part of the puzzle?

Follansbee: Only one part of the picture. At that point our treatment options were really still predicated on disease: on seeing clinical manifestations in somebody. We had really nothing to offer someone based on T-cell numbers per se at that point, and T-cell numbers were not by themselves adequate to make a diagnosis.

### The AIDS Antibody Test

#### Diagnosis and Treatment After Availability of the AIDS Antibody Test

Hughes: Once you had the antibody test, did disease-based diagnosis fade in importance?

Follansbee: Absolutely, particularly if you start to recognize the role of prevention--the role of prevention of Pneumocystis pneumonia, for example. We didn't have to wait any more for someone to come down with Pneumocystis and think about preventing a second episode, but instead could focus on primary prevention. So yes, once there was a test, I think we were motivated even more to monitor someone's clinical course using T-cells numbers.

Hughes: So then the T-cells really did become important?

Follansbee: Right. I think that my statement in the 1984 document and my point about not relying solely on laboratory criteria--which I think was being made by a number of people--was really an attempt to temper the hysteria. There were lots of tests being done: sedimentation rates, and beta-2 microglobulins, and all sorts of tests to find a surrogate marker for disease and to track the course of disease, because we didn't have an HIV test. Then there was a lot of pressure to tell a patient what was going to happen, predict the future. And we would find a fair amount of variability in the markers we had available at that time.

### Drawbacks

Hughes: Was there any down side to putting heavy reliance on the antibody test?

Follansbee: Well, there were several down sides. One is that obviously we were very concerned about issues of confidentiality in the medical record. We encouraged people--even the state encouraged people--to do anonymous testing. Frankly, I think anonymous testing has been very good and very important. There has also been a large number of anecdotes of errors, not probably in the testing, but somewhere between the hard copy of the test results and the person having the test, walking out of the clinic or test site. There's been a little misunderstanding of where testing may have fallen down.

The large number of times the patients have told me they were negative on multiple tests, and yet they clearly had HIV, is somewhat concerning. When we do it in a non-anonymous setting, the test in these cases is positive, and therefore, we've been sort of misled on the documentation.

Hughes: What went wrong there?

Follansbee: I think people sometimes heard, "You have HIV but you don't have AIDS." So they came out thinking, "Oh, thank god I don't have AIDS," and then forgot that they had HIV. Or maybe it's denial. They don't get a piece of paper with their name, the test, and the test results.

Hughes: Or maybe they didn't understand what it meant to have HIV but not AIDS?

Follansbee: Right, but I think most of the misunderstanding came from denial. Mostly, it was psychological. It wasn't like the counselor said, "Now, before you leave, I want you to write down the answer to this question: "My test result shows \_\_\_\_\_," to test and make sure people heard the right thing. And then there was just embarrassment: Yes, I found out I'm HIV positive, but I don't want anyone to know. Also, testing negative clearly didn't mean people were not infected, because of the lag time between infection and a positive antibody test. So there was the potential for some misunderstandings there. So there was clearly a down side to anonymous testing.

**BAPHR's Concerns about Antibody Testing**

Hughes: Were you involved in any of the BAPHR deliberations about the antibody test? For one thing, BAPHR came out with a brochure called, "Should I Take the Test?"

Follansbee: Yes, I was definitely involved. There was no treatment, and the question was, what do I do with the test results? And I've never been an advocate of doing tests that I can't do anything about. So there were clearly issues about the test. We recognized early on that following safer sex guidelines shouldn't depend on test results. So we weren't encouraging people to get tested so that they could then go out and have unsafe sex, or use their positive or negative test results as a way to seek partners. We recognized that early on.

Well, what's the purpose of getting the test, and what's my reason for getting tested, since it's not something that we can do anything about? We didn't have treatment when the test was first available. It was a test that was really informational. Obviously, gay men were already at risk. If they were taking the test in the first place, it was because they felt they were at risk, or they fit some criteria for risk. They shouldn't have been donating blood. They should be following safer sex guidelines. So the test wasn't a way to give the individual at risk the right to engage in activities they shouldn't be doing.

As you probably remember, when the test became available and blood banks began using it, by law there was about a six-month window during which the blood banks couldn't tell the infected person that he or she was infected because the staff at blood banks didn't want people going to donate blood in order to get the test before the test sites were up and running. So the blood banks were in the embarrassing situation of having to run the tests, screen blood, and not use blood. But then they could not by law divulge the results of their blood tests for six months. So this frustrating situation was just part of the thinking about, What are we really doing here?

CDC Definition of AIDS as a Guide to AIDS Diagnosis

Hughes: Well, getting back to the clinical aspects again, I'm wondering about the role of the CDC definition of AIDS in what you were doing in clinical practice.

Follansbee: I think there are two aspects to your question. One aspect is, did people who didn't identify themselves early on as being in a risk group perhaps get delayed care? Because they didn't say, "I'm a man having sex with men." I think clearly, that would be the case. By the time these patients got to me, usually some health professional had already suspected that a patient might have AIDS, and was thinking about that possibility.

And yes, we did see people who did not fall into risk groups, who denied that they engaged in risk-associated behaviors until their dying day. Of course, those people got reported, because they had the sentinel manifestation, whether it was Pneumocystis pneumonia or cryptococcal meningitis or whatever was on the list. That CDC list of AIDS-associated diseases expanded eventually. These patients reported, and as long as they fit the definition at the time, were included as an AIDS case, if they didn't have another underlying cause of their immunodeficiency. For every AIDS patient who didn't fall into a risk group, CDC would send an epidemiologist out to look into cases to find out what was going on and whether there were unidentified risk factors.

I supported the definition, because I think in order to track this epidemic the cases had to be proven (i.e. a diagnosis strictly defined and fairly rigid). If chronic fatigue became part of the definition without any of the other manifestations of severely impaired immune system--and you and I know there's been chronic fatigue since at least the 1930s--then we would never figure out what was going on with this disease, so the definition was necessarily rigid.

I think if people didn't tell us that they belonged to a risk group, then if they had pneumonia, they got tetracycline. By 1984, if we knew that they were in one of the risk groups, we wouldn't be giving them tetracycline as first-line treatment, we'd be trying to find the Pneumocystis.

I do know that very few of us, if a patient presented with a possible new AIDS-defining infection and belonged to an AIDS risk group, left our treatment to chance or "best guess." I think we pushed patients to establish a definitive diagnosis



because that was the only way we could truly say this patient really had, for example, Pneumocystis. I would say in the case of pneumonia, "I don't know what you have. This could be Pneumocystis. Or it could just be an atypical pneumonia. We need to know. It's not fair to you, since we don't have any other tests, to just treat you presumptively for Pneumocystis pneumonia. If you get better, you might have AIDS, but you might have gotten better anyway, say because of a viral pneumonia, and not have had AIDS." So we did push people into diagnostic and invasive procedures that we wouldn't have otherwise pushed.

### Lymphoma and Hairy Leukoplakia

Follansbee: The other side of your question was that there were diseases that we certainly recognized as being part of this syndrome that CDC didn't include in their guidelines. Were we frustrated? I think that the main example of that really fell to the oncologists, who recognized lymphoma in San Francisco, and were sort of told by other oncologists in other cities that this was not part of the epidemic. But oncologists at UCSF-SFGH knew that lymphoma was a manifestation of AIDS. And they knew they were seeing more of it. It took a little while, a few months, or whatever, to convince other people.

In terms of our treatment, we treated people with AIDS the best we could, for whatever they had, and didn't worry so much about whether this was a truly new and independent AIDS-defining diagnosis. So there wasn't frustration on my part in this area.

Hughes: Hairy leukoplakia, like lymphoma, which was also identified here in San Francisco, also took a while to be integrated in the CDC definition.

Follansbee: Well, hairy leukoplakia was never a serious enough opportunistic infection to say that someone had AIDS. There were these oral manifestations that were recognized more frequently. They weren't "AIDS-defining."

Hughes: Well, hairy leukoplakia, not on its own, but as part of a whole syndrome?

Follansbee: Right. I think hairy leukoplakia may have been more frustrating for patients than for physicians, in part because there's a lot of leukoplakia--caused by cigarette smoking,



dentures, funny-fitting teeth, this kind of thing. So the hairy leukoplakia per se, the kind of fuzzy leukoplakia associated with HIV, when it became recognized as part of the syndrome, would cause patients a lot of anxiety. People wanted to know, "Do I have hairy leukoplakia? I've got a little white patch." But since it wasn't life-threatening, physicians didn't really give it the same significance as, say, KS. I don't think there was high frustration level over the lack of recognition of hairy leukoplakia as part of AIDS epidemic by other authorities, in part because we were measuring T-cell subsets in patients at risk.

Hughes: If you saw lymphoma in a gay man, would AIDS be an automatic consideration?

Follansbee: Yes.

Hughes: Even though lymphoma was not part of the official CDC definition?

Follansbee: Yes, it would be a consideration. We would worry about it despite its being left out of the CDC definition because it signalled immunodeficiency. But it didn't bother me that I didn't have to report it to the CDC, just because it didn't fit the definition, because the reporting was really for epidemiology. It didn't affect my care or my patients' access to services at that point.

#### Other Diseases Outside the CDC Definition

Follansbee: There were other disease manifestations which were not part of the CDC definition, like recurrent endocarditis, i.e. bacterial endocarditis, or recurrent bacterial pneumonia, and wasting that ultimately were included in the case definitions. These were included finally because it was felt that IV drug users had different manifestations of impaired immunity compared to gay men. Well, I'd been seeing endocarditis in San Francisco in non-AIDS patients for a long time, and so I didn't really care whether endocarditis was included as a separate AIDS-defining diagnosis, nor think that it should be.

I saw a patient once when I was up at UC who had a fungal infection, Candida, in the muscle next to his kidney. Well, he was an IV drug user; he continued to use. He wanted to have that Candida called AIDS. I said, "Listen, Candida infections in IV drug users who continue to use are fairly

common. I don't know that this is AIDS, and I'm not going to say that you have it at this point." He was pissed at me, but from my standpoint, I was going to treat the Candida. This was probably '82-'83. Later on, he went down to Irvine, and later on, he developed something that was AIDS. Then I got a note saying, "See, I had it." Well, sure, but so what? At that level, I was going to treat what I was going to treat.

### AIDS-Related Complex

- Hughes: BAPHR published a definition of ARC, AIDS-related complex, in 1986 and offered it to the medical community at large. What role did ARC play in your clinical practice?
- Follansbee: I think that part of the issue--because I remember those discussions--was that people had what was called disabling ARC. In other words, they didn't have AIDS; they hadn't had Pneumocystis; they hadn't had KS. They hadn't had some of these sentinel infections, but they were clearly functionally disabled. So they fell into the ARC category because they were sick but didn't have the sentinel infections. And we were trying to grapple with: "How can we help people who clearly have HIV, clearly are as sick if not sicker than some of the people who have AIDS, but can't get services? This condition needs to be identified." So I think that the BAPHR definition of ARC was an attempt to establish some criteria for practitioners, and then hopefully for agencies.
- Hughes: Was there an understanding at that point, in 1986, that ARC patients would become AIDS patients?
- Follansbee: I think that's a good question. Did we think that everyone who developed ARC would automatically develop AIDS sooner or later? I think that that was early on a matter of some debate. Eventually people saw the numbers of ARC patients who developed AIDS increasing and realized that yes, there would be no one who would live a normal life but hung up in ARC per se. I don't know when that recognition came, but I think that AIDS practitioners and the gay community did see ARC as a prodrome. Or they saw ARC as early manifestations of AIDS and saw people sliding eventually into AIDS. Clearly, people could die of disabling ARC. They could die and never have an "AIDS definition" for whatever reason, but have died of disabling ARC. So the distinction began to blur pretty quickly.

The "Worried Well"

Hughes: Were you seeing in your practice in these early years people whom I've seen described as the "worried well"?

Follansbee: Oh, yes.

Hughes: And how did you approach them?

Follansbee: Well, you have to understand that it was frightening for people. Therefore, there needed to be some acknowledgement of what it was that prompted their concerns. Sometimes it was a cough. Having been to medical school, I've seen that nearly every medical student ultimately thinks that he or she has gotten some terminal disease, but then "miraculously" recovers. Sometimes it's a strictly psychiatric diagnosis when you're taking your psychiatry rotation. But the fact that medical students routinely worry about getting what they're studying shows that we're all subject to these concerns and fears.

So we acknowledged the concerns of the worried well by attempting to do whatever tests we could do at that point and show that they were okay. I certainly saw physicians who treated themselves for Pneumocystis and had no evidence of HIV, and to this day don't. Health care professionals in particular were apt to be among the worried well.

Some of the worried well had clearly psychosomatic problems. If it weren't AIDS, it would be something else that they would latch onto and be convinced that they had. As an intern before AIDS, one of my first patients in the emergency room at San Francisco General thought she had inhaled a poison gas and was going to die. She hadn't and didn't die. But she was at risk for thinking she had acquired AIDS. Also, what you couldn't do was play into their fears and treat them "empirically" for AIDS problems, "just to be safe," when they didn't have AIDS.

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Follansbee: It takes more time "not to treat", but you don't treat. So I had at least some background about how to deal with this and how to do reality testing and try to show people that they were okay. I'd promise them follow-up, assure them that this was not a one-shot evaluation or concern and that I would continue to follow them if things developed. I would tell

these worried well what symptoms to look for. So it was education, but yes, there was a lot of that.

### Alternative Therapies

Hughes: Do you have a comment to make about alternative therapies?

Follansbee: Certainly there are a lot of people who sincerely are looking to help people with HIV. Clearly there is a lot of alternative therapy offered in that vein. It's a frustrating disease; we don't have a cure. If we had a penicillin for AIDS and it worked every time, then I think there would be less interest in alternative therapies. You don't see a lot of people taking alternative therapies to treat syphilis, for example, and a lot of alternative therapists are not offering alternative therapies for syphilis. You know, the solution with syphilis is to take your penicillin. So I think the majority of alternative therapies are offered in sort of a sincere attempt to help people feel better, and sometimes for symptom control. So I think they're useful in that regard. I just try to make sure patients feel they can talk to me about trying alternative therapies.

And there have been a lot of alternative and nonalternative therapies that we have offered up that have gone by the wayside. We've been more successful, I think, in analyzing which medical interventions for AIDS should be abandoned than in analyzing which alternative therapies should be abandoned. So it distresses me sometimes to see the recrudescence of high-dose vitamin C use, for example. About every four years, we see vitamin C reappear as a cure-all, because no one seems to believe history. So I think that the alternative therapies need to be to some extent judged by their results, and their results need to be better established. But I encourage it.

Hughes: In the case of, say, vitamin C, would you discuss with a patient what you considered to be the drawbacks?

Follansbee: Yes, absolutely.

Hughes: So alternative therapy is a part of your discussion?

Follansbee: Absolutely. I don't initiate discussion about every alternative agent, but I ask people to tell me. For example, when I ask someone what medicines are they taking, they give

me the standard [ones]. I say, "What about vitamins?" because they don't think of vitamins as a medicine, but they tell me when I ask, "Are you taking any alternative medicines--herbs, or anything like that?" So at the point in taking my case history when I ask them about their medications, I include my inquiry about alternative remedies, and I have for a long time.

### Relationship with Patients

Hughes: Has the epidemic changed your relationship with your patients?

Follansbee: Because I wasn't in practice before AIDS and HIV, that's a tough question. I think that I'm doing things that I didn't anticipate I would be doing, and that I have relationships with patients that I didn't think I would have as an infectious disease doc.

Hughes: Please expand on that.

Follansbee: Well, I chose infectious diseases because it was the only branch of internal medicine that offered a cure. I didn't think that I would be having what looks like an oncology practice, with leukemics and with people who require as much care and attention as cancer patients. I didn't imagine I'd be dealing with patients whose every organ system is involved and who die ultimately--at least at this point based on a lack of effective therapy for the long term. I wasn't anticipating that.

In terms of my relationship with patients, I think that AIDS has offered me the opportunity to practice medicine the way I like to practice it. I like the relationships, and I like the role I've been able to assume. It's been a very positive experience in a lot of ways, because of the closeness. You've got to remember that these patients are peers. These people are colleagues. Some of them either have been friends or become friends as part of the interaction, because that's what they want, in part as part of their therapy. And that's not something that I think I would have anticipated, nor did I experience it necessarily through most of my medical training.

Hughes: Part of that training, if I understand it correctly, is to maintain a distance from your patients.



Follansbee: Right.

Hughes: And do you find that this disease fosters a closer relationship with patients? Do you find the line blurred between you as a practitioner and you as a private person, who, not incidentally, is also a gay man and has another link with at least some of your patients?

Follansbee: Yes, all that is true. Is that different than if I were, say, an oncologist and had a completely non-HIV practice? I think there would be patients who the line would be blurred with because they would be peers in that regard. And to some extent, I still maintain that professional distance and only allow myself to sort of break that if patients ask me to. Patients will ask to be hugged. I still will not sit--I cannot sit on a patient's bed. It is ingrained that you don't do this. And I still listen for clues from the patient. Do they want to call me Steve, do they want to call me Dr. Follansbee? Some of them call me Mr. Follansbee. [laughter] But whatever they want to do, I try to take the clues from them.

I expect that would, in fact, be true in any disease. I treat lesbians with osteomyelitis. And when they figure out that I'm gay--and I don't advertise it, because I don't think that's the key to my medical practice--then I think there is some special bond. I think that they identify with me. I think some of that's my personality, because I have straight patients who feel the same way, who say I'm the only doctor who will listen to them.

Now, did I hone those skills because of HIV? Maybe I did. Maybe those skills developed faster because I had to deal with the AIDS epidemic. Maybe I became much more aware of the importance of listening to patients because we had a patient population that had so many issues. And so many of them were so articulate about their concerns that yes, I developed those skills a lot more quickly than I would have otherwise.

#### Identification as an Infectious Disease Physician

Hughes: You said last time that you did not identify yourself as an AIDS physician. That you identify yourself as an infectious disease specialist.



Follansbee: Yes, you're right. One of my titles is I'm medical director of Davies [Medical Center] Institute for HIV Research and Treatment, so it doesn't take long for people to realize that that's what I do. But there's an emotional distance that I maintain to this disease which is: I want to be able to practice infectious diseases once we've cured AIDS, and I want not to hang up my hat. I feel I have skills in infectious diseases. Those include the infectious diseases associated with HIV infection at this point, but I don't certainly advertise myself in the yellow pages as an AIDS doctor and don't intend to. And part of it is because I want to see this disease go away.

Hughes: Is that a common perspective amongst your private-practice colleagues in San Francisco?

Follansbee: I don't know. Certainly a lot of people do advertise--who feel they have to advertise--their skills in the immunodeficiency syndrome. The majority of docs don't advertise and therefore don't have it on their stationery. They don't have their pictures in the gay press, and so they just don't highlight it in that regard. I think part of it is because they want to see general internal medicine patients or establish a family practice or whatever.

Hughes: It has struck me that not only the medical profession but many professions, advertising in the gay press, use a photograph. Why is that?

Follansbee: Oh, I think it's a little narcissism. Occasionally, you drive up 101, and you'll see some attorney or physician advertising on a billboard with a picture. I think it's narcissistic: to think that people are going to want to use that professional because they've seen his or her picture. And certainly the plastic surgeons use them all the time in the pink section of The San Francisco Chronicle. In the gay press in particular, I think that there's this feeling that it will attract patients if they've got a good picture.

#### Communication Between Community and University Physicians

Hughes: Before the County Community Consortium was formed, which was in 1985, did you as a private practitioner feel in the loop regarding new drugs that were coming out for this disease?

Follansbee: I think I was definitely in the loop, and it was because I was at San Francisco General. One of the reasons why I worked there and one of the reasons why I continue to work there is to stay in the loop. Plus, I think San Francisco General benefits from having me there, because to some extent they need to stay in the loop and know what's happening in the private community in terms of clinical practices. Sometimes some of the practices at San Francisco General followed some of the practices in the community logistically.

Hughes: Can you give an example?

Follansbee: Oh, transfusions. Everyone used to be hospitalized for transfusions at San Francisco General Hospital, and I said, "Gee, do you need to do this? We've been giving transfusions to the outpatients for quite a while." Everyone was hospitalized with Pneumocystis regardless of how well they were. I said, "Gee, most of the Pneumocystis isn't hospitalized in the private community. We treat it as an outpatient."

Hughes: So the information was flowing both ways.

Follansbee: Exactly, I think in both directions. I don't want to take credit for being the reason why Pneumocystis is treated as an outpatient condition at the General, but I think that the practitioners there needed to hear about it. One way they could hear that was because I was in the clinic.

So I didn't feel so much out of the loop, but clearly, other community docs, other physicians did. And as much as BAPHR tried to take a role in keeping information flowing into the community there was not a strong forum for private physicians in BAPHR. The KS Clinic by then, [ca. 1985] had pretty much gone by the wayside, and there had been a lot of private docs who went to the KS Clinic, as we talked about before. When the KS Clinic folded, then there was a lacuna.

The County Community Consortium<sup>1</sup>

## Foundation and Early History

- Hughes: Was there a model for community-based research prior to the County Community Consortium?
- Follansbee: The oncologists had a model for oncology research in these networks, Northern California Oncology Group or Southwest Oncology Group or whatever. Other than that, I would say no. And if you remember, the consortium didn't start as a research group. It started really as a forum for San Francisco General Hospital-based physicians to talk with physicians in the community about research, but also to talk about AIDS treatment and other concerns. Sometimes there were political concerns about what was happening at the health department. So the meetings were much more discussions and sometimes case reports or whatever.

The consortium only got into the research because of aerosolized pentamidine. The community physicians were seeing a lot of patients who couldn't tolerate trimethoprim-sulfamethoxazole prophylaxis for PCP. Aerosolized pentamidine was an idea that came out of UC/San Francisco General Hospital.<sup>2</sup> So the community physicians asked, "Why don't we try to do a study with this?" And everything went backwards. We did the study, and then the company that made pentamidine decided they wanted to try to apply to the FDA for an expanded indication. The drug company then went and hired this large clinical research organization to come in and look at all our data and to put our records into some sort of format that would be suitable for a submission to the FDA.

- Hughes: The company was Lyphomed?
- Follansbee: Right. So the Consortium didn't start with the idea that we would do community-based research.
- Hughes: Oh, that's interesting.

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<sup>1</sup> For more on the consortium, see the oral history in the AIDS physicians series with Donald I. Abrams, M.D.

<sup>2</sup> For more on PCP prophylaxis, see the oral history in the AIDS physicians series with Constance B. Wofsy, M.D.

Follansbee: Oh, absolutely. No, it was not a research organization initially.

Hughes: So the consortium was really for information exchange?

Follansbee: It was really information exchange. I think that people went to [Mayor] Dianne Feinstein and said, "There's a problem here: we don't have communication between SFGH and the community physicians. There's a gap. We're not talking." And Dianne Feinstein essentially mandated to Paul Volberding that there be some dialogue. The first meeting chaired by Volberding occurred in the San Francisco Medical Society board room. I don't remember the exact date.

Hughes: From what I understand, there was a fiscal reason for that mandate as well. Her intention was to share knowledge through the consortium so that the knowledge base became firm in the community, and patients did not have to be referred to San Francisco General and hence become a county or city expense.

Follansbee: That may be; I honestly don't know. I think that there was a fair amount of concern in the private community that they were taking care of patients but didn't know what was happening at the university. They felt they were just hearing rumors about what was happening at San Francisco General. In San Francisco, unlike a lot of the communities where primary care doctors said, "Let's get the AIDS patients to the university," there were a lot of docs who wanted to take care of these people themselves. They were getting more concerned that they weren't in the loop.

So Dianne Feinstein may have certainly seen it as a fiscal issue. I don't think the docs saw it as a fiscal issue. They just saw it as, "We want to take care of these patients; they're our patients."

Hughes: I'm probably being unfair to her in implying that that's the only reason that she was trying to set up the consortium. I'm not at all sure that she saw it only as a fiscal issue.

Follansbee: I'm not sure that it was.

Hughes: Paul Volberding called the initial meeting of the consortium, at the behest of Feinstein.

Follansbee: Right.

Hughes: And then very quickly, Volberding dropped out.

Follansbee: Right, he dropped out very quickly. I recall that he asked me to head the consortium, because I was a community-based physician and we had had some contact. I just didn't have the time, and there was no money to do this. So it became something that Donald Abrams took on and has done incredibly well. It would have probably killed the consortium if I had taken on that kind of responsibility. It was one of the few times I was able to say no.

Hughes: Why do you say it would have killed it?

Follansbee: Because I didn't have the time. In private practice, my commitments and responsibilities were such that I could not have had the vision or the time to really develop things.

Hughes: In your correspondence, I found a letter dated 1991 from Donald Abrams and Leonard Simpson, who was chairman of the social policy and action committee of the Community Consortium, written to Kenneth Kizer.<sup>1</sup> The opening paragraph says: "The Community Consortium is an association of over 180 Bay Area providers caring for patients with HIV disease. One of our primary goals is education of each other regarding important issues in HIV medicine." The original purpose.

Follansbee: Right.

Hughes: "Another aim is to conduct community-based clinical trials. Our study of inhaled pentamidine as prophylaxis for Pneumocystis pneumonia led to FDA approval and a New England Journal of Medicine publication. Our third major objective is to respond as a group to social and political issues that concern providers of care to patients and patients with HIV disease." Was the third goal always part of the consortium's goals, or does that become more important over time?

Follansbee: I think it becomes more important because BAPHR saw this as an important issue for them, in terms of responding to the epidemic, because it was initially an epidemic of gay men. Not coincidentally, by the way, Lenny was the chair of BAPHR for quite a while. But I think BAPHR got subsumed by the consortium as a forum for these kinds of discussions of political and social issues. I think this was not the original intent of the consortium, but developed over time. I don't remember Dianne Feinstein's office necessarily looking to the Consortium for expertise on these social or political

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<sup>1</sup> This document is reproduced and cited in the appendix of this volume.



issues. Her committee was made up of other people who gave her more direct input.

Hughes: Her AIDS Advisory Committee?

Follansbee: Her AIDS Advisory Committee, which was made up of people such as Merle Sande, who didn't belong to the consortium. So I think the social and political concerns occurred later and in response to specific legislative challenges.

Hughes: And are you saying that, with time, the CCC became the main authority for Feinstein and the AIDS Advisory Committee?

Follansbee: No, it never became the main authority, but with time, other groups became less authoritative, like BAPHR. So the consortium did become a forum for these social and political issues. Now, how much authority the consortium has even now is questionable. [laughs]

Hughes: In the beginning, which I guess would be 1986, was there an overlap in consortium and BAPHR membership?

Follansbee: There was a big overlap. Now, they certainly would have been candidates for BAPHR membership. Whether they actually belonged to BAPHR or not, I don't know.

Hughes: Meaning they were largely gay physicians?

Follansbee: Largely gay physicians.

Hughes: Is that still true?

Follansbee: For BAPHR or the Consortium?

Hughes: For the Consortium. One hundred and eighty Bay Area providers is quite a few.

Follansbee: Yes. When you look at the people who go to the meetings, there are a large number of gay providers there. It is certainly not a large majority. If it's a majority, it's a simple majority, and it may not even be a majority.

Hughes: When did you become associated with the CCC?

Follansbee: I went to the first meeting. It was in the board room of the medical society.

Hughes: How did you hear of it?



Follansbee: I guess I got a letter or a call.

Hughes: Would it have been from Donald Abrams?

Follansbee: No, it was from Paul. I think Paul chaired the first meeting?

Hughes: And that was the first and only meeting chaired by Volberding?

Follansbee: I don't remember whether he would have chaired a second meeting or not. But I don't think he chaired a lot of meetings.

Hughes: How did he present this new group?

Follansbee: I'm sure there were people there from the mayor's office, and that hasn't happened for years. I think that it was presented that an epidemic is going on throughout San Francisco, and we need to be talking with each other, communicating better about what's going on in our programs. We need to hear each others' concerns, and there just needs to be more formalized interaction. It was not presented as, We want to start research in the community. These were not issues that were talked about. We just want to hear what your concerns are and what's going on. I remember people like Jim Campbell and Bob Bolan being there.

Hughes: Bill Owen?

Follansbee: Bill Owen I think was there. I'm trying to remember what other docs in the community were there. Probably Steven Mehalko was there.

Hughes: Well, town-gown tensions have characterized medicine practically since its inception. Was it unusual to get academic and private practice medicine in one place dealing with one problem?

Follansbee: Yes, I think that it was unusual. In part because of the personality of Donald and Paul and Connie, it was always very respectful and collegial. I just may have my head in the sand, because I was going back and forth between the academic and private practice communities.

Hughes: Yes, you were a link.

Follansbee: But I don't feel that there was a tremendous tension between them. I don't feel that the medical community was being put down. Of the original five cases of Pneumocystis, only two came from the university, one from Moffitt Hospital and one

from San Francisco General; three came from the community. So I just don't feel that tension was characteristic of San Francisco, certainly not early on.

### The Proposed AIDS Public Health Hospital

Follansbee: Now, there have been tense times. The movement to set up a public health hospital was very tense, because it was anxiety-producing to docs to say, "Wait a minute, we're taking care of people with HIV. Is anyone asking us whether we want to move our practices to the public health hospital? How is this going to interact with the community? Why that location? That's not where patients are." The public health hospital was to be located in the inner Richmond area, about outer Richmond. And so there were tense moments throughout all of this.

Hughes: Who was behind that particular movement?

Follansbee: The health department, I think. Now, whether Merle Sande was involved as more than an advocate, I'm not certain. It's not something that I've heard talked about recently. But I think the health department was considering this project in part because there were monies available to set up demonstration projects. The government thought they wanted to get in the business of funding these demonstration projects. And there was still a little old-time thinking that hospital-based projects were the way to do that. Which of course didn't show a lot of foresight. So, in fact, there was some competition for the monies, and Davies got some of the money. It didn't all go to the public health hospital project.

Hughes: What killed the idea of an AIDS public health hospital?

Follansbee: I think that to some extent, it was the community and the community hospitals which didn't want this necessarily, although some hospitals didn't care so much. They didn't have a big AIDS presence and they said, "If they want to take care of [AIDS patients], let them. We don't want to."

Hughes: But that was never Davies' perspective?

Follansbee: No, it was never Davies' perspective, it was not Davies' perspective at all. So it was partly that the opposition of community hospitals, and I'm sure that there was politics involved in killing it. It was also going to be a tremendous

expense. The federal government really didn't necessarily want to get into special-fund projects. They felt they had been burned with dialysis and really didn't want to. And so I think that politically, it died.

### The Community-Based Trial of Pentamidine

Hughes: The pentamidine trial moved the CCC into quite a different dimension, and maybe the one that the public now knows best.

Follansbee: Oh, absolutely.

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Hughes: Tell me about some of the problems in getting such a program launched.

Follansbee: Yes, it was a major effort. I was not involved in the study design. I was the investigator at Davies, and I think we were one of the biggest enrollees, but it was really a multi-center project. St. Luke's had a site, and Davies had a site, and Presbyterian had a site, and Children's, and I think Kaiser and San Francisco General. Clearly, the pulmonologists at San Francisco General were interested in this and had been doing some research on drug delivery via aerosol. It was clear that patients couldn't tolerate trimethoprim-sulfamethoxazole, which was the only treatment at this point, and that primary and secondary prophylaxis for Pneumocystis were going to be important.

So doing a community-based trial, where the patients were, seemed like a good idea to get large numbers of participants. It was something that really got generated internally and was designed to really be in a sense a pilot study. It was never designed as a study that was going to lead to an FDA application or even a published article. We designed our own case report forms and all the research materials, and it was not something that we thought was going to go to the FDA for a package insert.

Hughes: And what about funding?

Follansbee: The funding came--I'm trying to remember--again, I'm vague about this. I think that Lyphomed clearly provided the funding for the drug delivery and for the drug itself. But I don't think that there was much funding for internal

infrastructure to do the study. I think that that was pretty much volunteer. There were probably some dollars available, but I don't think it was a lot, frankly, and it certainly didn't get seen out in the community. The money was probably seen by the consortium.

Hughes: What were the steps that you took to actually place patients in the study?

Follansbee: Well, the consortium told all the docs--not just in the consortium but in the community--that this study was available. The consortium explained that the study involved randomizing people to different doses of aerosolized pentamidine. I'll have to look at the New England Journal article to see how many doses, but I think there were three doses. It was a very low dose, and then about 150 and 300 milligrams once a month. We designed a simple consent form. Then, we went to all the institutional review boards at all the respective sites, because these experiments were all being done in hospitals.

I can speak to Davies in particular. Brian Christensen was the director of our research unit, and I can't remember what we called ourselves right at the very beginning. I think we called ourselves the Institute for HIV Research and Treatment, but it was just basically the two of us. We oversaw the research project in terms of making sure people made their appointments and kept their appointments and working with respiratory therapists here to organize all this. I think it was just pretty much a fair amount of volunteerism, is what I think it was. [laughs]

Hughes: And there weren't any glitches?

Follansbee: Well, I think there were a fair number of glitches, just from the standpoint that we didn't have clear criteria about how we were going to document Pneumocystis if it occurred. Also, getting people to make sure they were on time to their appointments and all that. It took a fair amount of volunteerism from personnel and staff and all that. So it was somewhat rough in terms of how we carried this out. I don't remember discussions about sample size, all the kinds of things we think about now.

So it took a fair amount of work for this outside agency, a CRO, a clinical research organization, to come in and spiff up the records to really get them into a format that could be reviewed.

Hughes: By the FDA.

Follansbee: By the FDA.

Hughes: What sorts of things are you talking about?

Follansbee: Oh, everything from making sure that there was an informed consent signed and dated, to case report forms filled out in black ink, to investigators' signatures, to documentation of events, to correspondence. Where is the correspondence over all of this, and how do you know you have IRB [Institutional Review Board] approval? There's just a lot of necessary book work. Confirmation that these patients really existed. The FDA needed to know that there was some source documentation that these patients were real. The case report form doesn't serve as the source document; it has to reflect a source document from somewhere else. So we had to create source documents that these patients really existed.

#### The Community Research Initiative

Hughes: Interesting. Well, I understand that the Community Research Initiative, the New York-based group, had from the start direct involvement by patients.

Follansbee: Yes.

Hughes: That, I understand, is not true of the CCC.

Follansbee: Right, in fact, there was a competing organization for a while. There was a Community Research Initiative in San Francisco that was going to do studies that were maybe more reflective of the patient community.

Hughes: Modeled after the New York group?

Follansbee: Modeled after the New York group. I think that Project Inform was somewhat involved in that movement early on and was sponsoring that or working with that.

Hughes: Were community-based physicians involved with that group as well?

Follansbee: There were some, with more episodic involvement, and it was clearly a competing group. They may have even applied, when



the request went out for community-based sites for the CPCRA, to be a site along with the Consortium application.

Hughes: And what happened to them?

Follansbee: They died out, because they didn't get the funding. The consortium now is really dominated by its CPCRA budget. The majority of the research that it's done has been through the CPCRA and its funding. The nucleus that was started by Dianne Feinstein and Paul Volberding is a peripheral wing to the current structure.

#### Community Advisory Board

Hughes: Talking about patient participation, I understand the CCC does have a community advisory board.

Follansbee: Yes, it does, and they review all the protocols, whether they were generated within the consortium or are CPCRA protocols. The community advisory board is quite active in that and has helped. We've looked at some pilot studies of viral load, for example, and have helped in their design in terms of what would be of interest to the community.

Hughes: Are there people on that advisory board that you would term activist? I'm thinking of people like John James.

Follansbee: Yes, there are people on the community advisory board who represent the activist community. But you have to remember that the activist community is a rather diverse group that doesn't always get along within the community. So it's probably communities.

Hughes: You have to catch me on that. [laughter] There is very seldom "a" community in anything.

#### Meeting at Lia Belli's about the Politics of AIDS

Follansbee: There was a meeting that went on at Melvin Belli's house that was organized very early, that Lia Belli was involved in, and I'm trying to remember the person who organized this. He was an aide to either the mayor or to [Art] Agnos up in Sacramento



at the time. That was a very big meeting, and I'm wondering if your history is covering this.

