



David Korn

Instantiating a Vision: Creating the New Pathology Department at Stanford Medical School

David Korn

Former Vice President and Dean of Medicine, Founding Chair of the New Department of Pathology, and Professor of Pathology (Emeritus), Stanford University, Stanford, California 94305

Annu. Rev. Pathol. Mech. Dis. 2012. 7:1–33

First published online as a Review in Advance on
August 12, 2011

The *Annual Review of Pathology: Mechanisms of
Disease* is online at pathol.annualreviews.org

This article's doi:
10.1146/annurev-pathol-011811-132447

Copyright © 2012 by Annual Reviews.
All rights reserved

1553-4006/12/0228-0001\$20.00

Keywords

Stanford Department of Pathology, creating, recruiting, Stanford
Blood Center

Abstract

This review represents my best effort to recreate and memorialize events that occurred 44 years ago, when I was invited to join the Stanford University faculty to create, essentially de novo, what rapidly became and remains today one of the very best and most admired departments of pathology in the world. That I was able to accomplish this challenging task I attribute to my holding fast to a somewhat inchoate vision of where the science and practice of pathology would go in future decades, a little bit to my gut instincts and innate ability to spot up-and-coming talent, but a lot to circumstances and good fortune in leading me to a small nucleus of wonderful young professionals of outstanding promise who were willing to join me in “betting the house” that, working together, we could pull off this once-in-a-lifetime opportunity—and we did.

PROLOGUE

The telephone rang in my home in Bethesda, Maryland, at about 8:00 PM on a Saturday evening in March 1967. My wife and I were enjoying dinner with our visiting good friend, William M. “Whitey” Thurlbeck, a pale blond South African of English descent, who had taken a year of U.S. residency training in pathology at the Massachusetts General Hospital (MGH) in 1956–57 and then become an acclaimed pulmonary pathologist, first on the faculty of McGill University, then as chair of pathology at the University of Manitoba, and finally at the University of British Columbia. Whitey was intelligent, boisterous, wonderfully witty, and sharp-tongued. No interaction with him, let alone a dinner well-lubricated with abundant wine, could ever be described as quiet! So, it was in a less than fully alert state that I rose from the table to respond to the unexpected, and frankly intrusive, ringing telephone.

The caller introduced himself as Robert Glaser, dean of the Stanford University School of Medicine. He told me that he would like to talk to me about the pathology department at the medical school and indicated that he would be visiting the National Institutes of Health (NIH) over the next few days. Would I be able to meet with him late on Tuesday afternoon at a nearby Bethesda hotel? After a moment of cloudy thought, and without a single follow-up question to the dean or an expression of even minimal interest, I answered casually, “Sure,” and we agreed on a time and place. The entire conversation could not have lasted five minutes.

When I returned to the dinner table and recounted what had happened, Whitey erupted in peals of laughter. “Stanford!” he roared. “That’s the worst pathology program in the United States! They’ve been trying unsuccessfully to recruit a chairman for years, and they’ve probably gone through every leading pathology chair in the U.S.—and several from the U.K.!” My wife, on the other hand, was mildly interested: Her only sibling, an older brother, after

receiving his PhD in high-energy physics from Massachusetts Institute of Technology (MIT) about 8 years earlier, had gone to Stanford for what he thought would be, at most, a couple of postdoctoral years, but he had accepted a faculty appointment and worked closely with Professor Wolfgang Panofsky to obtain Department of Energy (DOE) funding to help design and build the Stanford Linear Accelerator Center (SLAC, a DOE national laboratory). My brother-in-law, Burton Richter, would win the Nobel Prize in Physics in 1976 and serve as the director of SLAC for nearly two decades.

I met with Bob Glaser the following Tuesday afternoon in the lobby of his Bethesda hotel. He acknowledged that the Stanford medical school’s pathology program had fallen into deep disrepair since the school had relocated eight years earlier from its historic site in San Francisco to its new location on the university campus in Palo Alto; that the medical school had indeed talked with many present and prospective leaders of academic pathology; that my name had been brought before the search committee by Arthur Kornberg, chairman of the Department of Biochemistry, who had won the Nobel Prize in 1959 for discovering the first DNA polymerase (*Escherichia coli* DNA polymerase I) and who, as I would soon learn, was the most dominating and influential chairman in the medical school; and that the committee would like me to visit the medical school as soon as possible. I told Dean Glaser I was willing to visit, and we agreed on a date about three weeks later, in April. Prior to that visit, I went to the heavily attended Federation of American Societies for Experimental Biology (FASEB) annual meeting in Atlantic City (in the early 1960s, I had become a member of the American Association of Pathology, presently the American Society for Investigative Pathology, and shortly thereafter, of the American Society of Biochemistry, presently the American Society of Biochemistry and Molecular Biology—both founding societies of the FASEB), and I ran into many senior pathology academicians who, without exception, cautioned me that “Stanford pathology

is a pit!” Needless to say, my wife and I had many discussions about my upcoming visit and the possible issues of dislocation for ourselves and our three young boys.

WHENCE I CAME

I was born during the worst of the Great Depression, on March 5, 1933, one day after Franklin D. Roosevelt had been sworn into office for his first term as President and had immediately closed the banks, thereby preventing any of my mother’s greater New York City family from attending the ceremony of my “bris.” My father, the oldest of five siblings, and his next-older brother inherited what had begun as a fuel and grain company from my grandfather, who had been speculating heavily in the 1920s bull market and died suddenly on Black Monday in 1929, leaving his business and family heavily in debt. The two sons, working long hours seven days a week, managed to rescue the business and protect their families’ residences from foreclosure, but the experience deeply scarred my father in many ways, and it took him many decades to recover. I was brought up with my sole sibling, a brother who was three years younger, in a household that pinched every penny—there were few frivolities that I can remember from those years, or until well after World War II. My father was a first-generation immigrant, and like most of his cohort, he believed strongly that diligence and excellence in educational accomplishment were the path to a better life. He expected my brother and me to get straight As on every report card, whether in our public or religious schooling. It made no difference: A test was a test.

I went to Providence Classical High School, an elite public school modeled on the famed Boston Latin School, which at that time had classes of no more than 100 students, offered Latin and Greek, and required four years of study of one of those languages to graduate with a “Classical Diploma.” Because I elected to take a fourth year of mathematics, I graduated with an “English Diploma.” In the

middle of my junior year, I was appointed assistant editor of the school newspaper and became editor-in-chief in the second semester of my senior year. From the beginning, the two co-editors and I became good friends and spent many hours after school and weekends working together in one another’s homes to write news articles and editorials, edit and plan layouts, and seek advertisers to help support the costs of publication. Editing the *Classical Review* brought me much happiness and satisfaction; one of the high points was sitting in a Providence hotel room with a small number of other Rhode Island high school newspaper editors and numerous professional reporters to participate in an interview with Eleanor Roosevelt.

In the spring of my junior year, my guidance counselor had a conversation with me about my college plans. Surprisingly, I had given little thought to them, other than being confident that if I maintained a straight-A average, I would certainly get into Brown; but I was feeling more and more determined to go to college outside of Providence, which I was increasingly finding to be small and suffocating. When I discussed this with my parents, they were taken aback, in large part because of costs, but in the end they agreed that I “could go as far away as Boston.” I then told my guidance counselor that I was thinking of MIT or Harvard, neither of which I really knew much about, and he replied that Harvard would be better because of reputed lingering anti-Semitism at MIT and in the engineering professions generally, and because there was a unique, full-tuition scholarship program (the George E. Smith Scholarship) exclusively for graduates of Providence public high schools who were admitted to Harvard College. This bit of information was, unsurprisingly, most welcome to my parents, and we jointly agreed that I would apply only to Brown (as a fail-safe) and to Harvard. In the end, five of my Classical classmates and I were admitted to Harvard’s class of 1954 with full-tuition scholarships, which some of us later learned would also pay for our graduate education at Harvard.

HIGHER EDUCATION AND RESIDENCY

I matriculated in September 1950, having placed out on the advanced placement examinations in chemistry, mathematics, and English (thereby exempting myself from Harvard College's dreaded required freshman writing course, English A), as well as having demonstrated proficiency in a foreign language (in my case, German). I had no idea what I might major in or, indeed, what I wished to be. Looking back on my undergraduate education, I remember it as a wonderfully broad and rich experience that afforded me opportunity for remarkable growth. When I was a freshman, I took a full year of mathematics (differential calculus) and inorganic chemistry and, in the spring, decided that I wished to major in what was then known at Harvard as the Program in Biochemical Sciences. This major had only recently been established and was overseen by an interdisciplinary committee chaired by the eminent protein chemist John Edsall, who was and would continue for nearly three decades to be editor-in-chief of the authoritative *Journal of Biological Chemistry*. My advisor was Paul Zamecnik, an eminent biochemist housed in the Huntington Laboratories at the MGH, who pioneered the *in vitro* study of protein synthesis and discovered transfer RNA as well as, much later, inhibitory RNA. There was not yet a distinct discipline of biochemistry—indeed, only very recently had it been shown that cells contained nuclear DNA that carried the genetic information and cytoplasmic RNA, the functions of which were largely unknown, and that the two classes of nucleic acids differed in terms of their chemical composition (DNA contained cytosine and deoxyribose, whereas RNA contained uracil and ribose). Nothing at all was yet known about the structure of either nucleic acid. That the new major was multidisciplinary, did not require certain advanced chemistry courses with time-devouring laboratories, but did require a laboratory thesis appealed to me.

An immediate problem was that the introductory course in biochemistry, taught by George Wald, was offered only in the fall

semester and had the absolute prerequisite of a full year of organic chemistry, then known at Harvard as Chem 20. Because I did not wish to wait until the fall of my junior year to take Wald's course, I decided to take both semesters of Chem 20 during Harvard's summer school immediately following my freshman year. I roomed in the Yard that summer with a fellow classmate who was also taking Chem 20. Every day, and well into the evenings, there were two lectures, each followed by hours in stifling, non-air-conditioned laboratories with windows closed because of the scores of Bunsen burners heating highly volatile materials. There was an hour-long exam each week and two final exams, one after the first four weeks and one at the end of the eight-week course. Every night was spent memorizing chemical reactions from the Fiesers' classic textbook (1). The course was a grueling and unforgettable experience.

In the fall, I took George Wald's course, which was one of the most seductive pedagogical experiences I have ever had. I fell in love with biochemistry and decided this was the area in which I would spend my career, although exactly how was unclear. Wald was a pioneer in elucidating the biochemistry of vision and won the Nobel Prize in 1967 for his work on the chemical transformations of rhodopsin. That same academic year, I took a full year of physics, taught by Edwin Purcell, who won the Nobel Prize in 1952 for his codiscovery of nuclear magnetic resonance. I also took a full year of biology, which I found boring and very disappointing. Only later did I recognize that the biology course had been an all-too-accurate foretaste of my first year at Harvard Medical School (HMS).

My third year included a year of physical chemistry, taught by George Kistiakowsky, who had directed the assembly of the ignition system for the first atomic bomb at Los Alamos, and who, many Saturday mornings, put on exhibitions of expert glassblowing of laboratory equipment (which we students had to try clumsily to imitate in our laboratory assignments), typically with a lighted cigarette dangling from his lips! In the first semester I was assigned my

thesis advisor, Boris Magasanik, a young assistant professor of microbiology at HMS. I met with Magasanik in his office in then–Building D on the HMS quadrangle, and for my thesis project he assigned me the elucidation of the pathway by which the bacterium *Acetobacter suboxydans* oxidatively catabolized glycerol as a sole source of carbon.

I began work on my project early in the second semester, learning my way around the research lab, meeting Magasanik’s faculty colleagues and graduate students, mastering fundamental microbiological techniques and the imperative of sterility—and realizing what a tiny amount of glassware, especially pipettes, I would have for my work. I learned to use my supplies of glassware prudently because when they ran out, my only recourse was to spend more than an hour and a half washing and then sterilizing them in the departmental autoclave several floors away. On the day I arrived, Boris introduced me to my cardboard-covered, bound Harvard Laboratory Notebook with sequentially numbered, cross-hatched pages. I was told that this notebook would contain the record—in ink—of everything I did in the laboratory, including detailed descriptions of every preparation and experiment, all data collected and analyzed, all data tables and graphs, all my interpretations of experimental observations, and so on; that if anything was crossed out, there must be—at the site—a description of what was deleted and why; and that if a page were ever missing, I would be dismissed from the laboratory. Whenever I think about the promulgation by the NIH and the National Science Foundation during the past three decades of increasingly prescriptive requirements, not only for handling allegations of scientific misconduct but for educating new and established scientists alike in what has come to be known the Responsible Conduct of Research, dubbed (RCR), I recall that brief and blunt conversation with my mentor that indelibly etched in my mind the fundamental meaning of ethical science.

The summer of 1957 would be my last of freedom. Earning full-course credits during

summer school meant that I had met the distribution requirements for my major and had to take only three full courses in my senior year, one of which would be my thesis research. That autumn, I took a semester of advanced calculus and the first offering of a course on poetry taught by the Boylston Professor of Rhetoric and Oratory, and former Librarian of Congress, Archibald MacLeish. During my final semester, I was one of only six registered undergraduates in a lecture hall overflowing with biomedical scientists from the greater Boston area to hear Fritz Lipmann, who only that fall had received the Nobel Prize in Medicine, present for the first time a semester-long series of lectures on the seminal role of high-energy phosphate bonds in the energy economy of living cells—a memorable experience.

I spent most of my senior year working on my thesis project, typically driving to the HMS in the early afternoon and working until the wee hours of the morning. I preferred to work at night because most of the neighboring labs were empty, and I was able to borrow needed glassware for my experiments, dutifully washing, autoclaving, and returning them precisely to where I had found them before departing for Lowell House to sleep. Fortunately, both of my other courses, both semesters, began at 10:00 AM or later! In early spring, I submitted my thesis, which, together with my grade point average and performance on my major’s comprehensive examination, led to my graduating summa cum laude. That spring, I was also accepted into HMS, from which I received a fellowship enabling me to continue my research full time in Boris’s laboratory during the summer of 1954.

