This interview was supported by a donation from Emory Children’s Center
PREFACE

Oral history has its roots in the sharing of stories which has occurred throughout the centuries. It is a primary source of historical data, gathering information from living individuals via recorded interviews. Outstanding pediatricians and other leaders in child health care are being interviewed as part of the Oral History Project at the Pediatric History Center of the American Academy of Pediatrics. Under the direction of the Historical Archives Advisory Committee, its purpose is to record and preserve the recollections of those who have made important contributions to the advancement of the health care of children through the collection of spoken memories and personal narrations.

This volume is the written record of one oral history interview. The reader is reminded that this is a verbatim transcript of spoken rather than written prose. It is intended to supplement other available sources of information about the individuals, organizations, institutions, and events that are discussed. The use of face-to-face interviews provides a unique opportunity to capture a firsthand, eyewitness account of events in an interactive session. Its importance lies less in the recitation of facts, names, and dates than in the interpretation of these by the speaker.

Historical Archives Advisory Committee, 2007/2008

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ABOUT THE INTERVIEWER

Richard R. Ricketts, MD, FACS, FAAP

Dr. Richard R. Ricketts is Professor of Surgery and Chief of the Division of Pediatric Surgery at Emory University School of Medicine in Atlanta, Georgia. He attended Stanford University as an undergraduate and then went to Northwestern University Medical School. He did his surgical internship and general surgery residency at the LA County-University of Southern California Medical Center in Los Angeles, California under Dr. Leonard Rosoff from 1973 – 1978. He did his pediatric surgery training at the Children’s Memorial Hospital in Chicago under the direction of Dr. John Raffensperger. Dr. Ricketts was recruited by Dr. W. Dean Warren to come and establish a Division of Pediatric Surgery at Emory University in 1980.

Dr. Ricketts gradually expanded the Division and by 1995 a training program in pediatric surgery was approved by the ACGME. He was the Program Director for 10 years from 1996 – 2006.

Dr. Ricketts has been a member of the American Academy of Pediatrics for 25 years. In 2003, he was elected to the Executive Committee and served as Chair of the Surgical Section of the American Academy of Pediatrics in 2008.

He and his wife Sandy have been married for 39 years. They have one daughter and two granddaughters, with whom they spend as much time as possible.
Interview of Thomas E. Starzl, MD

DR. RICKETTS: I’m talking to Dr. Starzl, and to let you know that we're here, we’ll test to be sure it’s working.

DR. STARZL: I’m here. My voice is bad today, but I’ll do the best I can.

DR. RICKETTS: Okay.

[Recording interruption.]

DR. STARZL: What has that got, about a 30-minute time span?

DR. RICKETTS: This has a 60-minute time span, and I have three of them. That doesn’t mean we have to be here for three hours, though.

DR. STARZL: Okay. [Laughs]

DR. RICKETTS: We have that much time if it turns out to be that much. So let me just say I’m Richard Ricketts. I’m here with Dr. Thomas E. Starzl at one of his offices, in Pittsburgh. The date is February 25, 2007. Dr. Starzl is the 1993 recipient of the [William E.] Ladd Medal, which is the highest honor given by the surgical section [Section on Surgery] of the American Academy of Pediatrics.

Dr. Starzl, thank you for meeting with me on this chilly day in Pittsburgh. I’d like to go over some of the aspects of your life and your career development, and talk something about the development of the field of transplantation, particularly as it applies to children, and then pick your mind for what you think may occur in the future with respect to transplantation. So let’s start off with your hometown. Tell me about your little hometown of Le Mars, Iowa.

DR. STARZL: It was a rather isolated town, with a population of 5,000. The nearest big city was Sioux City, Iowa, which had a population of about 70,000. This, of course, was all of 80 years ago. And Le Mars has still got a population of 5,000, and Sioux City still has a population of 70,000, so nothing much has changed. Beyond that about 60 miles was Omaha.

DR. RICKETTS: Do you ever go back to visit Le Mars?

DR. STARZL: Yes, I’ve been back a number of times, but not recently. I haven’t been back there for the last 13 or 14 years because all the people [I knew] out there have died, except for a half sister who now is a full-grown woman and has had children. But I never really knew her very well, so there isn’t much, hasn’t been much of an incentive.
DR. RICKETTS: And in high school, did you play sports?