Hughes: There was a meeting that occurred at Lia Belli's in association with the bathhouse issue.<sup>1</sup> Was that about the time?

Follansbee: Maybe that was it. I can't remember the details of the meeting. I remember going to this meeting, and I remember a lot of people being there. I thought that it was not specifically about the bathhouse issue, but it certainly would have come up. It was about the politics of HIV and AIDS, and I think it was prompted by the AIDS Foundation or the KS Foundation.<sup>2</sup> She had an attic in that house, and there was a buffet dinner, and then there was probably an attic meeting. There must have been fifty people there.

Hughes: You're probably right when you bring in the AIDS Foundation, which may at that stage have been the KS Foundation. Lia Belli was a board member for a period.

#### Presidential Commission on AIDS, 1987

Follansbee: All right, now you wanted to ask about the Presidential Commission?

Hughes: Yes, and just to fix the date, it was September, 1987, according to your papers.<sup>3</sup> [shows paper]

Follansbee: Yes.

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<sup>1</sup> Randy Shilts. And the Band Played On: Politics, People, and the AIDS Epidemic. New York: Penguin Books, 1987, p. 258. For Lia Belli's association with the AIDS epidemic, see the oral histories in this series with Marcus Conant, M.D., and Andrew Moss, M.D.

<sup>2</sup> The Kaposi's Sarcoma Research and Education Foundation became the San Francisco AIDS Foundation. For details, see the oral history in the AIDS physicians series with Marcus Conant, M.D.

<sup>3</sup> This document is reproduced and cited in the appendix of this volume.

- Hughes: Why were you invited to make a presentation?
- Follansbee: I suspect that I was invited probably through my activities at Davies, that the administration here has political ties, and that it was felt that if the task force was out here serving the community, that the private community needed to be involved in the presentation and have formal comments. So I suspect that I got involved via my link here as a community physician although I used stationery from when I still had an office on Army Street. I think I maybe still was on Army Street, but I was more involved at Davies in terms of AIDS.
- Hughes: Were you the only representative of the San Francisco private practice community?
- Follansbee: I don't remember. I remember sitting at a table and having the commission in front of us along this long table, but I don't remember even where it occurred, what building it was in. I remember people listening and all that, but I'm trying to remember if there were any questions. It's so vague.
- Hughes: Well, please skim this document. I interpreted it as your notes of what you were to say, because it is informally typed, let's put it that way!
- Follansbee: [reads silently] Terrible spelling! Yes, I think it was obviously an attempt at fundraising for the city and for the community, and trying to focus on the community nature of this illness and the kind of issues that were surfacing in 1987.
- Hughes: Yes, the community nature of the illness and the important role of the community practitioner is strong and clear. And you also make the point that the epidemic is hitting hard at the practitioners, not only those who are infected, but just the grueling nature of treating this disease. What were you hoping would come from that observation?
- Follansbee: I think I was hoping that there would be some legislation that would make the practice of medicine easier vis-a-vis HIV. The amount of time that we spent trying to get services, get access to medications, having to fight for social services was fatiguing. We couldn't even focus on the medical aspects necessarily without having to spend so much time with the other aspects of health care delivery. And so I think that it was an attempt to get the government to really commit itself to HIV care for everyone infected. And also, I think I meant to defuse any sense that there was a town-gown fight. The commission had to try to broaden their perspective and make sure they understood that HIV care was something that had to

get out into the community where patients were, and doctors were, and nurses were.

Hughes: Do you feel that your message was received?

Follansbee: Oh, yes, it was received; I just don't think that there was anything they could do about it. I don't think the commission ever had any real power, or even had much of an ear in 1987 at the federal level.

### Concluding Thoughts

Hughes: Would you like to sum up?

Follansbee: I've never read Randy Shilts' book, but I knew Randy, and I was involved in his care. And I liked Randy. I never read his book because this whole epidemic was so close to home. On the one hand, I think giving these interviews was somewhat useful just to show, number one, how little I remember. But also, I have the sense, despite some of the complaints about being called by investment brokers about new drugs and the monies to be made and the kind of fringe issues around HIV care and profit-taking and all that, that in fact the response by the San Francisco community was pretty amazing and pretty well organized, all things considered. There has been a lot of progress in this period of time, and there were a lot of heroes. Bill Kraus was the one who organized the meeting at Lia Belli's. And it's people like Bill who clearly recognized, for better or worse, the impact the epidemic was going to have on our community.

As part of this process with you, I did go back into my journal and was amazed at how little I wrote about what was going on, but I did find the obituary on Pat Cowley, who died in '82.

Hughes: Now, is this one of your very early cases?

Follansbee: My first patient at UCSF, who got into the Annals article, was not someone for whom I made the diagnosis, but the second patient was someone for whom I made the diagnosis of Pneumocystis. He taught me a lot about myself and really about the human side of this epidemic. Reading the obituary when this was already called AIDS, and he being an early casualty in the epidemic, I mean, that person is a hero to me, because some of the lessons that I got from him have stayed

true. The complexity of this epidemic, and what was in store for us all. It's not all as simple as black and white in terms of the evolution of this epidemic, the issues of civil rights, of behaviors, of treatment decisions.

I wish I'd been better about keeping a history as I went along, keeping notes so that this can be written about when the whole thing [AIDS] is conquered. Even in 1987 I still assumed that this epidemic was going to be conquered, and now almost nine years later, I still assume it's going to be conquered.

Hughes: What do you consider to be your greatest contribution?

Follansbee: I think that my greatest contribution was the fact that I took care of people with HIV, that I was one of the people who had the privilege, honor, fate to be in the right place at the right time, and to take care of people who really needed health care and who still need health care. It's that one-to-one contribution, with those patients, their partners, their family members, that is my greatest contribution.

Hughes: Thank you very much.



TAPE GUIDE--THE AIDS EPIDEMIC IN SAN FRANCISCO: THE RESPONSE OF COMMUNITY  
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## BIOGRAPHICAL SKETCH

Give the following information for the key personnel and consultants and collaborators. Begin with the principal investigator/program director. Photocopy this page for each person.

<b>NAME</b> Paul O'Malley	<b>POSITION</b> Project Manager		
<b>EDUCATION</b> (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)			
<b>INSTITUTION AND LOCATION</b>	<b>DEGREE</b>	<b>YEAR CONFERRED</b>	<b>FIELD OF STUDY</b>
Massachusetts State College, Worcester, MASS	BA	1968	BIOLOGY

**RESEARCH AND PROFESSIONAL EXPERIENCE:** Concluding with present position. List, in chronological order, previous employment experience, and honors. Key personnel include the principal investigator and any other individual who participate in the scientific development to execution of the project. Key personnel typically will include all individuals with doctoral or other professional degrees but in some projects will include individuals at the masters or baccalaureate level provided they contribute in a substantive way to the scientific development or execution at the project. Include present membership on any Federal Government public advisory committee. List, in chronological order, the titles, all authors, and complete references to all publications during the past three years and to representative earlier publications pertinent to this application. DO NOT EXCEED TWO PAGES.

### Professional Expedience:

1969 - 1973	Communicable Disease Control Counselor, Environmental Health Inspector, Occupational Medicine Specialist, United States Air Force, Florida and Michigan
1973 - 1977	Public Health Counselor, STD program, San Francisco Department of Public Health (SFDPH)
1978 - 1980	Project Coordinator, Hepatitis B Prevalence and Incidence Study, STD Program, SFDPH
1980 - 1989	Project Coordinator, Hepatitis B Vaccine Trial and Follow-up and Hepatitis B Booster Program, STD Program, SFDPH
1984 - Present	Project Manager, HIV Natural History Study, STD Program and AIDS Office, SFDPH
1992 - 1993	Project Coordinator, Health Care Access Study, AIDS Office, SFDPH
1992 - 1997	Program Manager, HIV Vaccine Preparedness Study, AIDS Office, SFDPH
1997 - Present	Program Manager, HIVNET HIV Prevention Clinical Trials, AIDS Office, SFDPH
1998 - Present	Program Manager, VaxGen Phase III HIV Preventive Vaccine Trial, AIDS Office, SFDPH

### Publications:

1. Buchbinder SP, Holmberg SD, Scheer S, Colfax G, O'Malley P, Vittinghoff E. Combination anti-retroviral therapy and incidence of AIDS-related malignancies. *JAIDS* 1999; 21:S23-6.
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TIME TABLE OF EVENTS IN UNDERSTANDING AND  
PREVENTING VIRAL HEPATITIS

- 1885 Lurman and Jehn connect outbreak of "catarrhal jaundice" with smallpox vaccination at Bremen shipyard and Merzig insane asylum
- 1908 MacDonald hypothesizes that "acute yellow atrophy" is caused by a filterable agent virus
- 1926 Flaum concludes that "epidemic jaundice" was being transmitted by spring-driven lancet used to draw blood samples from diabetics ("Schnepper Ikturus")
- 1937 Findlay marshalls evidence that hepatitis was being transmitted by human serum used in preparing yellow fever vaccine
- 1942 Outbreak of 28,000 cases of hepatitis occurs (with 62 deaths) among U.S. troops headed for Pacific theater who were vaccinated against yellow fever with a preparation containing human serum
- 1942 Voegt transmits hepatitis to human volunteers by blood and fecal filtrates
- 1943 First use of term "serum hepatitis" to distinguish hepatitis transmitted by vaccines from community-acquired "infectious" (or epidemic) hepatitis  
*Hep. B (blood)*
- 1944 Stokes and Neefe prevent infectious hepatitis by administering immune globulin  
*Hep. A (fecal)*
- 1950-70 Krugman and colleagues do series of experiments at the Willowbrook School for the mentally retarded that clarify the characteristics of the two types of hepatitis
- 1963 Blumberg finds the Australian antigen
- 1968 Blumberg determines the Australian antigen is the hepatitis B surface antigen (HBsAg)
- 1970 Hepatitis B serological assays available
- 1970 Dane identifies the HBV virion (Dane particle)
- 1970 Krugman demonstrates that heat inactivated serum of chronic HBV carriers can protect susceptibles from infection
- 1972 Hepatitis A serological assay available

TIME TABLE OF EVENTS IN UNDERSTANDING AND  
PREVENTING VIRAL HEPATITIS

- 1972 Screening of plasma donations for HBsAg begins
- 1973 Feinstone detects the HAV virion by immune electron microscopy
- 1974 Recognition of transfusion hepatitis cases not reactive to HBV assays. Centers for Disease Control begin reporting of unspecified hepatitis cases
- 1977 Rizzetto finds the delta agent (now HDV) in serum of an HBsAg carrier
- 1979 Provost grows HAV in tissue culture (rhesus monkey kidney cells)
- 1981 HBV vaccine licensed (made from HBsAg harvested from pooled plasma)
- 1982 Centers for Disease Control adopt non-A, non-B hepatitis as a category in national reporting, retaining unspecified hepatitis for other cases
- 1982 Beaseley prospective study in Taiwan provides conclusive epidemiologic evidence of link between HBsAg carriage and hepatocellular carcinoma
- 1984 Recombinant HBV vaccine shown effective (only licensed recombinant vaccine for humans) *transfusion*
- 1989 Houghton and colleagues identify hepatitis C virus (HCV), using molecular techniques
- 1990 Reyes and colleagues identify hepatitis E virus by the same technique used to find HCV
- 1990 Alpha-interferon is shown to be effective therapy for chronic HBV carriage
- 1990-92 Hepatitis A vaccine proves effective

*Chiron  
of  
Emeryville*

## UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

(Ward 86 correspondence, carton 5  
 folder: research studies in or related  
 to San Francisco, 1984, AIDS History  
 Project, Special Collections,  
 UCSF  
 Library.)

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SANTA BARBARA • SANTA CRUZ

Department of Epidemiology  
 and International Health

SAN FRANCISCO, CALIFORNIA 94143

April 25, 1984

Mervyn Silverman, M.D.  
 San Francisco Department of Public Health  
 101 Grove St. #306  
 San Francisco, CA 94102

Dear Merv:

In my contract with the City, the third topic which I was contracted to investigate was AIDS morbidity in the hepatitis screening cohort. In order to investigate morbidity in the cohort, I need access to it. As you know, the cohort is under study through the Bureau of Communicable Disease Control.

In my original proposal to Dr. Braff, I also suggested investigating prospective AIDS morbidity in eight hundred people in the hepatitis cohort on whom questionnaire information is available. In order to examine morbidity in this group, I need access to the questionnaires. They are on tape, and a tape should be available through Don Francis at the CDC.

I should be delighted to complete both these investigations. However, as you know, the cohort is under study by the CDC, and a request for access is likely to produce general paranoia. Furthermore, the information I propose to obtain may well be available from Harold Jaffe. Thus you may prefer simply to have the CDC generate this information from the hepatitis cohort. Or alternatively, I can meet with Jaffe and Darrow and attempt to determine what is available. I am at your disposal.

On a related issue, Jaffe has proposed to add to the CDC's studies through the Health Department a study of sexual partners of AIDS cases. As you know, we are already engaged in such a study, partly with funding from the City. I would like to object strongly to starting another sexual-partner study in San Francisco. Our experience is that only about half of all AIDS cases can be interviewed, and less than one interviewable sexual partner can be found per interviewed AIDS case. Consequently, we and the CDC would be competing for this scarce resource. I don't think it is necessary to start another partner study in San Francisco, and we have already offered serum from our study to the key virological investigators who are working with AIDS. It would be disruptive to our study for the CDC to start competing for the same people; it would be dangerous to our prospects for getting Federal funding; and it is furthermore unlikely that the CDC will pick up the clinical burden associated with following a high-risk group. I strongly request that you discourage them from this rather opportunistic proposition.

Sincerely,

  
 Andrew R. Moss, Ph.D.  
 Adjunct Assistant Professor  
 Department of Epidemiology & International Health

cc: D. Echenberg, N. Petrakis, W. Winkelstein, M. Conant, P. Volberding, H. Britt, W. Kraus ✓

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## LONG-TERM IMMUNOGENICITY AND EFFICACY OF HEPATITIS B VACCINE IN HOMOSEXUAL MEN

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ERWIN BRAFF, M.D., GREGORY F. SHIPMAN, M.D., PATRICK J. COLEMAN, Ph.D.,  
AND ERIC J. MANDEL, M.S.

**Abstract** To study the duration of antibody persistence and protection provided by the hepatitis B vaccine, we followed 773 homosexual men for five years after completion of vaccination. Among the 635 participants in whom antibody levels above 9.9 sample ratio units (SRU) developed after vaccination, 15 percent lost antibody altogether, and in another 27 percent, antibody levels declined below 10 SRU within five years. The extent of the maximal antibody response strongly predicted the persistence of protective antibody. Hepatitis B infection occurred in 55 men; 8 of these infections were clinically important (characterized by the presence of the hepatitis B surface antigen and elevation of liver-enzyme levels), and two of the patients became hepatitis B virus carriers. The long-term risk of hepatitis B infection was

inversely related to the maximal antibody response to vaccine. Most severe infections occurred among those who responded poorly or had no response to the vaccination. The risk of late infection with hepatitis B in those with an initially adequate vaccine response increased markedly when antibody levels decreased below 10 SRU, but only 1 of 34 late infections resulted in viremia and liver inflammation. A second series of vaccinations induced a moderate antibody response in 50 percent of the subjects who initially had no response or a poor response; however, the persistence of antibody was poor. Both antibody loss and the risk of severe disease should be considered when booster-dose strategies for the hepatitis B vaccine are being designed. (*N Engl J Med* 1986; 315:209-14.)

**C**ONTROLLED studies have shown that plasma-derived hepatitis B vaccines are highly effective in providing protection against hepatitis B virus (HBV) infection.<sup>1-6</sup> Protective antibodies (antibody levels above 9.9 sample ratio units [SRU] by radioimmunoassay) develop in 80 to 97 percent of normal adults who receive the recommended vaccine series. The efficacy of the vaccine in the prevention of viremic HBV infection, with or without elevation of liver-enzyme levels, is 85 to 95 percent, and protection is virtually complete for up to two years in those who respond to the vaccine. Although some HBV infections have occurred in persons who responded to vaccination, almost all such infections have been innocuous and have been detected only through the appearance of antibodies to the hepatitis B core antigen (anti-HBc). Infections that produce viremia or liver inflammation have been limited to persons with no response or a poor response to vaccination.

There are two remaining major questions relating to the use of the hepatitis B vaccine: How long will the protection last, and when should booster doses of vaccine be given? Preliminary studies have reported that 8 to 20 percent of persons in whom protective antibodies develop after vaccination lose antibody within five years.<sup>7-9</sup> Studies that have assessed the long-term protective efficacy of the vaccine have suggested that the risk of infection increases as levels of antibody to the hepatitis B surface antigen (anti-HBs) decline below 10 SRU (comparable to 10 mIU of antibody), but that persons who lose antibody and later acquire HBV infection do not have viremia or liver inflammation.<sup>7,8</sup>

In this study, we examined the duration of antibody persistence and the nature of protection against HBV infection afforded by the vaccine in a cohort of homosexual men whom we followed for five years. We also examined the effectiveness of revaccination of those with no response or a poor response to initial vaccination.

### METHODS

This study was a continuation of the Centers for Disease Control multicenter study of the efficacy of the hepatitis B vaccine in homosexual men, a double-blind, vaccine-placebo trial that was initiated

From the Hepatitis Branch, Division of Viral Diseases, and the Division of Sexually Transmitted Diseases, Centers for Disease Control, Atlanta; the Bureau of Communicable Disease Control, San Francisco Department of Public Health; the Denver Disease Control Service, Denver; and the Howard Brown Memorial Clinic, Chicago. Address reprint requests to Dr. Hadler at the Hepatitis Branch, DVD, CID, Bldg. 6, Rm. 154, Centers for Disease Control, Atlanta, GA 30333.



in April 1980 and in which the efficacy of the vaccine was demonstrated by October 1981.<sup>2</sup> At the conclusion of the original study, susceptible recipients of placebo received three doses (20  $\mu$ g each) of vaccine according to the usual schedule. Both the men who were vaccinated in the original trial and those who had originally received placebo but were vaccinated later were eligible for this long-term follow-up study, if they received three doses of vaccine according to the recommended schedule (second and third doses given one and six months after the first dose). The follow-up study was limited to three of the five original study sites — Denver, San Francisco, and Chicago — at which 1090 of the 1400 participants in the initial study had enrolled. All participants were given the vaccine by intramuscular injections in the arm.

Persons who had no response or a poor response (maximum antibody <10 SRU) to the three-dose vaccine series were offered revaccination with three doses (20  $\mu$ g each) of vaccine according to the standard schedule, starting 12 to 24 months after the initial series. Such persons were followed as a separate group from the time of the first revaccination dose.

Participants visited the clinic at six-month intervals. During the visits, they were asked about recent symptoms of illness or diagnosis of hepatitis, and blood samples were drawn for serologic testing for markers of HBV and serum alanine aminotransferase. Persons with evidence of hepatitis B infection had follow-up visits to confirm seroconversion and ascertain the degree of liver inflammation.

Serum specimens were tested for hepatitis B surface antigen (HBsAg), anti-HBs, and anti-HBc by radioimmunoassay (Abbott Laboratories). Anti-HBs levels were determined by the SRU method described by the test manufacturer. Serum alanine aminotransferase was tested with the manual kinetic ultraviolet spectrophotometric method, as modified by Henry (normal, <50 IU).

A participant was considered to have HBV infection if he had two consecutive serum specimens that were positive for HBsAg, anti-HBc, or both. An infection was considered clinically important (Type I) if the specimen was positive for HBsAg or the level of alanine aminotransferase was more than 2½ times the upper limit of normal. Participants who remained positive for HBsAg for six or more months were considered to be HBsAg carriers. Infections characterized by development of anti-HBc only (without a positive test for HBsAg), elevated transaminase levels, or symptoms were considered to be clinically unimportant (Type II).

The maximal anti-HBs response of a participant who had been vaccinated was considered to be the highest anti-HBs level observed within eight months after receipt of the third dose of vaccine. Participants with increases in anti-HBs levels who had not received additional doses of vaccine or acquired HBV infection were considered to have anamnestic antibody responses, or "booster" episodes. Only participants in whom two consecutive assessments showed anti-HBs levels increased by fourfold and twofold, respectively, above the lowest previous antibody level were considered to have had such an episode.

### Statistical Analysis

Statistical analyses were performed on the follow-up data to assess the following relations: the dependence of antibody persistence and risk of HBV infection on explanatory variables, including demographic features (age, race, and education level), sexual activity (number of steady and nonsteady sexual partners in the four months preceding entry into the trial), and maximal response to the vaccine. Univariate analyses to establish significant predictors were done on the two dependent variables (antibody persistence and risk of infection) with use of chi-square statistics on two-by-two or two-by-n tables. Multivariate analysis was then carried out with use of a stepwise logistic regression program (BMDP PLR). A life-table analysis (BMDP PIL) was performed to examine the time dependence of risk of infection and the maximal anti-HBs level after vaccination.

## RESULTS

Of the 1090 participants from the three clinics in the original vaccine trial, 773 were eligible for the follow-up study. Of these, 751 (97 percent) made at least

one follow-up visit after receiving the third dose of vaccine, and 70 percent were followed until within one year of this report or until they were found to have an HBV infection or were revaccinated. Persons who did not complete the long-term follow-up did not differ in age, race, education, or sexual activity from those who were followed successfully. The complete vaccination series induced maximal anti-HBs levels above 9.9 SRU in 635 participants (82 percent); 69 had a poor anti-HBs response ( $\leq$ 9.9 SRU), and 69 participants had no anti-HBs response.

Among the persons who responded to vaccination with maximal antibody levels above 9.9 SRU, antibody levels decreased slowly during the five years of follow-up. By the completion of follow-up, 15 percent of these men had no detectable anti-HBs, and an additional 27 percent had antibody levels below 10 SRU (Fig. 1). Only 14 percent maintained antibody levels above 100 SRU. Persistence of antibody was directly related to the maximal level of anti-HBs after vaccination (Fig. 2). Among persons whose peak antibody level was less than 50 SRU, 54 percent had lost antibody by three years after vaccination and 70 percent by five years; in almost all others, antibody levels had dropped below 10 SRU. In contrast, among persons whose peak antibody level was above 100 SRU, only 7 percent had lost antibody after five years ( $P < 0.01$ ). An examination by stepwise logistic regression revealed that no other variable was associated with the rate of antibody decline when the maximal anti-HBs level was included in the model.

### HBV Infection in Vaccinated Persons

During the follow-up period, 55 HBV infections were identified in participants who received three doses of the vaccine (Table 1). Eight of these were Type I infections, with all eight persons having both HBsAg positivity and elevated transaminase levels; two persons became HBV carriers. Forty-seven infections were Type II, detected only because of the appearance of anti-HBc antibody. The risk of HBV

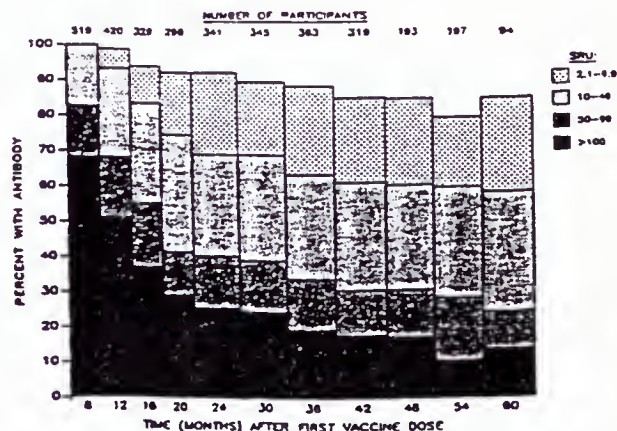


Figure 1. Long-Term Persistence of Anti-HBs in Persons in Whom Anti-HBs Levels above 9.9 SRU Developed after Standard Three-Dose Vaccination.

infection during the follow-up period was directly related to the extent of sexual activity. Persons who became infected had an average of 29 nonsteady sexual partners, as compared with 11.5 partners among those who did not become infected ( $P < 0.01$ ).

The risk of HBV infection was inversely related to the maximal anti-HBs response in the vaccinated persons, independent of the risk due to sexual activity. The risk of any HBV infection was highest in nonresponders (16.7 per 100 person-years); this risk was comparable to that observed in placebo recipients in the early phase of the trial (Table 1). Infection rates were also relatively high in persons with a poor (2.1 to 9.9 SRU) or moderate (10 to 49 SRU) maximal anti-HBs response, but they remained low for those with a good (50 to 99 SRU) response and minimal for those with a strong ( $>99$  SRU) response.

An analysis of cumulative infection rates by the life-table method confirmed these relations and suggested that the risk of HBV infection in vaccinees increased as anti-HBs levels declined, and that it approached the risk in unvaccinated persons as anti-HBs became undetectable (Fig. 3). Participants with no response to the vaccine had a consistently high infection rate between receipt of the third dose of vaccine and revaccination, whereas those with a poor response had a similarly high infection rate after a six-month delay. Persons with a moderate response (10 to 49 SRU) had a low infection rate for nine months after the completion of vaccination and then had an increasing infection rate, which paralleled that in those with no response or a poor response. Infection rates in participants with high peak antibody levels remained low throughout the follow-up period.

HBV infections were most likely to occur in persons whose antibody level before infection was below 10 SRU. Forty-two infections (76 percent) occurred in such persons, and most of the remainder occurred in persons whose antibody level before infection was less

Table 1. HBV Infection Rates in Vaccinated and Revaccinated Persons, According to the Maximal Antibody Response to Vaccine.

GROUP	MAXIMAL ANTI-HBs RESPONSE SRU	NO. OF PERSONS	TYPE OF INFECTION				
			ALL INFECTIONS		HBsAg		HBV CARRIERS
			no.	rate <sup>1</sup>	no.	rate <sup>1</sup>	
Placebo*		529	127	21.1	80	13.3	11
Vaccinated	773 <sup>‡</sup>	55	2.9	8	0.42	2	
	0	69	11	16.7	4	6.1	1
	2.1-9.9	69	6	8.9	2	3.0	1
	10-49	116	22	8.9	1	0.41	0
	50-99	94	7	2.7	0	—	0
>100	425	9	0.74	1	0.08	0	
Revaccinated		79	12	8.3	7	4.9	3

\*In the initial phase of the study (before vaccination).<sup>2</sup>

<sup>1</sup>Per 100 person-years of follow-up.

<sup>‡</sup>Total number of vaccinated men.

than 50 SRU. The estimated risk of infection for persons whose antibody level during a given six-month interval was less than 10 SRU was 6.9 infections per 100 person-years exposed, which was more than seven times higher than the risk for persons whose antibody level was above 9.9 SRU (their risk was 0.94 infections per 100 person-years;  $P < 0.01$ ).

Protection against clinically important, Type I, HBV infection was also inversely related to the maximal anti-HBs response to the vaccine. Six of eight such infections occurred in persons with no response or a poor response to vaccination. Both persons who became HBV carriers were in this group. Only two Type I infections occurred in persons who responded to vaccination. One occurred two months after receipt of the third vaccine dose in a person who had no response to the initial two doses and whose only positive anti-HBs response (23 SRU) occurred one week before HBsAg positivity developed. The other occurred in a person who had a maximal anti-HBs level of 234 SRU after the third vaccine dose, but whose anti-HBs level dropped below 10 SRU 18 months later. This participant had a mild HBV infection 4½ years after the completion of vaccination, with HBsAg positivity for 1½ months, a peak serum alanine aminotransferase level of 250 IU, and subsequent resolution of the infection with development of anti-HBs.

The chance that a late HBV infection would be Type I was markedly lower in persons who had responded to vaccination than in those who had no response or a poor response or in the placebo recipients in the earlier trial. Only 3 percent (1 of 34) of late infections (those occurring more than six months after completion of vaccination) in the persons who responded to the vaccine were Type I, as compared with 35 percent of the infections in those with no response or a poor response to the vaccine, 58 percent in revaccinated persons, and 63 percent in placebo recipients ( $P < 0.01$ ).

Booster Episodes

Sixteen persons who were vaccinated had booster episodes during the follow-up period. The booster re-

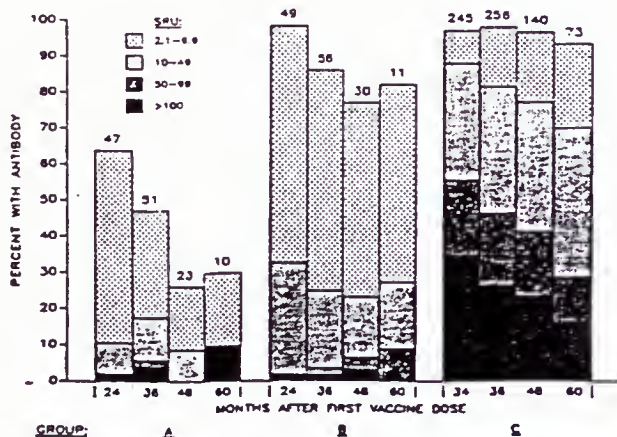


Figure 2. Relative Persistence of Anti-HBs, According to the Maximal Antibody Level after Three Doses of Vaccine.

The maximal anti-HBs level was 10 to 49 SRU in Group A, 50 to 99 SRU in Group B, and  $\geq 100$  SRU in Group C. The number of persons tested at each interval is given at the top of each column.



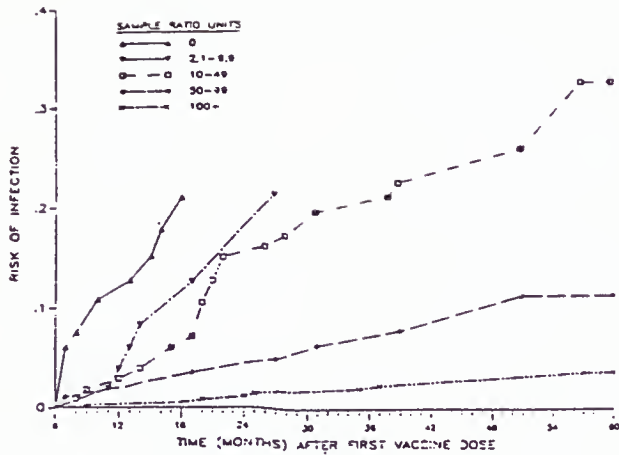


Figure 3. Life-Table Analysis of Risk of HBV Infection According to the Maximal Anti-HBs Level after Three Doses of Vaccine.

sponses occurred throughout the follow-up period in persons with all levels of peak anti-HBs response. Most such responses occurred in persons whose anti-HBs had dropped to a low level (mean, 8.0 SRU); anti-HBs levels immediately after the booster response were an average of 20-fold higher (mean, 184 SRU; range, 14 to 320 SRU). The persons who had booster events had an average of 24 nonsteady sexual partners—significantly more than among the persons without such events and comparable to the number of sexual partners among persons in whom HBV infection developed.

#### Revaccination of Nonresponders and Poor Responders

Of the persons with no response or a poor response to the initial vaccine series, 73 received two additional vaccine doses and 62 received three. The response to revaccination was moderate (Fig. 4). When the men were tested after receipt of the first two additional doses of vaccine, 33 percent were found to have antibody levels above 9.9 SRU, and 14 percent had levels above 49 SRU. After the third additional dose, 50 percent had antibody levels above 10 SRU; however, follow-up testing at 30 months showed that antibody levels remained above 9.9 SRU in only 14 percent.

The response to revaccination was better in those who responded poorly to the initial series than in those who had no initial response. Of the original nonresponders, only 41 percent had antibody levels above 9.9 SRU after the three revaccination doses, and only 8 percent had retained this level at the 18-month follow-up evaluation. In contrast, of the persons who had a poor response to initial vaccination, 79 percent responded to the three revaccination doses, and antibody persisted in 29 percent at 18 months.

Twelve HBV infections occurred among persons undergoing or completing revaccination. The rates for all infections, as well as those for Type I infections only, were comparable to those among persons who had no response or a poor one to the original vaccina-

tion (Table 1). Ten infections occurred before completion of revaccination, and two (both in persons with no response or a poor one to revaccination) occurred after complete revaccination. No infections were observed among persons who completed the revaccination and in whom antibody levels above 10 SRU developed.

#### HBV Carriage

Five persons became HBV carriers after infection. Two had received three doses of vaccine; three were undergoing revaccination. All five persons had had no response or a poor response to all doses of vaccine. The rate of development of the HBV carrier state after HBV infection in nonresponders, poor responders, and revaccinated persons was 18 percent—twice the rate (9 percent) observed in placebo recipients earlier in the trial; however, this difference was not statistically significant.

#### DISCUSSION

This study provides important information on the long-term efficacy of the plasma-derived hepatitis B vaccine now available in the United States. Such data will be critical in devising appropriate strategies for booster vaccination in persons who have received the primary vaccine series but remain at high risk for HBV infection.

Our data show that antibody levels in recipients of the hepatitis B vaccine decline substantially within five years after vaccination and that loss of antibody is closely related to the maximal antibody response of the vaccinated person. Most men with moderate antibody responses had lost all antibody within three years, whereas only a small proportion of those with high antibody responses had lost antibody after five years. These data are consistent with a study of antibody persistence in vaccinated health care workers, which also showed that loss of antibody was closely related to the maximal anti-HBs titer.<sup>9</sup> That study

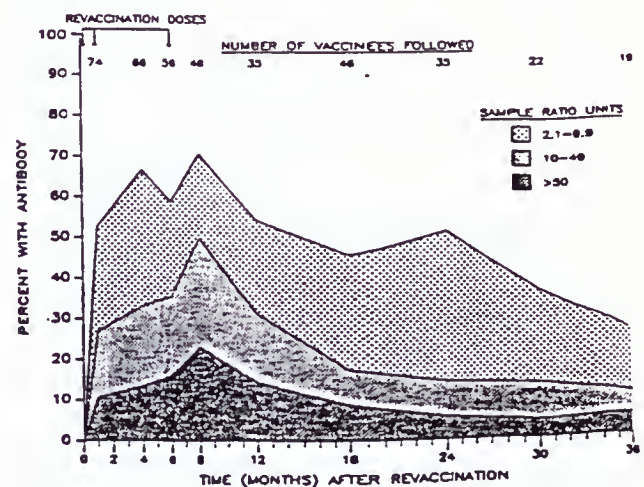


Figure 4. Response to Revaccination in Persons with No Response or a Poor Response to the Initial Vaccine Series.

demonstrated that the geometric rate of decline of antibody was independent of the maximal antibody response, further supporting the concept that the maximal anti-HBs titer is highly predictive of the longevity of anti-HBs.

The rate of antibody loss in our study was slightly higher than that reported in another study of antibody persistence in homosexual men who received twice the usual dose (40  $\mu$ g) of vaccine.<sup>7</sup> However, the immunogenicity of the vaccine in our trial was lower than in the other study, possibly because of the lower dose of vaccine we used (20  $\mu$ g) and an inadvertent partial freezing of some of the vaccine.<sup>10</sup> If the data are adjusted to compensate for the higher proportion of persons with the highest peak anti-HBs response in the other study, the results are more comparable. In recent trials that employed a 20- $\mu$ g dose of newer lots of this vaccine, higher immunogenicity has been observed, and we predict that the rate of antibody loss in these studies will also be lower than that in our trial.<sup>3,11</sup>

The applicability of data on antibody persistence in homosexual men to other groups may be questioned on the basis of two factors: the occurrence of boosts in antibody levels after exposure to HBV in high-risk groups, and the possibility that human T-cell lymphotropic virus Type III/lymphadenopathy-associated virus (HTLV-III/LAV), the organism that causes AIDS and is increasingly common in homosexual men, may modify the response to the hepatitis B vaccine.<sup>12</sup> Both our study and one other study in homosexual men showed clear evidence of booster events, and such episodes may lead to falsely high estimates of antibody persistence.<sup>7</sup> In our study, persons who had booster events shared several characteristics with persons in whom HBV infections developed, including low levels of antibody before the event and high levels of sexual activity. Booster events probably result from exposures to HBV that either do not produce infection or cause only low levels of viral replication, thereby stimulating an anamnestic boost of anti-HBs but failing to induce the appearance of anti-HBc. Nevertheless, in our study, the number of booster events was small, and their effect on estimations of true antibody persistence was minimal.