That summer, I was offered the opportunity to participate, with a select group of my classmates, in the HMS’s so-called new pathway for the first two years of medical school. The new pathway was centered on small-group teaching and intensive laboratory experience. Although the invitation was extremely appealing, I declined because I had decided to continue my thesis research that first year and realized that the structure of the new pathway would severely

constrain my ability to do so. As mentioned above, I found the standard HMS first-year curriculum to be overly focused on rote memorization, uninspiring, and very disappointing. In the spring of 1955, I was permitted to present the results of my research at the annual meeting of the Society of American Bacteriologists (which, in 1961, merged into the American Society of Microbiology; its proceedings are not accessible), and the following spring, I won the HMS's Soma Weiss Award for the best research paper presented at the annual Undergraduate Medical Assembly (2).

My second year at HMS was entirely different. At that time, pathology was the major second-year course. It ran for three full days each week for six months and was followed by three months of neuropathology. My laboratory section was fortunate to have deeply knowledgeable instructors who were fully engaged in the clinical practice of pathology at the MGH or the Peter Bent Brigham Hospital (PBBH); these instructors succeeded in imparting a deep clinical context to every gross and microscopic specimen we studied. The disappointment of my first year, during which I often considered whether to leave HMS to pursue a PhD degree, was replaced by enthusiasm and commitment, and I decided that academic pathology could be my career, providing me with a balance of clinical practice, in which I became keenly interested, with abundant opportunity and time for research.

That spring, Benjamin Castleman, professor and chief of pathology at the MGH, announced to the class he had acquired two U.S. Public Health Service (USPHS) predoctoral fellowship positions, which he was offering competitively to members of my class who wished to spend the following year pursuing research training in the MGH pathology program. The conception of the so-called Year-Out Medical Student Fellowship in Pathology originated with the University of Rochester's first pathology chair, George H. Whipple, who had hoped that it might attract medical students into academic pathology careers before they were seduced by their clinical clerkships. I

leaped at the opportunity and, with sophomoric grandiosity, proposed to isolate sufficient quantities of Bence-Jones protein from the urine of patients with multiple myeloma to examine whether and how it was related to the marked elevation of serum levels of immunoglobulins that was a hallmark of this disease. I did enlist as my research mentor professor of microbiology Albert Coons, who was pioneering the development of immunofluorescence microscopy, which became the foundation of immunopathology. Indeed, immunofluorescence and electron microscopy were the two major technological breakthroughs that propelled the science of pathology in the decades immediately following World War II.

My HMS classmate Karl Wegner and I began our fellowships in the James Homer Wright Laboratory of Pathology at the MGH in September 1956. Each of us was assigned a desk and microscope in the residents' room and provided with house officers' overly starched white uniforms, name tags, and meal tickets, as well as the MGH's engraved, leather-bound notebook, which was provided to all incoming house officers. We were shown the laboratory's facilities and put under the oversight of the chief resident, who informed us that he would treat us in most respects like new first-year house officers, albeit with our major service responsibilities initially limited to the autopsy service.

The learning environment in the pathology program was extraordinary. The MGH performed around 1,200 autopsies a year; every weekday morning at 8:00 AM sharp, Dr. Castleman presided over a conference for residents and faculty, during which the typically unfixed organs from each autopsy performed the prior day or night were presented seriatim by the responsible house officers, who were expected to have digested often multivolume medical records, including all procedures and therapeutics, and to provide a cogent summary of the deceased's medical history from minimal notes. Typically present were the rotating radiology resident and often a rotating general surgery resident, who were, respectively, expected to show and review any

films and add any relevant surgical details. In cases of unusual clinical complexity or interest, which were frequent in those days, attending clinicians also participated. The pathological materials were voluminous and amazingly diverse—the daily “gross conference” was an extraordinary learning experience.

Every day, at either 1:00 or 2:00 PM, Dr. Castleman presided over a second conference, during which slides of surgical specimens, which had been reviewed by the responsible resident alone and then with the attending faculty pathologist and found to be difficult to diagnose or of unusual interest, would be handed to Dr. Castleman as “unknowns.” Dr. Castleman would project the slide(s) on a screen, typically only at low power, and randomly choose one of the residents (or postsophomore fellows, as Karl and I soon discovered) to describe the specimen, its origin, and the pathological diagnosis. The expectation, of course, was that all of the selected slides would have been reviewed beforehand by all the residents and fellows prior to the conference—woe was s/he who had not! Not infrequently, Dr. Castleman would peer at the low-power projected image and rise from his stool to open one of the unlabeled glass-doored cabinets packed with unlabeled green slide boxes that lined three of the four walls. He would select a box, pull out a slide, and project it, explaining that he had once seen a similar section, perhaps 15–20 years earlier, and had diagnosed it as whatever, and that was how the unknown specimen would be signed out. None of us, staff or trainees, ever understood how “the Boss” managed these “homing” feats. Like the morning gross conference, this one also provided an exceptionally rich learning experience.

Very quickly, Karl and I became acclimated to the service’s routines and became regular members of the autopsy rotation. After a few months, we were permitted to become regular participants in the surgical desk rotation as well, although we only accompanied a resident responding to surgeons’ calls for frozen sections. In addition to the daily teaching exercises and heavy workloads, Dr. Castleman

had enormous files of published articles on just about every pathological manifestation that we encountered, and I spent many hours in the pathology library poring over relevant literature. The result—perhaps inevitable given the seductive environment—was that Karl and I became *de facto* indistinguishable from the pathology house staff. I performed around 50 autopsies that year and spent five months of increasing independence managing the surgical desk. It was during this fellowship that my wife and I became close friends of the Thurlbecks.

What about my research project? I began my fellowship by connecting with clinicians who cared for multiple myeloma patients and obtained consent from both the physicians and six or eight patients to collect 24-hour urine samples (there were no IRBs back then). I delivered sterile jugs to the participants’ homes and collected the filled jugs the following day. I reestablished contact with Paul Zamecnik, my undergraduate adviser, who provided me with refrigerator space to store the urine samples and access to standard and ultracentrifuges in the Huntington Laboratories. Working alone at night, I began to collect and freeze protein precipitates that I expected would contain Bence-Jones proteins. But as these laborious procedures continued, I asked myself, “What next?” I had by then, belatedly, read about the existing physical and chemical technologies for isolating purified proteins and recognized that the best method for me could be quantitative immunoprecipitation, but that seemed beyond my grasp. More important, I had at last recognized the overreach of my fellowship project and come to terms with the realization that I had become far more interested in mastering human pathology than in examining Bence-Jones proteins.

Although I abandoned my fellowship project, I did conduct a research project that year. I collected a small series of patients whose lungs at autopsy contained multiple, scattered, minute white nodules that had not previously been described. The histology revealed clusters of so-called cell balls reminiscent of the organization of chemodectomas, and Dr. Castleman

urged me to pursue these findings. We subsequently learned from Averill Liebow, professor of pathology at Yale University and a renowned pulmonary pathologist, that he had also observed and been puzzled by these nodules, and he assigned the problem to a young staff member in his department, Klaus Bensch. We agreed to perform a joint study of our combined 19 cases, and during the Christmas break of my third year, I spent nearly a week at Yale, where I got to know Liebow and worked closely with Klaus, with whom I formed a lasting friendship. In 1960, I was the first author of a paper, cowritten with Bensch, Castleman, and Liebow, entitled "Multiple Minute Pulmonary Tumors Resembling Chemodectomas," which was published in the *American Journal of Pathology* (3). At the MGH these nodules became known as kornballs, whereas at Yale they were dubbed benschomas.

My third and fourth years of medical school were notably pleasant because of my fellowship experience and my deep knowledge of the pathological manifestations of just about every disease I encountered during my clerkships. During my pediatrics clerkship at the Boston Children's Medical Center, I attended a young girl who had been diagnosed with cystinosis—a disease that I had seen a few times at autopsy and read about. One evening, while scanning the patient's many stained-blood smears under high power, I convinced myself and the attending physician that some of the child's circulating white blood cells contained crystalline deposits. I was encouraged to pursue this observation with scientists at the Massachusetts Eye and Ear Infirmary, who determined that the crystals were indeed cystine. This was the first description of cystine deposits in circulating blood cells or, indeed, in any cells other than the phagocytic cells constituting what was then known as the reticuloendothelial system and in retinal pigmentation cells and the cornea, where these deposits were diagnostic of the disorder. I was sole author on my first publication, in the March 1960 issue of the *New England Journal of Medicine* (4), which described the case and reviewed the literature.

The major bump in the road during these years was my diagnosis in January 1958 of pulmonary tuberculosis (TB), with which I became unwittingly infected during an autopsy I performed on an elderly walk-in patient at the MGH emergency room who died with a confluent bilateral necrotizing pneumonia that I failed to recognize in time was tuberculous. The TB cost me three months in a residential sanitarium close to the PBBH, where I was treated with bed rest and the newly developed triple-drug regimen. Because of that episode, I finished my last HMS clerkship in August 1959 and began my formal internship in pathology at the MGH on September 1. Simultaneously, I received a teaching appointment at HMS, and for the next two years I participated in teaching pathology to second-year medical students in the newly designed, integrated full-year course "Introduction to Disease," a product of the most recent revision of the HMS curriculum. I quickly learned that in such an integrated course, with exposure to clinician instructors and patients, students' interest in pathology was orders of magnitude less than it had been for me and my classmates—a lesson I would remember.

Because of the Korean War, I had received a student deferment from the draft during my college years, and when I was admitted to medical school, that changed to a medical deferment that delayed payback of military service obligation until completion of medical training. During my fellowship at the MGH, I learned it was possible to meet that obligation by becoming a commissioned officer in the USPHS and being selected for a two-year appointment as a research associate at NIH. Competition for these NIH appointments was fierce: They were sought after by every medical school graduate who was considering a career in academic medicine. Indeed, in the MGH, two years at NIH were routinely described as "the southern rotation." In my senior year, I began the process of applying for an NIH position and, after consultation with Boris Magasanik and several of his colleagues (Harold Amos was especially helpful), applied to the Laboratory of Molecular Biology at the National Institute of Arthritis

and Metabolic Diseases (NIAMD) to work with a young scientist, Arthur Weissbach, who had recently returned to NIH from a year at the Pasteur Institute in Paris and established a research program on the fascinating phenomenon of lysogeny.

I took a night train to Washington, D.C., and spent a long day at NIH interviewing with Weissbach, his four or five senior investigator colleagues in the Laboratory of Molecular Biology, and the associate director of NIAMD for Intramural Research, DeWitt “Hans” Stetten, who several years later became the founding dean of the new Robert Wood Johnson School of Medicine. The lab director was Leon Hoppel, a pioneer in RNA biochemistry; his deputy was Gilbert Ashwell, a renowned polysaccharide biochemist. To my delight, shortly after the interview Weissbach accepted me into his laboratory. My next hurdle, because of my history of TB, was the all-day medical examination required for appointment into the USPHS, but fortunately, my minimal right upper lobe scarring did not disqualify me. I received my letter of contingent appointment as a Research Associate at NIAMD in October 1959, and several months later, I received my appointment in the USPHS Commissioned Corps as lieutenant, junior grade, effective July 1, 1961. I accepted the commission, recognizing that I would be 14 months short of the minimum three post-doctoral years of training in anatomic pathology required for American Board of Pathology (ABP) eligibility.

My 22 months of formal residency training passed quickly. During those years, I managed to conduct and publish three studies (5–7), two of which were widely cited. I immensely enjoyed the challenges of autopsy and surgical pathology, the rich clinical interactions that in those days were attendant on both the research that I conducted and, especially, the always-stimulating learning environment in the MGH pathology laboratory. Ben Castleman remained a seminal figure in my life, and all of his staff were superb pathologists, caring and knowledgeable clinicians, and magnificent teachers. But at the same time, I recognized that the

transformative new era of molecular biology had begun: The pace of advancement in the basic biomedical sciences was increasing exponentially and, I feared, passing me by, and I thought I would never be fully satisfied peering at stained tissue slides and always wondering, “What is really happening in those cells?”

THE NATIONAL INSTITUTES OF HEALTH YEARS: BECOMING A SCIENTIST

I relocated my family to Bethesda and joined Art Weissbach’s laboratory in the then–still new NIH Clinical Center (Building 10) as a Research Associate on July 5, 1961. Scientific knowledge and technologies had indeed moved fast and far since my days in Boris’s laboratory: For example, I had never used radioisotopes or micropipettes, isolated proteins or DNA from cell extracts, or spent the night in a cold room purifying proteins by column chromatography. I spent most of July learning my way around the laboratory and reading everything I could find about bacteriophages and lysogeny. Weissbach was interested in the effects of lysogeny and lysogenic induction on DNA-metabolizing enzymes and assigned me to examine the effects of λ induction on the DNAses in *E. coli* K12 (λ). I began my work with a thymine-requiring mutant of this strain and soon discovered that abrupt removal of thymine from exponentially growing cultures of this lysogenic mutant triggered not only thymineless death but phage induction as well and that returning thymine to the medium supported robust phage replication and lysis of the culture. After intense characterization of this phenomenon, and while writing it up for publication, I discovered that I had been scooped by two months. Nonetheless, the paper was published (8). Returning then to my primary assignment, I discovered and characterized a novel DNase that appeared in K12 (λ) only after phage induction (9). Theretofore, the only new enzyme to have been specifically associated with bacteriophage infection or induction was the well-characterized, small, and stable lysozyme, which I believe was the

first enzyme structure to be crystallographically resolved.