DR. STARZL: Yes, I did.

DR. RICKETTS: What did you play?

DR. STARZL: I played everything. [Laughs]

DR. RICKETTS: You did?

DR. STARZL: But especially basketball and football.

DR. RICKETTS: And I noticed that you’ve been really quite active during your entire life, particularly with cycling. Can you tell me about some of the interesting cycling trips you have taken?

DR. STARZL: I did one huge one, which was a trip above the Arctic Circle in 1972. That involved flying up to the most northern place we could fly into, which was Alta [Norway], and then cycling north to places like Honningsvåg [Norway] and, best known, Hammerfest [Norway], which are way above the Arctic Circle. And then we did a sea voyage back down to a somewhat lower place, a more southern place. And then [we] cycled from there down to Helsinki [Finland], which was—it was a long trip, about 2,000 miles altogether.

DR. RICKETTS: Were there base camps along the Arctic?

DR. STARZL: No.

DR. RICKETTS: Or did you stay in little cities?

DR. STARZL: No, no, we camped out. We brought our own equipment. There were five of us. My two boys were with me, and a young surgeon-in-training, and one of the chief lab technicians, an African-American guy. We had these nice Arctic tents and split them up so we each had some component. We carried these on ten-speed bikes, and just cycled. We had to average 50, 70 miles a day.

DR. RICKETTS: So that took you how long?

DR. STARZL: It took us 30 days.

DR. RICKETTS: Thirty days.

DR. STARZL: Yes.
DR. RICKETTS: That’s a lot of cycling.

DR. STARZL: Yes, we were worn out by the time—worn out, and as we got farther south we were besieged by the mosquitoes that seemed to us the size of birds.

DR. RICKETTS: Is that right?

DR. STARZL: Yes.

DR. RICKETTS: Do you still ride your bike?

DR. STARZL: No, I gave that up, actually, after I came here [Pittsburgh], mainly because, although I started riding a bike—I had two bikes that were stolen. I didn’t have a safe place to keep them. Besides that, I thought it was getting more and more dangerous, so I stopped. Also, I live up the hill here only about three blocks [from here]. Although most of the people who work here [University of Pittsburgh Medical Center] live out in [the] suburbs, it was better for me to be close by, so I could walk down to the emergency room in five minutes.

DR. RICKETTS: What was your favorite subject in high school?

DR. STARZL: Well, I was pretty undifferentiated in high school. I think I was interested in everything. I had some thought of becoming a priest, because there had been so many priests in the family and, particularly, so many nuns. That generation of my maternal grandfather consisted of him (Thomas Fitzgerald) and his brother and four girls, two of whom became nuns. So I think my priestly ambition was an idea that would have been pleasing to certainly my mother, and probably my dad also who was an Austrian Catholic.

DR. RICKETTS: We discussed you’re from a Catholic family, then.

DR. STARZL: Yes, and the point [relative to thoughts of priesthood] is that I was a good student in Latin. If there was any subject that I probably concentrated on more than any other, it would have been Latin.

DR. RICKETTS: Do you think that had anything to do with your future career in medicine?

DR. STARZL: I think it had a pervasive effect, because it made it easy for me to read all those Latin languages. I can actually make sense out of any romance language, although I don’t speak any of them very well and can
get along in public only in France. But still, it established a vocabulary base, and so that was helpful.

**DR. RICKETTS:** What did your father do?

**DR. STARZL:** He owned a newspaper, a small-town newspaper.

**DR. RICKETTS:** Did you work with him on the newspaper?

**DR. STARZL:** Yes, he expected me to work. [Laughs]

**DR. RICKETTS:** [Laughs]

**DR. STARZL:** So I went to work as a paperboy and then graduated to a devil in the print shop. I learned how to do linotyping, and then I learned printing and then I did some composition. I wrote articles, did journalism work and created an atlas of the particular county that we lived in, and published that.

**DR. RICKETTS:** And you did that during high school?

**DR. STARZL:** Yes. Or actually quite a bit of that was done even before high school.

**DR. RICKETTS:** Really? Did you ever work in the newspaper business again?

**DR. STARZL:** Yes, I did. I worked for the *Chicago Tribune*. I did that just after the end of my freshman year of medical school.