In contrast, the presence of HTLV-III/LAV infection might be expected to reduce the response to the hepatitis B vaccine and possibly reduce the persistence of antibody. Although there are few data on the effect of HTLV-III/LAV infection on the response to the hepatitis B vaccine, available data suggest that antecedent HTLV-III/LAV virus infection was uncommon in study participants. In San Francisco, the study site with the highest incidence of acquired immunodeficiency syndrome (AIDS), only 6 percent of homosexual men seronegative for HBV who were participating in a study of AIDS at the clinic in 1980 had evidence of HTLV-III/LAV infection<sup>12</sup> (and Echenberg D: personal communication). The risk of HTLV-III/LAV infection at the Denver Clinic was 1,

percent (Judson F: personal communication) and that among the patients at the Chicago site was also presumably low because of the low incidence of AIDS in that location. Nevertheless, direct studies of antibody persistence in other adult groups, as well as in infants and children, are needed.

Because of possible sustained protection in the absence of detectable antibody, recommendations for vaccine booster doses should be based on the persistence of protection against disease rather than solely on the persistence of antibody. Our data provide several insights into the nature of the long-term protection offered by the hepatitis B vaccine. First, the long-term risk of HBV infection in vaccinated persons is closely related to the persistence of minimum levels of antibody. In particular, the risk of infection appears to rise markedly when the antibody level decreases below 10 SRU, and it approaches the risk in unvaccinated persons when antibody disappears. Because antibody persistence is strongly related to peak antibody response, the latter is an excellent predictor of the duration of protection against infection.

Second, the long-term risk of clinically important, Type I, infection remains extremely low in persons who respond to vaccination. Only one late Type I infection was observed among those who responded to the vaccine, in spite of the large number of such persons in whom HBV infection developed. These data strongly suggest that protection against Type I HBV infection (with viremia, liver inflammation, or infectivity to others) outlasts the presence of detectable humoral antibody and protection against HBV infection in general. This is most likely due to the persistence of immunologic memory, possibly cell-mediated immunity, which may be responsible for rejection or resolution of HBV infection. Because the only Type I infection occurred 2½ years after the vaccinated person's antibody level had dropped below 10 SRU, we believe protection may usually persist for several years after the decline in antibody.

Our data do not provide a promising picture of the effectiveness of revaccination of persons with no response or a poor response to the initial administration of vaccine. Although antibody levels above 9.9 SRU developed in 50 percent of persons who received three additional doses of vaccine, most acquired only moderate levels, and the majority lost antibody within 18 months. The rate of HBV infection in this group was not different from the infection rate before revaccination. Curiously, the risks of Type I infection and chronic carriage of HBV did not differ significantly from those in natural infections, and the pathogenesis of the disparate nonresponse to vaccine, as compared with the normal response to natural infection, remains a mystery. Finally, the minority of revaccinated persons in whom persistent antibody did develop appeared to be protected, since no late infections were observed in this subpopulation.

Given the above considerations, what is the best strategy for providing booster doses of hepatitis B vac-



cine? Alternatives include (1) not providing booster doses and relying on immunologic memory to protect against the serious effects of late infections; (2) giving booster doses to all vaccinated persons at regular intervals, without testing antibody levels; and (3) testing anti-HBs levels at appropriate times and giving boosters when protective antibody disappears. The first approach will be difficult to justify without more evidence that protection against clinically important infection outlasts antibody persistence by more than a few years. The second strategy is simple and inexpensive, and it has as a precedent the booster-dose strategies devised for all other inactivated vaccines. The long-term protection against clinically important infection shown in our study indicates that such a strategy is likely to provide good protection to all who respond to the initial vaccine series. Finally, strategies that involve testing of antibody levels and administration of boosters on the basis of the results are more complex than the other alternatives and are likely to be more costly; they may also present practical difficulties in the interpretation of commercially available antibody tests. Nevertheless, such strategies may provide the highest levels of long-term protection against clinically important disease. Clearly, a careful review of the cost effectiveness of the various alternatives is necessary; the data provided here should provide a useful basis for comparing approaches.

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SANFORD M. LEWIS, M.D.

# Map of AIDS' Deadly March Evolves From Hepatitis Study

## *Blood Samples of Gay Men Prove Invaluable*

By Christine Russell  
Washington Post Staff Writer

SAN FRANCISCO—In the late 1970s, thousands of homosexual and bisexual men came to San Francisco's public venereal diseases clinic to participate in a government-sponsored study of hepatitis B.

As they gave blood for the project, they had no way of knowing that a time bomb was ticking on a then-unknown disease far more threatening than anyone could have imagined and that the signs of the future epidemic—AIDS—lay hidden in the vials they filled.

By 1987, health authorities had learned at least 600 of the 6,700 participants had acquired immune deficiency syndrome. About 375 of them were dead from it. And a sample survey taken shows that about 70 percent of the participants tested positive for the AIDS antibody—indicating that they likely were carrying the virus.

For them, and for all of those involved in the study, the figures are tragic testimony to the speed and virulence of the epidemic some experts predict could become the worst in modern history.

But their participation in the study has brought at least one priceless benefit: The stored frozen blood has offered an unexpected opportunity to track the path of the AIDS virus over time in a large vulnerable group, teaching researchers more about the progress of the disease than any other single study. It is, as well, a harbinger of what can happen when the AIDS virus goes unchecked.

The study—which evolved into one focusing on AIDS—is showing, among other things, that the incubation period is much longer than many had earlier suspected; that the chance of becoming sick increases with time among those infected, with the likelihood of getting AIDS greater in the second five years after infection than the first.

The study has been more than science. The history of this re-

search project is the history of AIDS itself. It has been a very human study, staffed largely by gay men who have themselves been touched by the devastating disease they are studying.

"It was a potential gold mine of information," said Paul M. O'Malley, who has coordinated the study for the San Francisco Department of Public Health and the federal Centers for Disease Control (CDC) since its start in 1978. O'Malley has since watched close to 20 of his friends come down with AIDS and come to know dozens more with AIDS in the study.

Both professionally and personally, "we're in the front lines," he said.

"It is the longest followup study of any group of people in the United States or anywhere in the world for AIDS," said CDC official Dr. Harold W. Jaffe. "The study was done primarily because of the cooperation of the gay community, mainly by gay men. They are doing the study as scientists, but the findings are affecting them personally."

"It provided us with a picture window of what happened in a population before AIDS was known to exist and what has happened since then," added CDC researcher Dr. William W. Darrow. "I think that the value of the study and all research is in convincing other people of the real emergency of the situation."

A decade ago, San Francisco was a magnet for gay men, who came from all parts of the country, from all walks of life to explore their sexuality in a city where they were more accepted. Even then there were health consequences—gonorrhea, syphilis and other ailments—that led everyone from businessmen to bartenders to seek confidential medical help at the public clinic.

O'Malley, then a communicable disease investigator for the city, was concerned that the sexual re-

volution could breed more serious health problems for gay men "in the fast lane," but certainly nothing as deadly as AIDS.

"Looking back on that time, I used to think something was going to happen sooner or later. I worried about a resistant strain of gonorrhea or syphilis. I didn't think of some new virus that could decimate the community when people are in the prime of their life," recalled O'Malley, a gay man who had come from Massachusetts after a stint as a public health inspector in the Air Force.

In 1981, the hepatitis study produced a major victory: a hepatitis vaccine tested on the San Francisco group and in several other cities worked. But there was little chance to celebrate, as health officials began to worry about something far more ominous than hepatitis.

At a May 1981, meeting in San Diego, O'Malley heard of a mysterious new illness, one which had caused homosexual men in California and New York to die of rare forms of pneumonia and cancer. Something was apparently attacking their immune systems. A few weeks later the first five cases were cautiously announced to the world in the CDC's Morbidity and Mortality Report.

O'Malley got involved when a man in the hepatitis study called and said he had come down with Kaposi's sarcoma, a skin cancer associated with what would come to be known as AIDS. His interest intensified when many of the first AIDS cases in San Francisco—11 of the first 24 in 1981—turned out to be participants in the hepatitis project.

Many of the hepatitis participants had filled out elaborate questionnaires about their lives, from sexual habits to drug use. In 1983, CDC and the city health department agreed to contact a sample of the participants and question them again to see if they could isolate



special risk factors for AIDS.

By the spring of 1984, researchers at the Pasteur Institute in France and the National Cancer Institute had isolated the cause of AIDS, a new virus that knocked out key immune system cells. And CDC began using an experimental blood test to look for antibodies to the virus.

Using the test and samples of the frozen blood, the researchers tracked the footprints of the AIDS epidemic in the San Francisco men. They found that:

- Undetected, the virus had spread rapidly among this group of sexually active men. With the consent of the participants, CDC tested random stored blood samples and found about 3 percent of the gay men in the hepatitis study showed antibodies to the then-unknown AIDS virus in 1978, rising quickly to 12 percent in 1979, 20 percent in 1980 and 36 percent in 1981. By 1983, 62 percent were positive.

Although the rise has since slowed, preliminary results show that more than 70 percent of those tested in 1986 followup studies were positive on the AIDS antibody blood test.

- A positive antibody blood test generally signals long-term infection. Many people initially hoped that a positive AIDS antibody blood test meant past exposure to the AIDS virus but did not have serious long-term consequences. But CDC studies using data from the San Francisco project have since found that the virus can in fact be found in the blood of most of those who are positive. Because many people in the hepatitis vaccine study gave frequent blood samples, instances in which a person converted from negative to positive can often be pinpointed within months.

- For every case of AIDS, there are far more people who may be infected with the virus but show no symptoms. The government's oft-quoted projections that more than 1.5 million Americans may already be infected with the virus, in addition to over 29,000 reported AIDS cases nationally, is based in part on what has happened to these San Francisco men.

- In San Francisco, the gap is closing faster. In a selected sample of 66 infected men followed for an average of about six years, one-fourth

have already developed AIDS and more than one-third have developed symptoms of illness that sometimes precedes AIDS. Only 25 men—38 percent—remain free of symptoms, said Nancy Hessol, a statistical expert with the San Francisco study.

- There are exceptions. Four people who have been antibody-positive for at least nine years—based on stored blood samples from January 1978—still have not come down with AIDS, O'Malley said.

But most findings have been disturbing. "I hoped my worst fears would not come true. At first the data showed that the vast majority of people won't get sick. I held on to that for a long time. Now there is a very different picture," he added.

Dubbed "Father O'Malley" by a colleague who admires his protective concern for study participants, he said he is not infected with the AIDS virus but admits that "sometimes men like myself have feelings of survivor guilt. How come I slipped through?"

The AIDS project is reached through an unmarked door on Market Street, where a security guard keeps out vagrants. In the fourth-floor suite of offices overlooking city hall even the telephone operator answers with the street address and not the name. But once inside the confidential sanctuary, a giant red flag with bold white letters announces that it is the "S.F. City Clinic AIDS Research Study."

The flag is more than symbolic. O'Malley and many of the small staff of 10 or so carried the banner last June in the annual lesbian and gay rights parade in San Francisco, a parade filled with protest over an AIDS ballot initiative pushed by followers of political extremist Lyndon H. LaRouche Jr. The initiative—later soundly defeated in the November elections—raised fears in the gay community about possible discriminatory actions.

CDC and San Francisco health department officials credit O'Malley and his mostly gay staff with doing a remarkable job while juggling their roles as researchers, city employees and members of a community so dramatically affected by the epidemic.

"I don't think we made a conscious decision to use gay men as the investigators," said CDC's Jaffe. But, "it may make the study partic-

ipants more comfortable in participating. They may not trust the federal government. They may not trust the city government. But they do trust Paul and his staff. It means a lot of credibility."

Every participant in the study is guaranteed confidentiality. Perhaps most sensitive aspect of the study has been its use of the blood test that measures the presence of antibodies to the AIDS virus. Participants are offered the opportunity to learn the results of their blood tests or to choose not to, with all findings in the research statistics anonymous.

Doug Franks, now a 34-year-old research associate for the San Francisco AIDS study, has been exposed to and presumably infected with the AIDS virus.

He said that in the late 1970s he had numerous sexual relationships with both men and women, including a 1978 affair with San Francisco Supervisor Harvey Milk shortly before he was slain by a political foe. In 1979, Franks came to the city clinic for a checkup and became a participant in the hepatitis B study. In 1984, Franks heard of the new AIDS study and volunteered to be tested again.

Last year, he was hired by the health department and asked to work on the AIDS project, conducting interviews with study volunteers. He also decided to learn his own AIDS test results.

Tests of his stored blood showed that when he entered the hepatitis study in 1979 he was negative for antibody to the AIDS virus, but by the time he was retested in 1984 he was positive.

He feels fatigued at times and has shown signs of swollen lymph nodes. "When you see the statistics this study generates, it's not hard to project what's going to happen. It's going to be a pretty bumpy ride . . . . The possibility of coming down with AIDS in the future seems very real."

For Franks, the decision to learn his blood results was "a benefit . . . . Knowing your own antibody status can motivate you to make the necessary changes in your life style, to reduce the risk of coming down with AIDS as well as protect others from infection." He was able to inform former sex partners so they too could be tested and now has a

live-in lover. And the knowledge that he is positive makes him more eager to consider "aggressive action" as new AIDS virus therapies come along.

Concern about the impact of telling men in the study that they had been infected with a potentially deadly virus led staff member Michael A. Frigo to do a followup study on the psychiatric reactions. At the Second International Conference on AIDS in Paris last June, Frigo reported that in a sample of 800 men, about half of those tested elected to find out the test results—of them about two-thirds were indeed positive for infection.

Each was provided with counseling. Although there were no reports of suicide attempts or psychiatric hospitalization, Frigo reported "significant negative psychological impact," with "increased fear, anger and possible depression one month after receipt of results."

Long-term followup continues. But within two weeks of returning from Paris, Frigo was struck by a sudden illness and died of AIDS in a San Francisco hospital.

Meanwhile, the numbers of AIDS victims from among the San Francisco project's 6,700 volunteers account for about 20 percent of San Francisco's AIDS cases and one out of 50 cases in the United States.

"So far it's a pretty scary picture," says Torsten Weld Bodecker, 31, who updates the AIDS project's confidential computer data base. "Periodically what hits me when I print a list of code numbers with date of birth and date of death is to see, page after page, they are my age, they are my friends, they are people who I have interviewed. . . . I don't see those numbers decreasing; I see those numbers increasing."

Every day he must discreetly contact men to ask them to rejoin the study, to come in and undergo a complete physical as well as an extensive, hourlong interview that goes into the most intimate details of their lives—the kind and amount of sex they have, whether they use drugs, whether they drink—as well as memory tests to check their recall.

"The bottom line is that a lot of people are dying. I tell them they have an opportunity to do something about that. . . . I don't try to

harass them," he said. The fact that he is gay "helps break the ice. They know I'm not going to be judgmental and that I have the same kinds of concerns they have."

Bodecker too faces a very personal threat. He has been antibody-positive on the AIDS blood test for nearly four years and still feels completely healthy. But, just in case, he has "written a will and made it clear what I want done with my remains." He was willing to talk about himself "because this disease needs to have stigmatization taken away as much as possible. It's important to be upfront."

While many gay men have been reluctant to join the government-sponsored AIDS study, many of those who have are enthusiastic about their participation; for personal and altruistic reasons.

"It's like a confessional, only it's scientific and not in a church," said London Wildwind, a 40-year-old Arkansas-born gay "psychic healer" who has been in San Francisco for two decades. "If you want self worth and value, sometimes you have to be a volunteer. You have to be honest."

"It's a question of love your neighbor and help each other out. It's not a gay disease. It's a disease of the people. That's the only way we're going to lick this," said LaRon Gneiting, 37, a gay merchandise manager who came here from Blackfoot, Idaho, after a failed marriage.

There have been a few rays of sunshine in the gloomy news generated by the San Francisco study. Most significant, said CDC's Darrow, have been the changes in sexual behavior documented by the study.

He says that in 1978, the average number of different sexual partners over a four-month period among gay men in the hepatitis study was 30. When the group was reinterviewed in 1984, the number over the same time period had dropped to six. In 1985, it went down to four and in the last survey in 1986, down to two.

"The number who have decreased high-risk exposure, such as anal intercourse, has decreased as well," Darrow said.

"The vast majority of men interviewed are dealing with this epidemic in a realistic manner," O'Malley

said. "In fact, in the past three years San Francisco has dropped from second to thirteenth place among the nation's cities in the incidence of gonorrhea, and the rectal gonorrhea decline has been the most dramatic of all in this disease group." A decline in VD is considered evidence that unprotected sexual activity is declining.

While health authorities urge the use of condoms and sex that does not involve the exchange of bodily fluids to reduce the spread of AIDS, "monogamy and celibacy have been the alternative for a growing number of gay males," O'Malley said.

Over the last three years, about 2,000 men who participated in the original hepatitis study have been contacted and 1,200 of them have been included in the new AIDS study. Now the San Francisco AIDS project is starting an expanded nationwide effort to find the remaining 4,700 men who participated in the original study and confidentially ask for their help in hopes of finding more clues about why some people get AIDS and others don't.

Negotiations are under way with a drug company to offer some men in the study a chance to participate in a new study of the anti-AIDS drug AZT and its effect on those who have been infected with the AIDS virus but shown no symptoms of illness. Those who have received the highly publicized drug thus far have largely been AIDS patients.

The San Francisco researchers are sensitive to the need to offer the AIDS study participants whatever help should become available, rather than simply follow the men over time.

"We don't want to be accused of being the Tuskegee of AIDS," said Dr. George W. Rutherford, medical director of the San Francisco health department's AIDS office. The Tuskegee Institute study was a controversial federally funded study of untreated syphilis, begun in 1932, in which the effects of the disease in black men in rural Tuskegee, Ala., were followed for 40 years, even after penicillin treatment became available.

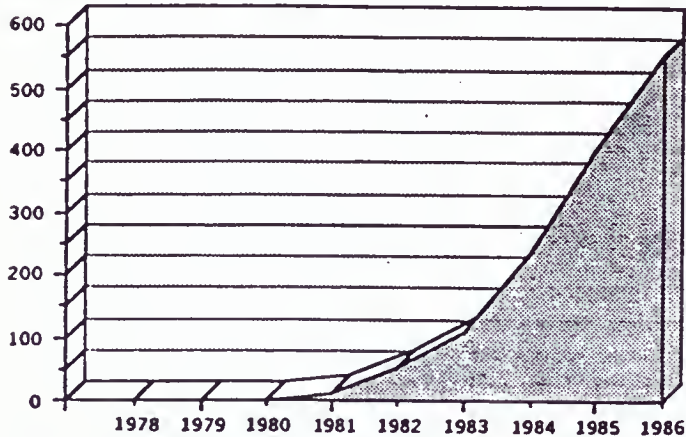
Added O'Malley: "We want to do something for these men, rather than just write them off."



# THE SAN FRANCISCO CITY CLINIC'S AIDS STUDY

## NUMBER OF PARTICIPANTS DIAGNOSED

This chart, based on information from San Francisco City Clinic's study of 6,700 homosexual and bisexual men, shows the number of men studied who were diagnosed with AIDS. Statistics are thought to be an underestimate, with more case reports for 1986 expected.

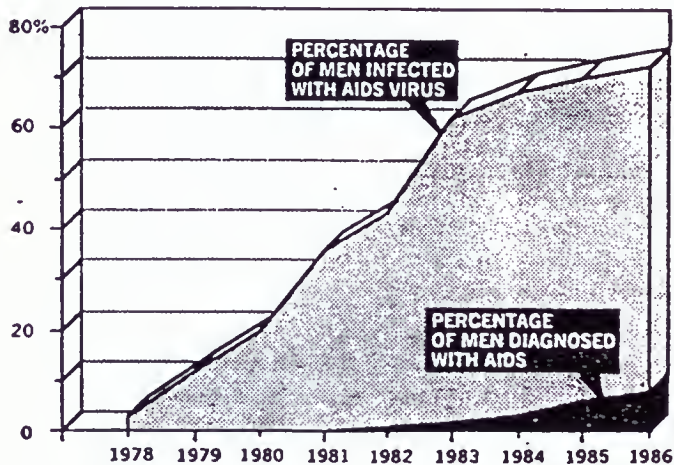


SOURCE: San Francisco City Clinic AIDS Research Study

## PERCENTAGE OF PARTICIPANTS INFECTED AND DIAGNOSED

This chart shows the percentage of men studied who became infected with the AIDS virus, and the percentage of those subsequently diagnosed as having the disease.

Statistics for infection are based on AIDS virus antibody tests performed on random blood samples, with positive findings presumed to show infection. Statistics for AIDS cases are based on actual diagnoses tracked by the San Francisco study.

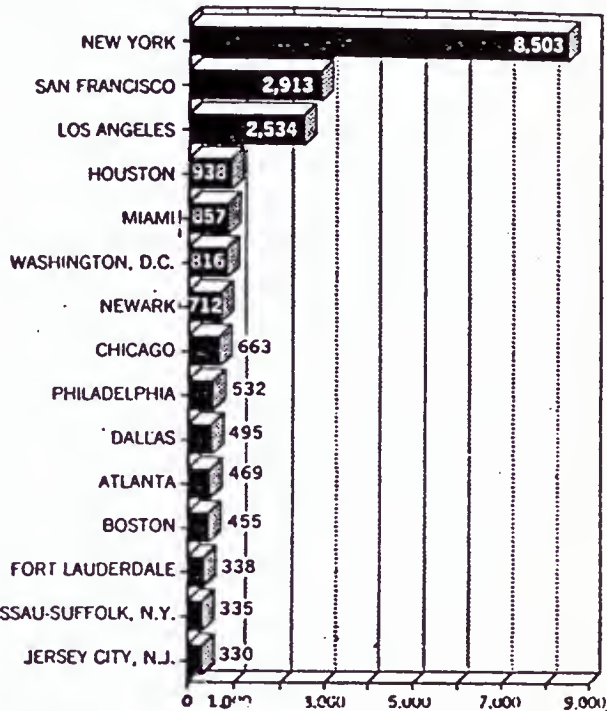


SOURCE: San Francisco City Clinic AIDS Research Study

THE WASHINGTON POST

# AIDS: THE NATIONAL PICTURE

METROPOLITAN AREAS WITH THE GREATEST NUMBER OF REPORTED CASES\*



\*As of Jan. 12, 1987

SOURCE: Centers for Disease Control

THE WASHINGTON POST

## MEDICAL WATCH

## 'Heroes' in the AIDS battle

By Judy Foreman  
Globe Staff

**S**AN FRANCISCO - They are, as the journal *Science* put it recently, the true "unsung heroes" of the AIDS epidemic.

Not the scientists in the headlines, or the doctors rationing out precious drugs, but gay men, who though more threatened by AIDS than any other group have refused to become its victims.

Nowhere more than in this city, where nearly 3,000 have been diagnosed with AIDS and 1,773 have died, have those most susceptible to AIDS offered themselves as human guinea pigs, putting up with frequent tests to track the spread of the deadly virus through their community and their bodies.

Across the city, which is bracing for an estimated 13,600 to 20,000 cases by 1992, their self-help efforts have won the respect, even the awe of doctors used to an un-mobilized, un-activist populace.

Together, the gay men of San Francisco created the country's first live-in hospice, Coming Home, a paint-still-wet, modern home for the dying which opened last month in a renovated convent across a quiet street from the Most Holy Redeemer Church in the city's Castro section, which has a large gay population.

Gay men have also helped turn ward 5A of San Francisco General Hospital into a surprisingly non-depressing place where volunteers offer emotional support for AIDS patients and staff alike, say ward physician Dr. Michael Clement and Dr. Paul Volberding, head of the AIDS service.

Gay men have focused the Shanti Project, a bereavement counselling group, on aiding people with AIDS, their families and friends. Gay men also run the AIDS Foundation, which raises funds, does counselling and provides a free food bank for victims of AIDS, or acquired immune deficiency syndrome.

#### Gays likely to remain pivotal

Even as AIDS spreads to heterosexuals, gay men are likely to play a pivotal role in studying and combatting the disease.

For the foreseeable future, California epidemiologist Andrew Moss testified in 1985 before Congress, "most of the sick people whom we are going to see in the clinic are going to be gay men."

"So, the antiviral therapies that the world is waiting for are going to be tested in clinical trials on sick gay men," he said. "The prevention trials, if we manage to get them started, will be done only with the support of [infected] gay men."

"And the vaccine, when it comes, like the Hepatitis B vaccine, will probably be tested only with the cooperation and assistance of gay men. So I urge you to remember as AIDS heads into its second, heterosexual, decade, that ironically, the cure and prevention the whole world is waiting for are going to come only with the support of its first and most stigmatized group of victims."

No one, of course, knows that better than the 70,000 gay men of San Francisco, half of whom



Globe photo/Doug Atkins

Torsten Weld Bodecker draws blood from Michael Sarfaty for research on AIDS.

are believed infected with the AIDS virus. For some, work is a respite from AIDS. But for others, like Torsten Weld Bodecker, 31, there is no escape from the disease that is both career and catastrophe.

One of 10 staff members at the discrete, unmarked offices of the San Francisco City Clinic AIDS Study, Bodecker has known for four years he carries the AIDS virus.

He is not one of the men being tracked by the City Clinic study and he remains symptom-free, but as he works with the study data day after day, he knows from the study data that his chances of avoiding AIDS get slimmer over time: The study shows that after five years, about 15 percent of those infected develop AIDS; after six years, more than 22 percent; after seven years, more than 32 percent.

Living in such nonstop contact with AIDS is "very hard," Bodecker acknowledges. One staff member has died of AIDS and staff members have lost dozens of friends to the disease.

Yet day after day, Bodecker, his blue eyes calm and his voice gentle, provides the sensitivity epidemiologists say is invaluable in enlisting the cooperation of sick and scared men.

He does not have an MD or PhD degree, but Bodecker's job is to draw blood from men exposed to the AIDS virus, to administer neuropsychological tests to see if they have developed symptoms of AIDS-dementia, to interview them about medical, sexual and drug histories and to help with the statistical number-crunching for projections of AIDS cases.

Contributions like that, says Dr. Harold Jaffe, an epidemiologist at the federal Centers for Disease Control, which is helping with the City Clinic study, greatly facilitate research.

"It is done primarily by gay men on a problem that is [crucial] for gay men," Jaffe says. "but it's very difficult to do a study when your friends and colleagues are dying."



## A SPECIAL REPORT

## LIVING WITH THE EPIDEMIC

## Clinic worker gains trust of clients

*Rewarding job has grim realities*

IT WAS always just "the clinic." There was no need to say more; everyone knew exactly what you meant.

"I was walking along Copacabana Beach in Rio 10 years ago," said Paul O'Malley. "Some guy came up to me and said, 'You work at the clinic, don't you?'"

The same thing happened during a blizzard in Denver, at a bar in long Kong and in a restaurant in Paris.

As many as 1,000 gay men a week visited the San Francisco City Clinic in the late '70s. Over the years, many of them came to know, trust or at least recognize O'Malley. A former Air Force public-health inspector, he joined the clinic as a disease-control investigator when he moved to The City from Massachusetts in 1973.

Now the 43-year-old program manager of the Clinic Study sees many of those same faces in his office — or on the obituary page of the Bay Area Reporter, a San Francisco gay weekly.

"The plague happened quickly," O'Malley said. "This is in slow motion. And it doesn't get any easier."

Although O'Malley is seronegative, he said he has some idea what people with AIDS or HIV go through because he had cancer in 1980.

His current lover of three years was diagnosed with AIDS in 1988



EXAMINER PHOTOS BY ELIZABETH MANGELSDORF  
Paul O'Malley, Clinic Study program manager, is recognized all over the world.

— three weeks after O'Malley's ex-lover died. Twenty-five friends have died or have been diagnosed. There have been seven cases of AIDS in his Castro apartment house in the past seven years. Four others were diagnosed while working at the Clinic Study; one is dead.

"Maybe it sounds like the Warsaw ghetto every day, where they're carting the bodies out of the gutter," O'Malley said. "But it's not."

Instead, the mundane and the morbid coexist. For O'Malley and his lover, life consists of gardening, dinner parties and injections of alpha interferon — of going to memorial services and going to the grocery.

"It would be nice to just look at the data, or to emotionally separate yourself from what you're looking at," he said. "But you can't."

O'Malley deals with his own stress by reading in a gym as he pedals a stationary bicycle.

"I don't read anything about AIDS," he said.

— Patricia Yollin

## Learning how to live a full life

*He triumphs over broken dreams*

YOU WILL have a very pleasant experience," the fortune cookie said.

It came at the end of a day in which Torsten Bodecker was told he had AIDS.

"It just blew me away," Bodecker said. "I'm going to keep it to remind me, because I don't see that this has to be a horrible experience either for me or for my family."

As a teen-ager, Bodecker entered a mental hospital twice, largely because he was gay. The first stay was voluntary; the second came after a suicide attempt. At age 20, he moved to the Bay Area to study art. Instead, he became the first man to graduate from the Women Studies program at San Francisco State. He volunteered early on at Shanti and the Names Project and joined the staff of the Clinic Study in 1985.

"Unintentionally, AIDS became the focus of my life in more ways than I ever expected," said Bodecker, 34. "That's what I have to look back at."

"There are a lot of broken dreams that I had. I was going to be an artist, I was going to be a writer, I was going to do all these things that I haven't done. But I have done this, and I can feel proud about the things I've done and will continue to do."

Last year, Bodecker's father died of cancer at age 66. He insisted



The death of Torsten Bodecker's father taught him a special lesson about life.

on dying at home — in the cold and snow of New Hampshire in February — in a house with no heat and no running water. Volunteers from the local hospice chopped wood; the neighbors brought food.

Bodecker and his two brothers were there, along with his father's best friend from Denmark. "We laughed and joked and sang songs and then he died," Bodecker said.

They removed the medical paraphernalia from the room, bathed the body in rosewater, rubbed it with almond oil and covered it with the Danish flag. Then they lit candles and read a poem his father had written.

"It was just so, so, so, so special," Bodecker said.

"And I feel like it was such a gift my father gave me in his dying because I don't know what will happen with me. But there's a model there of how to die and how to support the people who live. Because those are the people it affects — those who stay behind."

— Patricia Yollin



# 10 Years Later, Hepatitis Study Still Yields Critical Data on AIDS

In one group infected by 1980, 21 percent are free of disease.

By BRUCE LAMBERT

**M**ORE than 16 years ago, 4185 gay men here rolled up their sleeves and answered 175 samples of blood, urine and semen, including their sexual and drug habits. They had volunteered for a study on the spread of hepatitis B, a serious problem among gay men.

But when an even deadlier plague was found to be sweeping through the gay men in the 1980's, the researchers realized that the blood samples and questionnaire they had gathered about hepatitis B were also a source of new infections and the spread of death. Today the San Francisco City Clinic Cohort Study is continuing to provide new data. Researchers call it the most valuable research project on AIDS infection and disease.

Each year findings are updated, and each year they yield new clues about the disease.

**Unusually Detailed Records**  
This year researchers reported on 345 men, all of whom were infected when the study began and whose records are unusually detailed, because they also participated in a trial of a hepatitis vaccine. Although 61 percent of them have developed AIDS and 61 percent of those who developed it had symptoms. The other 21 percent have early indications of the disease.

The long-term survivors may simply have weaker strains of the virus than those men who got sick, or they may be protected by unusually strong immune systems, researchers speculate. Some of the men may also have benefited from taking experimental medications.

The researchers keep in close touch with these men. At one time, they traveled around the country to draw blood samples and administer further tests to men who had moved away. Now the men are periodically flown here for full physical examinations.

**'A Face and a Life'**

As the project continued, bonds developed between the health department staff members and the study participants. "Each of the 4,705 is a face and a life," said Dr. Alan R. Lifson,



Dr. Alan R. Lifson, the director of the continuing San Francisco City Clinic Cohort Study.

researcher would be "see by now. Why the rate is slowing is uncertain, Dr. Lifson said. One reason might be the onset of symptoms. But most of the men who have survived with no signs of illness are not taking the drug, leading researchers to suspect that other factors may be at play.

The prognosis for the 21 percent who as yet have no symptoms of AIDS is also unclear, Dr. Lifson said. In a blood analysis done this year for the first time, a third of this group showed abnormally low counts of immune system cells called T cells. A decline in these cells is an indicator of impaired immunity, and often precedes AIDS.

### Shock and Uncertainty

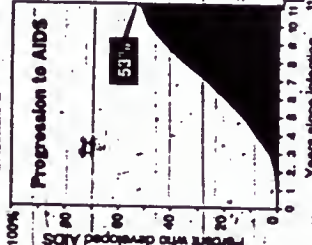
One force behind the analytics is Eric C. Warden, the 39-year-old computer applications programmer for a long time, he did not expect to be an organizer. The permission they need under California law to test this blood samples for the AIDS virus. But in 1987 he agreed. "Tania showed he had been infected since 1980, when he first volunteered."

Even though he expected that Warden said he is "a little shocked," Mr. Warden said he is "a little shocked" because it is the most common lethal complication of AIDS, and he remains healthy. "I'm excited I'm not sick," he said. "On the other hand, now that more and more people are getting sick, I wonder if I will too, and when."

Another volunteer, Howard A. Moffet, 32, said he agreed to participate "because it seemed worthwhile." Like many others, he waited a few years before requesting his test results. His multivitamin was a decision he faced on going to graduate school. Prepared for the worst, he learned he

was not infected after all. "It was great news," he said. Now he is finishing his studies at the American College of Traditional Chinese Medicine and has become associate director of the Alan Yin Healing Arts Center.

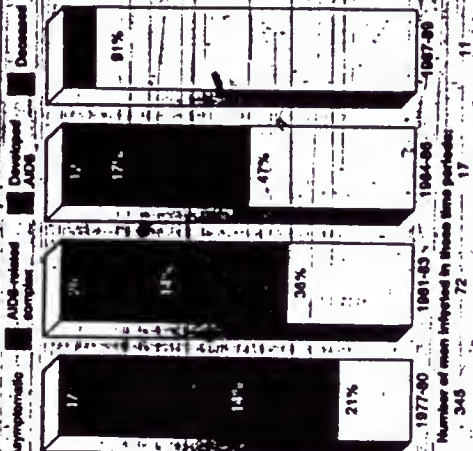
"It continues as a study volunteer," he said. "This is some of the most important research being done." Dr. Gordon Gross, 40, a former U.S. programmer, was another pioneer in the study. He volunteered "one of a sense of helping my community" and because he himself had suffered a severe episode of hepatitis.



Source: San Francisco City Clinic Cohort Study

### Men Outcomes After AIDS Infection

A group of 448 homosexual men from San Francisco has provided the strongest data on how long it takes after infection with the AIDS virus through sexual intercourse to develop disease and die. In 1977-80, 346 of the men became infected, of whom 81% have since developed or died from AIDS, and 21% are still free of symptoms.



Source: The New York Times, Illustrated by Susan Hagenstein

At first he declined to get his AIDS test results, because there was no treatment then. He did not want to be distressed by learning he was infected and knowing he was unable to do anything about it. When AZT became available as an experimental drug four years ago, he changed his mind. The test result showed he had been infected when he entered the study in 1979. His lover was also tested, but to his surprise, the result was negative. "I still go in for the exams and answer all kinds of questions," Mr. Gross said of his continued participation in the project. He has had so many blood samples drawn, as often as every four weeks, that the jokingly refers to the researchers as "vein-pipers."

Today, by all indicators, Mr. Gross remains in excellent health. Researchers are watching to see that occasionally happens in people who are infected.

"I know I'm unusual," he said, "and I hope I stay that way."



## HIV survivors may offer clue to treatment

### Blood of long-term patients shows cells that fight virus

By Lisa M. Krieger  
EXAMINER MEDICAL WRITER

In the 13 years that Stan has lived with the AIDS virus, 6,941 San Franciscans have died of the disease. Yet he's as robust as the day he was infected.

"I am healthier now than I've ever been," he said. "As much as anything, I want to prove to scientists that you don't have to die of this."