Although standard appointments as research associates were for two years, I obtained permission to remain for a third year to continue my research with Weissbach, during which I was offered a permanent staff appointment and my own laboratory space in a neighboring laboratory, with which the Heppel laboratory often held conjoint Journal Club meetings. During my three years with Weissbach, I published another seven papers (10–16), most of them in the top-ranked *Journal of Biological Chemistry* and *Virology*, and became an independent scientist working in one of the frontier areas of molecular biology. In those days, the total number of scientists working on lysogeny was small; we all knew each other, and we freely shared our findings by fax and in small international workshops. Certainly we were competitive, but without the intensity and perversity that would, regrettably, within the decade come to be the norm in this and other areas of basic biomedical research.

During these years, I became a colleague and friend of Alan Rabson, a pathologist/scientist in the National Cancer Institute (NCI) and deputy chief of the Clinical Center's pathology service, and his wife, Ruth Kirschstein, also a pathologist and scientist, who at that time chaired the pathology section in the Division of Biological Standards (which later became the U.S. Food and Drug Administration's Center on Biologics Evaluation and Review, located on the NIH campus); in 1974, Ruth became Director of the National Institute of General Medical Sciences and the first woman to direct an NIH institute. Fearing total loss of my anatomic pathology skills when I decided to remain at NIH for a third year, I discussed with Rabson and Louis Thomas, chief of the pathology service, the possibility of my obtaining a staff appointment on a one-day-a-week basis to assist in signing out surgical specimens and autopsies. They were agreeable, provided that I become certified by the ABP. Because I had not looked at a pathology specimen or section for nearly three years, I plunged into rapid refreshment by

borrowing every week from the Armed Forces Institute of Pathology (AFIP) as many boxes of glass teaching slides as they would permit and spending nearly every night, into the wee hours, poring over the slides. Over the course of several months, I believe I reviewed every teaching slide in the AFIP's collection.

I also had to negotiate with the ABP: first, to count my predoctoral year at the MGH; second, to count my first two years of research training at NIH; and third, to grant me eligibility to take the examination even though my residency at the MGH was only 22 months, leaving me 2 months short of the required 36 months of training in Anatomic Pathology. I assumed the ABP would receive strong letters of support from Ben Castleman and his staff, as well as from Louis Thomas. To my first great relief, the ABP granted me eligibility. In November 1964, I flew to San Francisco (only my second trip west of the Mississippi) to take the examination and discovered among the candidates my former coauthor, Klaus Bensch, whom I had not seen since I moved to Bethesda. Klaus, like me, was a walker, and during our two evenings in San Francisco, we hiked through much of the central city and sipped drinks at the Top of the Fairmount atop Nob Hill. I was utterly captivated by the beauty of the city and its surroundings. To my second great relief, I learned shortly thereafter that I had passed the exam and, as Ben Castleman later informed me, with the highest score in the cohort.

With credential in hand, I began serving one morning every week as a staff pathologist, signing out surgical specimens from the Clinical Center's surgical service, as well as reviewing the many outside slides sent to the pathology service for consultation. I also took operating room calls for frozen sections, some of them memorable. In those years, the NCI surgical service was led by Alfred Ketcham, a product of M.D. Anderson and the Braunschweig school of massive resection of otherwise inoperable neoplasms, such as forequarter and hindquarter amputations, pelvic exenterations, and so on. Ketcham's special interest was head and neck cancers, especially nasal sinus cancers that

invaded posteriorly into the midbrain. The latter excisions consisted of a quarter-cranium that included an eye and upper jaw, and Ketcham always had to know when his posterior margin was clean. Typically, one or two dozen frozen sections would be demanded seriatim as Ketcham gingerly shaved into the midbrain; the OR call could consume much of a day. One pathology publication (17) emanated from this service: a case study in the *New England Journal of Medicine* describing a thymoma associated with aplastic anemia, immune deficiencies, and a most unusual thymic infiltrate that had not been previously described.

During these years, I was invited to visit and consider joining a number of pathology departments, typically as an assistant professor, and I knew that Ben Castleman was still expecting me to return to the MGH. But I was greatly enjoying my research and appreciated the unique environment NIH provided, which allowed one to focus entirely on one's science without the intrusions and distractions inherent in academic faculty life. In addition, my wife and three young sons enjoyed living in Bethesda, where there were so many aspiring young scientists who had come to NIH or the Walter Reed Army Institute of Research (WRAIR) to fulfill their military obligations and decided to remain—for awhile. In these years, the NIH appropriation was still growing rapidly; academic medical institutions and university life sciences departments were expanding apace; and faculty opportunities for young researchers, especially physician scientists with demonstrated research accomplishment and promise, were abundant. We were still basking in the glow of the post-World War II “NIH golden age,” when the agency was funding construction and equipping facilities to expand biomedical research capacity in universities, medical schools, and major teaching hospitals, and newly appointed chairs, especially clinical chairs, could request sufficient “below the line” funds on training grants to provide core support facilities for their reoriented research divisions and departments. Few, if any, of us realized that this golden age was soon to end (18, 19).

I was also shaping a vision of the kind of pathology department I would like to become part of: one that would nurture and facilitate the kinds of fundamental, cutting-edge molecular biology research in which I and those around me were so passionately engaged. But how could such research fit comfortably within departments typically laden with service and education and perforce focused on excellence in meeting these demands? All of us were operating on evolutionary faith that our research on bacteria and their viruses and other primordial model organisms so far removed from humans and their ills would in fact contribute to deeper understandings of the etiology and pathogenesis of human diseases, but would they? And if so, when? How could one robustly nurture such research and training alongside then-contemporary pathology research, clinical service, and clinical training in a coherent departmental structure committed to excellence in all? I became aware, from my visits, of several departments of pathology respected for their commitment and accomplishments in experimental pathology, but in most the research and service programs were highly dissociated, and none offered precisely the model I was seeking.

I started my new NIAMD laboratory in July 1964 and remained there for four years. The research conducted by my fellows and me would continue to focus with increasing sophistication, and continuing recognition, on bacteriophage systems (20–26, 27, 28), but in my last two years at NIH, I became very interested in the biochemistry of DNA replication in human cells. This major turn in research direction led to my career-long effort to identify and characterize the human DNA polymerases, using only cultured human KB cells because I had had no experience with tissue culture and because large quantities of these cells were readily obtainable from a nearby virology laboratory. I quickly learned how much more difficult it was to work with cultured human cells than with bacteria and gained even greater appreciation of the genius of the molecular biology pioneers in choosing bacteria and their phages as initial models. Notwithstanding, my fellow and I

persisted, and our first publication in this line of research (29) described our isolation and preliminary characterization of what later became known as human DNA polymerase β .

STANFORD I

What I Found

I made my first visit to Stanford in April 1967, enjoying the convenient but short-lived helicopter service between the San Francisco airport and Palo Alto. I met twice with Dean Glaser and interviewed with each member of the search committee, a large fraction of which was composed of relatively young, research-oriented department chairmen newly appointed after Stanford relocated its medical school to the university campus in 1959. Notable exceptions were Avram Goldstein, chair of pharmacology, and Henry Kaplan, the iconic radiation oncologist and chair of radiology, both of whom had joined the medical school in San Francisco and played seminal roles in the recruitment of Nobel laureates Joshua Lederberg and Arthur Kornberg—all of whom then became key instigators of the controversial relocation. I spent considerable time in the pathology department, which I learned was responsible only for autopsies and student teaching. There were nine faculty members, six based at Stanford and three at the closely affiliated Palo Alto Veterans Affairs Medical Center (PAVAMC), a newly erected structure opened almost contemporaneously with the medical school's relocation. Of the nine, the three at the PAVAMC and five at Stanford were ABP-certified pathologists. Leland Rather, a long-time Stanford faculty pathologist who, in the past decade, had completely lost interest in the discipline and had become a noted medical historian; three assistant professors, congenial and hardworking, who carried the brunt of the department's teaching and autopsy load but lacked the kind of academic distinction I was seeking; and the highly respected neuropathologist Lucien Rubinstein, who had been recruited to Stanford in 1964, and whose textbook, *Pathology*

of Tumors of the Central Nervous System, coauthored with Dorothy Russell and first published in 1959, was becoming the classic in this field. David Glick, one of the world's leading histochemists, controlled much of the department's meager and minimally equipped research space and owned its only electron microscope (EM). Contained within the department's academic space were the autopsy room and the tissue-processing laboratory, as well as the residents' offices. My recollection is that all of the few residents were international graduates.

The PAVAMC faculty consisted of three assistant professors. Two of them, Jon Kosek and Luis Fajardo, competently directed the laboratory service, which encompassed both anatomic and clinical pathology, and the third, Lysia Forno, was a neuropathologist who was already recognized as an authority on the pathological manifestations of Parkinson's disease. The contributions of the PAVAMC pathologists to the Veterans Affairs service, resident training, and medical student education were considerable and well regarded, and I was grateful to be able to rely on this component of the program without near-term changes.

The current incumbent in an annual procession of acting chairmen was Morgan Berthrong, pathologist-in-chief of the Penrose Hospital in Colorado Springs; the prior year (1965–1966), it had been Kenneth Weinbren, a highly respected academic pathologist and educator from the Hammersmith Hospital in London. The last regularly appointed chairman, dating from the San Francisco years, had been Alvin Cox, a leading dermatopathologist who, shortly after the medical school's relocation, decided to give up his chair and abandon the department to join the Department of Dermatology full time. Berthrong was exceptionally gracious and helpful in providing me with his assessment of the department's circumstances, including the contributions of the faculty members.

The Surgical Pathology Service was run by a single pathologist, Stanton Eversole, a Johns Hopkins graduate who spent all of his time in a tiny office, reeking of formalin, in the hospital across from the operating theater.

Eversole had no relationship to the pathology department and desired none. Because he was a full-time, always-available provider of service to the surgeons, they valued him highly, even though ophthalmology, obstetrics/gynecology, and urology all operated their own histopathology laboratories within their academic space, and Henry Kaplan sent all his lymphoma patients' specimens to the University of Chicago to be read by Henry Rappaport, one of the leading hematopathologists of the era. The clinical laboratory, which provided routine blood, urine, and microbiology testing, was directed by a competent and personable clinical pathologist based in the Department of Medicine; the lab had never sought College of American Pathologists (CAP) accreditation. All the specialty laboratories were operated by faculty in the Departments of Medicine and Pediatrics in their personal academic space. It was made clear to me that the clinical laboratory was not intended to be part of a pathology recruitment package.

Severely complicating the situation were the peculiarities of structure and governance of the hospital, which was built to be the medical school's principal teaching hospital as part of the Edward Durell Stone medical quadrangle and was physically contiguous with the medical school. To understand these requires a bit of history. Stanford's medical school was founded in San Francisco in 1908, 17 years after Stanford opened its own doors, and its teaching partners were essentially community hospitals. The relocation of the medical school to the university campus in 1959 was not welcomed by the Palo Alto Medical Clinic (PAMC), which, since its founding immediately after World War II, had become the dominant provider of medical care in the area and was politically very influential. To assuage the concerns of the PAMC and the City of Palo Alto, it was agreed that the new hospital would be co-owned by the city and named the Palo Alto-Stanford Hospital Center, that it would be overseen by a third-party administrator acceptable to both owners, and that it would be governed by a board of directors composed of prominent area residents and community physicians; only two of the board's

seats were awarded to the university to be filled, respectively, by a university trustee and the vice president for medical affairs, who at that time was Dean Glaser.

The operational manifestations of this awkward arrangement included a contractual agreement between Stanford and Palo Alto that guaranteed to community physicians a majority of the hospital's beds and permitted the PAMC to operate its own laboratory services within the hospital. Thus, when I first entered the hospital, I was struck by prominent signs with opposing arrows directing patients and physicians to Stanford radiology or community radiology, to community EKG, EEG, and EMG, or to the corresponding Stanford laboratories, and so on. Although there were no separate pathology laboratories, the PAMC physicians and surgeons, who were by far the predominant users of the hospital's facilities, including the operating rooms, routinely sent their tissue specimens to the pathology laboratory in the PAMC.

Whitey Thurlbeck and others had understated the situation—to describe the pathology problem as “a pit” was a euphemism! Nevertheless, the opportunity had some attractions for me. Stanford's physical setting was extraordinarily attractive—almost resort-like to a New Englander—and promised to be a strong asset in recruitment. I met with all the chairs, and all had been enthusiastic, strongly committed to research, and welcoming, as had key faculty in the powerful biochemistry department, with whom I had already developed cordial professional relationships while pursuing my research on bacteriophages, DNAses, and now DNA polymerases. I felt confident that my own research program and those of like-minded faculty to be recruited would find a very supportive environment at Stanford. The bright side of the severe impoverishment of the pathology department suggested that the required near-total rebuilding might be accomplished relatively quickly and with few casualties. The small size of the hospital and the Stanford clinical services, notwithstanding the hostile political climate and the need for major organizational changes, was perversely appealing

in suggesting that a revitalized pathology department need not become overwhelmed with clinical service demands, as happens to so many fine academic pathology departments. Finally, my visit had convinced me that the time might be ripe: There was strong support in the faculty leadership as well as the administration finally to address Stanford's notorious pathology problem.

I was invited back to Stanford in June 1967 for a second visit and further discussions, to deliver a formal seminar, and to meet with the leadership of the PAMC. The seminar went very well, except for several senior surgeons who demanded, understandably, to know what studies of bacteriophages had to do with pathology. I was generally aware that the most clinically active surgeons considered me another "lab rat" and were leery of my clinical commitment and my plans. I knew that after my initial visit Stanford had made many phone calls to check out my credentials and accomplishments, one of them to Ben Castleman, who despite his keen disappointment that I would not be returning to the MGH, called me soon thereafter to discuss the Stanford situation. Castleman was especially concerned, as I had been, with the Stanford-Palo Alto contract that permitted competing laboratory services in the hospital, and he strongly advised me, beyond any other demands I might make, to insist that failure to guarantee a single and exclusive academic pathology service in the Palo Alto-Stanford hospital would be a deal breaker. He also told me that he had delivered this message bluntly to Dean Glaser. Before returning to Stanford, I had prepared a letter to the dean that described the resources of space and personnel I thought would be essential to begin the rebuilding effort and underscored the imperative of a single service.