**DR. RICKETTS:** Did that help you learn how to write scientifically, do you think?

**DR. STARZL:** Well I think it did, yes. But it put a potential stain on my scientific writing, or maybe you might even call it a potential advantage. I always liked to write in a way that normal people could understand. A lot of people who do science writing seem to try to confuse the reader or at least, perhaps, cover up whatever the message is with jargon that is comprehensible only to the learned, to the insiders. And I was always determined to try to say things in ways that non-science people, non-professional people could understand. That involved the use of phrases that you might hear in the marketplace or in a casual conversation. You have no idea how much trouble that got me into. I was constantly being criticized by reviewers.
DR. RICKETTS: Right. Well, so it seems like that work was beneficial to you.

DR. STARZL: Well, I think in the long run—it was probably beneficial.

DR. RICKETTS: Now, what did your mother do?

DR. STARZL: She was two things. She started out being a schoolteacher, as did all of her sisters, and she had five of those. They were all educators - teachers. And then, secondarily, she became a nurse.

DR. RICKETTS: Do you have any siblings?

DR. STARZL: Yes, I did. I had a brother and two sisters. One sister is still alive. She’s coming up to 80 now. But the others are dead.

DR. RICKETTS: Sorry. Can we adjust to the present for just a minute? Tell me about your wife, Joy, and your three children.

DR. STARZL: Well, yes, this is my second wife. My first wife divorced me in 1975 and promptly remarried. I was quite surprised when that happened, and it was quite devastating, not only for me but also for the three children. Several years after that I met Joy, my present wife, and we’ve been married now for 27 years.

DR. RICKETTS: How did you meet her?

DR. STARZL: She was a laboratory technician at the University of Colorado, working at the Clinical Research Center [where we were doing] important work on growth factors. It [the research] was related in one way or another to liver physiology and liver transplantation. So when we got into that, we needed a full-time technologist. We recruited her, and that’s how I met her.

DR. RICKETTS: I actually heard that she hit you when you were riding your bicycle to work. [Laughs]

DR. STARZL: That story is true.

DR. RICKETTS: [Laughs] Knocked you off your feet!

DR. STARZL: It very definitely brought her to my attention!

DR. RICKETTS: What about [your children] Rebecca, Tim [Timothy] and Tom[Thomas]? What are they doing now?
DR. STARZL: They’re floating around. They’re in the west doing various things. Tim right now is in India. I think he’s setting up a new company. He has been an entrepreneur, and he has formed several companies, and right now he’s starting a new one. Tommy [his younger brother] has never focused on a specific professional objective, but has always been one of the nicest people I know.

DR. RICKETTS: [Laughs]

DR. STARZL: And Becky’s got some health problems right now.

DR. RICKETTS: How were you able to balance your extremely busy career with raising three young children?

DR. STARZL: That was a tremendous problem because the children ranged from only a few weeks [Tommy] to 4 years of age when I began the work in liver transplantation [in 1958] that was to occupy me for the rest of my life. I was not in a good position to manage the professional problems and to pay sufficient attention to either my first wife or the children. This already was apparent when I moved from Northwestern University (Chicago) to the University of Colorado in 1961.

By then, I had been working on experimental liver transplantation for about four years, and this had become somewhat of a passion. Long before we went to Colorado, I had concluded that the real problem [that faced liver transplantation] was going to be immunosuppression. We had worked out all the components of the complex liver transplant operation, but there still remained the big question, and that was: How do you prevent rejection?

I had talked to [Francis D.] “Franny” Moore as early as 1959 and 1960 about coming up to the Brigham [Peter Bent Brigham Hospital, now Brigham and Women’s Hospital] and seeing what could be done about controlling rejection, because they were working on that. But he [Franny] had already made arrangements for [Sir] Roy Calne to come there [as a research fellow]. This was a fairly small group, and they didn’t have room for me. Besides that, Franny may have been annoyed because [laughs], I was developing the same operation that he was [liver transplantation in dogs]. We had done this completely independently, as far as I can tell.