Long-term survivors like Stan — people who live with the infection for 10 years or more, with no illness and with vigorous immune systems — have important clues in their blood that suggest the reasons for their survival.

Their blood has yielded the first evidence that genetic and biological differences may explain the mysterious variations in life span, and it could open new avenues to treatments or vaccines.

Researchers at UC-San Francisco are looking closely at a particular type of immune system cell, called CD8 lymphocyte cells. An abundance of these cells could help delay or prevent the spread of the virus, according to UCSF virologist Dr. Jay Levy.

"It adds hope (for) the ability of your own immune system in controlling infection," he said.

Survivors "hold some secrets that others could benefit from," said Paul O'Malley, director of a San Francisco study that is following these men.

Scientists have long been puzzled why the AIDS virus, which is normally dormant and harmless for

### ◆ SURVIVORS from A-1

## HIV survivors' blood yields clues

years, suddenly turns deadly.

They don't understand why some people succumb quickly, while others seem to peacefully coexist with the virus for a decade or longer. Statistics show that half of all people infected by the virus get AIDS within 11 years.

Stan (not his real name) and eight other long-term survivors were discovered because they were original participants in a huge hepatitis B vaccine study conducted by The City. The study of 489 gay men began in 1978 — when the human immunodeficiency virus that causes AIDS first began sweeping the gay community. Their blood samples were subsequently thawed, providing "snapshots" of HIV infection over time.

Because the study contains the oldest AIDS-infected blood stored in the country, it has been prophetic of trends in AIDS longevity. Its data show that AIDS tends to follow a relentless continuum of infection, AIDS, then death.

Of the men, like Stan, infected before 1980, 47 percent are dead, 32 percent have either AIDS or ARC and 6 percent have not experienced symptoms, but have weakened immune systems.

Yet 15 percent of these men have robust immune systems and have yet to experience their first symptom of disease.

"There may be people who live way out there — 20 to 25 years — but we just haven't gotten there yet," said O'Malley, director of the Hepatitis B study.

"They know how unique they are," said O'Malley. "They've seen

people infected at the same time they were, but who have been dead for years. They ask: 'Why me?'"

"They are employed and healthy, leading active lives," said Dr. Susan Buchbinder, also of the study. "They do whatever they can to help our research."

Stan leads a reflective life. For exercise, he strides the hills of his San Francisco neighborhood. A professional artist, he enjoys flower gardening in his sunny yard. Weekends are spent touring museums and hosting dinner parties.

### The CD8 cells

Every six months, he donates blood for testing. The UCSF research shows that blood of survivors like Stan has an abundance of CD8 cells. In lab studies, CD8 cells in this blood are able to suppress the spread of the virus. If CD8 is removed, the virus proliferates. If CD8 is added back, the virus is once again suppressed.

"This cell doesn't kill the virus, but keeps it in check," said Levy.

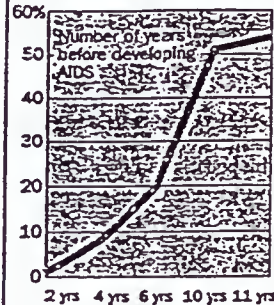
It is probably a substance produced by the CD8 cell that actually shuts down production of the virus, Levy explained. Yet it doesn't seem to be a protein like interferon or interleukin, as first suspected. "It's a brand new factor — and we're after it," said Levy.

For genetic or other reasons, individual people produce different levels of CD8 cells, Levy said. "Some make a lot. Some make a little, and the virus escapes. Some make none at all."

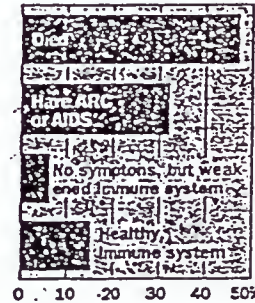
A related area of research suggests that another type of immune system cell, called a cytotoxic lymphocyte, may also contribute to survival. These cells, whose mission it is to kill any virus-infected cells in the body, are very active in the blood of long-term survivors —

### AIDS CASES

"489 gay men, all infected with HIV, studied since 1978"



Of these infected with HIV between 1977-'80



EXAMINER GRAPHICS

and inactive in people with AIDS, according to new test tube research by Buchbinder and Dr. Alison Mawle of the Centers for Disease Control. Does that mean that these cells control HIV in the body? "It is suggestive, but not conclusive," Mawle said.

Lifestyle factors have not been ruled out. It is known that infection by sexually transmitted disease, such as gonorrhea or herpes,

can accelerate disease. But more research is needed to understand what role, if any, is played by diet, exercise, smoking, attitude, recreational drug use, meditation, and an array of other behavioral factors, according to Nancy Hess, who is also studying the long-term survivors.

For Stan, who has lost several dozen friends and acquaintances to AIDS, the passing years have not been easy. He is inspired by his lover, who is also strong and healthy. But like the hanging sword of Damocles, the worry illness "doesn't ever completely leave me," he said.

"I spend a lot of time psyching myself up," he admits. "I remind myself that being exposed to the AIDS virus is like being exposed to the flu — some die, some don't. I try turning my thoughts around — and remember what I've got live for. I can't die now."

**COFACTORS FOR HIV DISEASE PROGRESSION  
IN HOMOSEXUAL MEN: 1978-1991**

**Nancy Hessol\*; Robert Fusaro\*\*; Peter Bacchetti\*\*\*; Jennifer Liu\*;  
Susan Buchbinder\*; Paul O'Malley\*; Scott Holmberg\*\*\*\*; Mitchell Katz\*.**

**\* San Francisco Department of Public Health; \*\* UC Berkeley School of Public Health; \*\*\*  
San Francisco General Hospital, CA; \*\*\*\* Centers for Disease Control, Atlanta, GA, USA.**

**Presented at the VIII International Conference on AIDS, Amsterdam, The Netherlands, July  
19-24 1992.**



## INTRODUCTION

Progression from infection with human immunodeficiency virus (HIV) to the development of acquired immunodeficiency syndrome (AIDS) has been evaluated in a number of prospective studies. However, progression rates and initial AIDS diagnosis vary, suggesting that there may be cofactors which alter both the rate of disease progression and the initial AIDS-defining illness. Additionally, since the risk of AIDS in HIV-infected persons is not constant over time, the duration of HIV infection at the time of recruitment in a study will directly affect a person's rate of progression. In this analysis we evaluated cofactors for progression to AIDS and to disease-specific AIDS diagnoses in a cohort of homosexual men for whom the time of HIV seroconversion can be reasonably approximated and for whom extensive questionnaire information is available.

## METHODS I

The San Francisco City Clinic Cohort study has prospectively evaluated 6,704 homosexual and bisexual men originally recruited for hepatitis B studies in 1978-1980. In 1980 we began conducting hepatitis B vaccine trials in a subsample of this cohort and in 1983 we initiated AIDS and HIV follow-up studies in the entire cohort. With the participants' consent, stored serum from the hepatitis studies and current sera from follow-up studies were tested for HIV antibodies. Prospective evaluations included a blood draw, an interview regarding behavioral and demographic risk factors for HIV infection and AIDS, and an examination and medical history by a physician.

## METHODS II

The HIV-infected men included in this analysis were 1) HIV-positive on entry into the hepatitis study (1978 to 1980) [n=352], or 2) had intervals of HIV seroconversion  $\leq 24$  months [n=193], or 3) participated in the vaccine trial, with an HIV seroconversion interval of  $>24$  months [n=42]. We estimated the month of HIV seroconversion by appropriate methods for doubly censored data, conditional on the individual's last negative and first positive HIV antibody tests. For men who were positive on entry into the study, the last HIV negative date was assumed to be January 1977.

We examined the association between various cofactors and the progression to AIDS and AIDS-specific diagnoses by estimating relative hazards (RH) and 95% confidence intervals (CI) using multivariate Cox proportional hazard models. To determine the incidence of AIDS in the cohort, we cross-matched with San Francisco and national AIDS surveillance registries, using January 1991 as the censoring date. Initial AIDS diagnoses were categorized as Kaposi's sarcoma (KS), Pneumocystis carinii pneumonia (PCP), or other opportunistic infections (OOI) (includes all other diagnoses except lymphomas). Age at seroconversion (in 10 year units) and race (dichotomized as white or non-white) were included as fixed covariates. Calendar time (dichotomized as being before or after June 1987) was included as a time-dependent covariate. Histories of sexually transmitted diseases, excluding herpes simplex, were treated as time-dependent covariates indicating the cumulative number of episodes over time. Since herpes simplex is a chronic disease, we modeled its history as a binary time-dependent covariate assuming a value of one following the initial manifestation. We also incorporated oral/anal contact data as a fixed covariate indicating whether the participant engaged in that behavior after January 1980.

**TABLE I**  
(Relative Hazard and 95% CI)  
[N=587]

<u>Variable</u>	<u>AIDS</u>	<u>KS</u>	<u>PCP</u>	<u>OOI</u>
Age (10 years)	1.28 (1.04,1.58)	1.15 (0.80,1.65)	1.09 (0.79,1.52)	1.85 (1.23,2.80)
White/Non-white	0.83 (0.58,1.19)	0.84 (0.46,1.52)	0.79 (0.46,1.35)	0.85 (0.37,1.97)
Calendar time	0.59 (0.41,0.85)	0.71 (0.39,1.30)	0.41 (0.24,0.72)	1.10 (0.52,2.33)

## DISCUSSION

In this multivariate model, older age at seroconversion was associated with increased progression to AIDS and, in particular, with OOI as a first AIDS diagnosis. In this predominantly white cohort, race was not strongly associated with progression to AIDS or specific AIDS-defining illnesses. Progression to AIDS appears to have slowed after June 1987. This slowing was strongest for the diagnosis of PCP and may be due to the introduction and widespread use of antiviral therapy and prophylaxis against PCP. The lack of a slowing after June 1987 for OOI may be due to several factors: 1) the expansion of the AIDS case definition in 1987 which increased the number of AIDS defining OOs; 2) improvement in the diagnosis of specific OOs (such as MAI and CMV); and 3) an increase in OOs due to prevention of PCP through effective prophylaxis.

TABLE II

(Relative Hazard and 95% CI)

[N= 587]

<u>Variable</u>	<u>AIDS</u>	<u>KS</u>	<u>PCP</u>	<u>OOI</u>
Age (10 years)	1.33 (1.06,1.66)	1.13 (0.76,1.67)	1.22 (0.86,1.74)	1.82 (1.13,2.91)
White/Non-white	0.84 (0.57,1.23)	1.06 (0.53,2.09)	0.78 (0.43,1.39)	0.61 (0.26,1.45)
Calendar time	0.62 (0.41,0.93)	0.80 (0.40,1.57)	0.40 (0.21,0.77)	1.30 (0.55,3.07)
Syphilis	0.89 (0.68,1.17)	0.85 (0.53,1.37)	0.84 (0.55,1.29)	1.20 (0.64,2.27)
Gonorrhea	0.90 (0.83,0.99)	0.92 (0.80,1.07)	1.02 (0.91,1.14)	0.51 (0.35,0.76)
Giardia	0.97 (0.72,1.32)	1.08 (0.75,1.54)	0.50 (0.21,1.17)	0.89 (0.36,2.21)
Shigella	1.02 (0.62,1.68)	1.63 (0.85,3.12)	0.88 (0.37,2.08)	1.04 (0.25,4.35)
Amebiasis	1.26 (1.05,1.52)	1.44 (1.11,1.86)	0.95 (0.65,1.40)	1.50 (0.90,2.49)
Herpes simplex	0.56 (0.41,0.76)	0.70 (0.43,1.12)	0.44 (0.27,0.72)	0.75 (0.39,1.45)
Herpes zoster	0.56 (0.37,0.85)	0.49 (0.22,1.08)	0.66 (0.36,1.18)	0.74 (0.36,1.52)

## DISCUSSION

In this multivariate model, older age at seroconversion is still significantly associated with AIDS and OOI and calendar time (>6/87) is associated with a slower progression to AIDS and PCP. Amebiasis is associated with faster progression to AIDS and strongly associated with progression to KS. Herpes simplex is associated with a slower progression to AIDS and PCP. The reason for the slower progression associated with history of herpes simplex and herpes zoster is unclear. Perhaps the use of acyclovir for treatment of herpes may, either by itself or in combination with other antivirals, slow disease progression.

**TABLE III**  
(Relative Hazard and 95% CI)  
[N= 349]

<u>Variable</u>	<u>AIDS</u>	<u>KS</u>	<u>PCP</u>	<u>OOI</u>
Age (10 years)	1.79 (1.25,2.57)	1.44 (0.80,2.57)	1.69 (0.91,3.12)	3.04 (1.65,5.60)
Enterics	1.46 (1.26,1.70)	1.60 (1.30,1.98)	1.25 (0.94,1.66)	1.26 (0.84,1.89)
Oral/anal	1.96 (1.28,3.02)	1.65 (0.88,3.10)	2.51 (1.22,5.17)	1.53 (0.59,3.96)

## DISCUSSION

Due to the apparent association between enteric diseases, especially amebiasis, and KS, we added a behavioral variable on oral/anal (rimming) sexual practices into a multivariate model. This analysis was restricted to men interviewed in 1987 or later, due to missing information on oral/anal behavior for the earlier interviews. Although oral/anal behavior data was strongly associated with progression to AIDS, it was more strongly associated with PCP than KS. The weaker association between oral/anal contact and KS may be due to the exclusion of the men who were only interviewed prior to 1987, many of whom developed KS. By grouping giardia, shigella, and amebiasis as one enteric variable, the association with AIDS and with KS remains strong.

## SUMMARY

Identifying potential cofactors for HIV disease progression is important for understanding the pathogenicity of the virus and may lead to better interventions and treatments. Our analysis supports findings from previous epidemiological studies which found associations between the following:

- 1) older age and more rapid progression to AIDS, especially to the development of opportunistic infections as an initial AIDS diagnosis;
- 2) history of enteric diseases and the development of KS as an initial AIDS diagnosis;  
and
- 3) calendar time and a decline in AIDS and PCP after the introduction of therapy (June 1987).

These results should be interpreted with caution. The association of herpes simplex and herpes zoster with slower progression to AIDS is puzzling and warrants further investigation. Also the association of enteric disease with KS but the lack of association between oral/anal contact and KS deserves further attention.





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(1) Pt. ID \_\_\_\_\_  
Last Initial      Last 4 digits SS#      Leave Blank

(2) 1st Rpt. Yes - Age \_\_\_\_\_ yrs. (35) Rds. avail. for \_\_\_\_\_ yrs.

Follow-up report: Yes

(3) Report Date \_\_\_\_/\_\_\_\_/\_\_\_\_ (4) Weight (Kg) \_\_\_\_\_ (=lbs/2.2)  
                  day    mo.    year

Diagnosis or Signs: (5) Wt. loss \_\_\_\_\_ (Kg) over \_\_\_\_\_ months

(6) Fatigue    Y    N      Anorexia    Y    N      Fever        Y    N  
    Diarrhea    Y    N      Night sweats Y    N      Neuritis     Y    N  
    Other: \_\_\_\_\_

(7) Infection:    Y    N      Record agent with site:      Confirmed?

(8) Agent 1 \_\_\_\_\_ (9) Site 1 \_\_\_\_\_ Y    N  
(10) Agent 2 \_\_\_\_\_ (11) Site 2 \_\_\_\_\_ Y    N  
(12) Agent 3 \_\_\_\_\_ (13) Site 3 \_\_\_\_\_ Y    N  
(14) Agent 4 \_\_\_\_\_ (15) Site 4 \_\_\_\_\_ Y    N

(16) Lymphadenopathy Y    N      Biopsy    Y    N      Site(s):  
(17) \_\_\_\_\_ (18) \_\_\_\_\_  
(19) \_\_\_\_\_ (20) \_\_\_\_\_

(21) Malignancy:      Kaposi's Sarcoma    Y    N      Biopsy    Y    N  
(22) Other malig.    \_\_\_\_\_ Biopsy    Y    N

Site(s): (23) \_\_\_\_\_ (24) \_\_\_\_\_

Other clin. findings: (25,26) \_\_\_\_\_  
\_\_\_\_\_

Other lab. findings: (27,28) \_\_\_\_\_  
\_\_\_\_\_

Drug or other interventions since last report: (29,30) \_\_\_\_\_

\_\_\_\_\_ (31) Dead    Y    N  
(32) In your opinion, is this

condition AIDS?    Y    N      (33) Physician: \_\_\_\_\_

(Leave blank) \_\_\_\_\_

# ID/IC BULLETIN

Infectious Diseases and Infection Control, University of California, S.F.

Vol. 2, #3  
October, 1982

## ACQUIRED IMMUNODEFICIENCY SYNDROME

The appearance of a profound immunodeficiency syndrome in previously healthy homosexual males, Haitians, and hemophiliacs has raised considerable interest in the medical community and American press.

The University of California San Francisco (U.C.S.F.) first began treating these patients in August 1981. The number of patients has been steadily increasing and questions concerning treatment modalities and infection control measures have been frequently posed. This bulletin concentrates on the infectious aspects of this syndrome and presents guidelines for infection control in U.C.S.F. Hospitals and Clinics.

### EPIDEMIOLOGY

Between June 1, 1981 and September 3, 1982 the Centers for Disease Control received reports of 578 cases of Kaposi's Sarcoma (KS), *Pneumocystis carinii* pneumonia (PCP) and other serious opportunistic infections (OI) occurring in previously healthy persons. Of the 578, 435 (75%) were homosexual or bisexual men, 83 (14%) heterosexual males, 28 (5%) males of unknown sexual orientation and 32 (6%) women. Almost 90% of the cases were 25-49 years old. Approximately 60% were white not Hispanic, 20% black not Hispanic, and 13% Hispanic.

Cases have been reported from 27 states; however, approximately 50% of the reports have come from New York City, California, Florida, New Jersey and Texas have also reported a large number of cases. (CDC, MMWR, 31:294, 1982). Nine foreign countries have reported at least one case. Some of these individuals had been to New York City prior to becoming ill. There also appears to be a large number of seemingly similar cases occurring in Haiti and there have been 34 diagnosed cases in Haitian refugees in the United States.

Table 1 is a breakdown of disease and mortality of United States cases as of September 3, 1982. (CDC, Task Force on Acquired Immunodeficiency Syndrome, weekly report).

Table 1. Disease and Mortality, United States

	Cases	Percent of total	Deaths (percent)
KS without PCP	177	30.6	34 (19.2)
PCP without KS	291	50.4	137 (47.1)
Both KS and PCP	43	7.4	29 (67.4)
OI without KS or PCP	67	11.6	33 (49.3)
Total	578	100.0	233 (40.3)

The San Francisco Public Health Department first received reports of cases with onset in 1980. There have been 100 cases reported as of September 5, 1982. Ninety-nine were male homosexuals and 1 was a heterosexual male. Fifty-one of the 100 cases have had onset in 1982 and the number of reported cases is continuing to increase. Table 2 summarizes the cases in San Francisco. (personal communication, Selma Dritz MD Disease Control, San Francisco Public Health Dept)

Table 2. Disease and Mortality, San Francisco

	Cases	Percent of total	Deaths (percent)
KS without PCP	45	45.0	7 (15.6)
PCP without KS	37	37.0	15 (40.5)
Both KS and PCP	8	8.0	5 (62.5)
OI without KS or PCP	10	10.0	5 (50.0)
Total	100	100.0	32 (32.0)

Initially reported cases were in bisexual or homosexual males; however, there has been a small but continuing increase of cases in heterosexual males and in



women. In one study, 63% of the heterosexual men and 57% of the women reported using intravenous drugs, compared with 14% of the homosexual men (CDC, MMWR, 31:294, 1982). In July the Centers for Disease Control released reports of 3 cases of PCP in patients with hemophilia A but no other underlying disease. All 3 patients had received multiple doses of factor VIII concentrate but none of the patients had received concentrate from the same lot (CDC, MMWR, 31:365, 1982).

### IMMUNOLOGY

The common denominator explaining the multiplicity of presentations in these patients appears to be a severe disorder of immunoregulation, termed acquired immunodeficiency syndrome (A.I.D.S.) These findings have recently been described (AS Fauci, *Ann Intern Med*, 96:777, 1982). Patients present with a spectrum of immune dysfunction. This is most often characterized by anergy to skin testing, lymphopenia, and an often profound reversal in the ratio of two populations of T-lymphocytes, termed the helper-cell to suppressor-cell ratio or OK-T4 to OK-T8 cell ratio. Immunoglobulin levels are most often normal, although in some cases elevated IgA and/or IgG levels have been noted. In addition, at least in a few patients, the B-lymphocytes do not appear to be responding normally; this may be a secondary manifestation of abnormalities in the T-lymphocyte population.

This disorder of immune function impairs the body's "surveillance" to both infectious and non-infectious illnesses. The appearance of Kaposi's sarcoma, Burkett's lymphoma, Hodgkin's disease and some mucocutaneous malignancies represents a defect in these patient's ability to recognize or to eradicate malignantly-transformed cells. The recently reported observation of several cases of autoimmune thrombocytopenic purpura probably represents an additional manifestation of this disorder of immunoregulation.

The underlying cause of this disorder is unclear and it is difficult to sort out which risk factors (e.g. environmental, use of street drugs such as nitrites, genetic predisposition, etc.) may be contributory rather than causative. The most current theories include an infectious agent, possibly a virus which is transmitted like hepatitis B virus. It is suggested that this agent might infect T-lymphocytes and be responsible for immunosuppression. This may be a new agent or a mutated form of an old virus. Currently there is some speculation that cytomegalovirus may be involved. Others postulate a "chronic antigenic stimulation" theory which hypothesizes that repeated infections with gonorrhea, syphilis, hepatitis, amoeba and cytomegalovirus (CMV) "exhaust" the immune system.

### CYTOMEGALOVIRUS INFECTIONS

Cytomegalovirus is a herpes DNA virus which is found ubiquitously. It is well recognized as a cause of a mononucleosis-like syndrome in adults, neonatal and congenital infection, and illness in immunosuppressed patients. Patients receiving organ transplantation, in particular, are at risk. This infection may manifest as a pneumonitis, hepatitis, gastroenteritis, or retinitis. Recently it has been noted that 93% of sexually-active gay men have cytomegalovirus IgG antibody and approximately 50% have IgM antibody (LW Drew et al., *J Infect Dis*, 143:188, 1981). In addition, a number of these patients have been found to excrete CMV in their urine and semen. The diagnosis of CMV usually is made on clinical, serological, culture, and often pathological criteria.

A.I.D.S. patients have serological evidence of past infection. The virus has been found in cultures from biopsy specimens and microscopic evidence has been seen in biopsies of lung, bowel and tumor (LW Drew et al., *Lancet*, 11:125, 1982). Speculation regarding the role of this virus as a possible etiological factor in this syndrome remains controversial. The association of this virus and Kaposi's sarcoma was reported in African patients prior to this current epidemic (Geraldo G and Beth E, *The Role of Viruses in Human Cancers*, Elsevier - North Holland, 1:57, 1980). In addition, the laboratory evidence for a transient immunosuppressive state following CMV mononucleosis in otherwise healthy patients has been reported independently. (CR Rinaldo Jr., *J Infect Dis*, 141:488, 1980).

No efficacious therapy has been defined to date. However, interest in a number of antiviral drugs and interferon has led to ongoing clinical trials with a variety of agents.

### PNEUMOCYSTIS CARINII PNEUMONIA

*Pneumocystis carinii* is a protozoan also found ubiquitously worldwide. By the age of 4, approximately 80% of immunocompetent children have serological evidence for past infection. However, this organism has been identified as a cause of often fatal pneumonitis in several subpopulations. These include protein-malnourished children in European orphanages and immunosuppressed patients. The appearance of this infection and Kaposi's sarcoma in homosexual adult males alerted the medical communities in New York City, Los Angeles, and San Francisco to this syndrome. The organism is most often limited to the lungs. It presents as a diffuse pneumonitis with few findings on physical examination, but often profound hypoxia and dyspnea. Occasionally the organism has been found to disseminate to multiple sites, including the retina. Diagnosis is made by silver methenamine staining of tissue obtained from lung biopsy. To date, attempts at culture and serological diagnosis have been disappointing.



prim-sulfamethoxazole (TMP-SMX), given either orally or intravenously, or with pentamidine, administered intravenously or intramuscularly. No comparative trials in this population have been reported, and in some cases a combination of these drugs has been given. Although the mortality from this infection in patients with hematological malignancies has decreased with the advent of appropriate chemotherapy from over 50% to 5-10%, the mortality from pneumocystis in patients with A.I.D.S. has been in the range of 50% despite drug therapy. In addition, a number of cases of recurrence have been described. Therapy with oral trimethoprim-sulfamethoxazole should include documentation of adequate absorption by obtaining peak serum concentrations of either trimethoprim or sulfamethoxazole. Problems with both drugs are common. Skin rash and fluid overload with intravenous TMP-SMX are not absolute contraindications to therapy, but must be monitored. The usefulness of prophylactic TMP-SMX in this syndrome has not yet been investigated.

### OTHER INFECTIONS

A number of additional infections of varying severity have been reported. Thrush (candida pharyngitis and esophagitis) is nearly universally seen. This infection is unusual in patients not taking antibiotics or known to have an underlying chronic illness. Although occasionally difficult to manage, a number of treatment options are available. These include oral nystatin, mycostatin oral tablets, ketoconazole, and intravenous amphotericin B.

Perianal ulcerations secondary to herpes simplex virus have been described. These can be quite extensive, causing the patient a great deal of discomfort. On occasion this has required the antiviral drug acyclovir for control.

A few cases of disseminated *Mycobacterium tuberculosis* and atypical mycobacteria have been reported. These have presented as diffuse tissue involvement with poor host cellular response. Treatment outcome with as many as five anti-tuberculosis medications has generally been poor.

Disseminated cryptococcus and cryptococcal meningitis have also been seen. These infections may present as pulmonary infiltrates, skin rash, or diffuse headache and photophobia, suggesting meningitis. Treatment with amphotericin B and 5-flucytosine has cured the infection in many cases, but some of these patients have gone on to relapse.

Other infections noted include recurrent bacterial sepsis, follicular impetigo, herpes zoster, *Nocardia* pneumonitis, and cryptosporidiosis colitis. Severe and fatal toxoplasmosis has also been seen. In summary, each new sign or symptom in these patients must be taken seriously and investigated fully for the broadest spectrum of possible infectious agents.

193 The following guidelines were derived through discussion with members of the U.C.S.F. Kaposi's clinic and personnel caring for the patients and have been approved by the Infection Control Committee. They were established in consideration of a potentially transmissible agent being responsible for this syndrome and may be changed as further information becomes available.

**All Patients:** Private room not necessary (see exceptions below); however, A.I.D.S. patients are immunocompromised and should not be placed in a room with infected patients. Patients should be placed on "Blood" Precautions; i.e. blue sign; gloves required for contact with blood, secretions, excretions; gown and mask not necessary. A needle box should be placed at the bedside for immediate disposal of used sharps. Pregnant women should avoid direct patient contact because many of these patients are excreting cytomegalovirus.

**Pneumocystis Patients:** Private room not necessary but patients cannot be in the same room with high-risk patients or other A.I.D.S. patients because of the theoretical potential for *P. carinii* airborne spread to immunosuppressed patients. High-risk patients are defined as persons receiving radiation therapy, or other immunosuppressive treatments, cortisone derivatives in a dose greater than 100mg hydrocortisone equivalent per day or diagnosed as having leukemia, lymphoma, aplastic anemia, etc.

**Kaposi's Sarcoma Patients:** A private room not necessary but it is desirable to have private room and bath if the patient has diarrhea.

**Compromised Host:** Patients with WBC <1000 or PMN <500 should be placed on Compromised Host Precautions.

**Material Services:** Instruments and other items grossly contaminated with blood or excreta should be rinsed under running water in the utility room and sent to Material Services in a plastic bag labelled "contaminated."

**Environmental Services:** Rooms need not routinely be high-cleaned; questions concerning exceptions should be directed to Infection Control.

**Dietary:** Isolation trays are not necessary.

**Linen:** Double bag linen only if grossly contaminated with blood or excreta.

**Laboratory:** Blood specimens should be handled with caution as with known hepatitis B carriers; however, it is important to remember that blood from any patient may be infectious and appropriate infection control measures should always be followed.

**Addendum:** Frequently patients present with other infectious processes (e.g. hepatitis, herpes, enteric pathogens) and should be placed on appropriate precautions. *NOTE: Individuals who appear to have this syndrome can be seen in the Infectious Disease - Tropical Disease Clinic on the U.C.S.F. campus (x5787).*

## An Outbreak of *Pneumocystis carinii* Pneumonia in Homosexual Men

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*Pneumocystis carinii* pneumonia has rarely been reported in previously healthy persons over the age of 6 months. Five cases of *P. carinii* pneumonia in adult homosexual men, confirmed by biopsy results, are reported. All five patients were seropositive when tested for antibodies to cytomegalovirus and four had evidence of active concurrent cytomegalovirus infections. Kaposi's sarcoma was shown in two of the patients and one had possible *Pneumocystis* infection of the central nervous system as well as *P. carinii* pneumonia. Three patients had second episodes of *Pneumocystis* pneumonia. Four of the five patients have died. Past or concurrent cytomegalovirus infection and homosexuality were the only common epidemiologic features in all five patients.

THE OCCURRENCE OF *Pneumocystis carinii* pneumonia was first reported in the United States in 1955 as interstitial plasma cell pneumonia (1). Pulmonary disease due to this protozoan can be divided into three distinct categories. The commonest is a usually self-limited, asymptomatic or mildly symptomatic infection shown by IgG-IgM seroconversion in 75% to 90% of healthy children before the age of 4 years (2, 3). The second is a severe, often fatal, diffuse pneumonitis seen in premature or malnourished infants (4, 5). The third category is pneumonia occurring in older children and adults in association with immunosuppressive therapy, neoplastic disease, or serious underlying illness (6). Dissemination outside the lung has been rarely reported (7). *Pneumocystis carinii* pneumonia has been rarely reported in previously healthy persons over the age of 6 months (8-11). Analysis of 163 cases of *P. carinii* pneumonia, confirmed by the Centers for Disease Control, in the 3-year period from November 1967 to December 1970, yielded no cases in otherwise healthy persons (12).

Recently, clusters of cases of *P. carinii* pneumonia have been noted in New York City and Los Angeles (13-15). Most cases occurred in homosexual men. We report five cases of *P. carinii* pneumonia, confirmed by biopsy, in San Francisco between November 1980 and June 1981.

### Methods

All patients were hospitalized in the San Francisco Bay Area from November 1980 through November 1981. Consultation was provided by one or more of the authors for each patient during his hospitalization. Diagnosis of *P. carinii* pneumonia was made by methenamine silver staining of lung biopsy tissue in each case. All patients were treated with trimethoprim-sulfamethoxazole at dosages of 20 mg and 100 mg/kg body weight intravenously in divided doses, unless otherwise stated.

The methods for throat and urine cultures have been described previously (16). Throat swabs were transported in modified Stuart's medium (Culturette, Marion Scientific Corp., Rockford, Illinois) and inoculated directly into tubes of diploid fibroblast cultures (WI-38, Microbiological Associates, Cockeysville, Maryland; Flow 2000 or IMR-90, Flow Laboratories, Inglewood, California) within 2 to 4 hours of collection. After 1 hour at room temperature for virus elution, the swabs were removed, and 0.2 mL of medium was used to inoculate a companion diploid fibroblast tube. Two-tenths of a milliliter of urine was inoculated directly into each of two tubes of diploid fibroblast cells after treatment of the sample with high concentrations of gentamicin and amphotericin. After overnight incubation at 35 °C, the medium in tubes inoculated with urine was replaced with 3 mL of fresh maintenance medium. Peripheral blood leukocytes were separated and prepared for culture according to the Ficoll Hypaque method of Boyum (17). Biopsy specimens were prepared for culture by grinding the tissue and preparing a 10% suspension in antibiotic-containing media.

All viral cultures were checked daily for 4 weeks for the development of distinctive cytomegalovirus cytopathic effect. Isolates were subcultured in diploid fibroblast, HEP-2, and primary monkey kidney cells to establish their selective ability to grow only in human diploid cells.

Serum IgG antibody to cytomegalovirus was measured by a modified anticomplement immunofluorescent antibody assay (18). The presence of IgM antibody to cytomegalovirus was ascertained by immunofluorescent assay using cytomegalovirus-infected diploid fibroblast cells that were treated with distilled water to obtain "naked nuclei" (much like the procedure for the IgG antibody assay). All sera tested for specific cytomegalovirus IgM antibody were negative for rheumatoid factor by a latex agglutination method. In both tests antibody titers of greater than or equal to 1:8 were considered significant.

### Case Reports

#### PATIENT 1

A 44-year-old white homosexual man was hospitalized 8 April 1981 for evaluation of fever and abdominal complaints. In November 1980 the patient had developed intermittent fever, nonbloody diarrhea, and crampy abdominal pain. In January 1981, a stool specimen showed *Entamoeba histolytica* and the symptoms resolved after treatment with diiodohydroxyquin and metronidazole. In March the symptoms recurred. The patient was given quinacrine hydrochloride for presumed giardiasis and had slight improvement in the abdominal pain and diarrhea. A rapid plasma reagin test was positive and treatment with tetracycline was started. The fever continued for 5 weeks and the patient was hospitalized for further evaluation. The patient had had hepatitis in 1963 and primary syphilis in 1973. There was no history of foreign travel or occupational exposure to inhalants or animals.

On admission, the patient's temperature was 39.7 °C. Physical examination showed no abnormalities except a few plaques of oral candidiasis. There was no adenopathy, skin rash, or abnormality on funduscopic examination. The erythrocyte sedimentation rate was 76 mm/h but the complete blood count, results of urinalysis, serum electrolyte levels, blood urea nitrogen level, serum creatinine level, and SMA-12 chemistry panel were all within normal limits. A chest roentgenogram showed no abnormalities. Bacterial cultures of the blood, urine, and

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stool were negative as were examinations of the stool for ova and parasites. Delayed hypersensitivity skin testing in response to tuberculin purified protein derivative, mumps, candida, and spherulin antigens gave no reaction.

On the third hospital day, a dry cough developed. A chest roentgenogram showed diffuse reticular infiltrates. Progressive dyspnea, tachypnea, and hypoxemia developed and on day 8 a fiberoptic bronchoscopy examination was done. Transbronchial biopsy, stained with methenamine silver, was positive for *P. carinii*. Culture of the lung for bacteria, including *Legionella*, acid-fast bacilli, and fungi, were negative. Culture of lung tissue for cytomegalovirus and immunofluorescent microscopy for cytomegalovirus were also negative. Serum titers for cytomegalovirus-specific IgG and IgM antibodies were 1:1024 and 1:8, respectively. On day 10, treatment with trimethoprim-sulfamethoxazole was begun. The patient's symptoms, arterial blood gas measurements, and findings on chest roentgenogram improved steadily; however, on day 18, therapy was discontinued because of neutropenia ( $1900/\text{mm}^3$ ) and development of an erythematous rash.

Repeat skin tests, after resolution of the *Pneumocystis* infection, remained unreactive. Computed tomographic scan of the abdomen and chest showed no abnormalities. The hepatitis B surface antigen test was negative. Quantitative measurement of immunoglobulins showed an elevated IgA concentration of 680 mg/dL (normal is less than 392). The CH50, C3, and C4 concentrations were within normal limits. At the time of discharge on day 28, the patient was fatigued but otherwise felt well. The chest roentgenogram showed no abnormalities and the arterial oxygen content was within normal limits.