During my second visit, I had two discussions with the PAMC leadership that were pivotal. The first was with the PAMC pathologist John Dieferding, whom I found congenial and competent; he assured me that he had no vested interest in continuing to provide pathology services to PAMC patients in the hospital and

would support the creation of a single, academic pathology department and service at Stanford. The second was with surgeon Robert Jamplis, the politically powerful president of the PAMC, who assured me that he, too, would support the single department and service, requiring only that I assure him a high quality of clinical service and that Dieferding receive a voluntary faculty appointment and be welcomed as a colleague. With doubtlessly visible relief, I gave Jamplis my word that I would do so. With this potentially deal-breaking barrier overcome, Dean Glaser explained to me that creating a single pathology service required a change in the city contract and that Stanford University's president, Wallace Sterling, had agreed to open the contract to address this and other chafing matters.

The dean and I had several discussions about the elements of an offer letter and finally agreed to terms. Key elements were exclusivity of the pathology service; surgical pathology, but not the clinical laboratories, becoming part of the department; commitment to creating new research space contiguous with the pathology department, which would require relocation of present inhabitants, to be ready for occupancy within the next 12 months; provision of core research equipment and funding for the transition of my personal research program; and the modest number of faculty positions that I could recruit at the outset. I made certain the dean understood that I would not retain the three Stanford-based assistant professors beyond the end of the 1967-1968 academic year, that neither Rather nor Glick would be of any help to me, and that I regarded Rubinstein as my sole Stanford-based asset. To allow a smooth winding down of my NIH laboratory, we agreed that my formal appointments as professor and executive head of the department and chief of the pathology service would not become formally effective until July 1, 1968, but that I would assume strategic direction of the department and begin active recruiting and so on immediately upon approval of my faculty appointment by the Stanford University Board of Trustees. Specifically, I agreed to be at Stanford twice each

month, with one of my visits timed to enable my full participation in the monthly meetings of the medical school executive committee. All expenses would, of course, be covered by Stanford. We agreed that Lucien Rubinstein would serve as acting chairman until my relocation.

In addition to my semimonthly visits to Stanford, I spent much time, including evenings and nights, during my final 11 months at NIH in identifying and recruiting an initial cadre of faculty, soliciting their reference letters, preparing their long-form Stanford University appointment packages, defending their appointments before the Executive Committee of the Stanford University School of Medicine, and so forth. Stanford agreed to pay for my membership in the Harvard Club of New York City to provide me a quiet space in which to meet and interview candidates. Concurrently, I continued to direct my research and bring my laboratory to an orderly close.

Shortly before the end of 1967, Stanford University succeeded in rewriting its contract with Palo Alto to complete its purchase of the hospital and change its name to the Stanford University Hospital (SUH), albeit retaining the original allocation of beds, the right of the PAMC to continue to operate its existing nonpathology diagnostic laboratory services in the hospital, and the excessively community-weighted composition of the hospital board. The contract contained a new precedential clause asserting that SUH would have a single pathology service directed and operated by the academic department. The contract also stated that Stanford University would henceforth manage the hospital—the era of third-party administration, with all of its clumsiness and adverse symbolism, was over.

STANFORD II

Laying the Foundation

In the summer of 1967, even before receiving my formal letter of appointment on July 20, I realized that to create the department I envisioned I would have to overcome two major

antithetical hurdles. The first was the immediate need to establish clinical credibility with the surgeons and win over the many clinician skeptics who believed that the culture of the relocated medical school was far too focused on research excellence and not nearly enough on clinical excellence. Indeed, to these skeptics, many of the new clinical chairs had been chosen almost exclusively for their research accomplishment and commitment rather than for their clinical commitment or even competence. The second was that to build the basic research component of the department, the faculty I appointed and the research they produced would have to meet the very high standards exemplified in the newly restructured basic science departments in the medical school, and especially in the Department of Biochemistry, which was led by Arthur Kornberg and widely regarded at the time to be the top biochemistry department in the world. Beyond that, my training at the MGH had convinced me that demonstrating consistent excellence in delivering pathology services would be the essential—and probably sufficient—condition for establishing the fundamental research programs I desired.

I had already faced the first hurdle. For many reasons, Stanton Eversole had to go: During my freighted second visit, he had tauntingly told me he had not “read a paper in more than a decade” and asked what I intended to do about that. When I told him he should look for another job, he laughed and said, “The surgeons will never let you get away with that.” So, upon returning to Bethesda, I immediately began to seek recommendations of surgical pathologists when, purely by chance, I mentioned my search to Vincent Marchesi (who had begun his studies of membrane proteins from red blood cell ghosts in the Laboratory of Experimental Pathology at NIH and would later become chairman of pathology at Yale) and his fellow, Tom Tillack (later to become chairman of pathology at the University of Virginia), who had recently joined Marchesi from his pathology residency at Washington University in St. Louis (WUSTL). Tillack quickly mentioned the names of two very highly regarded

upcoming faculty in Lauren Ackerman's internationally acclaimed surgical pathology program at WUSTL. I visited with both men on a sweltering day in August, and we talked for several hours about my plans for the new department. I took an immediate liking to both of them, recognized that they perfectly complemented one another and enjoyed working together, and offered them the opportunity to continue working together as co-directors to build a new surgical pathology program at Stanford. Ronald Dorfman, a South African émigré educated at the renowned University of the Witwatersrand in Johannesburg and a rising star in hematopathology, and Richard Kempson, a superb general surgical pathologist with special interest in gynecological pathology, agreed to visit Stanford during my upcoming visit several weeks later for a heavy schedule of interviews. Together, they completely captivated the surgeons, and Ron, with his already strong reputation in the pathology of lymphomas, especially delighted Henry Kaplan and his influential medical counterpart, Saul Rosenberg, chief of medical oncology. After an intense but brief courtship, during which I assured Ron and Dick of pretty much a free hand in building their program, they agreed to come to Stanford as first-term associate professors and co-directors of surgical pathology effective July 1, 1968.

Although I was indescribably delighted—and relieved—by their decision, I did feel a tiny pang of sorrow because I realized that in hiring Dick and Ron I was buying into the WUSTL tradition of separating surgical pathology entirely from autopsy pathology, a tradition not unique to that institution but foreign to the culture and structure of unified pathology programs in Boston, which I had found to have significant clinical as well as educational and training value. I was also creating a problem for myself in providing direction of the autopsy service, which I did not fully resolve until my colleagues and I succeeded in recruiting Charles Carrington to this role in 1975. Carrington had been a protégé of Averill Liebow at Yale and was already a leading pulmonary pathologist,

as well as a superb autopsy pathologist who had no desire to participate in general surgical pathology.

During that same time period, I called Klaus Bensch and offered him the opportunity of partnering with me in building the new Stanford program as a professor of pathology, the only tenured position I was to offer in creating the program. My interests in Klaus were that I knew him and had worked easily with him on the pulmonary chemodectomas project; I believed he was deeply principled and that I could depend on him; he was a broadly experienced academic general pathologist working in a first-rate department, with deep knowledge of the discipline; Lucien Rubinstein knew and thought very highly of him; and he had a well-regarded research program in electron microscopy. Klaus had been the first author on a recent, widely cited paper describing the marked advantages of tissue fixation with glutaraldehyde for EM studies; this paper quickly became the standard in that field. Klaus was cordial but cautious and said he had no immediate desire to leave Yale but would think about it. Unbeknownst to me, Averill Liebow had only recently accepted an appointment as professor and founding chairman of the Department of Pathology in the newly established medical school at the University of California, San Diego, also effective July 1, 1968, and he had invited Klaus to join him. It would be some time before Klaus let me know that he would join me at Stanford.

Throughout the summer and fall of 1967, I took every opportunity to consult with colleagues about promising young investigators successfully engaged in contemporary research who were aspiring to pathology careers. I discussed possible interest in Stanford with, among others, Vince Marchesi and Peter Ward (who was then fulfilling his military obligation at the WRAIR), but both were my contemporaries and uninterested. For my first success, I was indebted to David Goldthwait, professor of biochemistry at Case Western Reserve University and a pioneer in the biochemistry of DNA damage and repair, who alerted me

to Errol Friedberg. Errol was a South African who had completed his medical education and a portion of his residency in anatomic pathology at the University of the Witwatersrand, come to Cleveland for a third year of residency, and then taken a postdoctoral fellowship in the Goldthwait laboratory, where he was completing a productive two years and establishing his career-long engagement with DNAses and their roles in the repair of damaged DNA. Errol visited me at NIH and made such a positive impression that I straightaway offered him an assistant professorship in the new department effective July 1, 1968, which Errol equally quickly accepted. A short time afterward, Errol called me, crestfallen, to tell me that upon his decision to remain in the United States and seek citizenship, he had been drafted and would be reporting to the WRAIR on July 1. He was elated when I told him I would hold his position until he could arrive, which he did in January 1971.

Another stroke of fortune occurred when Henry Kaplan, who had been extremely helpful to me after I agreed to come to Stanford and was especially pleased with the recruitment of Ron Dorfman, urged me to talk to a 1965 Stanford medical school graduate who had forsaken further medical training to pursue his research interests in Kaplan's division of radiation biology. I interviewed Irving Weissman soon thereafter and learned that he had committed to a research career while in high school after spending a summer working in the laboratory of the pathologist in his local hospital in Bozeman, Montana. Irv's interests were already focusing on fundamental questions regarding the development, maturation, regulation, and functioning of the mammalian cellular immune system. His passion for research and his creativity shone through in that interview, and I had no hesitation in offering him an assistant professorship in the new department.

The third young appointee to the research unit two years later, and also serendipitously, was David Clayton. David received his PhD from Caltech in 1970 where, using his mentor, Professor Jerome Vinograd's newly invented

methodologies, he discovered by electron microscopy that some mitochondrial DNA (mtDNA) in acute myelogenous leukemia cells was twice the length of that in normal cells, and that the percentage of cells with elongated mtDNA increased as the disease progressed, approaching 100% in so-called blast crisis. At that time, the very existence of mtDNA was highly controversial, and David's observation, the first example of a change in mtDNA associated with a pathological condition, generated a host of fundamental mechanistic questions and was widely discussed. During his one-year postdoctoral fellowship at the City of Hope Medical Center, Clayton explored in mouse-human fusion cells whether mtDNA could replicate in the presence of heterologous nuclear DNA, and he was considering where to go next to pursue his career. That year, Vinograd, while on sabbatical leave at Yeshiva University, met Stan Cohen (later of Cohen-Boyer patent fame), whom I had befriended at NIH. Cohen informed Vinograd about the program I was trying to build at Stanford, which led Clayton to contact me to arrange a visit, during which he presented a seminar and interviewed with my departmental colleagues and several professors in the Department of Biochemistry. All the interviewers were positive, as was I, and I offered David a faculty appointment. Clayton was indeed early in his career development, although not that much earlier than Weissman had been when I first appointed him, but for the first time I was questioned by the dean about "the relevance of this appointment to the pathology program."

STANFORD III

The Clinical Laboratories

Sometime in late October or November of 1967, I received a (typical) late-night phone call from Dean Glaser informing me that Stan Eversole would be leaving Stanford on January 1 to become chief of pathology at the El Camino Hospital, a well-regarded community hospital about three miles south of Palo Alto, and that

he had recruited the director of the SUH clinical laboratory to join him. When I reminded Glaser that the clinical labs were not my responsibility, he informed me that medicine chair Halsted Holman had abruptly decided he no longer desired the responsibility, and the dean made clear that he expected me to deal with both problems. I appreciated the urgency of the matter but was confident that, together, Lucien Rubinstein, Ron Dorfman, Dick Kempson, and I could find a solution for surgical pathology, which we achieved rather promptly in Paul Miller, a senior pathologist at El Camino Hospital, who did not get along with Eversole and was happy to agree to cover Stanford's surgical pathology service for six months until Dorfman, Kempson, and I would be on-site. The clinical laboratory, however, was a major concern, totally unexpected and unplanned for, and a specialty area in which I had neither past experience nor contacts. I had begun rather frantically to ask around when I received a telephone call from Paul Wolf, who told me that he was a professor of pathology at Wayne State University and either director or associate director of the clinical laboratory at the Detroit Medical Center, and that he would be interested in coming to Stanford on short notice to help me resolve the clinical laboratory problem. I knew no one at Wayne State whom I could immediately contact, so I asked Wolf to send me his CV and to meet with me in Bethesda for an interview, which he promptly did. There was always a glibness about Paul that troubled me (and others), but his references were supportive of his experience and competency, and in the urgency of the moment, I told him I would be willing to appoint him as Stanford's director of the clinical laboratories but only as a first-term (non-tenured) associate professor. That he so willingly accepted this demotion further troubled me, but the situation was acute, and we agreed to proceed.

Around that same time, I met Howard Sussman, an MD with a masters degree in chemistry who, after a year of rotating internship, spent his two years of deferred military obligation in Bernard Brodie's laboratory at

NIH; followed by a year of anatomic pathology training at Columbia University; and then four additional years at NIH in the Clinical Center's Department of Laboratory Medicine, first with George Brecher, chief of hematology and director of the department, then for most of his stint with Ernest Cotlove, chief of chemistry and deputy director of the department. Howard came to talk to me with a strong recommendation from Al Rabson and made a very good impression. I offered him an assistant professorship conditioned on his taking another year of residency training in pediatric pathology so that he could serve our department as a pediatric pathologist and oversee pediatric autopsies, which Howard accepted. Howard was strongly research oriented and would be a continuously funded investigator and member of the department's evolving research unit.