Up to this time, my thought had been, “Well, if they develop the immunosuppression, I’ll work on other problems.” These [other problems] were technical and physiologic: for example, what was the proper way is to revascularize the liver, and where should you put [transplant] it. Other examples were how to preserve organs and what to do about the coagulation problems and specific details of surgical technique. I thought at first that
after dealing with these issues, we would just borrow the immunosuppression that was being developed by Joe [Joseph] Murray at the Brigham for kidney transplantation. But as the time came to move from Chicago to Colorado, we were getting grim messages from Boston about their results using Imuran for the first time in a clinical kidney transplant trial. Although they had a single patient who eventually lived with a functioning kidney graft for about 17 months, there were about a dozen others who all died in less than six months. At the time that I decided to go to Colorado, we were poised to go forward in Denver with a clinical trial of liver transplantation. This Boston experience with Imuran caused a drastic change in my plans.

In Colorado, I got a supply of Imuran of my own and tried, at first, to use it for liver transplantation in dogs. It was a difficult objective. This was too complex an operation with which to study immunosuppression. So I made a switch and said, “We will develop immunosuppression ourselves and do it in dogs with the simpler kidney model. The road to the liver has got to be through our own kidney program.” Actually, that was the reason for starting the kidney transplant program in Colorado. After doing extensive experiments using Imuran in dogs, we did our first human kidney transplant case in March, 1962.

By early 1963, the combination of Imuran and prednisone that we had worked out in the dog lab had allowed us to churn out a large group of kidney recipients. I would say that even we were surprised how good the outcomes were in humans. In recipients of kidneys from live related donors, we had an 80 percent one-year survival. This series began at a time when, in the world, there had been, over [the preceding] period of about four years, only six examples of survival as long as one year after kidney transplantation. Five of these six recipients had been treated with total body irradiation—the first in Boston by Joe Murray and the next four in Paris. The sixth recipient was the Brigham patient I mentioned a moment ago who was treated with Imuran. When we started our kidney program, the field of clinical organ transplantation seemed to be dead in the water.

When we reported our results—hardly anybody could believe them except Dave [David M.] Hume [Chairman of Surgery, Medical College of Virginia, Richmond], and Will [Willard] Goodwin [Chief of Urology at UCLA]. Goodwin visited us in Colorado in May 1963, and saw first hand what was going on as described in The Puzzle People. During 1962-63, I was in almost daily telephone contact with Dave Hume and shared with him all of the rapidly accumulating information about the use of the double drug treatment [Imuran and prednisone]. He was just starting his program in Richmond, used the two drugs, and also got good results. The Brigham people were delayed in combining prednisone with Imuran, primarily because Roy Calne—now in Boston, having come over from England—had actually tested Imuran and prednisone in dogs, using both drugs together from the day of
kidney transplantation. Survival in dogs using the two drugs in this way was worse than using Imuran alone. They remained convinced that the prednisone should be avoided until sometime pretty late in 1964.

DR. RICKETTS: You said earlier on that the road to the liver was [through] the kidney. I think that’s because you have an extra kidney to deal with if the transplant fails, and it’s easier to study.

DR. STARZL: It wasn’t so much that. My assumption—and it was a correct one—was that whatever immunosuppression treatment worked with the kidney was going to be applicable to every organ. In my book of 1964 [Experience in Renal Transplantation], I have a whole chapter explaining why I thought that had to be the case. That view was not initially shared by Franny Moore who suspected that every organ was going to have its own rules. My conviction was that if you simply could make kidneys work, all you had to do [for other organs] was, with some probably rather minor modifications, to use the same immunosuppression.

That proved to be true, but the explanation was not worked out completely until 1992. This was the migratory passenger leukocyte story.

DR. RICKETTS: I want to get to that, I think. But let’s go back to your career development a little bit.

DR. STARZL: But anyway, the reason for saying that the road to the liver had to go through the kidney was because I thought if you could work out the immunosuppression for the kidney, then surely all those lessons would be applicable to every organ. That was the main reason.

There was another theoretical reason why kidney transplantation was more defensible. Although chronic dialysis was not available as a realistic option in 1962, you already could make the argument that it was more ethical to do a kidney [transplant] because you had an artificial organ backup.

DR. RICKETTS: You have a fallback.