The patient was followed for recurrence of oral candidiasis and persistent fatigue. On 29 August 1981, he was readmitted because of a 2-week history of intermittently spiking fevers to  $40^\circ\text{C}$ , progressive dyspnea, and worsening fatigue. On admission, the temperature was  $38.3^\circ\text{C}$  and the patient appeared moderately ill. Physical examination showed candidiasis and bibasilar dry rales. Laboratory tests showed a leukocyte count of  $6700/\text{mm}^3$  with 9% lymphocytes and hypoxemia. Cytomegalovirus-specific IgG and IgM antibody levels were not significantly changed. Transbronchial biopsy of the lung tissue showed *P. carinii* cysts, and trimethoprim-sulfamethoxazole therapy was instituted. Cytomegalovirus was not recovered from the lung biopsy tissue. On day 6, trimethoprim-sulfamethoxazole was discontinued and pentamidine (4 mg/kg body weight · d, intramuscularly) was begun because of persistent fever and progressively deteriorating respiratory function. The patient needed mechanical ventilation. Pentamidine was discontinued 12 days later when the patient's condition stabilized, and repeat bronchial washings showed no *Pneumocystis*. However, the patient continued to need mechanical ventilation with positive end-expiratory pressure and high concentrations of inspired oxygen. Repeat bronchial washings 5 days after discontinuation of pentamidine therapy showed cysts of *P. carinii*, and pentamidine was reinstated on the 24th day. The remainder of the hospital course was complicated by progressive respiratory failure, ventricular irritability, and hypotension. The patient died on the 31st hospital day.

Immunologic analysis (done by Dr. Arthur Ammann, Pediatric Immunology Laboratory, University of California, San Francisco, California) before death showed 40% T-cell rosettes (normal level is greater than 60%), depressed response to mixed lymphocyte culture (1336 counts/min; normal level, greater than 5800), and depressed phytohemagglutinin stimulation (265 counts/min; normal level, greater than 1150). Post-mortem findings included chronic organizing interstitial pneumonitis with microscopic findings of diffuse intranuclear and intracytoplasmic inclusion bodies consistent with cytomegalovirus infection and intra-alveolar fluid showing occasional *P. carinii* cysts. Also, cytologic findings consistent with cytomegalovirus infection in tracheal mucosal cells and adrenal cells were seen. Analysis of multiple lymph nodes yielded only mild hyperplasia and siderosis, but otherwise no pathologic diagnosis. Viral cultures of lung were positive for cytomegalovirus at 5 days.

## PATIENT 2

A previously healthy 31-year-old white homosexual man was hospitalized on 20 March 1981 because of a 3-week history of increasing dyspnea, fever, and pleuritic chest pain. A chest roentgenogram 4 days earlier had shown minimal bilateral interstitial infiltrates. He was treated with tetracycline but the symptoms persisted. The patient had had hepatitis in 1971 and one episode of gonorrhea. He had a chronic rectal fistula. In early March, male sexual partner was ill with fever, and lymphadenopathy and cytomegalovirus was recovered from this person's urine. The patient reported that he used nitrate inhalants occasionally.

The patient's temperature was  $38.5^\circ\text{C}$ . Scattered bilateral inspiratory rales were heard. The remainder of the physical examination, including the fundoscopic examination, showed no abnormalities. Hematocrit was 39% and no lymphopenia was present. The chest roentgenogram showed increased bilateral interstitial infiltrates, and arterial blood gas measurements showed hypoxemia.

The patient was not taking antibiotics and remained febrile and the pulmonary infiltrates worsened over the first 5 days. Oral thrush was noted. Blood and urine bacterial cultures were sterile. An open lung biopsy was done on day 6. Histopathologic examination showed a limited interstitial mononuclear cell infiltrate with eosinophilic material in the alveolar spaces. Silver methenamine stain showed cysts of *P. carinii*. Bacterial and fungal cultures of the lung were negative; however, viral culture of biopsy tissue yielded cytomegalovirus. Serologic evaluation for cytomegalovirus was reactive at a titer of 1:32 for IgM specific antibody and 1:256 for IgG specific antibody.

Treatment with trimethoprim-sulfamethoxazole was begun orally. After 2 weeks the patient was afebrile and the chest roentgenogram showed only minimal residual infiltrates. At the time of discharge, delayed hypersensitivity skin tests were unreactive in response to tuberculin purified protein derivative, trichophyton, streptokinase-streptodornase, mumps, candida, and mixed respiratory vaccine (Hollister-Stier Laboratories) (19). T-cell testing (done by Dr. Lynn Spitzer, Children's Hospital, San Francisco, California) showed a total T-lymphocyte percentage of 56% with an active T-cell percentage of 40%. There was a normal response to stimulation with phytohemagglutinin. Quantitative immunoglobulin levels were IgG, 960 mg/dL (normal range, 564 to 1765); IgM, 142 mg/dL (normal range, 45 to 256); and IgA, 142 mg/dL (normal range, 85 to 135).

Fever and dyspnea recurred within 24 hours after hospital discharge. The patient was readmitted and had a temperature of  $39.3^\circ\text{C}$ . Examination of the chest showed no abnormalities, but the chest roentgenogram showed a recurrence of bilateral pulmonary infiltrates. Treatment with trimethoprim-sulfamethoxazole was resumed, but the patient's fever persisted and the hypoxemia and pulmonary infiltrates worsened. A transbronchial lung biopsy was done. Pathologic examination of the lung specimen showed a limited round-cell interstitial infiltrate without evidence of cytomegalovirus or *P. carinii*. Cytomegalovirus was again recovered from the lung tissue in culture. Repeat serologic evaluation for cytomegalovirus showed an IgG titer of 1:128 and an IgM titer of 1:8. Treatment included continued trimethoprim-sulfamethoxazole, erythromycin, and mechanical ventilation. The patient's overall course was one of gradual improvement and slow clearing of the pulmonary infiltrates. The recovery was complicated by prolonged fever, liver function abnormalities, and a pyopneumothorax, from which *Pseudomonas aeruginosa* was cultured.

After discharge from the hospital, in July 1981, the patient had intermittent low grade fever and failed to gain weight when not on antibiotic therapy. Oral candidiasis was again seen and needed suppression with topical antifungal agents. In October 1981, he was hospitalized briefly because of transient vomiting, which resolved, but nausea, bifrontal headache, and severe mental depression developed 1 day after discharge. The patient was readmitted to the hospital. The temperature was  $38.5^\circ\text{C}$ . The neck was supple. Lumbar puncture on two occasions showed protein levels of 45 and 47 mg/dL, and 0 to 3 mononu-



clear leukocytes/mm<sup>3</sup>. On the second specimen, an India ink preparation was positive and a culture grew *Filobasidiella (Cryptococcus) neoformans*. Bronchoscopic biopsy on a left lower lobe infiltrate showed yeast forms after staining; *P. carinii* was identified. Bronchoscopic culture grew *Candida albicans* but no *F. neoformans*. The bronchoscopy specimen also grew cytomegalovirus; the cerebrospinal fluid did not. Cryptococcal antigen titer was 1:2048 in serum and 1:256 in cerebrospinal fluid.

Treatment with intravenous amphotericin B for 6 weeks and oral 5-fluorocytosine for 2 weeks resulted in clearing of fever, headache, and mental depression. The left pulmonary infiltrate also resolved. Culture and staining of cerebrospinal fluid were negative, and cryptococcal antigen titer fell to 1:4 in serum and 1:2 in cerebrospinal fluid. An intercurrent *Staphylococcus epidermidis* bacteremia was treated by removing a Hickman catheter and antibiotic administration. The patient developed perianal ulcerations, from which herpes simplex virus type II was recovered. Recurrent fever and a right middle lobe pulmonary infiltrate appeared in the last 2 weeks of amphotericin B treatment. In December 1981, a percutaneous needle lung biopsy specimen showed numerous polymorphonuclear leukocytes and cysts of *P. carinii*. *Nocardia* species was recovered from culture. Viral culture was negative. A 14-day course of trimethoprim-sulfamethoxazole resulted in defervescence and improvement of the infiltrate.

Additional laboratory testing in November 1981 showed cytomegalovirus IgG and IgM titers of 1:256 and 1:16, respectively. Cytomegalovirus was isolated from saliva but not from urine. Hepatitis B surface antigen was undetectable; antibodies to hepatitis B surface antigen and hepatitis B core antigen were present. Blood lymphocyte phenotype was DR3-DR5. Repeat T-cell testing (done by Drs. Lynn Spitzer and Dobri Kiprov, Children's Hospital, San Francisco, California) showed 75% T cells (normal 70% to 80%) with 33% activated. Stimulation by phytohemagglutinin was normal. Of the total T cells, 23% were helper/inducer cells (normal is 40% to 56%) and 61% were suppressor/cytotoxic cells (normal is 20% to 30%). The helper/suppressor cell ratio was 0.4 (normal ratio ranging from 1.3 to 4.0). Circulating immune complexes were detected by the CIQ solid phase assay. The patient remains hospitalized because of poor nutrition. He is receiving trimethoprim-sulfamethoxazole orally at 160 mg and 800 mg, respectively, twice a day.

#### PATIENT 3

A previously healthy 37-year-old white homosexual man was admitted on 21 March 1981 for evaluation of severe headaches. In November 1980, he experienced increasing anxiety, dizziness, nausea, anorexia, and weight loss. Physical examination at that time showed multiple small blue nodules over the upper body and one blue mass on the inner upper alveolar ridge. Diffuse lymphadenopathy was present. Laboratory tests showed normochromic normocytic anemia and abnormal hepatic enzymes.

A biopsy of the skin nodule was done and the specimen was interpreted as angioendothelioma. Biopsy specimens of the lymph node and liver were nondiagnostic. Cultures of the lymph node and bone marrow for bacteria, fungi, and acid-fast bacilli were all negative. Cytomegalovirus serology for IgM was significant at a titer of 1:16. Urine culture grew cytomegalovirus. No specific therapy was instituted. In March 1981, increasingly severe headaches, nausea, vomiting, and dizziness developed. He denied use of nitrite inhalants and had no history of intestinal parasitic disease. Temperature was 38.8 °C. Funduscopic examination showed papilledema. The skin and oral lesions and generalized lymphadenopathy were present. No other abnormalities were detected on physical examination.

Laboratory tests showed a hemoglobin of 11.6 g/dL and a leukocyte count of 4400/mm<sup>3</sup> with a differential count of 59% polymorphonuclear cells, 13% band forms, 10% lymphocytes, 6% mononuclear cells, and 12% eosinophils. Analysis of cerebrospinal fluid showed cryptococcal organisms, and the cryptococcal antigen titer by latex agglutination was 1:512. Results of

urinalysis, serum electrolytes, blood urea nitrogen, and SMA-12 panel were within normal limits. Additional laboratory tests included negative IgG and IgM antibody titers for toxoplasmosis and a normal percentage of T and B lymphocytes. Skin tests in response to purified protein derivative, coccidioidin, histoplasmin, and candida were all negative; but the patient did react to a respiratory vaccine skin test (19). The hepatitis B surface antigen test was negative. Biopsy specimens of skin nodule, lymph node, and liver taken in December 1980 were reviewed and were consistent with Kaposi's sarcoma.

Treatment with amphotericin B and flucytosine was initiated and there was improvement of fever, headaches, and anemia. Computed tomography of the brain, chest, and abdomen; intravenous pyelogram; barium enema; and upper gastrointestinal series were done and no abnormalities were detected.

Chemotherapy with vinblastine for Kaposi's sarcoma was begun during the 3rd week of hospitalization. The total leukocyte count dropped to 1400/mm<sup>3</sup> and fever, bilateral retinal infarcts with hemorrhage, and a new pulmonary infiltrate developed during the fourth week of antifungal therapy. A transbronchial lung biopsy was done. Silver methenamine stain of the lung specimen showed numerous cysts of *P. carinii*. Viral cultures of lung biopsy tissue were negative, but immunofluorescence antibody testing for cytomegalovirus was positive.

A 2-week course of trimethoprim-sulfamethoxazole was instituted and the fever and pulmonary infiltrates resolved. The patient was given 6 weeks of antifungal therapy. India ink examination of the cerebrospinal fluid remained positive, but *F. neoformans* could not be cultured. The patient was discharged but received vinblastine therapy weekly for Kaposi's sarcoma. Viral cultures of urine and blood were positive for cytomegalovirus at the time of discharge.

From May to September, the patient was hospitalized on five occasions for exacerbation of Kaposi's sarcoma, fever, and neutropenia. The oral sarcoma lesions responded to local irradiation. Chemotherapy was resumed with dactinomycin, dacarbazine, and vinblastine. Although *P. carinii* pneumonitis was not shown, the fevers responded to reinstitution of the high-dose trimethoprim-sulfamethoxazole. The patient's urine remained positive for cytomegalovirus, and he had persistent herpes virus perianal lesions and intermittent *Candida* stomatitis. Persistent budding yeast forms were seen in the cerebrospinal fluid, but fungal cultures were negative. The serum cryptococcal antigen titer continued to fall. Computed tomography of the head, multiple electroencephalograms, and multiple toxoplasma titer evaluations by indirect fluorescent antibody and Sabin-Feldman dye tests were done and all the results were negative. The patient took lithium carbonate and trimethoprim-sulfamethoxazole erratically as an outpatient.

On 29 October 1981, the patient was admitted to the hospital with fever, bilateral pulmonary infiltrates, obtundation, and complaints of worsening vision. Physical examination showed enlarging retinal infarcts with overlying hemorrhage and nuchal rigidity. Spinal fluid analysis showed nonviable budding yeast. A repeat lung biopsy showed cryptococcus and *P. carinii* on microscopic examination. Bronchial washings grew cytomegalovirus; however, direct immunofluorescence stains for cytomegalovirus on lung biopsy tissue were negative. Serum cryptococcal antigen titer had risen from a low of 1:64 in May 1981 to 1:8192 on 7 November 1981. Blood cultures grew cryptococcal species as well. Despite therapy with amphotericin B, 5-fluorocytosine, trimethoprim-sulfamethoxazole, and vidarabine, the patient died on the 31st hospital day.

Postmortem examination confirmed Kaposi's sarcoma involving multiple skin sites. In addition, a fibroangiomatous process similar to that seen in the skin was found to involve diffusely the bronchial mucosa, bronchial and retroperitoneal lymph nodes, lungs, spleen, bladder mucosa, epididymis, and adrenal glands. *Filobasidiella neoformans* was found in both lungs, bronchial lymph nodes, meninges, and spleen. In addition *P. carinii* was again present in the pulmonary parenchyma.

#### PATIENT 4

A previously healthy 35-year-old white homosexual man was





Figure 1. Representative computed tomographic scan from Patient 5 showing one of several enhancing lesions in the central nervous system.

admitted on 21 November 1980 for evaluation of dyspnea, fever, retinal exudates, and a 9 kg weight loss. The patient was a botanist, working in the central valley of California, and had an extensive travel history. Physical examination at the time of admission included a temperature of 39.5 °C, tachypnea, bilateral retinal "cotton wool" exudates without hemorrhage, and *Candida* stomatitis. The chest was clear to auscultation.

Laboratory test results included a hematocrit of 37.3%, a lymphocyte count of 270 cells/mm<sup>3</sup>, and an erythrocyte sedimentation rate of 88 mm/h. Results of urinalysis showed 2+ proteinuria and no microscopic abnormalities. Serum electrolyte, blood urea nitrogen, and serum creatinine levels were within normal limits. The SMA-12 panel results were within normal limits except for an aspartate aminotransferase level of 94 IU/L (normal is less than 30) and lactic dehydrogenase of 900 IU/L (normal is less than 170). An arterial blood gas measurement showed marked hypoxemia. The chest roentgenogram showed no abnormalities.

On day 1 a presumptive diagnosis of desquamative interstitial pneumonitis was made and treatment was instituted with corticosteroids and cyclophosphamide. On day 2 an open lung biopsy was done. Histopathologic examination of the tissue did not confirm interstitial pneumonitis. Silver methenamine stain of the lung tissue showed numerous *P. carinii* cysts. Cyclophosphamide was discontinued and the patient was treated with corticosteroids and trimethoprim-sulfamethoxazole. Over the next several days, the patient's clinical status improved with remission of the fever and correction of the arterial hypoxemia. After 14 days of therapy, the patient was asymptomatic and the chest roentgenogram showed no abnormalities.

Additional data taken before discharge from the hospital included the findings of a mild polyclonal gammopathy with an IgA level of 464 mg/dL (normal range is 60 to 330) but no abnormalities in cryoglobulin, angiotensin-converting enzyme, C3, C4, CH50, antidual double stranded DNA, and antismooth muscle antibody measurement. Routine bacterial cultures of

blood and sputum were negative. At the time of admission, tests in response to purified protein derivative, coccidioidin, toxoplasmin, and mumps antigens were negative. Serology tests for cytomegalovirus IgG and IgM antibodies were markedly abnormal with titers of greater than 1:4096 and of 1:128, respectively. No viral cultures were done.

After discharge on the 16th hospital day, as the corticosteroid therapy was being tapered, recurrent fever, *Candida* stomatitis, and fatigue developed. Blue nodular skin lesions appeared over the arms and legs. The patient was readmitted on 28 February 1981 for further evaluation. A biopsy of the skin done as well as a blind lymph node biopsy and both showed Kaposi's sarcoma. Culture of the sputum, lymph node, and a repeat bone marrow specimen grew *Mycobacterium avium* *tracellulare*. Antituberculous therapy with isoniazid, rifampin, and ethambutol was instituted, but the patient died 16 weeks later.

#### PATIENT 5

A 25-year-old white homosexual man was admitted March 1981 for evaluation of a new grand mal seizure disorder and weakness of his left side. He had been well until February 1981 when a nonproductive cough and nongonococcal epididymitis developed. The patient was treated with tetracycline. He had no history of fever, chills, gastroenterologic symptoms, local or foreign travel, occupational exposure, nitrite use, exposure to animals. The patient had had one episode of gonorrhea. On admission the patient was afebrile. Physical examination showed moderate left hemiparesis but no other abnormalities.

Laboratory test results included an erythrocyte sedimentation rate of 53 mm/h, but complete blood count, results of urinalysis, serum electrolytes, blood urea nitrogen, serum creatinine, and SMA-12 panel were within normal limits except for a lymphocyte count of 750 cells/mm<sup>3</sup>. The chest roentgenogram showed a hazy left retrocardial infiltrate and computed tomography of the brain showed multiple ring and solid enhancing lesions in both cerebral hemispheres (Figure 1). Strontium examination yielded a few cysts of *Giardia lamblia*.

Dexamethasone therapy, 24 mg/d orally, was initiated. Brain biopsy done on day 6 was nondiagnostic. On day 7 therapy with penicillin and chloramphenicol was started. Serial computed tomographic studies of the brain showed progression of the size and number of the lesions. On day 14 fever, tachypnea, and a nonproductive cough developed. Arterial blood gas measurements showed increasing hypoxemia, and a chest roentgenogram showed diffuse pulmonary infiltrates. A transbronchial lung biopsy done on day 18 showed *P. carinii* on silver methenamine stain. Penicillin and chloramphenicol were discontinued and treatment with high dosage oral trimethoprim-sulfamethoxazole was started.

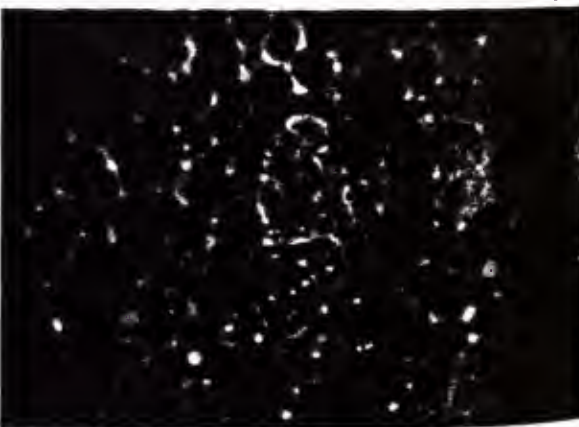


Figure 2. Touch preparation of a brain biopsy specimen from Patient 5 showing *Pneumocystis carinii* trophozoites. (Giemsa stain; phase contrast; original magnification,  $\times 1000$ .)



Table 1. Clinical Characteristics of Five Patients with *Pneumocystis carinii* Pneumonitis

Patient	Age	Therapy Before Diagnosis	Other Opportunistic Infections	Kaposi's Sarcoma	Skin Test	Outcome
1	44	Tetracycline × 10 days	Thrush; cytomegalovirus pneumonitis and adenitis	No	Nonreactive	Died
2	31	Tetracycline × 4 days	Thrush; cryptococcal meningitis; cytomegalovirus pneumonitis; <i>nocardia</i> pneumonia; herpes simplex virus proctitis	No	Nonreactive	Alive
3	37	Vinblastine × 7 days	Cryptococcal pneumonia, meningitis and fungemia; perianal and central nervous system herpes simplex; thrush; cytomegalovirus pneumonitis and viremia	Yes	Responsive to nasal respiratory vaccine	Died
4	35	Corticosteroids and cyclophosphamide × 24 hours	Thrush; atypical mycobacteriosis	Yes	Nonreactive	Died
5	25	Tetracycline; dexamethasone × 14 days	<i>Giardia lamblia</i> cysts in stool	No	Responsive to mumps antigen	Died

Histopathologic examination of a brain biopsy specimen taken on day 17 showed a cyst of *Toxoplasma gondii*. Serologic evaluation for toxoplasmosis included an IgM titer of less than 1:4 and an IgG titer of 1:2048, both of which were stable. A third brain biopsy done on day 24 showed only gliosis and non-specific meningeal inflammation. A probable cyst of *P. carinii* was seen on the Giemsa stain of the touch preparation (Figure 2). No additional *Toxoplasma* or *Pneumocystis* cysts were identified. Cultures of the lung tissue and brain biopsy specimens were negative for acid-fast bacilli, fungi, *Nocardia*, *Legionella*, viruses, and chlamydia.

There was no skin test response to purified protein derivative, but there was a skin test response to mumps antigen. Computed tomography of the chest and abdomen, and cerebral angiography yielded no abnormalities. Quantitative serum immunoglobulins measurement, hepatitis B surface antigen test, antinuclear antibody test, and rheumatoid factor test were within normal limits. Serologic tests for cytomegalovirus yielded an IgG titer of 1:256 and an IgM titer of less than 1:8. Cerebrospinal fluid analysis also showed no abnormalities.

Two weeks after initiation of trimethoprim-sulfamethoxazole therapy, the pulmonary symptoms resolved and the chest roentgenogram showed no abnormalities. After 3 weeks of therapy, computed tomography of the brain showed marked resolution of all lesions with residual postbiopsy artifacts. The patient was discharged on phenytoin therapy, and was asymptomatic except for minimal left hemiparesis.

Ten days after discontinuation of the trimethoprim-sulfamethoxazole, the patient was admitted to a hospital in Pennsylvania because of recurrent seizures and aphasia. Computed tomography of the brain again showed enhancing lesions in both cerebral hemispheres. The chest roentgenogram showed no abnormalities. High dosage trimethoprim-sulfamethoxazole and corticosteroid therapy was started and the neurologic symptoms cleared. There was no further evidence of *P. carinii* infection.

On low dosage trimethoprim-sulfamethoxazole (4 mg/kg body weight · d and 20 mg/kg body weight · d, respectively), the patient suffered a progressive downhill course. This was characterized by further neurologic deterioration despite stable but persistently abnormal computed tomographic studies of the head, profound lymphopenia, and recurrent pulmonary emboli. He died on 15 December 1981, 9 months after his initial hospital admission. Permission for postmortem examination was refused.

## Results

Table 1 shows the clinical aspects of the five cases of *P. carinii* pneumonia. All of the patients were sexually active homosexual men. None appeared to know each other. There were no uniform histories of drug abuse. Two admitted to the occasional use of inhaled amyl or butyl nitrite. Three of the patients had a definite past history of hepatitis. One of the five patients had a history of intestinal parasitic diseases. Only two patients had been receiving immunosuppressive therapy before signs or symptoms of *Pneumocystis* infection developed. Patient 5 received dexamethasone therapy for 18 days in tapering doses and Patient 3 received vinblastine for 7 days before the diagnosis of *P. carinii* pneumonia. Three patients had taken tetracycline before admission. Patients 3 and 4 had Kaposi's sarcoma. In Patient 3 the diagnosis of Kaposi's sarcoma was made 4 months before the episode of *P. carinii* pneumonia. In Patient 4 the diagnosis was made 3 months after the episode of *P. carinii* pneumonia.

Only two patients were admitted with respiratory complaints (Patients 2 and 4). In others, signs and symptoms developed during hospitalization (day 4 in Patient 1, day 14 in Patient 5, and the fourth week in Patient 3). Four patients had additional opportunistic infections (cytomegalovirus cultured from Patients 1, 2, and 3; cryptococcal meningitis in Patients 2 and 3; atypical mycobacteriosis in Patient 4; nocardiosis in Patient 2; *Candida* stomatitis in Patients 1, 2, 3, and 4; perianal herpes simplex virus infection in Patients 2 and 3). Evidence of past or current cytomegalovirus infection was found in all patients (Table 2). In four of the patients, positive cytomegalovirus cultures and elevated cytomegalovirus-specific IgM titers suggested recent or concurrent active infections.

All patients had negative hepatitis B surface antigen tests and no evidence of active hepatitis except for Patient 4, who had mildly abnormal hepatic enzymes on hospital



Table 2. Results of Culture and Serologic Testing for Cytomegalovirus\*

Patient	Cytomegalovirus Serology		Cytomegalovirus Cultures			
	Date	ACIF IgM	FA	Blood	Lung	Urine
1	1 May 1981	1024	8	ND	-	ND
	29 August 1981	512	16	-	-	-
	29 September 1981	ND	ND	ND	+	ND
2	8 April 1981	256	32	ND	+	-
	13 May 1981	128	8	ND	+	ND
	17 November 1981	256	16	ND	+	-
3	22 December 1980	128	16	ND	ND	+
	31 March 1981	32	8	ND	ND	+
	22 April 1981	32	8	ND	ND	+
	1 May 1981	16	8	+	-	+
4	22 November 1980	4096	128	ND	ND	ND
	2 March 1981	256	32	ND	ND	ND
5	19 April 1981	256	8	ND	-	-

\* ACIF = IgG measured by anti-complement immunofluorescent antibody assay; IgM FA = IgM measured by immunofluorescent assay; + = positive; - = negative; ND = not done.

admission. The only parasitic infestation was asymptomatic intestinal giardiasis in Patient 5. Lymphocyte testing was done in two patients; a normal response to phytohemagglutinin was found in Patient 2, but was markedly depressed in Patient 1. A relative excess of suppressor/helper T cells was found in Patient 2, whose T-cell subsets were measured. All patients had two or more delayed hypersensitivity skin tests; two patients reacted to one test and the others showed no reaction.

One patient (Patient 2) has survived, but remains seriously ill from problems currently unrelated to *Pneumocystis* pneumonia. He is receiving low dose trimethoprim-sulfamethoxazole (160 mg and 800 mg, respectively, twice daily), although its efficacy in preventing the recurrence of *P. carinii* pneumonia in this population remains unproven. Patient 1 died of progressive respiratory and cardiac failure, with evidence of disseminated cytomegalovirus infection and persistent *P. carinii* pneumonia despite more than 30 days of treatment. Patient 3 died of Kaposi's sarcoma, disseminated *F. neoformans* infection, persistent *P. carinii* pneumonia, *Candida* stomatitis, herpes simplex perianal infection, and cytomegalovirus. Patient 4 died during therapy for atypical mycobacteriosis. Patient 5 died without evidence for *Pneumocystis* infection on a dose of trimethoprim-sulfamethoxazole previously reported to be suppressive against *P. carinii* pneumonia in a pediatric population (20). The cause of his neurologic deterioration was not confirmed.

## Discussion

We report five cases of *P. carinii* pneumonitis in five homosexual men. *Pneumocystis carinii* is a protozoan distributed widely in nature and found in both humans and lower animals (21). The true incidence of infection in humans has not been ascertained. Small clusters of cases have been reported previously (22-25). It has been difficult to ascertain whether occurrence and identification of these clusters has been due to increased intensity of chemotherapy (26), aggressiveness of diagnostic eval-

uation, or person-to-person transmission in a susceptible population. The mode of transmission of this protozoan is not well defined. Limited attempts to document person-to-person transmission have shown that healthy persons who come in close contact with index cases have elevated antibody titers (24, 25). A murine model that closely resembles *P. carinii* pneumonia in humans has been developed (27, 28). The illness appears to develop in these animals by reactivation of latent infection.

Many infectious and noninfectious diseases have been reported with increased frequency in homosexual men (29). The infectious illnesses include gonorrhea, syphilis, condyloma acuminatum, meningococcal urethritis and proctitis, shigellosis, *Campylobacter* enteritis, typhoid fever, herpes simplex proctitis, hepatitis A and B, amebiasis, giardiasis, enterobiasis, strongyloidiasis, and cytomegalovirus infection.

Patient 5 had been receiving steroids during the 14-day interval during which his chest roentgenogram progressed from a small retrocardiac infiltrate to diffuse bilateral disease. The association of steroid administration and development of *P. carinii* in immunocompromised humans and the rat model is well established (24-26, 30-32). The mean duration of steroid therapy before onset of pulmonary symptoms in humans has ranged from 30 to 100 days (24). In this patient it is likely that pneumocystis pneumonia and possible central nervous system dissemination were established at the time of admission.

Tetracyclines were used by three of the five patients just before or concurrent with the onset of *Pneumocystis* infection. Tetracyclines are given in the rat model for *P. carinii* pneumonia to prevent fatal pneumonia caused by *Corynebacterium kutscheri* (27). Although tetracyclines have been shown to impair host defense mechanisms and are responsible for hypertrophy of rat type II pneumocytes, there is no evidence that they directly predispose to *Pneumocystis* infection (33, 34).

In all five patients there was evidence for either an acute or preceding infection with cytomegalovirus. The virus was recovered from the lung biopsy specimen of Patient 2 in only 2 days, indicating a high titer of virus in the specimen. Pathologic findings and viral cultures from autopsy specimens taken from Patient 1 showed disseminated cytomegalovirus infection. Cytomegalovirus was isolated from blood and urine cultures in Patient 3 during therapy for *Pneumocystis* pneumonia. All five patients had cytomegalovirus specific IgG antibody and four had cytomegalovirus specific IgM antibody. Because 93% of sexually active gay men have cytomegalovirus IgG antibody (35) and approximately 50% have IgM antibody, the significance of these serologic findings is uncertain. Nonetheless, all five patients had evidence of past or concurrent cytomegalovirus infection, which has been suggested as a predisposing condition for *P. carinii* pneumonia (36, 37).

Coexistent cytomegalovirus and *Pneumocystis* pneumonia has been described previously (38, 39). Rosen and associates (40) found histologic evidence of cytomegalovirus infection in the lungs of eight of 20 patients with *Pneumocystis* pneumonia. This may well be an under-



mate because the presence of characteristic viral inclusions is an insensitive marker of cytomegalovirus presence in the lung (41).

If cytomegalovirus infection predisposes persons to *Pneumocystis* infection, what is the mechanism? Cytomegalovirus is able to infect the alveolar macrophages of both man and mouse (42, 43); in the mouse model cytomegalovirus impairs phagocytosis of bacteria. Histologic studies (44) suggest that the macrophage is an important component of the host response to *P. carinii*. It may be that cytomegalovirus infection of macrophages impairs their ability to interact with *P. carinii*. Cytomegalovirus infection might also predispose to subsequent *P. carinii* pneumonia by impairing cell-mediated or humoral immune responses or both. Mononuclear leukocytes from the blood of patients with active cytomegalovirus mononucleosis are selectively hyporesponsive to pokeweed mitogen and concanavalin A (45). In the mouse, cytomegalovirus infection suppresses the primary and secondary antibody responses to foreign antigens (46). Still another explanation for cytomegalovirus-*Pneumocystis* interaction was proposed by Wang and colleagues (47) who suggested that *P. carinii* might serve as an intermediate host or reservoir for cytomegalovirus.

In preliminary studies other patients with *P. carinii* pneumonitis had profoundly depressed numbers of thymus-dependent lymphocytes cells (13-15). In addition, in-vitro proliferative responses to mutagens and antigens were profoundly depressed. In this series, three patients were nonreactive to skin tests and two reacted to a single antigen. Patient 2, whose skin tests were negative, had tests after his first course of trimethoprim-sulfamethoxazole therapy that showed normal T and B cell numbers and normal qualitative lymphocyte function. However, Patient 1, late in the course of his illness, had the depressed immunologic functions characterized in earlier reports.

An attempt was made to establish a number of possible epidemiologic factors predisposing to *P. carinii* pneumonitis in addition to cytomegalovirus infection, immunosuppressive therapy, and tetracycline use. There was no consistent history of amyl or butyl nitrite inhalation. These inhalants, or "poppers," are used chiefly "to enhance sexual activities, intensify meditation, make dancing a more vivid experience, and alter consciousness" (48). No previous association between these and any respiratory disease has been reported. Intestinal infectious diseases (included in the pattern of anorectal and colon diseases termed "gay bowel syndrome") are common in some homosexual men (29), but documentation of previous or concurrent infection could be established in only two of our patients. Although giardiasis has been associated with hypogammaglobulinemic disorders (49), there is no reported association between this or other protozoan infections and *P. carinii* infection.

Two of these patients had Kaposi's sarcoma. Recently, the appearance of an apparently fulminant form of this disease in young homosexual men has been noted (50). The association of Kaposi's sarcoma with cryptococcal pulmonary infection and *Toxoplasma* cerebritis has also

been noted (51). Until recently (52) Kaposi's sarcoma had not been reported in association with *P. carinii* pneumonitis. The nature of this association is unclear.

Finally, we believe that Patient 5 probably had *Pneumocystis* infection of the central nervous system. He presented with multiple enhancing central nervous system lesions; biopsy of these lesions showed a cyst form of *Pneumocystis*. The patient improved markedly on trimethoprim-sulfamethoxazole therapy for *Pneumocystis*, having failed to respond to steroids, chloramphenicol, and penicillin. An exhaustive evaluation including three brain biopsies failed to show any bacterial, fungal, granulomatous, viral, neoplastic, or other protozoan cause. Although a *Toxoplasma* cyst was also seen in one of the biopsy specimens, we think that this is an unlikely explanation of this patient's neurologic disease. Serum measurements were repeatedly negative for *Toxoplasma*-associated IgM and showed stable titers of IgG by Sabin-Feldman dye testing. In addition, toxoplasmosis has been reported in a chronic asymptomatic form in approximately 50% of the population of the United States (53).

Dissemination of *Pneumocystis* to the central nervous system has not been reported previously. Extrapulmonary dissemination has been shown in a small number of cases and has involved multiple organs including perihilar and other lymph nodes, spleen, pericardium, stomach, small intestine, and kidney (7, 54-57). None of the patients with widespread dissemination had focal neurologic signs or symptoms. All were severely immunocompromised. In only one case was a negative histologic search of the central nervous system reported (7). The mode of dissemination has not been elucidated but presumably is hematogenous because of the large number of organs involved and the histologic evidence of direct capillary invasion by *Pneumocystis* (55).

It is unlikely that many earlier cases of *P. carinii* pneumonia in previously healthy homosexual men would have escaped detection. Each of our patients presented with severe progressive clinical and roentgenographic findings. An explanation for the recent appearance of this illness in New York City, Los Angeles, and the San Francisco Bay Area has not been found. Does it involve a new method of spreading this common organism or a new stimulus to reactivate previous infection? Is there a new agent, either infectious or noninfectious, that can depress the host defenses in some people and therefore predispose to *P. carinii* pneumonia, other opportunistic infections, and Kaposi's sarcoma? It is likely that an agent not yet identified, an environmental factor, or multiple factors, are involved. Of the factors examined in our patients, all of whom were homosexual, only cytomegalovirus infection was consistently noted. In view of the known capacity of cytomegalovirus to depress various components of the immune reaction, it seems possible that repeated cytomegalovirus exposure or infection or both initiates a state of severe immunocompromise that in turn provides the necessary conditions for the emergence of opportunistic infections such as *P. carinii* pneumonia. Ongoing surveillance for similar cases and further research is warranted to attempt to clarify this problem.



## Addendum

Since this manuscript was submitted for publication in July 1981, we have seen an additional six homosexual men with biopsy specimens that showed *P. carinii* pneumonia, in San Francisco. All are white between the ages of 29 to 45 years. All had had a 2.3- to 4.5-kg weight loss associated with fatigue, intermittent fevers as high as 39.5 °C, and a respiratory syndrome associated with progressive exertional dyspnea and cough producing white sputum. The duration of these symptoms ranged from 10 days to 4 months. Only one patient has evidence of Kaposi's sarcoma. One patient has diffuse adenopathy; another has adenopathy limited to the periaortic and mesenteric areas, shown on computed tomography of the abdomen.