During his years at NIH, Howard had developed keen insight into clinical laboratory operations, and although he did not interfere with Paul Wolf's direction and clinical interactions, he began to focus on automating SUH's clinical laboratory operations. Working often with graduate students of professor of electrical engineering Bernard Widrow, Howard conceived and directed the successful development in the early 1970s of a computer-based information system to capture all clinical laboratory transactions; this system was adopted by the SUH and remained operational for nearly 25 years. Paul Wolf was not reappointed at the end of his term and went to the University of California, San Diego, in 1974, at which time Sussman became the laboratory director. We shortly thereafter received, and would maintain, CAP accreditation, and within the decade, with the enactment of CLIA I (the Clinical Laboratory Improvement Amendments I regulations) and stringent Medicare requirements for reimbursement of clinical laboratory tests, all of the medicine and pediatrics "splinter labs" became folded in. The last of these was the Pediatrics Cytogenetics Laboratory, which, years later, served as a departmental cornerstone in its development of genetic pathology.

STANFORD IV

The Early Years, Part I

When my small band of pioneers and I took up residence at Stanford in July 1968, we were greatly understaffed and would remain dependent for several more years on visiting professors and fellows to help us meet our clinical service and teaching obligations. I had given notice to the three inherited assistant professors and moved Lee Rather into a small office to suitably accommodate his history of medicine scholarship. David Glick understood that he could no longer control so much of the department's research space, which he graciously relinquished. However, sharing the department's sole EM, which he had purchased years earlier and was now being used largely by one of the departing assistant professors, with Kempson and Dorfman proved to be very contentious and was resolved only when I told David that if the EM could not be shared, it could no longer remain in the department's space. The instrument became an invaluable resource for the surgical pathology program and was our only EM until Klaus Bensch negotiated the transfer of his research EM from Yale.

Kempson and Dorfman swiftly set about creating a superb clinical service, and the reputation of the department climbed steadily. We were able to attract a host of top-notch visiting faculty, many from Commonwealth countries, especially South Africans, who found the Bay Area reminded them of "their Cape." Dorfman and Kempson brought with them from WUSTL their fellow, Marshall Kadin, who stayed with us for several years before accepting an academic appointment at Boston's Beth Israel Hospital, an HMS affiliate. Some of these visiting faculty, who were invaluable to our service and teaching programs and to whom I will remain forever grateful, were (with my deep apologies to those whose names I have forgotten) Robert Archibald from New Zealand; Mahendra Ranchod, a South African Indian, who became an assistant professor and later resigned to join a nearby private hospital practice;

Klaus Lewin from the United Kingdom, who stayed with us for six years as assistant professor before relocating to the University of California, Los Angeles, where he made professor and became internationally renowned for his work in gastrointestinal pathology; and Gerald Levine, another splendid product of the University of the Witwatersrand, an outstanding diagnostician and teacher who became assistant professor but, several years later, resigned to join a nearby hospital practice, where shortly thereafter he died from Burkett's lymphoma (in which, ironically, he was an expert). Also notable were our sabbatical leave visitors, Malcolm Mitchinson and his pathologist wife, Jean Arnaud, from the pathology faculty at Cambridge University, and Rosemary Millis, a pathologist from a major London teaching hospital. In our first year, we were greatly helped by a visiting professor from the University of Manchester (whose name, as I recall, was George Thompson), who directed our autopsy service and was a marvelous teacher and colleague. We tried hard to recruit George, but these were the years of the Vietnam War, and he had an 18-year-old son whom he would not put at risk of being drafted, so he reluctantly declined our offer and returned to England.

The quality of our residency applicants improved dramatically almost from the beginning, and within several years we were able to attract trainees of exceptional promise, including highly competitive MD-PhDs, whom we mentored into faculty positions in the Stanford department and elsewhere. To note just a few who joined our faculty: Roger Warnke, who succeeded Ron Dorfman as director of hematopathology; Michael Hendrickson, who succeeded Dick Kempson as co-director of surgical pathology and obstetrics and gynecology pathology; Richard Sibley, who became director of renal pathology and also co-director of surgical pathology; Jeffrey Sklar, an MD-PhD who, after two years of residency, took a two-year research fellowship with Paul Berg, returned to the pathology department as assistant professor, and became the first to use DNA restriction mapping to determine the

clonality of a variety of morphologically ambiguous lymphoproliferative disorders and thereby distinguish the malignant from the benign (Sklar was recruited to Brigham and Women's Hospital by my dear friend and fierce competitor, the late Ramzi Cotran); Michael Cleary, who trained with Sklar and became Stanford's associate chair for experimental pathology; Eugene Butcher, who trained with Irv Weissman and is now a professor and a leading expert on vascular adhesion biology; and Robert Rouse, who also trained with Irv Weissman and is now associate chair for the laboratory services at the PAVAMC.

A trainee among this group who merits special mention is Margaret Billingham, an English general practitioner who retired from practice to raise her children and then decided to pursue training in pathology when she and her husband emigrated to the San Francisco Bay Area. Her husband became a senior scientific program director at the National Aeronautics and Space Administration's Ames Research Center at Moffett Field outside San Jose, and she entered the pathology residency at the PAVAMC. Margaret developed a strong interest in cardiac pathology and worked closely with Norman Shumway's cardiac transplantation team to establish the endomyocardial biopsy as a dependable detector of very early cardiac rejection. Her meticulously documented studies of these biopsies led to the development and universal acceptance of the Billingham scale of histopathological changes and dramatically transformed the early detection and clinical management of cardiac rejection and, thereby, the survival of transplanted hearts.

During these early years, there were two islands of relative stability: the laboratory service in the closely affiliated PAVAMC, mentioned above, and the neuropathology laboratory, directed by the late Lucien Rubinstein, who was already well known and attracted outstanding neuropathology residents as well as rotating neurosurgery, and sometimes neurology, residents. Many of Lucien's trainees went on to distinguished academic careers; they include, among others, Sam Ludwin, professor

at Queen's University; Stephen DeArmond, professor at the University of California, San Francisco; and Bernd Scheithauer, professor at the Mayo Clinic. Lucien, as noted above, served as the acting chairman during my commuting year and continued to be a trusted colleague to whom I could always turn for wise and unvarnished advice. Lucien was assisted in directing his program by Mary Herman, who had completed her pathology and neuropathology training at Yale, come to Stanford as a neuropathology fellow, and then joined the faculty as assistant professor; she rose to associate professor and ultimately married Lucien and led him to the University of Virginia in 1981.

The Early Years, Part II: Medical and Graduate Student Education

When the Stanford medical school relocated to the university campus, it instituted a five-year curriculum, known as the five-year plan, for which it became renowned. Upon admission, each of the 60 medical students was assigned a desk in the teaching laboratories that would be his or her home for the first three years. The basic science courses were taught with substantial laboratory components that engaged the students in research problem solving requiring the use of contemporary methodologies, such as purification of DNA from cellular extracts by cesium chloride buoyant density ultracentrifugation, purification of proteins by column chromatography, isolation and study of the properties of microbial mutants, and so on. The pathology course filled the traditional hundreds of hours with substantial histopathology laboratory instruction as well as participation in autopsies. This was the curriculum that existed when I accepted my appointment, but during my commuting months, the Vietnam War was creating chaos on major university campuses, with daily demonstrations and sit-ins led by students and faculty aimed at overturning perceived institutional authoritarianism and long-established academic traditions and promoting an affirmative action agenda. Stanford was not exempted, and during 1967–1968, the Stanford

medical school faculty senate, for reasons that I never understood (other than to be fashionable), voted to abandon the acclaimed five-year plan, eliminate most laboratory instruction, and sharply reduce the number of required hours allotted to the basic science departments. Thus, upon my arrival pathology had been allocated a grand total of 50 hours of instruction for general and systemic pathology!

The faculty and I debated long and hard about how we would deal with this meager allotment and finally designed a course that we dubbed the 50-hour wonder. But at the same time we agreed to offer new elective courses in autopsy and surgical pathology, which proved to be very popular and attracted many students in each class. So while adhering to our 50-hour core allotment, we succeeded in exposing what became probably a majority of the students to in-depth, interactive pathology experiences. These experiences were enthusiastically received and conveyed, in our view, a much deeper and more relevant experience in pathology than had the earlier required curriculum.

Given the many challenges we faced, I decided that we did not have the resources to attempt to develop a PhD program in pathology. However, for those faculty who joined our research unit, Stanford already had in place a number of interdepartmental and interschool, federally funded training programs, some for both predoctoral and postdoctoral students. These programs included interschool programs in biophysics, immunology, and neurosciences, all of which granted PhDs. In addition, Stanford's medical school had been an early recipient of an NIH-funded medical scientist training program (MSTP) award that, over the years, attracted many of our very best medical school applicants. Pathology research faculty became very active participants in the MSTP: David Clayton joined the MSTP committee shortly after his arrival and directed the program from 1978 to 1996. In 1972, following enactment of the National Cancer Act that launched President Richard Nixon's war on cancer, I applied for and received Stanford's only tumor biology training grant,

which provided support for both predoctoral and postdoctoral students. Coincident with receipt of the award, I established the Laboratory of Experimental Oncology to embrace the department's basic research faculty and successfully petitioned the university senate to approve a new PhD-granting training program in tumor biology, which I directed with an executive committee for 12 years, and which we shaped to be as similar as possible to the MSTP. Indeed, over time, the graduate students in both training programs became well-nigh indistinguishable. In addition, the tumor biology program offered postdoctoral positions, perhaps as many as 12, that became intensely competitive. As a result of these two training programs, the pathology department became well stocked with superb graduate students, many but not all of whom sought double degrees, and postdocs, all of whom were typically highly interactive and greatly enriched the department's research culture, cohesiveness, competitiveness, and productivity.

The Early Years, Part III: Basic Research

All of the initial cadre of basic research faculty were very young and selected largely on the basis of their promise, and all of them exceeded my fondest expectations, becoming professors at Stanford and leaders in their respective fields. David Clayton's laboratory made major contributions to our understanding of the structure, replication, and repair of mtDNA and proved to be a popular attraction for graduate students, who themselves went on to illustrious scientific careers. David contributed important services to the medical school, serving as director of the MSTP for two decades, as deputy director under Paul Berg, and as my trusted decanal designate on the executive committee that oversaw the construction, programming, and occupancy of the Beckman Center for Molecular and Genetic Medicine, at the time the largest and most expensive research structure on the Stanford campus. David left Stanford in 1996 to join the Howard Hughes Medical Institute (HHMI),

where from 2001 to 2007 he was vice president and chief scientific officer. He has now returned to research, directing his own laboratory at the HHMI's Janelia Farm Research Campus, where he continues his studies of mitochondria. Errol Friedberg's laboratory made many seminal contributions to illuminating the molecular processes involved in the repair of DNA damage, and Friedberg's 1995 textbook, *DNA Repair and Mutagenesis* (30), has become a classic. In 1990, Errol left Stanford to become professor and chair of pathology at the University of Texas Southwestern Medical Center, where he built a first-rate program. Since 1992, Friedberg has authored seven books, most recently a well-received biography of Sydney Brenner, a giant in molecular biology; Errol recently stepped down from his chairmanship to become a full-time author. Irv Weissman, one of the most imaginative and creative scientists I have ever met, has arguably had the most spectacular research career of any of us, making seminal contributions in every area of study to which he turned and opening several new fields of scientific exploration. More than any of us, Irv trained not only graduate students and fellows but also pathology residents, several of whom are now professors at Stanford and elsewhere. Irv remains as engaged (and over-committed) as ever, serving Stanford as director of the Institute for Stem Cell Biology and Regenerative Medicine and, until very recently, as director of the Comprehensive Cancer Center.

My own laboratory at Stanford was initially funded with a grant from what was then known as NIAMD and focused on continuing my bacteriophage studies, and I was fortunate to have some terrific fellows and an excellent technician, but with so much of my time consumed by launching the new department, only a few publications resulted (31, 32). More importantly, my attention was increasingly shifting to human DNA polymerases. I did not attempt to renew the NIAMD grant, but rather in 1972 I applied for and received a substantial new grant from the NCI to fund the polymerase research. The seminal event for my future research was the arrival in my lab of a postdoctoral fellow

from Gil Ashwell's laboratory at NIH, Teresa S.-F. Wang, a superb biochemist, who stayed with me for my remaining 12 years as chairman. Teresa gradually morphed from trainee to colleague with faculty appointments in Stanford's research faculty line, mentored my students and fellows, and oversaw the day-to-day operations of my laboratory. After I became dean in the fall of 1984, I realized sadly within a few months, in spite of my determined efforts, that the demands of my decadal calendar were preventing me from maintaining a significant presence in my research lab, and I successfully petitioned the NCI to conduct a site visit to review my recommendation that Teresa become principal investigator and take over my grant. After especially careful consideration (in May 1984, I had been appointed chairman of the National Cancer Advisory Board), NCI agreed, and Teresa went on to lead the laboratory and mentor her own students and fellows with great distinction, until she decided to retire in 2010–2011. During the late 1980s, the department put Teresa up for promotion to full professor in the academic regular line, and I was very pleased to see her receive this well-deserved recognition. During my years as principal investigator, my laboratory purified both DNA polymerases α and β to homogeneity; extensively characterized their very different enzymatic properties; created the first monoclonal antibody to polymerase α ; and demonstrated that polymerase α was the mammalian "primase" responsible for the initiation of DNA replication, an assignment of biological function that has become generally accepted in the following years. The publications describing this body of work, and recognizing the invaluable contributions of my students and fellows, are References 33–76.