DR. STARZL: You’ve got a fallback. But with the liver or heart, you simply didn’t have anywhere you could go except transplantation. You either succeeded or failed. End stage liver disease was a death sentence without transplantation, but exactly when was not known. With a failed liver or heart transplantation, death could be on the day of operation.

DR. RICKETTS: So what about—let’s go back in your career development. Where did you go to medical school?
DR. STARZL: Northwestern [Northwestern University Medical School, now the Feinberg School of Medicine, Northwestern University].

DR. RICKETTS: That’s right, we both went to Northwestern.

DR. STARZL: Yes.

DR. RICKETTS: We talked about that. Who at Northwestern was most influential in your ultimate career?

DR. STARZL: By all odds, a guy named H.W. Magoun, Horace W. Magoun. He was a great neurophysiologist, a great neuroanatomist. In fact, as you know, I dropped out of [medical] school for almost two years—

DR. RICKETTS: You got a PhD.

DR. STARZL: Yes, in neuroscience. But there were some other people who always played a big role. One of them was a guy named Leslie B. Arey. I don’t know whether that name means anything—

DR. RICKETTS: Yes, yes.

DR. STARZL: —to you, but he was already about 90 years old when I was there.

DR. RICKETTS: He must have been over 100 when I was there.

DR. STARZL: Was he still around?

DR. RICKETTS: He was still teaching. He was still teaching human embryology.

DR. STARZL: I’m sure he’s dead by now. Is he?

DR. RICKETTS: I don’t know.

DR. STARZL: [Laughs]

DR. RICKETTS: I suspect so.

DR. STARZL: He’d be 130 if he wasn’t. But he was a fine teacher. I had an office next to him, and even when I was in neuroanatomy he was always there, so he was a big influence. I think Loyal Davis also was a significant influence.

DR. RICKETTS: How’s that?
DR. STARZL:   Well, it’s just—

DR. RICKETTS:   He was a neurosurgeon.

DR. STARZL:   Yes, he was a neurosurgeon. I don’t know, he was just a flamboyant character. I had a great deal of affection for him, and for Magoun. And incidentally, Magoun and Loyal Davis sometimes seemed to dislike each other. [Laughs]

DR. RICKETTS:   Had you ever thought about becoming a neurosurgeon?

DR. STARZL:   Oh, yes. Well, everyone thought I was slated for that. In fact, when I went to [Johns] Hopkins [Hospital], the other option was to go to the MGH [Massachusetts General Hospital]. They were interested in my coming up there because I had the PhD in neuroscience. There was a guy named [James C.] White who was head of their neurosurgery department, and the work that I had done with Magoun in neurophysiology was really quite influential. Even today it’s still quite influential. The Brain [Research] Institute logo at UCLA [University of California, Los Angeles] is based on the work that was done with Magoun in those days.

DR. RICKETTS:   So do you believe that getting the PhD and the research involved ultimately benefited your career?

DR. STARZL:   Oh, for sure. I think it dominated my career, because it established a real science base that was broadly applicable. I think, in some ways, it was good not to stay in neuroscience, because what happens if you stay in your first field is that at the beginning you learn some technology, and then you’re always seeking some way to exploit the technology. But if you establish a base—kind of an intellectual platform—and then leave the field behind, from that point onward, if you encounter a problem or something interesting in your practice you’re in a position to ask a question and figure out, “what technology do I need,” instead of being led around by the original technology—looking for ways to apply it. You’re not chained to the technology. The question and the curiosity drives you. It’s just the other way around.

DR. RICKETTS:   There is one interesting story I’d like you to tell me a little bit about, and that is you purchased the cadavers while you were in medical school.

DR. STARZL:   Yes.

DR. RICKETTS:   How did you ever get away with that?
DR. STARZL: You could just do it.

DR. RICKETTS: Yes?

DR. STARZL: Yes. My dad had to pay the bill [chuckles], but—

DR. RICKETTS: And you studied the anatomy yourself?

DR. STARZL: I did it myself, yes. I had gone through the course [two years earlier as a freshman]. I don’t know how they do it these days, but I had to share that cadaver with other people.

DR. RICKETTS: We had four when I was there.

DR. STARZL: Yes, we had four, too. Was there a guy named Scotty around when you were around?

DR. RICKETTS: Don't recall.