On admission, all patients had chest examinations that were either clear to auscultation or showed only soft inspiratory dry rales. Two of the six patients had "cotton wool" exudates on fundoscopic examination. Five of the six patients had abnormal chest roentgenograms. The sixth patient, who had a clear chest roentgenogram, had 4+ *P. carinii* on stain of his transbronchial biopsy specimen. Total lymphocyte counts on admission ranged from 50 to 1700 cells/mm<sup>3</sup>. All patients have been hypoxemic. Five of the six patients have had elevated serum IgA determinations. One patient responded to skin tests for mumps and coxioidin. The remainder did not respond to two to four recall antigens. The patient who reacted to skin tests had only slightly abnormal lymphocyte tests with a response to phytohemagglutinin stimulation that was 70% of normal. Other parameters were within normal limits and his *P. carinii* pneumonia responded to 11 days of high dose trimethoprim-sulfamethoxazole therapy.

All six patients had one or more additional opportunistic infections. These included *Candida* stomatitis or esophagitis in five patients; cytomegalovirus cultured from more than one source with elevation of IgM-specific antibody or a rising titer of IgG-specific antibody in five patients; herpes simplex infections involving the perianal area, lung, or central nervous system in three patients; and herpes zoster in one patient.

All patients received trimethoprim-sulfamethoxazole, pentamidine, or a combination for treatment of *Pneumocystis* infection for 11 to 30 days. Five are alive. One patient has renal failure with an IgA nephropathy in which cytomegalovirus-specific antibody has been identified on direct immunofluorescence stain of renal tissue, pancytopenia, and presumed herpes simplex encephalitis. One was discharged after 9 days of trimethoprim-sulfamethoxazole therapy and 21 days of pentamidine therapy, received sequentially. This patient has been followed for 2 months on no therapy after he developed an immediate hypersensitivity reaction to trimethoprim-sulfamethoxazole, shown on rechallenge. A third patient received 14 days of trimethoprim-sulfamethoxazole and 25 days of pentamidine therapy, which overlapped by 11 days. His condition began to improve on pentamidine, 4 mg/kg body weight · d, intramuscularly, 3 days after trimethoprim-sulfamethoxazole was discontinued and 1 day after high-dose glucocorticoid therapy was initiated. However, after 28 days of therapy, the presence of cytomegalovirus and the persistence of *P. carinii* in the lung was shown in the results of the transbronchial biopsy. The patient continues to receive high dose trimethoprim-sulfamethoxazole, intravenously. The fourth patient is receiving treatment for Kaposi's sarcoma, now 4 months after the episode of *Pneumocystis* pneumonia. The fifth patient is currently being treated for *P. carinii* pneumonia. The sixth patient died; there was postmortem evidence of overwhelming *Pneumocystis* and herpes simplex pneumonia, herpes simplex encephalitis, and oral and perianal herpes simplex infection, as well as angiomatous malformations of the distal esophagus and histiocytic infiltration of multiple lymph nodes with loss of the follicular architecture.

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CITY AND COUNTY OF SAN FRANCISCO  
DEPARTMENT OF PUBLIC HEALTH  
INTER-OFFICE MEMORANDUM

Date: January 11, 1982 [1983] Subject: (S.E. Follansbee, personal correspondence, folder #421).  
From: Aids Risk Reduction Sub-Committee  
S.F.P.H.D. Gay and Lesbian Health Services Coordinating Committee  
Lynn Eggers, R.N.  
Tom Smith, M.D.  
Glen Margo, M.S.W., Dr. P.H.  
To: Health Providers

Report on meeting held Wednesday, 22 December, 1982.

PRESENT: Pat Norman; Tom Smith, M.D., Richard Smith; Helen Schietinger;  
Bill Owen, M.D., Michael Gorman, Lynne Eggers, R.N., Glen Margo.

A very productive work-group focused on Aids Risk-reduction needs of several target groups, which included hospital and medical worker settings, dental workers, diagnosed patients and the general public, and mental health workers.

Discussion centered around what areas need to be explored in relation to the drafting of risk reduction guidelines. Four major work groups are set up to generate draft materials for further discussion and development by the whole group at the next meeting.

The Four working groups are:

- |  |  |
|--|--|
| <p>A. <u>Hospital and Medical Settings</u><br/> Physicians<br/> Dentists<br/> Nurses<br/> Lab Techs.<br/> Janitorial Staff<br/> Kitchen Staff<br/> Other Workers</p> | <p><u>Draft Work Groups</u><br/> Richard Smith,<br/> (and an infection control<br/> person to be designated)</p> |
| <p>B. <u>Diagnosed Patients</u></p>  | <p>Helen Schietinger, R.N.</p>   |
| <p>C. <u>Public-Social Settings</u></p>  | <p>Tom Smith, M.D.<br/> Glen Margo, M.S.W., Dr. P.H.</p>   |
| <p>D. <u>Mental Health Settings</u></p>  | <p>Lynn Eggers, R.N.<br/> Michael Lipp</p>   |

All of the ~~all~~ these groups need input on how people are currently proceeding around Aids Risk Reduction. Information can be mailed to Glen Margo, Office of Health Promotion and Education, Room 204, 101 Grove St., S.F. CA 94102.

## Information Sheet for Hospital Employees:

## AIDS Infection Precautions

<u>Precautions</u>	<u>Rationale</u>
1. Private room.	1. a) Protect patient from hospital infections. b) Protect other immunosuppressed patients.
2. Masks On patient outside of room. On susceptibles inside room.	2. a) Protect visitors and employees who might have some degree of immunosuppression already. b) Excretion rate of CMV from lungs is unknown.
3. Gloves and handwashing for contact with <u>all</u> body secretions.	3. <u>Protect employee from the transmissible agent of AIDS, herpes, CMV, hepatitis, etc.</u>
4. Blood Precautions - a. Wear gloves and wash hands for <u>all blood contact</u> . b. <u>Wear gloves to draw blood</u> (including needle aspirations and through stopcocks).	4. <u>Protection from the transmissible agents of AIDS and hepatitis.</u>
5. Needle Precautions Do not break needles. Place in needle box or impervious plastic container for Blood Precautions being careful not to puncture sides.	5. Accidental puncture of skin or splatter of mucous membranes or conjunctiva may constitute a significant exposure.
6. Pregnant women should not have direct contact.	6. Almost 100% of AIDS patients excrete CMV. CMV infection during pregnancy may result in birth defects.

based on Infection Control Committee Policy, revision 12/13/82

ISOLATION PROCEDURES FOR PATIENTS  
WITH KAPOSI'S SARCOMA AND OTHER  
ACQUIRED IMMUNODEFICIENCY SYNDROMES

Purpose: To prevent the possible transmission of an unknown infectious agent to susceptible individuals.

Introduction: Although an infectious etiology for the acquired immunodeficiency syndromes has not been proven, the epidemiology of the syndrome suggests a transmissible agent. Cytomegalovirus, hepatitis B, and Epstein-Barr viruses have all been considered as possible causes. Intimate contact with blood or secretions may be necessary for transmission.

Recommendations: Until the etiologic agent is identified, it seems prudent to institute certain precautions to protect individuals working with these patients. These should include at least isolation precautions appropriate for CMV and Hepatitis B viruses.

1. Patients should be admitted to private rooms.
- \* 2. Careful handwashing after patient contact is of utmost importance, even when gloves have been worn.
- \* 3. Excretion and blood precautions i.e. wearing of gloves for direct contact with blood, urine, stool, vomitus, and other body fluids should be instituted. Contaminated material should be placed in red or orange plastic bags and designated as "Infectious Waste". Needles should be rendered unfit for use and discarded as for all needles.
4. Pregnant women should ideally not care for patients with this syndrome.
5. Identification of patients requiring these isolation measures will be by the physicians caring for the patients and the head or charge nurses on each unit. In addition, oncology and infectious disease consultants seeing these patients will ask that isolation be instituted if it has not already been done. Key diagnoses that will alert medical staff to the need for isolation will include:
  - a) Acquired immunodeficiency syndrome or AIDS
  - b) Gay immunodeficiency syndrome or GRIDS
  - c) Kaposi's sarcoma
  - d) ITP in gay male
  - e) Gay lymph node syndrome
  - f) Gay male with fever and/or pulmonary infiltrate
  - g) Pneumocystis
  - h) Cytomegalovirus infection in an adult.

*Mary Anne Johnson*  
Mary Anne Johnson, M.D.  
Co-Chairman Infection  
Control Committee

*W. Keith Hadley*  
W. Keith Hadley, M.D.  
Co-Chairman Infection  
Control Committee



## Emergency Department

- Subject : Isolation precautions for patients identified or suspected with Kaposi's Sarcoma and other Acquired Immune Deficiency Syndromes.
- Rationale: To prevent the possible transmission of an unknown infectious agent to susceptible individuals.
- Policy : Although the infectious etiology for acquired immunodeficiency syndromes has not been proven the epidemiology of the syndrome suggests a transmissible agent. Cytomegalovirus, hepatitis-B and Epstein-Banviruses have been considered as possible causes. Intimate contact with blood or secretions may be necessary for transmission.
- Procedure: Until the etiologic agent is identified, it seems prudent to institute certain precautions to protect individuals working with these patients. These should include at least isolation precautions appropriate for CMV and Hepatitis B viruses:

I. Identification of patients

- A. Identification of patients requiring isolation precautions will be initiated by the triage nurse when possible, and with discretion and sensitivity, and by nurses and physicians caring for the patients in the clinical areas
- B. Key signs and symptoms are:
- 1) -gay male with fever
  - 2) -gay male with generalized lymph node
  - 3) -gay male with weight loss
  - 4) -gay male with fatigue
  - 5) -gay male with cough
  - 6) -gay male concerned about skin lesion
  - 7) -gay male concerned about new disease
- C. These precautions may also apply to IV drug abusers, Haitian entrants and Hemophiliacs.

II. Patient contact

Careful handwashing after patient contact is of utmost importance even when gloves have been worn

### III. Excretion and blood precautions

- A. Wearing of gloves for direct contact with blood, urine, stool, vomitus, and other body fluids should be instituted.
- B. As all trash from the Emergency Department is treated as "Infectious Waste", contaminated materials may be placed in regular trash receptacles.
- C. Needles should be discarded in the bin as for all needles.
- D. Laboratory specimens should be labeled with "blood precautions" or "AIDS precautions".
- E. Blood spills should be cleaned up promptly with a disinfectant solution.

### IV. Employees

- A. Pregnant women should ideally not care for patients with this syndrome.
- B. Gay male employees are advised to take precautionary measures when caring for patients identified or suspected with this syndrome.
  - 1) wearing of mask if cough is present
  - 2) wearing of gloves as above.

Approved by:

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Frank Lewis, M.D.  
Medical Director, E.D.

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Judy L. Spinella, RN, MS  
Clinical Coordinator, E.

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Constance Woolsey, MD  
Infectious Disease Clinic

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W. Keith Hadley, MD, PhD  
Co-Chairman Infection C  
Committee





INTER-OFFICE MEMORANDUM

DATE: January 4, 1983      SUBJECT:  
 FROM: NANCY HINDS 7-4  
 TO: GLEN MARGO

This is in response to the AIDS risk reduction committees' request for current health providers procedures for handling of AIDS patients at City Clinic. When nursing is screening an AIDS patient they use examining gloves, only when placing blood on the slides. When nursing is informed that the patient is a confirmed AIDS patient, it is currently at the discretion of the individual nurse to use examining gloves when drawing blood, or giving injections. The physicians use the same techniques as usual, examining gloves for the examination.

INFECTION CONTROL COMMITTEE  
PATIENT CARE GUIDELINES ON  
ACQUIRED IMMUNE DEFICIENCY SYNDROME

May 4, 1983

The Committee suggests the following guidelines be adopted by the Infection Control Committee at Moffitt Hospital for incorporation into its policies and procedures.

- 1) The definition of an AIDS patient is as outlined by the Centers for Disease Control in their category A designation; that is, "a disease at least moderately predictive of a defect in cell mediated immunity, occurring in a person with no known cause of diminished resistance to that disease. Such diseases include Pneumocystis carinii pneumonia, Kaposi's sarcoma and other serious opportunistic infections." It is recommended that patients with the generalized lymphadenopathy syndrome or those being evaluated for the possibility of AIDS should be included in the definition.
- 2) The responsibility for identification of the patient in the above categories should rest with the attending physician. It is also the responsibility of the attending physician to report the diagnosis to the Public Health Department since category A AIDS is now a reportable disease. The attending physician should also have the responsibility, in consultation with the Infection Control Unit, of determining when appropriate precautions and/or isolation measures can be discontinued.
- 3) Evidence to date indicates that the transmission of the AIDS agent is similar to the hepatitis B virus; that is, it requires direct contact with blood and body secretions. There is no evidence of airborne transmission; and, therefore, infection control policies and procedures appropriate for hepatitis B patients should be applied to AIDS patients.
- 4) The patients conforming to the definitions above should be placed on Blood and Excretion precautions. Specimens from the patients should be labelled "Blood Precautions" without mention of a specific disease.
- 5) Since there is no evidence for airborne transmission of an AIDS agents, mask precautions should only be applied as appropriate for patients with possible respiratory infection. That is, in patients with a productive cough mask precautions as presently practiced should be employed until the diagnosis of M. tuberculosis is ruled out. The Committee wishes to emphasize that this is a practice to prevent workers and visitors and other patients from organisms such as M. tuberculosis but not to prevent the transmission of an AIDS agent.
- 6) An isolation unit for AIDS patients is unnecessary and not appropriate at the Moffitt Hospital. Patients should be cared for on various floors utilizing policies and procedures outlined in this document.

Infection Control Committee  
Patient Care Guidelines on  
Acquired Immune Deficiency Syndrome  
May 4, 1983  
Page 2

- 7) Private rooms are not necessary for the care of patients with AIDS unless they have an additional illness which would normally require a private room. Therefore, patients should not be placed in a private room solely because they have or are suspected of having AIDS. Additionally, the Committee felt that a patient with AIDS should not be placed in a room with another immunocompromised patient.
- 8) Except as noted in Number 9, no special precautions should be instituted for AIDS patients being cared for in the outpatient setting. However, normal infection control measures should be taken for individuals with a productive cough.
- 9) Procedures for equipment used for AIDS patients in in-or outpatient settings should be as follows:
  - a) Because of the extreme difficulty in cleaning and refurbishing "Clinitron" beds, AIDS patients or suspected AIDS patients will not be using these devices.
  - b) Lensed instruments should be sterilized as recommended by the Centers for Disease Control.
  - c) Materiel Services should continue to pasteurize respiratory therapy tubing as it is presently doing.
  - d) Any instrument which comes in contact with blood, secretions or excretions must be sterilized before reuse. This includes anesthesia instruments, such as laryngoscopes and endotracheal tubes.
  - e) All contaminated (i.e. visibly soiled with potential infectious material) disposable items are to be considered "infectious waste" and must be red-bagged.
- 10) Employees who have needlestick injuries associated with the care of AIDS patients will be treated according to the present Hepatitis B Protocol. The Committee did not resolve the issue of follow-up of these patients and requested an opinion from the University attorney.
- 11a) There is no known contraindication for employees with AIDS who have recovered from intercurrent illnesses to return to work. There is no evidence to date that the "AIDS agent" or the AIDS-associated illnesses (CMV or Pneumocystis pneumonia) can be transmitted to patients.
- 11b) Because employees with AIDS are susceptible to infections, careful counselling on an individual basis should take place. Decisions should be based on the type of work the individual is trained to perform, the level of potential exposure and the willingness or ability of the employee to comply with careful technique.

Infection Control Committee  
Patient Care Guidelines on  
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July 4, 1983  
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- 12) The question of employee's refusal to work with an AIDS patient was discussed by the Ad Hoc Committee but not resolved. It was felt that this issue is more an administrative and ethical one rather than an infection control question. A letter has been sent to the Medical Ethics Committee, Hospital Administration and University counsel for an opinion on this situation.

This report prepared by:

John E. Conte, Jr., M.D.

Chairman, Ad Hoc Advisory Committee on Acquired Immune Deficiency Syndrome  
Chairman, Infection Control Committee



SCHOOL OF MEDICINE

Please address reply to undersigned at  
THE MEDICAL SERVICE  
Room 5 H 22  
San Francisco General Hospital  
San Francisco, California 94110

February 15, 1983

## Minutes of Infection Control Meeting re. AIDS

Present: Jensen (VA), Follansbee (UCSF), Hadley, Wofsy, Hopewell,  
Rankin, Volberding (SFGH),

This meeting was convened to discuss with representatives from each of the three major hospitals of the University of California at San Francisco issues concerning infection control precautions for patients with the Acquired Immune Deficiency Syndrome. There has been growing concern recently at SFGH because of the rapidly increasing number of AIDS patients, their distribution throughout the hospital, and a growing awareness on the part of medical and nursing staff of the problem these patients potentially represent. In response to these issues, and to optimize the care of AIDS patients, discussion has recently focused on possible values of an AIDS inpatient service/unit. The specific benefits which are hoped to accrue from this centralization of compliance with infection control guidelines for AIDS patient care include the following:

- (1) increased uniformity of compliance with infection control guidelines given the centralized nature of this unit and the availability of associated nursing personnel for in-depth education;
- (2) providing AIDS patients with nursing staff who are selected for work with this disease, who are interested in caring for these extremely ill patients, and who are felt to be at low personal risk for the acquisition of opportunistic infections;
- (3) centralization of inpatients should optimize the multi-disciplinary consultative care and improve the followup by medical subspecialty services including pulmonary, infectious disease and oncology;
- (4) similarly, centralization should improve the access of patients for organized psychologic support activities by such organizations as the Shanti Project.

These issues were discussed at this meeting and several concerns were raised, especially ones with regard to the possible potential psychologic stress such a ward might create for patients placed there, if this were seen as an isolation ward. The fear was expressed that patients would feel isolated from friends and support staff. Additionally, concern was raised that by concentrating AIDS patients in one area of the hospital, the medical and nursing staff would thus be exposed to more frequent contact with them, thereby potentially



increasing their risk for the acquisition of disease. These issues were discussed at length and it was the general consensus of the group that with such a unit, if care were taken to limit the isolation characteristics of the ward, optimal care for patients could be provided and should be supported.

An additional subject discussed at length was the type of isolation precautions which should be recommended for nursing personnel, whether on such an inpatient unit or on general medical wards. The approaches of the three hospitals isolation precautions were reviewed, and some differences were apparent. Specifically, while AIDS patients at the VA and SFGH are placed in private rooms, patients with AIDS at UC Medical Center are occasionally placed in semi-private rooms. While masks are recommended for certain personnel caring for AIDS patients at SFGH, no such recommendation has been adopted by the Infection Control Committee at UC Medical Center. These issues were discussed at length, and the following general isolation procedures were agreed upon:

- 1- Pregnant women should not care for AIDS patients;
- 2- Personnel may be excused from caring for AIDS patients only if they have themselves a documented immune deficient state (subject to approval by individual infection control committees);
- 3- Gloves should be worn when in contact with secretions, when performing phlebotomy or when placing intravenous catheters;
- 4- Gowns should be worn when in contact with patient secretions or wastes;
- 5- Masks should be worn only in rooms of patients who are actively coughing;
- 6- Masks need not be worn by patients when out of their hospital room unless they are actively coughing;
- 7- As much as possible all patient procedures should be in agreement with the above recommendations for inpatient care.



HOSPITALS AND CLINICS

SAN FRANCISCO, CALIFORNIA 94143

AD HOC ADVISORY COMMITTEE ON  
ACQUIRED IMMUNE DEFICIENCY SYNDROME

MARCH 8, 1983

PRESENT:

John E. Conte, Jr., M.D., Chairman  
Donald Abrams, M.D.  
David Altman, M.D.  
Arthur Ammann, M.D.  
Stephen Follansbee, M.D.  
Jeffrey Golden, M.D.  
Jane Hirsch, R.N.  
Jay Levy, M.D.  
Ronald Lipsy  
Jacqueline Octavio R.N.  
Linda Rosendorf, M.S.  
Helen Schietinger, R.N.  
Daniel Stites, M.D.  
Carol Viele, R.N.  
William West  
Betty Wimberly, R.N.

ABSENT:

Stephen Cohen, M.D.  
Marcus Conant, M.D.  
Richard Jacobs, M.D.  
Vincent Pons, M.D.  
Connie Wofsy, M.D.

Dr. Conte summarized the charge to this subcommittee of the Infection Control Committee. There has been increasing concern about issues surrounding the care of patients and the handling of specimens from patients with Acquired Immune Deficiency Syndrome (AIDS). This advisory committee was formed to reconsider guidelines to minimize the risk to staff and other patients and yet preserve an optimal environment for the care of AIDS patients.

The Committee reviewed the following documents:

- a) Centers for Disease Control guidelines for the care of AIDS patients.
- b) The Infectious Diseases and Infection Control Bulletin which reviewed AIDS policies and procedures at U.C.S.F.
- c) A synopsis of questions and problems that have arisen at U.C. related to care of AIDS patients.

Dr. Conte reported that a Task Force chaired by Dr. Merle Sande is meeting at San Francisco General Hospital to develop guidelines as well. They are considering strongly the creation of an "AIDS Ward" and a separate bronchoscopy room and equipment.

March 8, 1983  
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✓ Dr. Stites summarized the Committee on Human And Environmental Protection's decision that the AIDS agent be classified as a P2\* agent. This raises questions regarding the flow of unknown specimens through the laboratory and proper notification when a specimen is known to be from an AIDS patient. The P2\* designation also requires that ventilation hoods be provided for work areas which will receive "AIDS" specimens. The Committee recommended that a generic label be adopted to identify hepatitis and/or AIDS specimens.

The Committee agreed that evidence to date indicates that the transmission of the AIDS agent is similar to the hepatitis B virus; i.e. direct contact with blood and body secretions; it also concluded that there is no evidence for airborne transmission and that infection control policies and procedures appropriate for hepatitis B patients should be applied to AIDS patients.

A wide ranging general discussion was held addressing many aspects of the biology, pathogenesis and transmission of AIDS.

Other issues, such as sterilization of equipment, identification of patients, psychosocial implications of guidelines, were discussed but not resolved at this meeting.

The Committee recognized the need to have additional meetings and agreed to do so as soon as possible.


 AD HOC ADVISORY COMMITTEE ON  
 ACQUIRED IMMUNE DEFICIENCY SYNDROME

May 13, 1983

## PRESENT:

John E. Conte, Jr., M.D., Chairman  
 Donald Abrams, M.D.  
 David Altman, M.D.  
 Ed Chinn, M.D.  
 Stephen Cohen, M.D.  
 Linda Ferrell, M.D.  
 Stephen Follansbee, M.D.  
 Jay Levy, M.D.  
 Ronald Lipsy  
 Jacqueline Octavio, R.N.  
 Roger Pedersen, Ph.D.  
 Linda Rosendorf, M.S.  
 Kevin Welsh, M.D.  
 Karla West  
 Victor Yick

## ABSENT:

Arthur Ammann, M.D.  
 Marcus Conant, M.D.  
 Jeffrey Golden, M.D.  
 Bernie McLin  
 Edward Smuckler, M.D.  
 Daniel Stites, M.D.

## STAFF:

Karen Mah-Hing

Dr. Conte summarized the charge to the subcommittee. After this Committee made its recommendations on Acquired Immune Deficiency Syndrome to the Infection Control Committee, other concerns regarding AIDS transmission not previously addressed or resolved were raised. These concerns involved the Pathology Department including the morgue, clinical laboratories and dialysis machines.

Dr. Chinn reviewed the concerns of the Pathology Residents as outlined in their letter dated 5/10/83. (See attached) This summary lists the problems in two areas 1) the tissue processing area and 2) the morgue. Measures have already begun to correct some of these problems. Dr. Chinn stated that in the interim the residents would like to reduce their exposure to AIDS by performing "in situ" autopsies.

Dr. Conte, Ms. Octavio and Ms. Rosendorf toured the Pathology facilities and confirmed the problems with facilities, policies and procedures and housekeeping.

Ad Hoc Advisory Committee on  
Acquired Immune Deficiency Syndrome  
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Dr. Ferrell reported that the following corrections have been made or are under way:

1. A full time person has been approved to control a central specimen receiving area. The Infection Control Practitioners will assist in developing policies and procedures for handling specimens and appropriate cleaning of the tissue processing area.
2. The surgical pathology cutting area will be moved to a location with more space and where proper ventilation can be achieved. This project will begin as soon as the facilities plans are finalized by the pathology faculty and EKG Department moves to its new facilities.
3. The new facilities plan will provide a larger area for bone fracturing and sawing. In this case, the pathology faculty felt that more hand saws would be adequate.
4. A new refrigerator will be purchased to store specimens which are fresh or in fixative. In the new facilities plan, new non-corrosive and narrower shelves will be used to cut down the possibility of specimens being knocked over. New specimens containers with tighter lids will also be purchased.
5. Problems with pedestrian traffic will be somewhat alleviated by the presence of a specimen receiving person and the relocation of a door. Traffic control is being considered in the new plans as well.
6. Ms. West is working with the Housekeeping and Linen Department to improve housekeeping and linen handling. Policies and procedures are also being developed for duties by pathology staff which are not Housekeeping's responsibilities.
7. The equipment issues in the morgue have been resolved.
8. Mr. Yick reported that the hospital engineers and architects are working on the problem of flooding in the morgue and basement in general.
9. Insect control is not as serious a problem here as it is at SFGH.

Dr. Pedersen noted that there are very apparent differences in standards for handling infectious materials in the research laboratories and in the morgue and clinical laboratories of the hospital.

The group discussed the problems of labelling specimens and concluded that materials should be marked as potentially hazardous but not marked with AIDS. Specific procedures for specimen handling will be developed by Pathology in consultation with the Infection Control Unit and all departments submitting specimens to the Pathology Department; i.e. Operating Rooms, Outpatient Surgery and Diagnostic Clinics.



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Dr. Abrams expressed concern that reports made by the Pathology Department on his patients often contained information stating that the patient had AIDS or making reference to the sexual orientation of the patient. This information is added even though it was not included in the history. The Committee agreed that it was not appropriate for this information to be on the reports and asked that this concern be addressed by the Pathology Department.

The Committee discussed "in situ" autopsies versus full autopsies and felt that they could not add any information to assist the Pathology Department in making a decision. However, they would not favor the "in situ" method if quality suffered. In addition important information regarding AIDS could be obtained from autopsies. The Committee felt that it was always important for the clinician and the prosecutor to discuss the needs for pathologic information on an individual basis. If an "in situ" autopsy was agreed upon, it should be noted in the record. Since handling potentially infectious material have always been a risk in performing autopsies, a member expressed concern over the issue of efficiency in performing autopsies at this time and stated that the benefits of a limited autopsy is not known. A question regarding the amount of manipulation being done "in situ" and the potential for exposure was also raised.

Pathology and Hospital Administrative staff are satisfied with the progress to date and commitments to time tables for the future. Dr. Conte asked that this information be summarized and submitted for the Infection Control Committee's record.

#### CLINICAL LABORATORIES

At the request of the Clinical Laboratories faculty, Dr. Conte and Ms. Rosendorf surveyed the Clinical Laboratories's receiving area, chemistry, hematology, blood bank and immunology. They examined behavior and spoke with people regarding the handling of specimens. Overall, there were no major problems with the laboratories. The specimen receiving area was organized and policies and procedures were followed. A few minor deviations were noted 1) there was no uniform gloving policy, 2) one automatic pipetting machine was not fully contained and 3) lab coats were often worn out of the department. Both Ms. Rosendorf and Dr. Conte expressed concern regarding the handling, in the Clinical Immunology Laboratory, of tissue specimens from AIDS patients. Since the "AIDS agent" may be present in high concentration in such tissues, it was recommended that policies and procedures similar to those applicable to the research areas should be developed. The Committee discussed this concept at length and recommended that the Infection Control Unit collaborate with the Biosafety and Infection Control Committee to develop such policies and procedures.

Ad Hoc Advisory Committee on  
Acquired Immune Deficiency Syndrome  
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DIALYSIS UNIT

The members were asked to voice their opinion regarding the use of dialysis machines for AIDS patients. At the present time, these patients are being dialysed on the HBsAg+ machine. Should this practice continue or should a separate machine be set aside for AIDS patients. Can any machine be used? The Committee members were unable to make a recommendation.

The meeting was adjourned at 3:05 p.m.

Prepared by Karen Mah-Hing

Approved by



John E. Conte, Jr., M.D.  
Chairman, Ad Hoc Advisory Committee  
on Acquired Immune Deficiency Syndrome



(Archives, Ward 5A, San  
Francisco General Hospital)

HOSPITALS AND CLINICS

SAN FRANCISCO, CALIFORNIA 94143

Kaposi's Sarcoma Clinic, UCSF  
AIDS Clinic, San Francisco General Hospital

## INFECTION PRECAUTIONS FOR CARETAKERS OF AIDS PATIENTS

So far, no one has identified an organism which is causing AIDS. Most medical experts, however, believe that AIDS is caused by an infectious agent such as a virus. It is assumed, because of the groups of people who have gotten AIDS (gay men, IV drug abusers, hemophiliacs, babies whose mothers use IV drugs, and others), that this infectious agent is spread by blood and by body secretions shared in close, intimate (sexual) contact. These body secretions and excretions may include saliva, urine, feces, semen, mucus, and especially blood.

If you are careful with the above secretions and excretions while caring for an AIDS patient, you should be protected from the possibility of coming in contact with infectious agents, including the assumed organism thought to cause AIDS.

Here are specific precautions to take, which nurses are aware of in caring for any infected patient:

1. Wear disposable gloves when handling any secretions or excretions, especially when handling blood. Avoid direct contact with blood.
2. When your uniforms or clothing is likely to be in contact with secretions/excretions (such as when bathing or cleaning up a patient), wear a gown, lab coat, or smock.
3. Use plastic bags to dispose of soiled tissues, dressings, band aids, soiled gloves. Close and secure the bag tightly when discarding the bag.
4. Wash soiled linens and towels in a washing machine using hot water and detergent, and dry on high in the dryer. Wash dishes and silverware with hot soapy water.
5. Keep bedpans and urinals in a designated place; handle with gloves.
6. Diarrhea and vomitus: Using gloves, clean up patient and linens immediately, rinsing soiled surfaces with soap and water. Put linens which are soiled in a plastic bag until laundered.
7. Wash your hands well with soap and water after removing gloves, and before and after contact with AIDS patients. Use lotion on your hands to keep skin from becoming dry and cracked from frequent washing. If you have open sores on your hands, be especially careful to wear gloves, and wash hands.
8. Keep your hands away from your mouth and face while working. Wash your hands before eating.

AIDS stands for Acquired Immune Deficiency Syndrome. It is a relatively new and alarming disease that is mostly affecting male homosexuals and bisexuals, intravenous drug abusers, hemophiliacs, and Haitian immigrants. AIDS is a malfunction in a person's normal immune system. The cause of this disease has yet to be identified and there is no simple test for AIDS. Currently it is believed that the agent responsible for AIDS may be a virus which is transmitted in a fashion similar to that of hepatitis B. This would mean that it is spread in blood, semen, tears, saliva, and other body fluids.

The immune deficiency of AIDS makes a person susceptible to illnesses that a healthy immune system would fight off. These diseases include cancers (Kaposi's Sarcoma, non-Hodgkins lymphoma) and infections (pneumocystis pneumonia, toxoplasmosis, cytomegalovirus, herpes zoster, and many others).

The syndrome of generalized lymphadenopathy (lymph node swelling) may or may not be related to AIDS and the development of further health problems. Currently, people with lymphadenopathy are evaluated for any infection that may be causing the lymph node swelling. If no infection is found, they are seen every few months and followed for any health problems that may develop. Some people have been followed for years without any problems, while others have developed AIDS. There is no way to predict who will get AIDS.

No one knows what causes AIDS. Recommendations as to how you may reduce your chances of getting AIDS are based on certain theories about how AIDS might be spread. Until more is known about AIDS, it is recommended that you do the following:

1. Get medical insurance if you do not already have it.
2. Reduce or eliminate the number of anonymous sexual contacts that you have. Stay away from public bath houses or any other environment where "no-name" sexual contacts are apt to occur.
3. Reduce or eliminate your use of recreational drugs (including marijuana) and alcohol. Do not use any intravenous drugs.
4. Develop a program of regular physical activity.
5. Make sure you have a well-balanced diet.
6. Get an adequate amount of rest. Try not to run your body down.
7. Develop a close association with a health care provider who can provide ongoing follow-up of your health.
8. Know your body. Learn how to examine yourself for signs that may be suspicious for AIDS.

The symptoms to look for are usually very nonspecific and may be related to simple ailments. However, if these symptoms persist without getting better, see your health provider.

1. Unexplained weight loss
2. Painless rashes or skin discolorations that do not go away
3. Delayed healing of cuts and infections
4. Lymph node swelling that is not related to a known infection
5. Shortness of breath or a dry cough.
6. Persistent low grade fevers and soaking night sweats
7. Extreme fatigue





RECEIVED

1378 THIRD AVENUE  
SAN FRANCISCO, CALIFORNIA 94143  
(415) 666-5793

MAY 16 1983

SCHOOL OF MEDICINE  
DEPARTMENT OF MEDICINE  
Clinical Infectious Diseases and  
Hospital Infection Control Unit

John E. Conte, Jr., M.D., Chief  
Steven L. Barriere, Pharm.D.  
Jacqueline Octavio, R.N.  
Linda L. Rosendorf, M.S.  
Karen Mah-Hing, Administrative  
Coordinator

(Marcus Conant's Kaposi's Sarcoma Notebook,  
1-5/1983, AIDS History Project, UCSF Library).

May 13, 1983

Dear Dr. Conant,

Enclosed are two copies of a Letter to the Editor of JAMA regarding the two articles and editorial in the May 6, 1983 issue. Although I suspect the editors will be inundated with letters from around the country, I think it is important for those of us involved with AIDS patients in the San Francisco community to respond to the conclusions drawn in this issue. A companion letter stressing the psychological and social effects of these conclusions and the reaction of the press is being drawn up and circulated among other members of the AIDS Advisory Committee chaired by Florence Stroud.

*done 5/17/83*

Please read the enclosed letter. If you would like your name to appear on this draft, please return 1 copy to me at the above address and keep one copy for yourself. I expect all replies on or before Friday, May 20. I will be forwarding the letter with all signatures on Monday, May 23. Some of you had a chance to review the letter before this final draft. I am sorry I was unable to circulate it among everyone in the short time allotted.

Sincerely,

*Stent*

Stephen E. Follansbee, M.D.

Enclosure

cc: Donald Abrams, M.D., Arthur Ammann, M.D., Rick Andrews, M.D., Robert Bolan, M.D., David Busch, M.D., Marcus Conant, M.D., John E. Conte, Jr., M.D., George Matula, M.D., Jackie Octavio, R.N., Mark Oscherwitz, M.D., Linda Rosendorf, M.S., Merle Sande, M.D., Helen Scheitinger, R.N., Mervyn Silverman, M.D., Paul Volberding, M.D., Connie Wofsy, M.D.

*File - KS Notebook*



AGENDA FOR CREATING POSITIVE CHANGES IN SEXUALITY

## SECOND NATIONAL AIDS FORUM

(S.E. Follansbee,  
personal correspondence,  
folder #416).

I. RISK REDUCTION GUIDELINES

- A. STATEMENT OF ESSENTIAL INFORMATION VS. ELABORATIONS
- B. CRITIQUE OF CURRENT GUIDELINES
- C. TARGETING INFORMATION FOR SPECIFIC GROUPS

II. STRATEGIES FOR IMPLEMENTATION AND ACCEPTANCE

- A. WIDESPREAD DISTRIBUTION OF PRINTED MATERIAL
- B. TV AND RADIO PRESENTATIONS
- C. TRAINING WORKSHOPS
- D. INNOVATIVE COMMUNITY BASED PROJECTS
- E. OTHER STRATEGIES

III. RESTRUCTURING OF COMMUNITY TOWARDS POSITIVE CHANGES IN SEXUALITY

- A. ENVIRONMENTAL CHANGE
- B. SOCIAL CHANGE

Follansbee  
f #421  
[Denver, 6/83]

(S.E. Follansbee, personal  
correspondence, folder #421).