The initial research cadre put its unique stamp upon the program, and in subsequent years their legacy of quality and accomplishment would continue to attract outstanding basic scientists into the department. The first, as I recall, was Gerald Crabtree, who arrived in 1984–1985, shortly before I became dean. Gerry, whom I had first met during his two-year stint in the pathology laboratory at NIH, was on

the pathology faculty at Dartmouth University, where he had developed a splendid record of research achievement, including most recently being involved in cloning the human fibrinogen gene and closing in on the gene for protein C. Gerry dropped into my office one day and casually announced that he would like to relocate to Stanford, and I offered him a faculty position with the understanding (to ease his appointment) that in addition to pursuing his research, he would oversee the coagulation section of the clinical lab—which he did for a time. Many others followed, as is evident in the department's current roster, among them Andrew Fire, who in 2006 would share with his former University of Massachusetts collaborator, Craig Mello, the 2006 Nobel Prize in Medicine for their discovery of RNA interference.

STANFORD V

The Stanford University Blood Bank

When I arrived at Stanford in 1968, the SUH obtained most of its blood supply from two community blood banks to the north, whereas the PAVAMC obtained its blood from the regional American Red Cross (ARC) Blood Bank headquartered in San Jose. F. Carl Grumet had completed his residency in internal medicine at Stanford and trained as a clinical associate at NIH in transfusion medicine before returning to Stanford as an immunology research fellow with Hugh McDevitt. Observing that Stanford's endogenous blood components program was not up to the standards employed by NIH and other front-rank institutions, Carl in 1970 accepted Paul Wolf's invitation to become (part-time) director of the small blood component section of the clinical laboratory. In 1971–1972, while a senior fellow and instructor in hematology, Carl convinced the division head, Stanley Schrier, that Stanford should establish its own full-service blood bank for both clinical and research purposes. Negotiations ensued between SUH, the Division of Hematology, and the ARC that led to the opening in 1973 of the Stanford–Red Cross Blood

Center with salaries for both a medical director and a scientific director. Grumet, then an assistant professor in hematology, became medical director, as well as director of the Stanford Transfusion Service and of the newly created Stanford Tissue Typing Laboratory. These duties, combined with clinical attending responsibilities, proved too many, so Carl was very receptive to my suggestion that he accept a joint appointment in pathology, and in 1974 he joined the department full-time. It was by this convoluted sequence that blood banking, the transfusion service, and tissue typing became pathology laboratory services.

The Stanford–ARC partnership, launched in good faith with great hope, proved intolerably chafing because of the fundamental divergence of mission and goals. The ARC was entirely service oriented, not research oriented; indeed, local innovation was discouraged by the fact that no improvements in processes or procedures could be introduced into practice without going through a lengthy, tedious, and dulling process of national review and approval. For example, Stanford introduced testing for the first hepatitis B antigen (the Australia antigen) and for cytomegalovirus in blood intended for use in premature infants, both without ARC approval. Not surprisingly, by 1977, Carl had begun to press me to support Stanford's independence. During this same period, with my concurrence, he began to quietly recruit his former hematology faculty colleague, Edgar Engleman, who in mid 1978 joined the pathology faculty as assistant professor and assistant director of the blood bank and transfusion service.

The gestation of the Stanford University Blood Center proved to be extraordinarily difficult, unpleasant, and hurtful. If it were not for my own unwavering conviction that a fully academic program would provide pathology with a unique platform for top-rank research and training, as well as the opportunity to enhance the quality of patient services and be more responsive to some of the unique needs of Stanford's clinical programs, it never would have happened. When the ARC realized we were serious, they erupted in fury, attacking

Carl's integrity, as well as my character, competence, and judgment in standing by him. In those days, almost everyone's mother had rolled bandages or volunteered other services for their local ARC chapters during World War II, and the national ARC began repeatedly calling the president, the provost, and the wives of Stanford University trustees to complain that their pathology chair was leading a vicious campaign to defame and undermine that great American institution. The vice president for medical affairs, the provost, and the president, as well as key trustees and hospital board members, were shaken by the ARC assault, and I was repeatedly challenged to explain to them why creating a fully independent Stanford blood center was such an important academic objective to be worth all of this tumult, negative feeling, and adverse publicity. In this climate of hostility and threats, the challenge of developing a viable business plan for the independent operation and selling it both to the SUH and the Stanford University Board of Trustees naturally became even more challenging. But we persisted, and in the end we succeeded. Crucial to the deal was my agreement that all financial risks from operating the Stanford Blood Center would rest entirely with the department, but correspondingly, any net revenue generated would remain within the Stanford Blood Center. Accordingly, the Stanford Blood Center opened its doors in 1979–1980 in refurbished space on Welch Road as a wholly owned and operated service center of the Department of Pathology.

The importance and value of this academic investment probably would never be more visible or publicly apparent than during the early years of the AIDS pandemic, which was already erupting in the Bay Area during the late 1970s and early 1980s. At the time, the causative agent was unknown, as were its modes of transmission; the virus had yet to be isolated, and it would be several more years before it became conclusive to the skeptics (the very worst of whom were the blood bankers) that the disease was being transmitted by transfused blood and products. But basic research had identified the

devastation wrought by the causative agent on certain populations of lymphocytes and the resulting alteration of the T4/T8 ratio, and Ed Engleman, who in his research had prepared monoclonal antibodies specific for each lymphocyte type, convinced Carl and the SUH administration and board that it would be prudent for the blood bank immediately to begin assessing this ratio prospectively in all donors, as well as in all stored blood and components, and not to use any that showed a predetermined abnormal value. For a long time, the Stanford Blood Center was the only one in the world performing this test, and we were publicly derided by many competitors and commentators for charging transfused patients an added expense for a test of “merely academic value.” Several years later, with HIV isolated and a specific antibody test commercially available, Ed and Carl had all of the Stanford bank's stored aliquots retested and proved that we had not transfused a single HIV-positive specimen. No other blood bank in the world could make that assertion because, indeed, HIV had been transmitted stubbornly and promiscuously via transfusions to hundreds of thousands of unwitting recipients. Ed and Carl also demonstrated that our indirect screening test had been, as we would have hoped, overcautious because not all of the nonused samples proved to be HIV positive. (For those in the trade, the home-brew test proved 100% sensitive but less than 100% specific.) The work of the Stanford Blood Center was singled out for praise in the best-selling book *And the Band Played On*, published by Randy Shilts in 1987, which is the authoritative description of the eruption and the havoc wrought by the AIDS pandemic in the Bay Area.

STANFORD VI

Laying the Financial Foundation

Soon after our arrival, Dick, Ron, and I launched an intensive effort to raise awareness of our rejuvenating department among medical professionals in the Bay Area. We

became active participants in the monthly South Bay Pathology Society meetings that enabled us to interact with the pathology staffs of most of the hospitals in the Bay Area, and from those interactions we began to develop what became, over time, a very rich and robust surgical pathology consultation service that extended nationally and internationally. All of the consultation submissions were accessioned and used as teaching material, and the specimens added great diversity and complexity to the materials routinely submitted from the Stanford clinical services. My agreement with Dick and Ron was that the revenues accruing from the consultation service would remain in a surgical pathology consultation account under their control and could be used to enrich their program as they saw fit, but could not be used for faculty compensation or bonuses. This arrangement remained unaltered through my term as chair and was a wonderful resource for enhancements of the surgical pathology program.

My interactions with the South Bay Pathology Society soon got me involved with the California Society of Pathologists (CSP), and I agreed to serve on the CSP board for three years. In that role, I became very interested and enlisted Dick Kempson in the idea of redoing the current procedural terminology (CPT) charge codes for surgical pathology to make them more accurately reflect pathologists' actual effort in reading specimens (e.g., extra sections, special stains, cytochemistry, electron or fluorescence microscopy). The traditional CPT codes had never paid serious attention to this matter because the contracts of most practicing pathologists were structured as a percentage of clinical laboratory revenues, and they had no reason, or were forbidden by their contract, to charge for their tissue pathology work. Because the Stanford department did not have such a contract, Dick and I became very interested in trying to catalyze a major rethinking of the anatomic pathology CPT codes. To synopsize a huge amount of "extracurricular activity," we generated a totally revised set of charge codes and shepherded the revision through the South Bay Pathology Society and then, to our surprise,

through the CSP at its annual fall convention. At that point, we had won the support of the state's pathologists, and my next stop was to be at the annual meeting of the CAP, to which I had recently been elected a Fellow, where I had to meet with the CAP's CPT code committee. That proved to be a totally frustrating interaction with a group of ultraconservative, closed-minded practitioners who were earning good livings and were utterly opposed to consideration of any change in the current pathology codes or charging practices. They were doing just fine with percentage contracts, thank you, and would countenance no tinkering with the system.

With that reality check, soon after Howard Sussman took over as clinical laboratory director, I enlisted his help in building a professional fee structure for every clinical laboratory test. Earlier, I had consulted with the Blue Shield carrier for northern California and with Stanford's Medicare intermediary and learned that no pathologist in northern California had implemented such a charge system but that one pathology group had done so in southern California, and their charges were accepted by their Medicare intermediary and Blue Shield. Howard and I put together a very conservative charge schedule structured around what we thought was a reasonable measure of the pathologist's work; thus, high-volume automated tests had charges of pennies per unit, whereas tests requiring more direct professional oversight and interpretation received appropriately higher charges. The SUH administration found this schedule acceptable; my larger problem was selling it to the Faculty Practice Plan (FPP), on whose billing operation the burden of processing this very high volume of largely tiny charges would fall. There was a goodly amount of push back from the FPP administration and my fellow service chiefs, but in the end the charge system was implemented. For the first time, the pathology department began to have a dependable executive fund of substance, and in time, it became the largest executive fund of any clinical department. Upon its establishment, I took steps to have the

executive fund registered in the university's books as a departmental endowment to try to ensure that no medical dean or vice president for medical affairs would be able to invade it. This action certainly did not endear me to the medical school's administration!

Nearly a decade later, shortly before, unwittingly, I was to become dean, I met privately with then-SUH director Sheldon King and proposed that the department would be willing to cease charging separate professional fees on clinical laboratory tests, which confused patients and generated complaints to the hospital; eliminate the FPP billing burden and expense; and allow the SUH to fold the professional fee into a single hospital charge for a laboratory test—if King would guarantee me by contract that he would turn over to the department the percentage of SUH's total laboratory revenues that was represented by the current professional fees. Although my recollection is hazy, I think that the proposed deal, at the outset, would flow approximately 3.0–4.5% of the SUH's laboratory revenues to the department.

To my pleasure and utter surprise, King readily agreed, and in 1984, for the first time, Stanford's Department of Pathology obtained a percentage contract that would remain in effect at least throughout my ensuing deanship and generate a very substantial executive fund for the department's continuing academic investment and enrichment.

CONCLUDING COMMENTS

Reminiscences are by definition intensely personal. I imagine any one of the small group

of pioneer faculty who joined me in creating the new department of pathology at Stanford might remember the events I describe somewhat differently and possibly choose different emphases. My recollections of events that occurred 44 years ago were greatly refreshed by my visit to the department in early January, and I am grateful to Steve Galli for his arrangements and splendid hospitality.

As I look over what I have written, I am impressed by how many key pieces of the plan seemed to fall into place by happenstance and good fortune. Especially astonishing from this distance is that I was able to find such outstanding “youngsters” and, with but a single tenured appointment *ab initio*, assemble a collection of quite diverse individuals who bonded and worked together to create, essentially *de novo*, one of the top-ranking and admired departments of pathology in the world. Of course, the times were propitious: As David Clayton put it when I spoke with him recently (I paraphrase), it was a special time, a once-in-a-lifetime opportunity to do really fundamental work with small teams—and funding was accessible. The department was very close and very supportive, like a family. And Errol Friedberg felt similarly—the department was already gaining a great reputation, ahead of its time—being there when it was taking off was exhilarating. The program was small, agile, collaborative, and enjoyable; it broke out of the mold of service focused with a few token research PhDs; there was great esprit and excellent collaboration—we were like family. To all those who undertook the cross-continental voyage with me into the largely unknown, I remain forever grateful.

NOTE ADDED IN PROOF

After submitting this manuscript, I was notified by Steve Galli that on June 9, 2011, the Board of Trustees of Stanford University had approved the establishment, by the Stanford Department of Pathology, of the David Korn, MD, Professorship of Pathology in recognition of my “many contributions to the Department of Pathology, the medical school, and the broader pathology community and [that] pays tribute to his unwavering support of experimental pathology as a cornerstone of the discipline and its future advance.” I am truly honored, and delighted that the first incumbent will be Gerald Crabtree.