DR. STARZL: Scotty was this strange guy who was in charge of dead bodies. [Laughs] No, you could just go down there and ask if you could buy your own cadaver, and you could do it. And so I bought the cadaver [in my junior year]. I was dissatisfied [with the earlier freshman course] because I was with these three other people, and you’d take turns [dissecting]. Somebody would do an arm, and you’d come along and you’d see what they had messed up, and I just didn’t feel like I really learned anatomy. So I got my own cadaver during the period when I was taking formal classes in surgery, and we were using Christopher’s Textbook of Surgery. I don’t know whether that [book] was still around when you were there. Somebody here [on our Pittsburgh faculty] has my original Christopher’s Textbook of Surgery. For every single subject there is a layover page of my own drawings, which I did from my own dissections, planted right on top of whatever it [the text] was about: ganglions of the wrist, gastric resection, whatever. And I would use that cadaver to do my own anatomical studies to find out if there were variations.

DR. RICKETTS: So you would study the cadaver in relationship to the topics you were studying?

DR. STARZL: Yes, yes.

DR. RICKETTS: Did you end up doing the entire body?

DR. STARZL: I did the entire body that way. It created an anatomic knowledge base that served me tremendously well, because having acquired it and almost written my own textbook as I went along—which was a
coordinated one now—it wasn’t just anatomy, it was its application. I married surgery with anatomy right from the beginning. It was a huge job, but—I’m trying to remember what age I would have been at the time—that was all taking place when I was 22, 23 years old.

And then there was the research side. There was a period of time when I wasn’t going to go back to medical school. When I dropped out of medical school to do research with Dr. Magoun, I [worked with] clinicians from places like the Penfield Clinic [Montreal Institute of Neurology], where a guy named Wilder [Graves] Penfield—one of the pioneers and I would say even competitors of Loyal Davis—was doing a lot of electrophysiologic studies. He [Penfield] was doing brain extirpation and electrical stimulation, and studying the effects on brain function. He was exceptionally interested in Magoun’s research and sent people to our Northwestern laboratory. These Canadian fellows, and others from the United States were for the most part trained at a faculty level. I found, to my surprise that I could do experimental neurosurgical operations as well or better as they could. [Laughs]

DR. RICKETTS: They didn't know the anatomy as well, probably?

DR. STARZL: It wasn’t that. Some of them simply didn’t have the technical skills. In my early 20s, and as a medical student, I was operating on cats and then monkeys—first at Northwestern and then during a stay in 1951 at the still-budding new school at UCLA [School of Medicine at the University of California, Los Angeles; now the David Geffen School of Medicine at UCLA]. UCLA didn’t graduate its first class until 1952. I was out there [to complete my PhD] when their first class of 20 or 30 students was just completing their third year, the same medical school status as mine. At the end of the summer of 1951, I came within about 10 days of just leaving medical school and going to Stockholm to work with a guy named Ragnar Granit, who later won the Nobel Prize [1967]. I had a fellowship lined up to work with him. Instead of going from LA to Stockholm, I went back to Northwestern in September, 1951 and started my senior year classes.

DR. RICKETTS: It would be interesting to speculate what the field of neurosurgery would be now if you had been in there instead of in general surgery.

DR. STARZL: I don’t know about that. The studies that Magoun and I were doing had exposed a different way for sensory input to get to the brain. Then, as today, it was known that sensory input is routed mainly via lemniscal pathways, those straight pathways that involve a single synapse and go to specific areas of the [cerebral] cortex. What I had discovered was a second system in which sensory input went by neural collaterals to the primitive parts of the brain such as the reticular formation of the basal diencephalon. From there, projections went all over the cerebral cortex, not
just to the specific sensory areas. So basically what this “extralemniscal” system did was to connect the whole cortex of the individual—the human, if you will, or the cat, or the monkey—with the environment. It informed the whole brain when something happened. If you get burned or cut or have some other sensory experience, it’s not perceived just in the specific sensory area of the cortex. Electrical signals light up all over the brain.