THE PHYSICIAN IN THE AIDS CRISIS

Risk Reduction Factors  
Stephen E. Follansbee, M.D.  
Saturday, June 25, 1983

I. The scope of RISK REDUCTION includes:

1. Patients with AIDS.... at risk for opportunistic infections.
2. Gay male patients.... at risk for AIDS.
3. Health care workers and friends and family.... at presumed risk for AIDS.

II. RISK REDUCTION cannot be defined until the RISKS are elaborated. To date, the risk of acquiring AIDS has been associated with:

1. Drug abuse-most notably needle-associated.
2. Male homosexual activity, in particular
  - a. passive rectal sex
  - b. multiple partners
  - c. anonymity
  - d. oral-anal sex (anilingus)
3. Transfusion of blood and blood products.
4. Geographical background.

## Risk Reduction Factors (2)

III. The DATA are scant and based on the model of Hepatitis B virus transmission, association of other sexually-transmitted disease, and clinical suspicion. For some of the difficulty arising during the tabulation of data on sexual practices, see the article by Willcox (1980).

1. Hepatitis B.
2. Hepatitis A.
3. Intestinal parasites
4. Gonorrhea and syphilis
5. AIDS

## IV. Recommendations for Risk Reduction

1. To patients with AIDS.
  - a. Other acquired infections may be immunosuppressive (e.g., syphilis, intestinal parasites.)
  - b. Guidelines for "protective isolation."
2. To gay male patients worried about AIDS (see BAPHR's "Guidelines for AIDS Risk Reduction.")
  - a. Guidelines regarding sexual practices designed to avoid the direct exchange of bodily fluids but encourage intimacy and healthy sexual practices.

### Risk Reduction Factors (3)

- b. Guidelines regarding other activities of healthy living.
  
  3. To the health care workers and family and friends in contact with the AIDS patient (see U.C.S.F. Infection Control Committee Patient Care Guidelines on AIDS, May 4, 1983.)
- V. The CHALLENGE: Our focus with gay patients tends to remain on guidelines for healthy sexual activity. It has recently been stated that "the education of gay men to limit the nature and numbers of their sexual partners is unlikely to be productive on a large scale" (Handsfield, 1981.) This is in part because many of the proposed changes may go against current subculture sexual customs and threaten the sense of intimacy within the gay community. Recommendations for risk reduction need to acknowledge the fear of AIDS and other sexually-transmitted diseases and at the same time reinforce the concept of fun and healthy sexual activities.

## Risk Reduction Factors (4)

Bibliography

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(S.E. Follansbee, personal correspondence, folder #416).

RISK REDUCTION GUIDELINES

SLIDE ONE:  
MICROBIOLOGY

IT IS SOBERING TO ASSESS THE CURRENT STATE OF KNOWLEDGE REGARDING AIDS. WE HAVE NO AGENT: WE HAVE NO TEST FOR THAT AGENT. THE INDUCTION PERIOD (OR INCUBATION PERIOD IF YOU BELIEVE THE CAUSE IS INFECTIOUS) NOW DISCUSSED BY C.D.C. EXTENDS FROM FIVE MONTHS TO TWO YEARS OR MORE. THAT MEANS THAT IF THE TRANSMISSION OF THIS AGENT IS STOPPED TO DAY, WE WILL CONTINUE TO SEE PERSONS PRESENT WITH ~~THIS ILLNESS~~ <sup>AIDS</sup> WELL INTO 1985. ALREADY AIDS IS CONSIDERED THE LEADING CAUSE OF DEATH IN GAY MEN AGES 21 to 50 IN S.F. AND NEW YORK CITY. IF WE DELAY A YEAR, CASES WILL PRESUMABLY BE EXTENDED A YEAR. DESPITE OUR EXTREME LACK OF KNOWLEDGE, WE ARE BEING CHALLENGED TO ASSESS WHAT PRELIMINARY DATA WE DO HAVE, AND FORULATE GUIDELINES TO MINIMIZE THE RISK OF ACQUIRING AIDS.

SLIDE TWO  
POSSIBLE CAUSES

THE TASK IS TREMENDOUS. THE "SEXUAL MORES" WORKSHOP OF THE SECOND NATIONAL AID FORUM HELD IN DENVER JUST TWO WEEKS AGO BROUGHT HOME TO ME HOW DIFFICULT THIS IS--A MESSAGE I SOMEHOW DID NOT GET FULLY WHILE WORKING THREE AND FOUR MONTHS AGO, WITH OTHER MEMBERS OF THE SCIENTIFIC AFFAIRS COMMITTEE OF BAPHR ) PRODUCING THE GUIDELINES INCLUDED IN YOUR SYLLABUS.

WHAT ARE THE HURDLES WE NEED TO TAKE INTO ACCOUNT WHILE DISCUSSING RISK REDUCTI

1. AS PHYSICIANS WE MAKE LOUSY EPIDEMIOLOGISTS AND APPEAR TOO EASILY CONVINCED BY PRELIMINARY DATA. WE HAVE USED THIS EARLY INFORMATION REGARDING ASSOCIATION OF CERTAIN SEXUAL PRACTICES WITH RISK OF DISEASE TO REINFORCE OUR OWN SEXUAL BIASES AND THEORIES OF "DISEASED." OUR REPRESENTATIVES IN THE A.M.A. HAVE ALLOWED SHODDY DATA IMPLICATING CASUAL CONTACT TO BE PUBLISHED WITHOUT CRITICAL REVIEW, IN FACT REINFORCED IN ITS EDITORIAL COMMENT.

2. AS PHYSICIANS WE MAKE LOUSY SOCIAL SCIENTISTS. OUR RECOMMENDATIONS HAVE BEEN ACCUSED OF THREATENING THE SOCIAL STRUCTURE OF THE GAY COMMUNITY.

SLIDE THREE  
ISOLATION SIGN

ISK REDUCTION (2)

OUR STATEMENTS HAVE BEEN ACCUSED OF SPEARHEADING THE MOVEMENT OF ~~MEMBERS OF~~ <sup>of persons with</sup> ~~OUR COMMUNITY WITH AIDS~~ <sup>as</sup> AS WELL AS THOSE WHO ARE PERCEIVED AT BEING

AT RISK FOR AIDS INTO NOT ONLY SEXUAL, BUT ALSO SOCIAL ISOLATION. <sup>FRINDS STAFF</sup> <sup>visit them persons who are, throw them out of their hands, deliver food on paper plates left on</sup>

SIDE 4  
MECHANISM

(3) AS PHYSICIANS WE MAKE LOUSY SEX COUNSELORS. IF WE ARE ASKED "WELL, <sup>the danger is that sex can be</sup> WHAT CAN I DO?", WE APPEAR BEFUDDLED. <sup>comparing it as easily as</sup> OUR LITANY OF PROHIBITIONS BEGIN

TO EXTEND UP THE SLEAVES OF OUR SAFE WHITE COATS. WE SEEM TO ~~PASS OFF~~ <sup>DISTRIBUTE</sup> THE MESSAGE <sup>only that</sup> THAT SEX IS DANGEROUS. IT BECOMES ALMOST RELIGIOUS TO SOME AS WE APPEAR TO SAY "WELL IT IS OKAY TO BE GAY AS LONG AS YOU DO NOT HAVE SEX." WE PLAY ON GUILT BY TALKING ABOUT "TOO MANY DIFFERENT PARTNERS"

BUT ARE SURPRISED WHEN PATIENTS ASK "HOW MANY PARTNERS WOULD BE SAFE?" <sup>our terminology has moral overtones as we describe "victims" "contamination" "play"</sup>

(4) AS PHYSICIANS WE OFTEN CANNOT ACKNOWLEDGE OUR OWN PERSONAL LIMITS AND ANXIETIES. PERSONAL FEARS ABOUT ACQUIRING THE DISEASE LEAD US TO THE BRINK OF HYSTERIA. TO THE POINT WHERE WE FURTIVELY RUN CMV ANTIBODIES ON OUR SELVES, KNOW OUR OWN HELPER-TO-SUPPRESSOR CELL RATIOS, BUT THEN poo-poo PATIENT REQUESTS FOR THESE TESTS AS SIMPLY HYSTERICAL OR NOT-COST-EFFECTIVE IN THE ABSENCE OF DISEASE. SOME PHYSICIANS REFUSE TO SEE AIDS PATIENTS, AND THOSE OF US WHO DO, <sup>are unable/unwilling</sup> REFUSE TO CONFRONT THOSE WHO DO NOT.

(5) AS PHYSICIANS WE HAVE APPEARED "HOMOPHOBIC" TO OUR BROTHERS AND SISTERS AS WE ATTEMPT TO PREACH "THE SCIENCE" OF THE DISEASE WHILE DISTANCING OURSELVE FROM THE POLITI CAL, SOCIAL, AND PSYCHOLOGIC ISSUES AND RAMIFICATIONS OF OUR ACTIONS AND STATEMENTS. OUR GUIDELINES, WHEN THE FINALLY APPEAR, ARE OFTEN "TOO TECHNICAL" AND EASILY MISINTERPRETED, AND MISUSED, SOMETIMES INNOCENTLY, OTHERTIMES BY <sup>FRINDS</sup> ~~FOCES~~ THAT WOULD MAKE AIDS A "GAY DISEASE," OR A "DRUG-RELATED DISEASE", OR A BLACK DISEASE, OR EVEN A SWINE DISEASE. WE PLAY INTO THE HANDS OF THOSE WHO WOULD USE THE LAW TO KEEP

## RISK REDUCTION GUIDELINES (3)

us "IN OUR PLACE" AND PRESUMABLY OUT OF SUCH PLACES AS RESTAURANTS, BANK BUILDINGS, THE POLICE FORCE, AND EVEN THE HEALTH CARE PROFESSION, ALL UNDER THE GUISE OF "RISK REDUCTION." *Police officers Association guidelines for SF*

I HEARD ALL THESE ISSUES AND MORE IN THE MEETING ROOM IN DENVER. 125 OF US WERE FILLED WITH OUR OWN NAIVETE, FEARS, AND HIDDEN AGENDAS AS WE DISCUSSED, LECTURED, AND EVEN SHOUTED DURING THOSE FIVE <sup>H</sup>OURS. A THIN VENEER OF SCIENTIFIC OBJECTIVITY WAS QUICKLY WIPED AWAY IN THAT MEETING. THE LEADERS DID A TREMENDOUS JOB TRYING TO FORM ORDER OUT OF CHAOS AND THE WORK DONE IN THAT ROOM BY EVERYONE WAS EQUALLY IMPRESSIVE.

Yet, THE FACT IS THAT GROUP COULD COME UP WITH LITTLE CONCRETE RECOMMENDATIONS EITHER FOR SEXUAL GUIDELINES FOR RISK REDUCTION, HOW TO DEVISE THEM, HOW TO DISTRIBUTE THEM, HOW TO IMPLEMENT THEM, HOW TO JUDGE THEIR EFFECTIVENESS.

I CAN TELL YOU THAT I DO NOT PLAN TO TAKE THE REMAINING <sup>25</sup> @% MINUTES TO PROPOSE, IMPOSE, OR EVEN SUGGEST MY OWN GUIDELINES. RATHER, MY GOALS ARE HOPEFULLY LESS AMBITIOUS. HAVING SUGGESTED SOME OF THE PITFALLS, I WANT TO LAY OUT THE SCOPE OF THE PROBLEM -- TO WHOM OUR COMMENTS NEED TO BE ADDRESSED, TO DEFINE THE MODEL OF TRANSMISSION OUT OF THE SCANT AMOUNT OF DATA THAT EXIST, AND TO OFFER THE B.A.P.H.R. GUIDELINES AS A STARTING POINT FOR THE DEVELOPMENT OF YOUR OWN GUIDELINES APPLICABLE TO YOUR OWN PATIENT POPULATION AND COMMUNITY.

## THE SCOPE

THE SCOPE OF RISK REDUCTION IS BROAD. TO MOST OF US IT INCLUDES RISK REDUCTION THROUGH MODIFICATION OF SEXUAL PRACTICES. THIS IS NOT SURPRISING GIVEN THE EARLY ASSOCIATION OF AIDS WITH MALE HOMOSEXUAL ACTIVITY, THE "FAST LANE" LIFESTYLE MANY OF THE EARLIEST PATIENTS CLAIMED, AND THEIR HISTORY OF MULTIPLE EPISODES OF OTHER SEXUALLY TRANSMITTED DISEASES. *As well as common STDs with gay men* HOWEVER, IF THE B.A.P.H.R.

## RISK REDUCTION (4)

SLIDE 5,  
 Many  
 symptoms.

GUIDELINES SUFFER FROM ONE MAJOR FLAW (AND THERE MAY BE MORE THAN ONE), IT IS THAT "RISK REDUCTION" WAS USED IN TOO NARROW A SENSE. THE TITLE DID NOT SPECIFICALLY LIMIT THE INTENT OF THE MESSAGE TO SEXUAL ACTIVITY. CERTAINLY PERSONS WITH AIDS AND THE "WORRIED WELL" GAY MAN ARE CONCERNED ABOUT SEXUAL TRANSMISSION. HOWEVER, NO LESS FEARFUL ARE OTHER MEMBERS OF OUR MORE GENERAL COMMUNITY--MANY OF WHOM ARE, SHALL WE SAY, "NOT EXACTLY THRILLED WITH OUR PRESENCE." THESE INCLUDE HEALTH CARE WORKERS WHO ARE NOW CONCERNED AND ARE EVEN BEING TOLD BY SOME OF US THAT "THEY ARE MEMBERS OF THE NEXT HIGH RISK GROUP" TO BE IDENTIFIED, BY VIRTUE OF <sup>direct</sup> CONTACT WITH AIDS PATIENTS IN HOSPITALS, CLINICS, AND OFFICES, AS WELL AS <sup>person</sup> THEIR TISSUE AND FLUID SPECIMENS IN THE LABORATORY. THE WORRIED INCLUDE CO-WORKERS OF PERSONS WITH AIDS IN BANKS, RESTUARANTS, AND EVEN GAS STATIONS, WHERE A GAY EMPLOYEE MAY BE ASKED NOT TO RIDE THE ELEVATOR, PREPARE THE FOOD, OR SHARE THE EMPLOYEE BATHROOM. FAMILY MEMBERS, ROOMMATES, AND LOVERS ARE CONCERNED ABOUT THEIR RISK WHEN PERSONS WITH AIDS RETURN HOME FROM THE HOSPITAL.

THE MESSAGE IS INDEED CLEAR--WHATEVER WE SAY MUST BE CLEAR, ITS INTENT TO DEFINE RISKS AS BEST WE CAN MUST BE CLEARLY UNDERSTOOD, THE SITUATIONS WE ARE ADDRESSING MUST BE CLEAR AND THEIR SCOPE WELL-DEFINED, AND THEY MUST BE CONSISTENT. WE MUST NOT ALLOW ONE SET OF GUIDELINES TO BE OKAY FOR ONE SITUATION (SAY THE HOSPITAL) AND FAIL TO UNDERSTAND THE IMPLICATIONS OF IT FOR THE HOME. WE MUST BE WILLING TO IMPLEMENT THESE GUIDELINES IN OUR OWN LIVES. IF WE SAY "SWEAT IS OKAY," WE MUST BE WILLING TO TOUCH OUR PATIENTS WITHOUT GLOVES. IF WE SAY AIDS IS NOT TRANSMITTED BY THE RESPIRATORY OR DROPLET ROUTE, WE MUST BE WILLING TO SHED OUR MASKS AND BREATHE THE SAME AIR. IF WE SAY THEY CAN PREPARE FOOD, WE MUST SHARE THAT FOOD WITH THEM.



RISK REDUCTION (6)

ON RETROSPECTIVE SUMMARIES OF SEXUAL PRACTICES THROUGH INTERVIEWS CONDUCTED IN A VARIETY OF FORMATS. THE VALIDITY OF MUCH OF THE DATA MUST BE CONSIDERED AGAIN PRELIMINARY, BUT THE BEST WE HAVE.

HEPATITIS B

*SCUD 4  
SIX  
WpB.*

THE CONVERSATIONS STARTS WITH HEPATITIS B--IN PART BECAUSE HEPATITIS VIRUS IS USED AS A MODEL--NOT THE AGENT OF DISEASE--BUT MODEL FOR AIDS TRANSMISSION SINCE THE HIGH RISK GROUPS IDENTIFIED ABOVE ARE ALSO HISTORICALLY AT HIGH RISK FOR HEPATITIS B. A LARGE STUDY PUBLISHED UNDER THE AUSPICES OF CDC (SCHREEDER, J.I.D.) EXAMINED 3816 gay men in five s.t.d. CLINICS IN U.S.A. SEROPOSITIVITY FOR HEPATITIS B VARIED TO A HIGH OF 75.8% IN THE S.F. COHORT. THE MEAN NUMBER OF NONSTEADY SEXUAL PARTNERS OVER 4 MONTH RETROSPECTIVE INTERVIEW INTERVAL WAS 35.9 (AGAIN THE HIGHEST WAS RECORDED IN S.F.) SEROPOSITIVITY WAS MOST STRONGLY RELATED TO DURATION OF REGULAR HOMOSEXUAL ACTIVITY (AND DURATION OF ALL HOMOSEXUAL ACTIVITY) AS WELL AS THE NUMBER OF NON-STEADY SEXUAL PARTNERS. CERTAIN SPECIFIC PRACTICES WERE ASSOCIATED WITH AN INCREASED CHANCE OF SEROPOSITIVITY.

1. PASSIVE ANAL-GENITAL WITH NONSTEADY PARTNER WAS MOST LIKELY TO BE ASSOCIATED WITH POSITIVE SEROLOGY BY LINEAR LOGISTIC REGRESSION ANALYSIS.

2. INTERACTION OF ACTIVE ANAL-GENITAL AND ACTIVE ORAL-ANAL INTERCOURSE WITH NONSTEADY PARTNERS WAS ALSO HIGH.

3. RECTAL DOUCHING INDEPENDENT OF ITS ASSOCIATION WITH ANAL-GENITAL AND ORAL ANAL CONTACT, *was a risk for the A seropositivity (serology)*

PRACTICES NON-ASSOCIATED INCLUDE PASSIVE ORAL-GENITAL, ACTIVE ORAL-GENITAL AND ORAL-ORAL IF DURATION OF ALL HOMOSEXUAL ACTIVITY AND NUMBER OF NON-STEADY PARTNERS WERE ACCOUNTED FOR. OBVIOUSLY DATA

*Supp. from  
WpB.*



## RISK REDUCTION (7)

BASED ON RETROSPECTIVE ANALYSIS OF SEXUAL ACTIVITY OVER ONLY A FOUR MONTH PERIOD OF time NEED TO BE INTERPRETED SOMEHWAT CAUTIOUSLY. HOWEVER, SIMILAR CONCLUSIONS HAVE BEEN REPORTED IN TWO OTHER ARTICLES. <sup>(1)</sup> THE FIRST BY SZMUSNESS ET AL IN THE ANNALS OF INTERNAL MEDICINE, 1975, IN FACT SUGGESTED THAT THE SEROPOSITIVITY RATE IN HOMOSEXUAL MEN WHOSE SEXUAL PRACTICES EXCLUDED ANAL-GENITAL ACTIVITY APPROACHED THE LEVEL IN THE HETEROSEXUAL POPULATION DRAWN FROM AN S.T.D. CLINIC. <sup>(2)</sup> ALTHOUGH THE ARTICLE BY COREY AND HOLMES IN N.E.J.M., 1980 dealt WITH HEPATITIS A PRIMARILY, AND NO ACTUAL DATA ON HEPATITIS B WAS REPORTED, IN THEIR DISCUSSION IT WAS POINTED OUT THAT IN THEIR POPULATION STUDIED PROSPECTIVELY, THE ACQUISITION OF HEPATITIS B (10 cases in 102 men over <sup>6.1</sup> ~~12~~ months) was correlated with frequency of passive anal-GENITAL INTERCOURSE AND WAS NOT CORRELATED WITH ORAL ANAL INTERCOURSE, AS CONCLUDED BY THE C.D.C. STUDY.

A WORD ABOUT KISSING... KISSING AND THE SITUATIONS WHERE SALIVA IS POTENTIALLY TRANSMITTED OR EXCHANGED REMAINS CONTROVERSIAL. HEPATITIS B SURFACE ANTIGEN CAN BE FOUND IN HIGH TITER IN UP TO 76.% OF PATIENTS EARLY IN THE COURSE OF ACUTE HEPATITIS B INFECTION AND INTERMITTENTLY IN OVER 85% OF CHRONIC HEP B SURFACE ANTIGEN CARRIERS. HOWEVER, TO MY KNOWLEDGE KISSING HAS NEVER BEEN IMPLICATED IN THE TRANSMISSION OF HEPATITIS B.

A WORD OF CAUTION... IN THAT IN MOST STUDIES SUFFER BECAUSE FEW SEXUALLY ACTIVE PERSONS DO NOT KISS, SO THE BETA-ERROR IS HIGH. THERE HAVE BEEN A NUMBER OF REPORTS OF INSTANCES WHERE INDIVIDUALS INCUBATING HEPATITIS B HAVE SHARED RESUSA-ANNIE WITH OTHER C.P.R. STUDENTS AND NOT PASSED THE INFECTION TO THEM--AGAIN SUGGESTIVE THAT SALIVA IS NOT A PARTICULARLY HIGHLY INFECTIOUS VEHICLE FOR THIS AGENT.

SO MUCH FOR HEPATITIS B. ~~REMEMBER WHY WE FOLLOW THE HEPATITIS B MODEL.~~

↳ ALSO REMEMBER THAT HEPATITIS B WAS NOT AN INDEPENDENT RISK FACTOR FOR THE ACQUISITION OF AIDS IN THE EARLY C.D.C. STUDY SO TOO STRICT AN ADHERENCE TO

## RISK REDUCTION (8)

*SLIDE 7  
MPT*

~~THIS MODEL~~ MAY NOT BE ENTIRELY APPROPRIATE. HEPATITIS A ANTIBODIES WERE HOWEVER, ASSOCIATED WITH AN INCREASED RISK OF AIDS IN THAT C.D.C. CASE CONTROL STUDY. WHAT ABOUT THE ASSOCIATION OF HEPATITIS A AND VARIOUS SEXUAL PRACTICES? FROM DIARIES KEPT BY <sup>67</sup>~~102~~ GAY MEN STUDIED PROSPECTIVELY BY COREY AND HOLMES FOR CLINICAL AND SEROLOGIC EVIDENCE OF ACQUISITION OF HEPATITIS A, <sup>was seen of b.i. number</sup> ACTIVE ORAL ANAL CONTACT WAS THE HIGHEST RISK FACTOR, ALTHOUGH IT WAS ALSO THE LEAST COMMON SEXUAL ACTIVITY RECORDED BY THEIR SUBJECTS. OTHER ACTIVITIES SUCH AS RECTAL INTERCOURSE (WHETHER ACTIVE OR PASSIVE), ORAL GENITAL ACTIVITY AND KISSING WERE NOT ASSOCIATED WITH AN INCREASED RISK OF ACQUISITION OF HEPATITIS A. IN ADDITION, AS FOR HEPATITIS B, ACQUISITION WAS ALSO ASSOCIATED WITH A HIGHER NUMBER OF SEXUAL PARTNERS (68 versus 47; p less than 0.05). OBVIOUSLY HEPATITIS A OUTBREAKS HAVE BEEN ASSOCIATED WITH FOOD HANDLERS AND FECAL CONTAMINATION OF FOOD. IN ADDITION, URINE CONTAMINATION HAS BEEN IMPLICATED EPIDEMIOLOGICALLY ALTHOUGH HEPATITIS A IN URINE HAS NOT BEEN DEMONSTRATED. M

*SLIDE 4  
CMV*

THAT CMV IS ASSOCIATED WITH GAY MALE SEXUAL ACTIVITY IS WELL RECOGNIZED. I WILL NOT BE RE-EMPHASIZING THAT. THE FACT THAT THIS VIRUS REACHES HIGHEST TITRES IN SEMINAL FLUID AND CAN BE FOUND IN THE URINE OF 13% OF GAY MEN IN A STD CLINIC MAY HAVE IMPLICATIONS FOR TRANSMISSIBILITY. DESPITE THE UBIQUITIOUSNESS OF THIS VIRUS, THE OCCASIONAL AIDS PATIENT WITHOUT EVIDENCE FOR THIS AGENT'S SUGGESTS IT IS NOT A SINE QUA NON OR THE CAUSE OF AIDS. INTERESTINGLY, THERE IS NO PUBLISHED EVIDENCE ON THE RISKS FOR ACQUISITION OF CMV <sup>with respect to</sup> AND PARTICULAR SEXUAL PRACTICES. DATA IN PRESS BY LARRY DREW ET AL AT MT. ZION MEDICAL CENTER IN S.F. (annals of internal medicine, september 1983) WILL RECTIFY THAT IN AT LEAST A PRELIMINARY WAY. THEY WERE ABLE TO IDENTIFY 87 MEN FROM THE LARGE SCREENING OF SEVERAL THOUSAND IN THE MID SEVENTIES FOR THE HEPATITIS B VACCINE TRIAL WHO WERE CMV SERONEGATIVE

RISK REDUCTION (9)

AND SUBSEQUENTLY SEROCONVERTED. THE HISTORY OF SEXUAL PRACTICES WAS THEN OBTAINED RETROSPECTIVELY ON THIS RELATIVELY SMALL COHORT OF MEN. NO ASSOCIATION WITH ANY ACTIVITY OTHER THAN PASSIVE ANAL-GENITAL INTERCOURSE COULD BE ASSOCIATED WITH SEROCONVERSION (98% in participants, 74% in non-participants).

Activities such as KISSING AND SURPRISINGLY PASSIVE ORAL GENITAL INTERCOURSE WERE <sup>not</sup> FOUND TO BE STATISTICALLY ASSOCIATED WITH INCREASED RISK. <sup>(enriches)</sup> ALTHOUGH

I HAVE NOT SEEN THE STUDY, THE SMALL NUMBERS OF MEN AND RETROSPECTIVE NATURE OF THIS STUDY, SUGGEST THE DATA ARE PRELIMINARY. AT LEAST ONE PROMINENT SET OF GUIDELINES FOR ~~SEXUAL GUIDELINES FOR RISK REDUCTION~~ (NEWS FROM THE FRONT--  
HOW TO HAVE SEX IN AN EPIDEMIC: ONE APPROACH) RELIES HEAVILY ON THE CMV-

MODEL FOR AIDS TRANSMISSION. <sup>They emphasize far as to suggest say may be true for CMV (by anal/oral transmission) to know if they + their partner are safe.</sup>

Study 9  
Epid. intestinal diseases

INTESTINAL PARASITES ARE MENTIONED AGAIN BECAUSE OF THE WILLINGNESS EARLY ON TO ATTRIBUTE AIDS TO 1. PARASITES, 2. THE DRUGS THAT TREAT PARASITES, AND 3. VIRUSES OR SOME TOXIN ASSOCIATED WITH PARASITES. AGAIN THE LITERATURE HAS ASSOCIATED PREVALENCE OF ENTERIC PROTOZOAL INFECTION WITH

1. MALE HOMOSEXUAL ACTIVITY (highest correlate)

2. ORAL-ANAL SEX (analingus). <sup>A</sup> Although infection

Study 10  
intestinal parasites

RATES IN SOME STUDIES DID NOT APPEAR TO INCREASE WITH INCREASING NUMBER OF PARTNERS, IN OTHERS THIS APPEARS TO BE AN IMPORTANT FACTOR. AGAIN, CASUAL CONTACT SUCH AS LIVING IN THE HOUSEHOLD HAS NOT BEEN ASSOCIATED WITH INCREASED TRANSMISSION OF THESE ORGANISMS IN THE U.S.A.,

Study 11  
Syphilis + # sex partners

I WILL NOT SPEAK MUCH TO THE OTHERS LISTED EXCEPT TO REMIND AGAIN THE ASSOCIATION OF AIDS WITH SYPHILIS. ~~AGAIN~~, THIS APPEARS TO BE A MARKER FOR THE DURATION OF HOMOSEXUAL ACTIVITY AND NUMBER OF DIFFERENT SEXUAL PARTNERS AND NOT THAT SYPHILIS IS IMPLICATED IN AIDS OR FOLLOWS A SIMILAR MODE OF TRANSMISSION. NEITHER SYPHILIS NOR GONORRHEA NOR HERPES ARE TRANSMITTED

Am. J. Publ. Health

## RISK REDUCTION GUIDELINES (10)

via casual CONTACT OR FOMITES. THIS IS EVEN TRUE FOR HERPES, WHICH SOME RESEARCHERS AT U.C.L.A. HAVE FOUND CAN REMAIN VIABLE ON TOILET SEATS. NONE-THE-LESS, THIS IS NOT A RISK FACTOR FOR ACQUISITION OF THIS DISEASE SINCE TOILET SEATS HAVE NEVER BEEN DIRECTLY IMPLICATED. THEREFORE GUIDELINES SUCH AS THOSE PUT OUT BY THE PRESIDENT OF THE NY P.H.R. WHICH SUGGEST THAT VOLUNTEERS FOR THE GAY MENS HEALTH CRISIS UNIT NOT SHARE TOILET SEATS WITH CLIENTS OR <sup>OR</sup> AT LEAST CLEAN THE SEAT WITH SOAP AND WATER BEFORE USE ARE, IN MY ESTIMATION, INAPPROPRIATE AND INFLAMMATORY.

*summary: + six partners + a q. sexually transmitted disease*

IF YOU THINK THE INFORMATION DISCUSSED SO FAR IS INCOMPLETELY DOCUMENTED ON DISEASES THAT HAVE BEEN RECOGNIZED FOR TEN YEARS AS HYPERENDEMIC OR EPIDEMIC IN THE MALE GAY COMMUNITY, WAIT UNTIL YOU HEAR THE INFORMATION ON SEXUAL PRACTICES AND AIDS OR IMMUNODEFICIENCY.

THE TWO STUDIES THAT HAVE DRAWN THE MOST ATTENTION ARE THE EARLY CASE CONTROL STUDY OF AIDS PATIENTS, PERFORMED BY THE C.D.C. AND SLOWLY LEAKED OUT OVER THE LAST SEVERAL MONTHS AND MICHAEL MARMOR'S STUDY ON K.S. PATIENTS IN NEW YORK, RECENTLY REPORTED DURING THE MARCH MEETINGS IN NYC.

IN OCTOBER 1981, CDC INTERVIEWED 80% OF ALL AIDS PATIENTS IDENTIFIED TO THAT DATE, WHO WERE ALIVE AND NOT TOO ILL TO RESPOND. THE NUMBER OF RESPONDS WAS 50. I WILL NOT GO THROUGH ALL THE ATTEMPTED CONTROL GROUPS. THE TWO COMPARISON GROUPS REPORTED WERE "OVERMATCHED" SEXUALLY ACTIVE MEN SELECTED THROUGH AN STD CLINIC AND ~~IN PRIVATE PRACTICE, IN MASSACHUSETTS.~~ PRIVATE PRACTICE. THE ONLY STATISTICALLY SIGNIFICANT FACTORS BETWEEN PERSONS WITH AIDS AND THE CONTROL GROUPS WERE A HISTORY OF SYPHILIS (68% vs 36 vs 36%), larger NUMBER OF SEXUAL PARTNERS PER YEAR (61 vs 27 vs 25), AND A HISTORY OF NON-B

*slide 12  
all 1 dx  
+ soap  
partners*



RISK REDUCTION GUIDELINES (11)

HEPATITIS. THE DIFFERENCES BETWEEN GROUPS <sup>with</sup> RIMMING (ORAL-ANAL, ACTIVE) WAS ~~XXXXXXXXXXXX~~ 78 vs 64 vs 62% and fisting was 52 vs 33 vs 38%. MANY OF US THEN INTERPRETED THESE TO BE "RISKY" ACTIVITIES FOR AIDS ACQUISITION. I SUSPECT THAT THESE VARIABLES SUGGEST NOT THAT AIDS IS TRANSMITTED THROUGH <sup>v</sup> SYPHILIT SORES, OR EVEN NECESSARILY THROUGH RIMMING, BUT OFFER CONFIRMATION ABOUT THE INCREASED NUMBER OF SEXUAL PARTNERS. THIS STUDY IS PRELIMINARY, NOW DATED, AND RETROSPECTIVE. TO MY KNOWLEDGE IT HAS NOT BEEN REPEATED, ALTHOUGH NUMEROUS INVESTIATORS ARE ATTEMPTING TO LEARN FROM THESE EARLY MISTAKE AND PLAN ADDITIONAL STUDIES.

MICHAEL MARMORS STUDY WAS DONE WITH K.S. PATIENTS ALONE. CASES WERE TWENTY OF THE FIRST TWENTY ONE MEN WITH BIOPSY CONFIRMED K.S. AT N.Y.U. MED CENTER THROUGH AUGUST, 1981. THE CONTROLS WERE TWO HOMOSEXUAL MEN, MATCHED in addition for raace and AGE, FROM A MANHATTEN PRIVATE PRACTICE. THE KS PATIENTS WERE INTERVIEWED IN PERSON, THE CONTROLS BY MAIL.

SLIDE 13  
ES F sucking.

ALL SEXUAL PRACTICES WERE RETROSPECTIVELY RECORDED. THE RISK OF ACQUIRING K.S. APPARENTLY INCREASED WITH THE PASSIVE ROLE IN ORAL-GENITAL INTERCOURSE, AND INCREASED FURTHER IF THE PARTNER EJACULATED, AND EVEN FURTHER IF THE EJACULATE WAS SWALLOWED. IN GENERAL, ANAL-GENITAL INTERCOURSE SHOWED GREATER RISK, INCREASING WITH INCREASED EXPOSURE TO SEMEN.

SLIDE 14  
KS + fisting

ACTIVE ORAL-ANAL CONTACT AND THE NUMBER OF SEX PARTNERS (a month per year) before DISEASe ALSO WAS ASSOCIATED WITH INCREASED RELATIVE RISK. HOWEVER, THE NUMBER OF DIFFERENT SEX PARTNERS PER MONTH WITH WHOM SUBJECTS REPORTED THEY HAD PASSIVE ANAL GENITAL INTERCOURSE WITH

SLIDE 15  
K or fisting

EJACULATION OF THE ACTIVE PARTNER WAS ANALYZED WITH A MULTIPLE REGRESSION MODEL, AND ALL OTHER VARIABLES DROPPED OUT. THIS OCCURRED IRRESPECTIVE OF EJACULATION OF THE ACTIVE PARTNER. THE STUDY ALSO RAISED AGain THE SPECTRE OF AMYL NITRIE USE WHICH APPEARED INDEPENDENTLY SIGNIFICANT (although life time use of all inhalants or butyl nitrite was not significant.)



RISK REDUCTION (12)

IF WE ACCEPT THE DATA AT FACE VALUE, PASSIVE ANAL GENITAL INTERCOURSE WITH INCREASED NUMBER OF PARTNERS IS RISKY FOR ACQUISITION OF AIDS/KS.

AGAIN, THE DATA WHILE OFFERING SOME SUPPORT OF THE HEPATITIS B MODEL OF TRANSMISSION, MUST BE CONSIDERED PRELIMINARY SINCE

1. does not distinguish exposure to "agent" from exposure to semen
2. was retrospective (covering sexual practices during the "short end of the induction period" and persons probably more likely to remember activity one week ago than one year ago
3. and was carried out differently for the two groups.