DISCLOSURE STATEMENT

The author is not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

LITERATURE CITED

1. Fieser LF, Fieser M. 1944. *Organic Chemistry*. Boston: D.C. Heath
2. Harvard Med. Soc. 1956. Notices. *New Engl. J. Med.* 254:773–74
3. Korn D, Bensch K, Liebow AA, Castleman B. 1960. Multiple minute pulmonary tumors resembling chemodectomas. *Am. J. Pathol.* 37:641–72
4. Korn D. 1960. Demonstration of cystine crystals in peripheral white blood cells in a patient with cystinosis. *New Eng. J. Med.* 262:545–48
5. Korn D, Gore I, Blenke A, Collins DP. 1962. Pulmonary arterial bands and webs. An unrecognized manifestation of organized pulmonary emboli. *Am. J. Pathol.* 40:129–51
6. Korn D. 1962. Congenital hypoplastic thrombocytopenia. Report of a case and review of the literature. *Am. J. Clin. Pathol.* 37:405–13
7. Korn D, DeSanctis RW, Sell S. 1962. Massive calcification of the mitral annulus: a clinicopathological study of 14 cases. *New Engl. J. Med.* 267:900–9
8. Korn D, Weissbach A. 1962. Thymineless induction in *Escherichia coli* K12 λ . *Biochim. Biophys. Acta* 61:775–90
9. Weissbach A, Korn D. 1962. The effect of lysogenic induction on the deoxyribonucleases of *Escherichia coli* K12 λ . *J. Biol. Chem.* 234:3312–14
10. Weissbach A, Korn D. 1963. The deoxyribonucleases of *Escherichia coli* K12 λ . *J. Biol. Chem.* 238:3383–89
11. Korn D, Weissbach A. 1963. The effect of lysogenic induction on the deoxyribonucleases of *Escherichia coli* K12 λ . I. The appearance of a new exonuclease activity. *J. Biol. Chem.* 238:3390–94
12. Korn D, Weissbach A. 1964. The effect of lysogenic induction on the deoxyribonucleases of *Escherichia coli* K12 λ . II. The kinetics of formation of a new exonuclease and its relation to phage development. *Virology* 22:91–102
13. Weissbach A, Korn D. 1964. A study of the deoxyribonucleases and the deoxyribonucleic acid polymerase of *Escherichia coli* K12S after infection with the bacteriophage T4r. *Biochim. Biophys. Acta* 87:621–30
14. Korn D. 1964. Some results of phage superinfection during thymineless induction of *Escherichia coli* K12 λ thy-. *Virology* 23:438–41
15. Korn D. 1964. Study of the development of resistance to lambda ind-repression during thymineless induction of *Escherichia coli* K12 λ thy-. *Virology* 24:570–77
16. Korn D, Weissbach A. 1964. Purification and properties of a deoxyribonucleic acid–exonuclease associated with the formation of phage 434. *J. Biol. Chem.* 239:3849–57
17. Korn D, Gelderman A, Cage A, Nathanson D, Strauss AJL. 1967. Immune deficiencies, aplastic anemia, and abnormalities of lymphoid tissue in thymoma. *New Eng. J. Med.* 276:1333–39
18. U.S. Pres. Natl. Inst. Health Study Comm. 1965. Biomedical science and its administration: a study of the National Institutes of Health. Report to the President (Woolridge Committee report). Washington, DC: Gov. Print. Off.
19. U.S. Congr. Comm. Gov. Oper. 1967. The administration of research grants in the public health service. Ninth report by the Committee on Government Operations (second Fountain Committee report). Washington, DC: Gov. Print. Off.
20. Korn D, Protass JJ, Leive L. 1965. A novel effect of actinomycin D in preventing bacteriophage T4 maturation of *Escherichia coli*. *Biophys. Biochem. Res. Commun.* 19:473–81
21. Protass JJ, Korn D. 1966. Impairment of temperate phage adsorption of brief treatment of *Escherichia coli* with dilute solution of EDTA. *J. Bacteriol.* 91:143–47
22. Korn D. 1966. Relation between the CI region of prophage and propagation of bacteriophage T4rII. *Virology* 28:477–79
23. Protass JJ, Korn D. 1966. Inhibition of lysozyme synthesis by actinomycin D in bacteriophage T4-infected cells of *Escherichia coli*. *Proc. Natl. Acad. Sci. USA* 55:832–35

24. Protass JJ, Korn D. 1966. Function of the *N* cistron of bacteriophage λ . *Proc. Natl. Acad. Sci. USA* 55:1089–95
25. Protass JJ, Korn D. 1966. Control of exonuclease and endolysin synthesis during development of bacteriophage λ . *J. Biol. Chem.* 241:4175–79
26. Korn D. 1967. Inhibition of bacteriophage DNA maturation by actinomycin D: the accumulation of “replicating DNA.” *J. Biol. Chem.* 242:160–62
27. Greene RJ, Korn D. 1967. The instability of T4 messenger RNA. *J. Mol. Biol.* 28:435–43
28. Yarmolinsky MB, Korn D. 1968. Evidence for independent segregation of the *Escherichia coli* chromosome and non-replicating bacteriophage λ -b2. *J. Mol. Biol.* 32:475–79
29. Greene RJ, Korn D. 1970. The purification of DNA polymerase from KB cells. *J. Biol. Chem.* 245:254–61
30. Friedberg EC, Walker GC, Siede W. 1995. *DNA Repair and Mutagenesis*. Washington, DC: Am. Soc. Microbiol.
31. Hohn B, Korn D. 1969. Cosegregation of a sex factor and the *Escherichia coli* chromosome during curing by acridine orange. *J. Mol. Biol.* 45:385–95
32. Korn D, Thomas M. 1971. Control of plasmid replication in *Escherichia coli*. I. Correlation of the membrane site of DNA replication with the bacterial segregation unit. *Proc. Natl. Acad. Sci. USA* 68:2047–51
33. Sedwick WD, Wang TS-F, Korn D. 1972. Purification and properties of nuclear and cytoplasmic DNA polymerases from KB cells. *J. Biol. Chem.* 247:5026–33
34. Sedwick W, Wang TS-F, Korn D. 1974. The DNA polymerases of KB cells. *Methods Enzymol.* 29:89–102
35. Wang TS-F, Sedwick W, Korn D. 1974. Nuclear DNA polymerase: purification and properties of the highly purified enzyme from human KB cells. *J. Biol. Chem.* 249:841–50
36. Sedwick WD, Wang TS-F, Korn D. 1975. Cytoplasmic DNA polymerase: structure and properties of the highly purified enzyme from human KB cells. *J. Biol. Chem.* 250:7045–56
37. Wang TS-F, Sedwick WD, Korn D. 1975. Nuclear DNA polymerase: further observations on the structure and properties of the enzyme from human KB cells. *J. Biol. Chem.* 250:7040–44
38. Wang TS-F, Fisher PA, Sedwick WD, Korn D. 1975. Identification of a new DNA polymerase activity in human KB cells. *J. Biol. Chem.* 250:5270–72
39. Weissbach A, Baltimore D, Bollum F, Gallo R, Korn D. 1975. Nomenclature of eukaryotic DNA polymerases. *Science* 190:401–2
40. Eichler DC, Fisher PA, Korn D. 1977. The effect of calcium on the recovery and distribution of DNA polymerase α from cultured human cells. *J. Biol. Chem.* 252:4011–14
41. Fisher PA, Korn D. 1977. DNA polymerase α : purification and characterization of the near homogeneous enzyme from human KB cells. *J. Biol. Chem.* 252:6528–35
42. Mills LB, Stanbridge EJ, Sedwick WD, Korn D. 1977. Purification and partial characterization of the principal DNA polymerase from mycoplasmatales. *J. Bacteriol.* 132:641–49
43. Wang TS-F, Eichler DC, Korn D. 1977. The effect of Mn^{2+} on the in vitro activity of human DNA polymerase β . *Biochemistry* 16:4927–34
44. Eichler DC, Wang TS-F, Clayton DA, Korn D. 1977. In vitro replication of mitochondrial DNA: specific elongation of the endogenous primer sequence in D-loop mitochondrial DNA by human DNA polymerase β . *J. Biol. Chem.* 252:7888–93
45. Korn D, Eichler DC, Fisher PA, Wang TS-F. 1978. Structure and catalytic properties of human DNA polymerases α and β . In *Proceedings of the NATO Advanced Study Institute on DNA Synthesis, Present and Future*, ed. I Molineux, M Kohiyama, pp. 517–58. New York: Plenum
46. Korn D, Fisher PA, Battey J, Wang TS-F. 1978. Structural and enzymological properties of human DNA polymerases α and β . *Cold Spring Harbor Symp. Quant. Biol.* 43:613–24
47. Fisher PA, Wang TS-F, Korn D. 1979. Enzymological characterization of DNA polymerase α : basic catalytic properties, processivity and gap utilization of the homogeneous enzyme from human KB cells. *J. Biol. Chem.* 254:6128–35
48. Fisher PA, Korn D. 1979. Appendix. Theoretical basis for the measurement of average template lengths with DNA polymerases possessing intrinsic 3'-5'-exonuclease activity. *J. Biol. Chem.* 254:6136–37
49. Boxer LM, Korn D. 1979. Structural and enzymological characterization of the homogeneous deoxyribonucleic acid polymerase from *Mycoplasma orale*. *Biochemistry* 18:4742–49

50. Fisher PA, Korn D. 1979. Enzymological characterization of KB cell DNA polymerase α : II. Specificity of the protein–nucleic acid interaction. *J. Biol. Chem.* 254:11033–39
51. Fisher PA, Korn D. 1979. Enzymological characterization of KB cell DNA polymerase α : III. The polymerization reaction with single-stranded DNA. *J. Biol. Chem.* 254:11040–46
52. Wang TS-F, Korn D. 1980. Reactivity of KB cell DNA polymerase α and β with nicked and gapped DNA. *Biochemistry* 19:1782–90
53. Boxer LM, Korn D. 1980. Structural and enzymological characterization of a DNA-dependent ATPase from KB cell nuclei. *Biochemistry* 19:2623–33
54. Fisher PA, Korn D. 1980. KB cell DNA polymerase α : mechanism of primer template recognition by the core catalytic unit. *Mech. Stud. DNA Replication Genet. Recomb.* 19:655
55. Korn D, Fisher PA, Wang TS-F. 1981. Mechanisms of catalysis of human DNA polymerase α and β . *Prog. Nucleic Acids Res.* 26:63–81
56. Fisher PA, Chen JT, Korn D. 1981. Enzymological characterization of KB cell DNA polymerase α . Regulation of template binding by nucleic acid base composition. *J. Biol. Chem.* 256:133–41
57. Fisher PA, Korn D. 1981. Ordered sequential mechanism of substrate recognition and binding by KB cell DNA polymerase α . *Biochemistry* 20:4560–69
58. Fisher PA, Korn D. 1981. Properties of the primer-binding site and the role of Mg^{2+} ion in primer-template recognition by KB cell DNA polymerase α . *Biochemistry* 20:4570–78
59. Wang TS-F, Korn D. 1982. Specificity of the catalytic interaction of human DNA polymerase β with nucleic acid substrates. *Biochemistry* 21:1597–608
60. Filpula D, Fisher PA, Korn D. 1982. DNA polymerase α : common polypeptide core structure of three enzyme forms from human KB cells. *J. Biol. Chem.* 257:2029–40
61. Tanaka S, Hu S-Z, Wang TS-F, Korn D. 1982. Preparation and preliminary characterization of monoclonal antibodies against human DNA polymerase α . *J. Biol. Chem.* 257:8386–90
62. Bensch KG, Tanaka S, Hu S-Z, Wang TS-F, Korn D. 1982. Intracellular localization of human DNA polymerase α with monoclonal antibodies. *J. Biol. Chem.* 257:8390–96
63. Philippe M, Wang TS-F, Hanawalt PC, Korn D. 1982. DNA synthesis on UV irradiated model templates using human DNA polymerases α and β : primer slippage to account for evident transdimer continuity in product. *Biochimie* 64:783–88
64. Korn D, Fisher PA, Wang TS-F. 1983. Enzymological characterization of human DNA polymerases α and β . In *New Approaches in Eukaryotic DNA Replication*, ed. AM de Recondo, pp. 17–55. New York: Plenum
65. Yagura T, Tanaka S, Kozu T, Seno T, Korn D. 1983. Tight association of DNA primase with a subspecies of mouse DNA polymerase α . *J. Biol. Chem.* 258:6698–700
66. Wang TS-F, Hu S-Z, Korn D. 1984. DNA primase from KB cells: characterization of a primase activity tightly associated with immunoaffinity-purified DNA polymerase α . *J. Biol. Chem.* 259:1854–65
67. Hu S-Z, Wang TS-F, Korn D. 1984. DNA primase from KB cells: evidence for a novel model of primase catalysis by a highly purified primase/polymerase α complex. *J. Biol. Chem.* 259:2602–9
68. Lan NC, Wang TS-F, Johnson LK, Korn D. 1984. Monoclonal antibodies against human DNA polymerase α do not cross-react with glucocorticoid receptors. *J. Steroid Biochem.* 20:251–54
69. Miller MR, Ulrich RG, Wang TS-F, Korn D. 1985. Monoclonal antibodies against human DNA polymerase α inhibit DNA replication in permeabilized human cells. *J. Biol. Chem.* 260:134–38
70. Wang TS-F, Pearson BE, Suomalainen HA, Mohandas T, Shapiro LJ, et al. 1985. Assignment of the gene for human DNA polymerase α to the X chromosome. *Proc. Natl. Acad. Sci. USA* 82:5270–74
71. Wong SW, Paborsky LR, Fisher PA, Wang TS-F, Korn D. 1986. Structural and enzymological characterization of immunoaffinity-purified DNA polymerase α –DNA primase complex from KB cells. *J. Biol. Chem.* 261:7958–68
72. Wong SW, Wahl AF, Yuan P, Arai N, Pearson BE, et al. 1988. Human DNA polymerase α gene expression is cell proliferation dependent and its primary structure is similar to both prokaryotic and eukaryotic replicative DNA polymerases. *EMBO J.* 7:37–47
73. Miller MA, Korn D, Wang TS-F. 1988. The evolutionary conservation of DNA polymerase α . *Nucleic Acids Res.* 16:7961–73

74. Wahl AF, Geis AM, Spain BH, Wong SW, Korn D, Wang TS-F. 1988. Gene expression of human DNA polymerase α during cell proliferation and the cell cycle. *Mol. Cell. Biol.* 8:5016–25
75. Wang TS-F, Korn D, Wong SW. 1988. Human DNA polymerase α : protein structure and molecular genetic characterization. *Cancer Cells* 6:385–92
76. Wang TS-F, Wong SW, Korn D. 1989. Human DNA polymerase α : predicted functional domains and relationships with viral DNA polymerases. *FASEB J.* 3:14–21