The discovery of the extralemniscal system caused a paradigm shift in neuroscience. As I said, it became the basis for the UCLA Brain [Research] Institute. Their logo in 2007 is a picture that Magoun and I published in 1951 [Starzl TE, Taylor CW, Magoun HW: Collateral afferent excitation of reticular formation of brain stem. J Neurophysiol 14:479-496, 1951]. And so by leaving neuroscience, I abandoned a huge discovery area that has been a hot area of research ever since. I think the central issue was what is the real basis for consciousness, unconsciousness, and cognition? I was right in the middle of all of that. Of course, nobody yet knows the basis.

DR. RICKETTS: [Laughs]

DR. STARZL: If I’d stayed in neuroscience, I think that I might well have spent 50 years just flubbing around like everybody else has done and at the end of it all, [had] nothing definite for an answer.

DR. RICKETTS: Or the opposite could be true.

DR. STARZL: Or I might have actually found out something.

DR. RICKETTS: We might have known the answer. So then you went on to general surgical residency.

DR. STARZL: Yes, I came back to Chicago, finished it [medical school], and went to [Johns] Hopkins [Hospital].

DR. RICKETTS: Tell me about Hopkins. How was that in those days?

DR. STARZL: I just got back from Baltimore. I was over there two days ago for a visiting professorship, and I’ve been there other times over the years. I’ve always said it was the most educational four years of my life but also the most miserable four years of my life.

DR. RICKETTS: [Laughs] How was that?

DR. STARZL: I sort of disapproved of the system that they had. They had this steep pyramidal system, in which they took in 16 interns and ended up with two that made it all the way out to the chief residency in a six- or seven-year training program. But because they chopped off the other 14, it
caused an unusual degree of internecine strife and competition. I didn’t like it, and I made that well known. I just think that I was a very uneasy fit with the system.

DR. RICKETTS: Okay. So what about the conditions, the work hours and so forth there, and how would you compare them to our work hours now?

DR. STARZL: You were on call 24 hours a day for the whole year and you had a one-week vacation; that was it. And the pay was zero.

DR. RICKETTS: [Laughs]

DR. STARZL: They had to change that. But at that time, they were in the driver’s seat because they had done those blue babies [blue baby operations], and that was really the beginning of cardiac surgery big time.

DR. RICKETTS: Were any pediatric surgeons at Hopkins at the time?

DR. STARZL: You know, I don’t think so. I think that specialty really didn’t exist there.

DR. RICKETTS: So, then, what about your chief residency? I understand that was in Miami.

DR. STARZL: Yes, those were two tough years, too, because—in fact, I never actually had a chief residency at Hopkins. I think you’ll have to be careful how you approach this subject. People over at Hopkins even this past week were asking me all the same questions about: What happened after those four years at Hopkins? What actually happened was that the pyramid had come down to two people, and I was one of them. Once you got into a competition like that, you really couldn’t just quit. The other survivor was a guy named Bob [Robert A.] Gaertner. We lived up on the fourth floor of an old building there that was dominated by a huge statue of Christ. Huge. It’s still there. My roommate for much of the time during the four years was Jim [James V.] Maloney. You probably know him.

DR. RICKETTS: I know of him.

DR. STARZL: Gaertner had a room next to ours. Jim was a tremendous help to me because I had gotten, by this time, into cardiac physiology, doing some work in the lab. He knew that technology, and taught me. At the end of the fourth year, I went to see Dr. [Alfred] Blalock for one of those year-end meetings, and he told me that Gaertner and I were slated for the next residency year. He said that he had already promised Gaertner a fifth and sixth year. He also said that he was offering me a fifth
year. But he also said, “Well, we’ll wait and see about the sixth year.” And I was deeply offended by that.

So I told him, “Dr. Blalock, I won’t be available for the fifth year.” “Why not?” he asked. I said, “Because I don’t want to stay here anymore.” He asked me why, and I told him. A couple of things, maybe more, but the central issues I told him were, first, [that] I disapproved of the system, and secondly, [that] I was uneasy about what I perceived to be the motivations that were being taught. I said, “You’ve got all these bright guys coming here, and you send them over, like me, for example, to a lab.” But I said, “Most people who are going over and doing research are just doing it as a professional stepping stone, a career advancement move.” And I said, “You can’t ever really learn anything that way.”