ONE ADDITIONAL STUDY REPORTED IN THE LANCET, MARCH 19, 1983 SHOULD BE ADDRESSED BRIEFLY.. HERE LYMPHOCYTE FUNCTION OF 89 YOUNG GAY MEN WERE STUDIED. 27% (25) WERE FOUND TO HAVE HELPER TO SUPPRESSOR CELL RATIOS OF under 0.8, an ABNORMALITY THEY TERMED "IMMUNE AUGMENTATION" SINCE THE REVERSAL WAS ATTRIBUTED TO A MILD FALL IN THE TOTAL HELPER CELL POPULATION BUT A RISE IN THE SUPPRESSOR CELL POPULATION. THE GREATEST CORRELATION BETWEEN THIS PHENOMENON AND A SEXUAL PRACTICE WAS FOUND WITH PASSIVE ANAL-GENITAL INTERCOURSE. EVEN CONTROLLED FOR NUMBER OF SEXUAL PARTNERS PER MONTH AND DURATION OF HOMOSEXUAL ACTIVITY, THIS PRACTICE WAS ALLEGEDLY FOUND TO BE SIGNIFICANT FOR INCREASED RISK OF THIS LABORATORY PHENOMENON, <sup>in "cryptorchidism"</sup> <sup>which</sup> WELL, I HAVE TRIED TO GIVE THE SCIENTIFIC BACKGROUND UPON ~~EACH~~ ANY SPECIFIC SEXUAL PRACTICE RECOMMENDATIONS WILL NEED TO BE BASED. OBVIOUSLY, THE REACTION CAN BE TO IGNORE THE CURRENT INFORMATION AND MAKE NO SUGGESTIONS, TO IGNORE THE CURRENT INFORMATION AND RECOMMEND STRICT ISOLATION OF EVERYONE, OR TO ATTEMPT SOME RATIONAL SYNTHESIS OF THOSE POSITIONS, ACKNOWLEDGING THE LIMITS OF OUR KNOWLEDGE.

SLIDE 14  
Immune Augmentation  
AIDS

SLIDE 17, 18, 19  
Don't, Don't, Don't.

I believe the rational model was Hepatitis B virus transmission.

## RISK REDUCTION GUIDELINES (13)

*Specific guidelines are - Basil Pringler: Disputed: - intention, however, casual transmission given and IC Pringler all persons in no to.*

THE FIRST GROUP I WANT TO MENTION IS THOSE PERSONS WITH AIDS. I DO THIS FOR FOUR REASONS:

1. IT IS ONE GROUP THE BAPHR GUIDELINES IGNORED
2. IT IS NOT ENOUGH TO TELL THESE PERSONS TO BE CELIBATE. MOREOVER, TO DO SO WOULD BE UNHEALTHY, UN ETHICAL, AND INHUMANE.
3. I HAVE TRIED TO EMPHASIZE CONSISTENCY EARLIER. THERE IS THE SUGGESTION THAT AIDS MAY BE TRANSMITTED DURING THAT INDUCTION OR INCUBATION PERIOD *THIS MEANS THAT DURING THE ST. PETER BULLER, ALL PERSONS WHO WORK IN A SICK* IF WE TELL PERSONS WITH AIDS "NO SEX" OR ONE SET OF GUIDELINES, WE BETTER BE TELLING ALL HIGH RISK PERSONS THE SAME GUIDELINES. WE CANNOT GIVE THE MESSAGE THAT IT IS OKAY TO ACT ONE WAY TODAY, BUT IF YOU ARE GIVEN THE DIAGNOSIS OF AIDS TOMORROW YOU CAN EXPECT TO BE TOLD TO ACT IN ANOTHER WAY.
4. LIKEWISE, IF OUR GUIDELINES FOR HEALTH CARE WORKERS OR VOLUNTEERS SAY " NO SEX WITH CLIENTS" AS DO THE GUIDELINES WRITTEN TO THE VOLUNTEERS FOR THE G.M.H.C. IN NYC, AND SAY THEY ARE BASED ON "INFECTION CONTROL" MEASURES, AREN'T WE GIVING THE SAME MIXED MESSAGE? IF YOU THINK YOU ARE <sup>SAFE</sup> SAY, OR TELL YOUR PATIENTS THEY ARE SAFE, AS LONG AS THERE IS " NO SEX" WITH PERSONS WITH AIDS, BUT IT IS OKAY TO GO TO SWEATER BARS OR BAPHR MEETINGS TO PICK UP SOMEONE, WE ARE ONLY DELUDING OURSELVES AND OUR PATIENTS. AT THE SAME TIME WE ARE DIVIDING THE COMMUNITY IN A WAY I HOPE WE ARE NOT INTENDING TO DO.
5. ON THE OTHER SIDE, PERSONS WITH AIDS NEED TO BE AWARE OF THE FACT THAT INFECTIONS ARE <sup>I</sup>DEED IMMUNE SUPPRESSING. ADDITIONAL INFECTIONS MAY FURTHER COMPROMISE AN OTHERWISE COMPROMISED DEFENSE SYSTEM. THEREFORE, GUIDELINES MUST INCLUDE RECOGNITION OF THIS RISK. SPECIFICALLY, I SAY <sup>11</sup> WASH YOUR HANDS AND MAKE SURE OTHERS WHO ARE GOING TO HAVE CONTACT DIRECTLY HAVE WASHED THEIR HANDS. IN ADDITION, LIMIT EXPOSURE TO PEOPLE WITH ACTIVE INFECTIONS.

RISK REDUCTION GUIDELINES (14)

FOR GAY MEN,

*at the ...*

SEXUAL MORES WORKSHOP ONE CONSENSUS WAS THAT EDUCATION ABOUT RISK REDUCTION NEEDED TO BE "SEX POSITIVE AND GAY AFFIRMATIVE." APPROACHES MAY VARY AND WILL NEED TO VARY. BAPHR'S APPROACH WAS TO:

EDUCATION ABOUT TRANSMISSION OF DISEASE *in general*

*(emphasizing  
age &  
role)  
promotion  
barriers  
natural  
needs*

DISCUSS HOW AIDS MAY BE TRANSMITTED.

TRANSLATE THAT INTO SEXUAL PRACTICES, *reducing trauma,*

*enhancing sex...*

SUGGEST ALTERNATIVE PRACTICES, *ways to bring up sex + health  
with prog-ct partner.*

RAISE THE ISSUE OF INDIVIDUAL AND COMMUNITY RESPONSIBILITY

A PAMPHLET MAY BE GOOD FOR REACHING LARGE AUDIENCES, BUT SOME WILL RESPOND BETTER TO DISCUSSION IN SMALL GROUPS, FOCUS ON ISSUES SUCH AS COMPULSIVE SEXUALITY, SUGGEST ALTERNATIVE ACTIVITIES (SOCIAL), MASTURBATION ETC.

*2100 20. Holistic approach. Discuss alternative approaches  
+ ways to do red: discuss setting for sex + health effects risk  
with a commitment that enhance pleasure + enhance safety & identify  
about condom.  
The challenge. SUID 21.  
14262-1000000*

(S.E. Follansbee, personal  
correspondence, folder #421)

## GUIDELINES FOR THE EVALUATION OF THE "WORRIED WELL" IN THE CONTEXT OF A.I.D.S.

Offered by the Scientific Affairs Committee, B.A.P.H.R.  
January 15, 1984

### INTRODUCTION

These guidelines have been devised for the medical evaluation of the patient concerned about the possibility of A.I.D.S. This is not meant to replace the standard evaluation and care of the gay male patient (which would include routinely such tests as the RPR, hepatitis B serology, GC screen, stool examinations for ova and parasites.) Rather, these guidelines are designed to offer an outline for the screening of these patients with regard to A.I.D.S., including their potential risk for acquisition.

### HISTORY

#### General

fever: unexplained and greater than 100° for two weeks  
night sweats: recurrent, drenching  
weight loss: unintentional and unexplained, greater than 10 lbs.  
fatigue: interfering with work, normal activities  
drug allergies: particularly to trimethoprim-sulfamethoxazole

#### Skin/Mucous membrane

herpes zoster (shingles)  
slowly healing, frequently recurrent herpes simplex (labial/genital)  
candidiasis (oral/perianal)  
dermatitis: diffuse, chronic or recurrent (may be folliculitis,  
bullous impetigo, seborrheic, "dry skin")  
cold urticaria  
Kaposi sarcoma-like lesions

#### Respiratory

cough: usually dry or minimally productive, worse with deep  
inspiration or exertion  
dyspnea on exertion  
shortness of breath

#### Gastrointestinal

unexplained diarrhea  
dysphagia/odynophagia  
gas  
anorexia

#### Lymphatic

lymphadenopathy: unexplained, lasting more than 3 months  
duration, involving more than two extra-inguinal chains.

Central Nervous/Peripheral Nervous Systems

headaches  
paresthesias/dysesthesias  
depression/mood swings of new onset  
coordination difficulties  
visual complaints: scotomata, diplopia  
memory and concentration difficulty  
seizures  
weakness (focal or diffuse)  
speech difficulties

Social

drug use  
STD history: GC, syphilis, intestinal, pediculosis, hepatitis (A, B, and non-A, non-B), NSU, anal warts, mononucleosis, scabies, CMV  
sexual history: practices, locations, frequency, anonymity, contact with person with A.I.D.S., drug use during sex, loss of libido

Hematologic

easy bruising  
spontaneous bleeding  
transfusion history (receipt and donation)

PHYSICAL EXAMINATION, complete, but with particular attention to:

vital signs: weight, temperature, respiratory rate  
general appearance  
skin/mucosal lesions: K.S., thrush, herpes simplex, zoster, petechiae (include an anoscopy).  
lymphadenopathy: check and record occipital, cervical, auricular, submental, submandibular, anterior and posterior cervical, axillary epitrochlear, supra- and sub-clavicular, inguinal, femoral, and popliteal nodal chains  
funduscopy: "cotton wool" spots, hemorrhages  
organomegaly: hepatosplenomegaly

LABORATORY EXAMINATION, assuming the history and physical examination do not mandate more extensive system evaluation:

complete blood count with differential and platelet estimate  
erythrocyte sedimentation rate  
complete chemistry panel: with particular attention to low cholesterol, low albumin, elevated globulin, elevated LDH

Consensus about other tests varied, such as skin testing, lymphocyte population subsets, etc. Most consultants agreed that these are not indicated for the "worried-well" with no other indications on history or physical examination for opportunistic diseases.

COUNSELING



## ACQUIRED IMMUNODEFICIENCY SYNDROME (Part I)

## OVERVIEW AND INFECTION CONTROL

## I. Definitions

- A. Case definition (Centers for Disease Control): Presence of reliably diagnosed disease at least moderately indicative of underlying cellular immunodeficiency in absence of any other cause of reduced resistance or increased susceptibility to that disease.
1. malignancy
    - a. Kaposi's sarcoma in persons under age of 60 years (26%)
    - b. lymphoma limited to the brain
    - c. diffuse poorly differentiated non-Hodgkin's lymphoma
  2. opportunistic infection
    - a. Pneumocystis carinii pneumonia (52% all cases, additional 7% with K.S.)
    - b. candida esophagitis
    - c. disseminated mycobacterial disease
    - d. CNS toxoplasmosis
    - e. active CMV disease
    - f. diarrhea associated with cryptosporidiosis (greater than one month duration)

N.B. The diagnosis is established by disease, not simply laboratory criteria of immunodeficiency.

- B. Other related conditions (variously termed "AIDS-prodrome," "pre-AIDS," "AIDS-related conditions")
1. chronic lymphadenopathy (greater than 3 months in at least two extra-inguinal sites)
  2. thrombocytopenic purpura
  3. progressive multifocal leukoencephalopathy
  4. ?? squamous cell carcinoma of the tongue and cloacogenic carcinoma of the rectum.
  5. syndromes of fevers, weight loss, diarrhea, fatigue, night sweats in variable combinations of greater than 3 months duration.

## II. Epidemiology

## A. Total number of cases

USA 3339 mortality 42.5% 1/31  
 Day max 497 (see 348) mortality 132  
 number: see Jan 40 AS 23

## B. High risk groups

1. homosexually active men (72%)
2. intravenous drug users (17%; greater than 50% of female cases)
3. hemophiliacs (1%)
4. others
  - a. sexual partners of 1,2, and 3 (1%)
  - b. Haitians (5%)
  - c. blood product recipients (1%)
  - d. no information (3%)
4. additional possible cases not counted include infants--either blood product recipients or born of mothers in AIDS-risk groups.

## AIDS (2)

N.B. To date the absence of documented cases in health care workers (without other risk factors) and "general" population since first cases recognized in 1978-9.

C. Mortality and prognosis

D. Geographic and racial background; age distribution.

## III. Immunology

A. Cellular immunologic defects

1. reversed helper-to-suppressor (OKT4-OKT8) cell ratio
2. decreased blastogenesis
3. decreased mixed lymphocytic culture response
4. decreased delayed-type hypersensitivity
5. decreased specific and nonspecific cytotoxicity

B. Humoral immunologic defects

1. polyclonal B-cell activation
2. immune complex circulation
3. inappropriate humoral response (decreased de novo response to antigen such as pneumovax<sup>R</sup> or heptavax<sup>R</sup>)
4. increased number of spontaneously secreting Ig cells
5. refractoriness to normal in vitro signals for B cell activation.

C. lymphopenia

## IV. Theories

A. viral

1. multiple candidates (a new virus, CMV, retrovirus, adenovirus, hepatitis B virus or a co-virus, EBV virus.)

2. "antigen-overload"

3. toxin(s)

4. miscellaneous

## V. Clinical

A. symptoms nonspecific and systemic

## VI. Diagnosis and Treatment of non-CNS disease

A. Pneumocystis carinii pneumonia

1. requires tissue documentation
2. trimethoprim-sulfamethoxazole (20/100 mg/kg/day p.o. or i.v.)
3. pentamidine (4 mg/kg/day; available from Parasitic Disease Drug Division of C.D.C.)
4. newer agents

B. Kaposi's sarcoma

1. chemotherapy: single agent versus multiple-drug

2. Immunotherapy
  - a. alpha-interferon
  - b. gamma-interferon
  - c. interleukin-II
3. newer agents

- C. Cytomegalovirus
  1. chemotherapy
  2. immunotherapy

- D. Mycobacterium avium-intracellulare
  1. "standard therapy"
  2. ansamycin
  3. clofazimine

- 
- E. Difficulties in diagnosis and therapy
    1. symptoms nonspecific and overlap
    2. many patients present with multiple conditions simultaneously
    3. many organisms are cultured slowly or not cultured
    4. standard histologic response may be absent
      - a. M. avium-intracellulare
      - b. cryptococcal meningitis
    5. therapy may be prolonged, toxic, inadequate
    6. recurrence or relapses occur
    7. "test-of-cure" may be necessary and associated with morbidity

#### VII. Infection Control issues

- A. A well-written packet entitled "AIDS: An Infection Control and General Information Packet for Health Care Providers" may be obtained for \$10 (check or money order, payable to AIDS/KS Foundation, SF) from the AIDS/KS Foundation, SF, PO Box 14227, San Francisco CA 94114.
- B. Based on hepatitis B precautions for the AIDS-"agent" and standard precautions for other associated conditions.
  1. blood/secretion/excretion precautions
  2. do not include respiratory unless patient has M. tuberculosis pulmonary disease. Do not include disposable dietary trays or separate bathroom facilities.
  3. Tissue/laboratory specimens should be marked, respecting patients rights to privacy (i.e., avoid labels that advertise the diagnosis.)

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## AIDS (5)

## QUESTIONS

1. AIDS has been reported in each of the following, EXCEPT:
  - a. homosexually active men
  - b. male contacts of female AIDS patients
  - c. blood transfusion recipients
  - d. infants of high risk mothers
  - e. Nevada.
2. AIDS transmission has been implicated by each of the following routes, EXCEPT:
  - a. sexual contact
  - b. Factor VIII concentrate
  - c. aerosol
  - d. contaminated needle sticks
  - e. vertically, from mother to infant
3. Which of the following is LEAST helpful in diagnosis CNS toxoplasmosis in AIDS:
  - a. new onset seizure
  - b. CSF examination
  - c. ELISA IgG titer to toxoplasma
  - d. Heat CT scan
  - e. positive ELISA IgM titer to toxoplasma.
4. Infection control precautions for disseminated *M. avium-intracellulare* include:
  - a. mask
  - b. gown for all patient contact
  - c. prophylactic INH
  - d. quarterly PPD testing
  - e. none of the above.
5. Which of the following have been associated with immunosuppressive states prior to AIDS:
  - a. *P. carinii* pneumonia
  - b. candidiasis
  - c. disseminated mycobacterial disease
  - d. CNS lymphoma
  - e. all of the above
6. The basic immunologic defect is probably:
  - a. cell-mediated
  - b. humorally mediated (antibody deficiency)
  - c. local defense
  - d. immune overload
  - e. reversible if patients would just "live right."
7. Each of the following is true for the hepatitis-B vaccine, EXCEPT:
  - a. it is the first vaccine made from a human source
  - b. it has not been shown to be associated with AIDS because it has not been used long enough for anyone to know
  - c. it undergoes formalin, urea, and pepsin/acid treatment designed to inactivate every known human and animal virus
  - d. it has been recommended by several national immunization boards for high risk patients.
  - e. it costs about \$100 for three injections.



## AIDS (6)

## Questions, continued

8. The number of patients with AIDS who have been found to have spontaneous or therapeutically induced normalization of their immunodeficiency state is:
- a. 5
  - b. 10
  - c. 50
  - d. 500
  - e. none of the above (and none)
9. Each of the following disease has been associated with AIDS, EXCEPT:
- a. P carinii pneumonia
  - b. Cytomegalovirus enteritis
  - c. cryptococcal meningitis
  - d. disseminated herpes virus infections
  - e. multi-lobar pneumococcal pneumonia
10. Infection control precautions in AIDS include each of the following, EXCEPT:
- a) stickers and signs that clearly state "AIDS" for all medical and nonmedical personnel to see
  - b. needle precautions
  - c. private rooms if the patient has profuse and uncontrolled diarrhea
  - d. respiratory precautions for patients with M. tuberculosis pulmonary disease.
  - e. excretion and secretion precautions.

Stephen E. Follansbee, MD  
March 2, 1984

EPIDEMIOLOGY AND ETIOLOGY OF AIDS  
Transmission--Parallels to other infectious agents (Hepatitis B virus)  
(abstract)

Evidence that AIDS is a transmissible disease was quickly recognized. However, identification of the "AIDS agent" has been elusive, although many candidates have been suggested. Speculation about the route(s) of transmission remains important to attempt interruption of its spread among members of the identified high risk groups, as well as prevention of acquisition by health care personnel and the general population.

Recognition of the similarities between the epidemiology of the "AIDS agent" and hepatitis B virus has provided valuable basis for "Risk Reduction Guidelines," including infection-control guidelines for the medical community (1). The similarities have also suggested to some that hepatitis B virus may be the actual cause of AIDS (2) or a "Trojan horse" carrying the agent (3).

This discussion will review the body fluids in which hepatitis B virus and surface antigen may be found. Blood appears to contain the highest number of infective particles. The potential importance of these body fluids in transmission has been partially evaluated. Although the percutaneous inoculation of some fluids including blood and saliva are clearly associated with increased risk for acquisition of hepatitis B, other routes such as oral-oral (including activities such as kissing and sharing glasses) remains much more speculative. There is no evidence for fecal-oral or respiratory acquisition of hepatitis B. Literature on transmission within households (4) and institutionalized persons (5) remains somewhat vague and discrepant. Some discussion of sexual practices and hepatitis B transmission for both heterosexuals (6) and homosexual males (7) will be provided, although the exact nature of the risk for the former group is still not clear. Finally, the association of AIDS transmission and various male homosexual sexual practices will be discussed, although the data are preliminary.

Although the transmission of AIDS appears to follow that of hepatitis B virus most closely, "over-extrapolation" must be avoided. Absolute statements about transmission, risk of acquisition and prevention await identification of the agent, serologic testing for acquired immunity, and hopefully development of active/passive immunization products.

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EPIDEMIOLOGY AND ETIOLOGY OF AIDS  
Parallels to Hepatitis B virus (continued)

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GUIDELINES FOR THE EVALUATION, THERAPY, AND FOLLOW-UP OF PATIENTS WITH THE EPIDEMIC IMMUNODEFICIENCY SYNDROME (ESPECIALLY PNEUMOCYSTIS CARINII PNEUMONIA)

Consider the diagnosis in a patient with unexplained fever, lymphadenopathy, weight loss, or respiratory complaints, especially in gay men.

Patients with the reported syndrome have had a variety of malignancies (Kaposi's sarcoma, Burkitt's lymphoma, Hodgkins' disease, and squamous - cell carcinoma of the tongue) as well as multiple (sometimes simultaneous) infections (Pneumocystis carinii pneumonia, disseminated CMV, chronic herpes simplex infection, cryptococcal meningitis, tuberculosis, atypical mycobacterial infection, mucosal candidiasis, and toxoplasmosis). The evaluation and care of these patients should be undertaken with this experience in mind.

BASIC EVALUATION

History:

- . Sexual preference
- . Street drug use - types, routes, frequency
- . Cities of residence; occupation
- . Contact history - sexual partners, roommates, coworkers with Kaposi's, Pneumocystis pneumonia
- . Duration and nature of constitutional complaints
- . Duration and nature of respiratory complaints
- . Dysphagia or odynophagia, substernal burning
- . Visual changes, headache, change in mental function
- . Prior history: venereal diseases, parasitic infections, drug treatment, herpes simplex infections, infectious mononucleosis
- . Animal exposure, pets

Physical Examination:

- . Thorough cutaneous examination
- . Lymph nodes
- . Funduscopic exam with pupils dilated
- . Oropharyngeal exam
- . Meningismus
- . Chest exam
- . Abdominal exam: hepatosplenomegaly, masses (lymph nodes)
- . Perianal region: herpes, Kaposi's
- . Neurologic exam; especially mental status

Ancillary Studies:

- . Arterial blood gases
- . Chest X-ray
- . WBC with differential, platelet count *ESR*
- . Skin tests (PPD, mumps, Candida, trichophyton)
- . Pulmonary function testing with diffusing capacity
- . Cultures of urine, throat washings, blood for CMV

- Cultures of mucosal/cutaneous lesions for herpes virus
- Serum frozen for later (convalescent) antibody titers - *also pneumonia serotype as well + immunization pneumonia, K.H.*
- Optional, depending on clinical situation:
  - 67Gallium scan of lungs; serum and CSF cryptococcal antigen;
  - CSF india ink prep; CT scan of abdomen, cranium

### Definitive Diagnostic Studies:

- Skin biopsy of suspicious lesions
- Bronchoscopy with transbronchial biopsy = *specimen handling. Bronchial washing*
- Open lung biopsy
- People to call for help:
  - Infectious Disease fellow (Beeper #335)
  - Pulmonary consult fellow (Beeper # )
  - Oncology fellow (Beeper # )
  - Clinical Microbiology - Dr. Hadley, X 8576
  - Anatomic Pathology - Dr. Margaretten, X 8215

### DRUG THERAPY

#### Pneumocystis carinii pneumonia:

Trimethoprim/Sulfamethoxazole 20mg TMP + 100mg SMZ/kg/day in 4 divided doses. We advise parenteral therapy in order to exclude poor absorption of the drugs as a cause of treatment failure.

Addition of folinic acid (citrovarum factor) 5-10mg/d (IM or IV) should be considered for patients with granulocytopenia and/or thrombocytopenia prior to or developing during therapy with TMP/SMZ.

Pentamidine isethionate 4mg/kg/d IM for sulfa-allergic patients or patients failing therapy with TMP/SMZ.

#### Evaluation of Therapy:

- Temperature curve
- ABG's q 1-2 d if stable, otherwise more frequently
- Chest X-ray 1-2 X/wk
- Pulmonary function testing weekly

Most patients who respond to therapy show evidence of doing so in 5-7 days after beginning appropriate therapy. In a patient that appears to be failing, consider:

- Another diagnosis: CMV, bacterial superinfection, O<sub>2</sub> toxicity, pulmonary embolus, tuberculosis, atypical mycobacteriosis, cryptococcosis, other opportunistic pathogens
- Changing drug therapy
- Hyperalimentation if nutritional state is poor

Talk to K.H.  
 re: *Pneumocystis carinii*



## MISCELLANEOUS

1) Isolation:

Though the route of transmission and population at risk are not identified, experience with hospital outbreaks of P. carinii pneumonia suggests that patients with suspected or proven P. carinii pneumonia be placed in respiratory isolation.

2) Prophylaxis:

*immunosuppressed pts*

Since P. carinii pneumonia has a high (15-20%) relapse rate in these patients, we recommend prophylaxis with TMP/SMZ to the patients who recover or are otherwise at high risk. The dose is 5mg TMP + 25mg SMZ/kg/day in two divided doses. For a 55-80kg adult, this is two regular-strength SEPTRA or BACTRIM tablets twice daily.

3) Advice to Patients:

Though the activity, exposure, or agent(s) responsible for immune deficiencies in this group are unknown, circumstantial evidence suggests a transmissible agent that is acquired by sexual contact. Patients should be informed of the evidence for a transmissible agent and advised that it may be prudent to inform sexual partners of their history and to consider use of condoms during sexual activity to attempt to prevent transmission of an as yet unknown agent. In addition, patients should be advised to seek medical attention for any persistent symptoms that are not readily explained.

## OUTPATIENT MANAGEMENT AND FOLLOW-UP

On discharge from the hospital, patients should have a primary clinic identified for follow-up (Infectious Disease, Chest Clinic, or Oncology Clinic are suggested, depending on the patient's major problem[s]).

In clinic, follow-up evaluations should include:

- Temperature taken at each visit
- Weight at each visit
- Assessment of persistent or new symptoms
- Assessment of tolerance and toxicity of TMP/SMZ prophylaxis, if applicable (CBC with differential, platelet estimate or count, hepatic panel)
- Arterial blood gases + chest X-ray to assess for recovery and to give basis for comparison and/or an early clue to recurrence.

EVALUATION AND TREATMENT OF RECURRENCE OF P. CARINII PNEUMONIA

Recurrent fever, weight loss, or respiratory symptoms may be predominant symptoms of recurrence. Worsening of previously stable or improving arterial blood gases may also give early evidence of recurrence. Transbronchial + open lung biopsy are necessary steps in the evaluation of recurrences just as they are for initial diagnosis. For patients not taking TMP/SMZ prophylaxis, initial drug therapy of proven recurrences should be TMP/SMZ in the previously mentioned dose. For patients that have taken TMP/SMZ prophylaxis, we recommend Pentamidine for initial drug therapy of recurrences.

It should be kept in mind that this syndrome is poorly understood, and that further complications of depressed cellular immunity are likely to occur (Listeria infections, for example, occur in patients with abnormal CMI, but have not yet been reported in this group). While the preceding guidelines are offered to attempt to provide some consistency in the evaluation and therapy of these patients, close observation and thoughtful creativity are important in making further observations on the causes and complications of this complex of diseases.



## INFECTIOUS DISEASES ASSOCIATES MEDICAL GROUP INC.

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(415) 641-4328

SEPTEMBER 3, 1987

## COMMENTS TO PRESIDENTIAL TASK FORCE ON AIDS

The private practice community involved with AIDS is proud:

proud of its level of expertise  
 proud of its quality care  
 proud of its hospitals and hospital workers  
 proud of its home care agencies and dedicated nurses, pharmacists,  
 social workers, attendants, and other support persons  
 proud of its ties with the academic and research-based community,  
 here in San Francisco at SFGH, at UCSF, and at Stanford University.  
 They have been available for specialized procedures and testing.  
 They have been available for clinical research protocols.

However, we are also concerned:

We are concerned about our professional colleagues who are becoming increasingly fatigued. Some have taken no new AIDS patients in the last several months or few years. They are exhausted by workload and intensity of care required.

we have lost physician associates in this community through early retirement. Some have belonged to high risk groups and have succumbed to AIDS.

we are concerned that we have trouble recruiting new physicians to join us in practice when we need help, or to open their own practices.

we are concerned that the university training programs which provide primary care physicians suffer in their recruitment ability because of the perceived heavy training experience with AIDS.

we see a nursing shortage, not just at SFGH, but in many of the private hospitals and home based agencies.

we see the available attendant care dwindle in numbers so that patients who wish to remain at home are forced to allow marginal individuals into their homes as "sitters"--i.v. drug users, alcoholics, etc.

we see the volunteer agencies stressed beyond measure as they heroically provide services to fill the large gaps in medical coverage and arrangements--housing, food, counseling and bereavement support.

we see the commitment and availability of concerned friends and lovers dwindle and die as more and more people succumb to this disease, More and more gay men and others at risk no longer ask AIDS will develop, but only when will it develop.

We are concerned that patients who have benefited from the incredible network of care available are now threatened by a breakdown of that system. They lack effective and humane alternative sources of care.

## INFECTIOUS DISEASES ASSOCIATES MEDICAL GROUP INC.

3620 ARMY STREET SAN FRANCISCO, CA 94110

(415) 641-4328

*We also see  
 some of the  
 clients to our  
 patients: DHP  
 Gancichori*

PAGE TWO

Our patients suffer from lack of available research protocols that remain limited to other communities or centralized in university settings.)

We are concerned that the educational efforts of SF, while maybe the most intensive and sophisticated available in the country, still fall short. We've seen surgeons fearful of continuing marital relations with their spouse because of the fear of contagion and fearful of being testited, that they may be positive and lose their professional life.

We see physicians referring patients for physical examination because they are fearful of direct patient contact.

We see physicians in SF chastise nurses for their dedication to patient care of people with AIDS.

Clearly there are no simple answers to these problems. However, I think the private community of physicians feels strongly on several issues.

1. Success in depoliticizing this disease will make available resources to continue and improve existing services and programs. It will allow more intensive, personalized education of all elements of our community, both medical and nonmedical and relieve some of the burdens discussed.

2. success in removing the stigma of this illness will allow earlier diagnosis and more intensive and complete support. Families are afraid to visit their brother, or son for fear that the word will get out back home. Wives have nowhere to turn for support. once their husbands fall ill, and mothers are no longer allowed their grandchildren once they have cared for their dying child.

3. clearly recognizing the need for various tiers of support and urging third party payers to recognize such will open up options for people with AIDS to get humane and adequate care in supportive environments.

4. supporting the research protocols so that patients no longer have to travel around the world to Paris, across the country to Bethesda, or across town to SFGH for experimental drugs will more rapidly advance our knowledge and progress AIDS, or relieve the taxed and overstressed institution, while providing more continuous, compassionate, personalized medical care through decentralization.

The private community of health care workers and services would like to see AIDS industry put out of business by a cure. I do not like being called for investment brokers to render opinions about some new drug just announced in the SF Chronicle that morning. We feel that this is possible to conquer AIDS. However, it will take a massive, coordinated and humanistic approach, with leadership that has been slow in developing. Hopefully, this commission will turn the corner in conquering this truly awful disease.

David F. Busch, M.D.

Stephen L. Follansbee, M.D.

Shelley M. Gordon, M.D.





An Association of  
Bay Area HIV  
Health Care Providers

257 (S.E. Follansbee, personal correspondence,  
folder #418).

4 February 1991

Kenneth Kizer, MD  
Director, Dept. of Health Services  
714 "P" Street, Room 1253  
Sacramento, CA 95814

Dear Dr. Kizer,

The Community Consortium is an association of over 180 Bay Area providers caring for patients with HIV disease. One of our primary goals is education of each other regarding important issues in HIV medicine. Another aim is to conduct community-based clinical trials. Our study of inhaled pentamidine as prophylaxis for Pneumocystis pneumonia led to FDA approval and a New England Journal of Medicine publication. Our third major objective is to respond as a group to social and political issues that concern providers of care to patients and patients with HIV disease.

Most recently an obvious issue that has attracted much attention and discussion among Consortium members is that of invasive procedures performed by HIV positive health care providers. We have opposed on numerous occasions the imposition of any restrictions on providers with bloodborne infectious diseases providing prescribed infection control guidelines are followed. We commend the New York State Health Department for issuing a position statement that came out against that of the American Medical Association and the American Dental Association, policies which we feel were premature, especially in view of the fact that the Centers for Disease Control will be holding an open consensus meeting to address the issue later this month. We have received a copy of correspondence to you from ACT UP/ Golden Gate suggesting that the California Department of Health consider taking a stand "against making public health decisions on the basis of hysteria." We commend our associates at ACT UP/Golden Gate for recognizing the potential effectiveness of having California issue a statement similar to that which emanated from New York State. We are writing at this time to urge that you very seriously consider such an action. Current data does not support that restrictions which have such far reaching social and political implications should be enacted.

If we could be of any further assistance or participate in further discussion of this matter, please feel free to contact us at (415)476-9554.

Sincerely Yours,

Donald I. Abrams, MD  
Chairman, Community Consortium  
Assistant Director, AIDS Activities  
San Francisco General Hospital  
Associate Professor of Clinical Medicine  
University of California, San Francisco

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Chairman, Social Policy and Action Committee  
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Hughes: Did you see cases in the clinic that you thought might be related to the new disease?

O'Malley: That happened six months later in was early '82. As I mentioned last time, an individual that I knew who had been in the hepatitis study had been diagnosed with Kaposi's sarcoma in November of '81. So he was one of that first handful of [AIDS] cases here in San Francisco.

One thing that helped motivate the CDC to do a research study in this [hepatitis B] group is we started collecting information on [AIDS] cases diagnosed in '81. I think by the end of '82, early '83, we realized that of the twenty-four cases, I ~~think it was~~ that were <sup>reported</sup> ~~diagnosed~~ with AIDS in 1981 in San Francisco, eleven of them were amongst men we had screened for this hepatitis B cohort study. ~~So it was just one~~ ~~shy of 50 percent.~~ If almost half the men were screened by us, then clearly, ~~we had the right population here (in our~~ ~~hepatitis B study,~~ ~~if we wanted to start looking at what the causative agent, might be.~~ <sup>was the place to</sup> ~~start~~ <sup>for a</sup> It was probably going to be found in the blood of these men. Since the blood we had on these men was from '78, '79, and ~~some from~~ 1980, and this was now 1982, early '83, the question was whether we had serum close enough to the time that they <sup>several</sup> ~~got~~ ~~infected~~ ~~with~~ ~~whatever~~ ~~was~~ ~~causing~~ ~~this~~ ~~immune~~ ~~[deficiency]~~ ~~syndrome.~~ <sup>were</sup>

diagnosed with AIDS in 1981

I got a few calls from CDC like, "What do you think is going on?" They ~~were~~ ~~valuing~~ ~~my~~ ~~opinion,~~ ~~at~~ ~~that~~ ~~point~~ ~~about~~ ~~what~~ ~~I~~ ~~thought~~ ~~might~~ ~~be~~ ~~happening.~~ ~~But~~ ~~at~~ ~~that~~ ~~point,~~ ~~they~~ <sup>As to what</sup>

~~the CDC article?~~ the Conte article~~2~~ that they could say really became the standard first. You know, obviously, a physician-heavy committee with Merle Sande as the chair is going to overshadow the groundwork <sup>of other organizations.</sup> ~~[of these nurses?]~~. But, I think it was simply groundwork, and there was not a consistent UC policy that covered all campuses equally [before the Sande committee came up with their infection control guidelines~~8~~]. This was the committee that did that in a formalized way to make sure that there was a unified and uniform response.

#### Refining Hepatitis B Guidelines for AIDS care

[AIDS Task Force's New England Journal of Medicine article?,  
~~1982? correct?]~~ 1983

Hughes: Yes. So in one sense, what the *New England Journal* paper does is refine what the *MMWR* has come out with, which was basically the hepatitis B model, and make the infection control procedures more closely attuned to the needs of AIDS.

Follansbee: Right. Again, I haven't looked at this November '82 <sup>direction</sup> ~~direction~~ <sup>new</sup> ~~explain?~~. I haven't ~~looked at~~ our article, but I remember principles like needle boxes for all the rooms, and gloves in all the rooms. All <sup>that</sup> ~~this~~ kind of ~~stuff~~ precautions~~2~~ were the nitty-gritty ~~2~~ of AIDS infection control~~2~~ that had to be worked out. How can patients be assigned ~~to the AIDS ward?~~ <sup>to</sup>

an AIDS unit without ~~these~~ details established!

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