RELATED RESOURCES

1. Anderson RE, Benson ES, Korn D, Martin GM, Robbins SL, et al. 1977. Carcinogenesis testing and the pathologist. *Hum. Pathol.* 8:353–54
2. Korn D. 1988. What is peer review? *Stanford Univ. Campus Rep.* 20:18
3. Korn D. 1989. The history and present status of the School of Medicine. *Stanford Univ. Campus Rep.* 21:16
4. Korn D. 1989. Dealing with scientific fraud and misconduct. *Issues Sci. Technol.* 5:21–22
5. Korn D. 1989. The culture of the research university. In *Project on Scientific Fraud and Misconduct. Report on Workshop Number Three*. AAAS-ABA National Conference of Lawyers and Scientists, pp. 43–48. Washington, DC: Am. Assoc. Adv. Sci.
6. Korn D. 1989. Report on the implications of physician supply issues on programs for the education of biomedical scientists. In *Supplying Physicians for Future Needs, The Report of the Task Force on Physician Supply*, pp. 4.1–4.35; Washington, DC: Assoc. Am. Med. Coll.
7. Korn D. 1991. Comments on “A Plan for Managing the Costs of Biomedical Research.” *Stanford Univ. Campus Rep.* 23:13
8. Korn D. 1991. Scientific integrity and scientific misconduct: the interface between research institutions and journals. In *Peer Review in Scientific Publishing*, ed. DT Kirkendall, pp. 205–12. Chicago: Counc. Biol. Ed.
9. Korn D. 1992. Cancer screening and public education: commentary. In *Promoting Health and Presenting Disease*, ed. DJ Scherl, J Noren, M Osterweis, pp. 167–73. Washington, DC: Assoc. Acad. Health Cent.
10. Korn D. 1992. A statesman reflects: David Korn discusses biomedical research. *J. Natl. Cancer Inst.* 84:664–66
11. Korn D. 1993. Conflict of interest—a university perspective. In *Ethical Issues in Research*, ed. D Cheney, pp. 113–25. Frederick, Md: Univ. Publ. Group
12. Korn D. 1993. Human subjects: protection, policies, and practice. Commentary. In *Emerging Policies for Biomedical Research*, ed. WN Kelley, M Osterweis, ER Rubin, pp. 197–203. Washington, DC: Assoc. Acad. Health Cent.
13. Korn D. 1995. Preface. In *Academic Health Centers in the Managed Care Environment*, ed. D Korn, CJ McLaughlin, M Osterweis. Washington, DC: Assoc. Acad. Health Cent.
14. Korn D. 1995. Academic medicine in an era of resource constraints. In *The Academic Health Center in the 21st Century*, ed. R Snyderman, VY Saito, pp. 70–75. Durham: Duke Univ. Med. Cent. Health Syst.
15. Korn D. 1995. The JIM interview: David Korn MD. *J. Investig. Med.* 43:108–15
16. Fogelman AM, Goode LD, Behrens BL, DeAngelis CD, Forsyth JD, et al. 1996. Preserving medical schools’ academic mission in a competitive marketplace. *Acad. Med.* 71:1168–99
17. Korn D, Jones RF. 1996. *The Financing of Medical Schools. A Report of the AAMC Task Force on Medical School Financing*. Washington, DC: Assoc. Am. Med. Coll.
18. Korn D. 1996. Bioethical issues. *Science* 272:1248–49
19. Korn D, Jones RF. 1996. More on compensation for teaching. *New Eng. J. Med.* 335:1537

20. Houpt JL, Goode LD, Anderson RJ, Aschenbrenner CA, DeAngelis CD, et al. 1997. How medical schools can maintain quality while adapting to resource constraints. *Acad. Med.* 72:180–85
21. Korn D. 1997. Medical school funding. *Science* 275:1863–64
22. Korn D. 1998. FDA reform: unintended outcome? *Science* 279:5354
23. Korn D. 1997. Genetic privacy and the use of archival patient materials and data in research. In *Proceedings of the Public Health Conference on Records and Statistics and the Data Users Conference on Partnerships, Technologies and Communities: Evolving Roles for Health Data*. CD-ROM no. 1. Washington, DC: Dep. Health. Hum. Serv.
24. Korn D. 1998. Genetic privacy and the use of archival patient materials in research. <http://ASIP.uthscsa.edu/Korn.html>
25. Korn D. 2000. Contribution of the human tissue archive to the advancement of medical knowledge and the public health. In *Research Involving Human Biological Materials: Ethical Issues and Policy Guidance*, Vol. II, *Commissioned Papers*, ed. HT Shapiro, P Backlar, A Brito, AM Capron, EJ Cassell, et al., pp. E1–30. Rockville, Md.: Natl. Bioeth. Advis. Comm.
26. Korn D. 1999. Genetic privacy, medical information privacy and the use of human tissue specimens in research. In *Genetic Testing and the Use of Information*, ed. C Long, pp. 16–83. Washington, DC: Am. Enterp. Inst.
27. Korn D, McCabe C. 1999. Confidentiality of medical records. AAMC report on the Minnesota experience. <http://www.aamc.org/advocacy/issues/research/minrepot.htm>
28. Abramson SB, Flexner C, Snyderman R, Dieterich DT, Korn D, et al. 1999. Patients, physicians, and clinical trials: the other side of the coin. *J. Investig. Med.* 47:343–57
29. Holmes EW, Burks TF, Dzau V, Hindery MA, Jones RF, et al. 2000. Measuring contributions to the research mission of medical schools. *Acad. Med.* 75:304–13
30. Korn D. 2000. The NIH guidelines on stem cell research. *Science* 289:1877
31. Korn D. 2000. Scientific due process. *Issues Sci. Technol.* 17:5–6
32. Korn D, Meyer RE. 2000. Patents for intellectual property. *Lancet* 356:2015
33. Korn D. 2001. Conflicts of interest. *Science* 292:639
34. Korn D. 2002. Protecting patients' privacy. *Lancet* 359:84
35. Korn D, Heinig SJ, eds. 2002. Public versus private ownership of scientific discovery: legal and economic analyses of the implications of human gene patents. *Acad. Med.* 77:1301–401
36. Korn D. 2002. Scientific misconduct: The state's role has limits. *Nature* 420:739
37. Meyer RE, Korn D. 2003. Clinical-trial agreements between medical schools and industry. *New Engl. J. Med.* 348:476
38. Heinig S, Korn D. 2003. Agricultural IP and the public sector. *Science* 302:781–82
39. Schneider B, Korn D. 2004. NIH report card. *New Engl. J. Med.* 350:515–16
40. Heinig SJ, Korn D. 2004. More money, more results. *Chron. High. Educ.* 2004:B17–18
41. Korn D. 2004. The postdoctoral workforce. *Science* 304:516
42. Korn D. 2004. Managing conflicts of interest. *Health Aff.* 23:286–87
43. Alpert JS, Shine KI, Adams RJ, Antman EM, Kavey RE, et al. 2004. The ACCF and AHA codes of conduct in human subjects research. *J. Am. Coll. Cardiol.* 44:1724–28
44. Ehringhaus SH, Korn D. 2003. U.S. medical school policies on individual financial conflicts of interest. <http://www.aamc.org/research/coi/coiresults2003.pdf>
45. Korn D, Stanski DR, eds. Drug development science: obstacles and opportunities for collaboration among academia, industry and government. <http://www.aamc.org/research/initiatives.htm>
46. Hakimian R, Korn D. 2005. Ownership and use of tissue specimens in research. *J. Am. Med. Assoc.* 293:1325–26

47. Korn D, Ehringhaus SH. 2005. NIH conflicts rules are not right for universities. *Nature* 434:821
48. Korn D, Heinig SJ. 2005. Biotechnology products and university-based science. *J. Am. Med. Assoc.* 293:2862–63
49. Dickler HB, Korn D. 2005. The costs of institutional review boards. *New Engl. J. Med.* 353:315–16
50. Fang D, Dickler HB, Heinig SJ, Korn D. 2007. Recruitment of new physician investigators in clinical research: findings from a survey of clinical department chairs at U.S. medical schools. *Anal. Brief* 7:5. <http://www.aamc.org/juniorclinicalinvestigators>
51. Heinig SJ, Korn D. 2007. Missing the mark on biomedical research. *Nature* 450:27
52. Ehringhaus SH, Korn D, eds. 2007. *The Scientific Basis of Influence and Reciprocity: A Symposium*. Washington, DC: Assoc. Am. Med. Coll.
53. Ehringhaus SH, White P, Korn D, eds. 2008. *Protecting Patients, Preserving Integrity, Advancing Health: Accelerating the Implementation of COI Policies in Human Subjects Research. A Report of the AAMC-AAU Advisory Committee on Financial Conflicts of Interest in Human Subjects Research*. Washington, DC: Assoc. Am. Med. Coll.
54. Ehringhaus SH, Korn D, eds. 2008. *Report of the AAMC Task Force on Industry Funding of Medical Education*. Washington, DC: Assoc. Am. Med. Coll.
55. Korn D, Heinig SJ. 2009. The ecology of physician scientists in academic medicine. In *The Vanishing Physician Scientist?*, ed. I Andrew, MD Schafer, pp. 50–66. Ithaca: Cornell Univ. Press
56. Korn D, Heinig SJ. 2009. Restoring and invigorating an endangered species: initiatives by the Association of American Medical Colleges. See *Relat. Res.* 55, pp. 208–26
57. Bienenstock A, Korn D. 2010. A faster path from lab to market (letter). *Harvard Bus. Rev.* April:16
58. Korn D. 2010. The challenge of public expectations. *Boston Rev.* 35:11
59. Korn D. 1989. Funding for cancer centers: A challenge of scarce resource allocation. *J. Natl. Cancer Inst.* 81:1870–73
60. Korn D. 1996. Reengineering academic medical centers: reengineering academic values? *Acad. Med.* 71:1033–43
61. Korn D. 1996. Dangerous intersections. New proposals to protect genetic privacy may collide with the public interest in fostering medical research. *Issues Sci. Technol.* 13:55–62
62. Jones RF, Korn D. 1997. On the cost of medical student education. *Acad. Med.* 72:200–10
63. Korn D. 1997. FDA under siege: the public at risk. *Science* 276:1627
64. Korn D. 1998. Academic medical centers: whence they came, where they went. *J. Soc. Gynecol. Investig.* 5:227–36
65. Heinig SJ, Quon ASW, Meyer RE, Korn D. 1999. The changing landscape for clinical research. *Acad. Med.* 74:725–45
66. Jacobs CD, Bergen MR, Korn D. 2000. Impact of a program to diminish gender insensitivity and sexual harassment at a medical school. *Acad. Med.* 75:465–69
67. Korn D. 2000. Medical information privacy and the conduct of biomedical research. *Acad. Med.* 75:963–68
68. Smith BLR, Korn D. 2000. Is there a crisis of accountability in the American research university? *Minerva* 38:129–45
69. Korn D. 2000. Conflicts of interest in biomedical research. *J. Am. Med. Assoc.* 284:2234–37
70. Korn D. 2001. Medical information privacy and the conduct of biomedical research. In *Biomedical Ethics Reviews: Privacy and Health Care*, ed. JM Humber, RF Almeder, pp. 101–29. New York: Humana

71. Kulynych J, Korn D. 2002. The effect of the new federal medical-privacy rule on research. *New Engl. J. Med.* 346:201–4
72. Andrew JTG, Gale R, Winston R, Korn D. 2002. Research governance at the crossroads. *Nat. Med.* 8:99–101
73. Korn D, Rich RS, Garrison HH, Golub SH, Hendrix MJC, et al. 2002. The NIH budget in the “postdoubling” era. *Science* 296:1401–2
74. Kulynych J, Korn D. 2002. Use and disclosure of health information in genetic research: weighing the impact of the new federal medical privacy rule. *Am. J. Law Med.* 28:309–24
75. Korn D. 2002. Industry, academia, investigator: managing the relationships. *Acad. Med.* 77:1089–95
76. Kulynych J, Korn D. 2002. The effect of the new federal medical-privacy rule on research. *New Engl. J. Med.* 346:201–4
77. Kulynych J, Korn D. 2002. The new federal medical privacy rule. *New Engl. J. Med.* 347:1133–34
78. Korn D, Heinig SJ. 2002. Patents, genomics, and academic medicine. *Acad. Med.* 77:1301–8
79. Ehringhaus S, Korn D. 2002/2003. Conflicts of interest in human subjects research. *Issues Sci. Technol.* 19:75–81
80. Kulynych J, Korn D. 2003. The new HIPAA (Health Insurance Portability and Accountability Act of 1996) medical privacy rule: help or hindrance for clinical research? *Circulation* 108:912–14
81. Mallon WT, Korn D. 2004. Bonus pay for research faculty. *Science* 303:476–77
82. Korn D, Heinig S. 2004. Recoupment efforts threaten federal research. *Issues Sci. Technol.* 20:26–30
83. Hakimian R, Korn D. 2004. Ownership and use of tissue specimens in research. *J. Am. Med. Assoc.* 292:2500–5
84. McKinney R, Korn D. 2005. Should an institution which has commercial rights in a new drug or device be allowed to evaluate the technology? *PLoS Med.* 2(1):e9
85. Korn D. 2005. Maintaining the integrity of scientific research. *J. Law Policy* 13:7–15
86. Korn D, Ehringhaus S. 2006. Principles for strengthening the integrity of clinical research. *PLoS Clin. Trials* 1(1):e1
87. Dickler HB, Korn D, Gabbe SG. 2006. Promoting translational and clinical science: the critical role of medical schools and teaching hospitals. *PLoS Med.* 3:e378
88. Dickler HB, Fang D, Heinig SJ, Johnson E, Korn D. 2007. New physician investigators receiving NIH research project grants: an historical perspective on the “endangered species.” *J. Am. Med. Assoc.* 297:2496–501
89. Heinig SJ, Krakower JY, Dickler HB, Korn D. 2007. Sustaining the engine of U.S. biomedical discovery. *New Engl. J. Med.* 357:1042–47
90. Alexander H, Heinig SJ, Fang D, Dickler H, Korn D. 2007. Contributions of international medical graduates to U.S. biomedical research: the experience of U.S. medical schools. *J. Investig. Med.* 55:410–14
91. Psaty BM, Korn D. 2007. Congress responds to the IOM drug safety report—in full. *J. Am. Med. Assoc.* 298:2185–87
92. Korn D. 2011. Financial conflicts of interest in academic medicine: whence they came, where they went. *Indiana Health Law Rev.* 8:3–42