He then went down a list of his own faculty members. He asked, “How about X, Y, Z?” And I told him, “X—that’s a perfect example of what I’m saying.” [Laughs] And he got to Hank [Henry T.] Bahnson, and I said, “Well, now, that is a real exception. If Hank goes to the lab, he has a real question. It has some real relevance to the practice of medicine, and you can count on the integrity of whatever the answer is.”

So I quit. And he was shocked by it. The people that were grilling me last week [chuckles], nearly 60 years later, were guys that were, like me, 80 years old or more, and were there at the time, like Bricks [R. Robinson] Baker and [J.] Alex Haller [Jr.]. They wanted to know—what happened at that meeting.

But in some way that I don’t fully understand, I resolved never to discuss what went on in that conference with anyone. And in his own way, Dr. Blalock also made the same decision. To my knowledge, he never spoke to anyone about the meeting, and I never did, either. Sixty years later, I guess it’s okay.

Blalock always was a great supporter after that meeting, and offered to help me at the time. He had scrupulously looked for, and found, great places for people that were clipped off that Hopkins pyramid. He would send these bright people out to UCLA, Vanderbilt, Barnes [Hospital, St. Louis], or other centers where they were gratefully received. The best people were coming there [Johns Hopkins] because of the blue baby and other breakthroughs in heart and vascular surgery. But I also realized that that windfall was coming to an end. So when Dr. Blalock told me during this conversation that he would find me a position, I told him, “That won’t be necessary. I’ll find my own place.”

And so I did. I looked around for a vacuum, and the great vacuum was [the University of] Miami. Here was another [new] school. I had just witnessed
the starting of UCLA, I saw the opportunities that were being presented in Los Angeles, and I was pretty sure Miami would be just like that. It turned out to be, in one respect, and that was the presence there of an enormous amount of material. And also Miami was a city that was, at that time, really under-developed in surgery. So I was bringing down from Baltimore things that no one in Miami could do.

DR. RICKETTS: So you were teaching the teachers.

DR. STARZL: It was sometimes like that. That created some problems, but it created a huge torrent of material. I did a thousand majors each of the two years I was down there, and they were always genuine majors. That meant three big operations a day on average—no small things. In fact, there was way too much work. I was exhausted by the whole thing, and never really enjoyed operating [laughs] anymore. I was worn out by it.

DR. RICKETTS: You were tremendously busy clinically, it sounds like, but yet you were still doing a lot of research in Miami, too.

DR. STARZL: Well, I did. I set up a little lab in a garage and borrowed or stole, I guess, whatever fluids and equipment were needed and did some experiments that were really directly relevant to important questions of liver physiology, and liver transplantation. I tried to do some liver transplants—some liver replacements. Also when I was in Miami, one of the visiting professors was [C.] Stuart Welch, who had published a paper in 1955 in Transplantation Bulletin about auxiliary liver transplantation. [C.S. Welch, A Note on Transplantation of the Whole Liver in Dogs, Transplantation Bulletin 2:54, 1955] He tried to put [an extra] liver in dogs. So I met him and talked to him extensively. He was an interesting guy. Overall, it [the two year stay in Miami] was a great experience, and during this time [1956-58] I did experiments on liver, insulin and carbohydrate metabolism, all of which were central to the development of liver transplantation.

DR. RICKETTS: So you must have really put in the 80-hour work week.

DR. STARZL: Oh, yes.

DR. RICKETTS: [Laughs]

DR. STARZL: When I was younger and for a great number of years, I really didn’t require very much sleep at all. There was a little change in that requirement when I had hepatitis in 1964 and was laid up for several months. The sleep requirement then jumped up to maybe five or six hours. But I could always find time, yes, to sleep.
DR. RICKETTS:  You just mentioned, and I was going to ask about that, Dr. Starzl, you are really considered to be one of the greatest technical surgeons ever, but you’ve said that at an early age you thought you weren’t emotionally equipped to be a surgeon. Can you explain that to me?

DR. STARZL:  The reason was that even to this day—and right now I’m engaged in some patient management activities—

DR. RICKETTS:  We’ll probably get to that.

DR. STARZL:  But because I tended to get too closely tied up with the patients, failures bothered me more than most people. I think that some surgeons tend to remember their successes and sweep those others under the rug. For me it was the ones that went under the rug that I was permanently [focused on].

DR. RICKETTS:  You concentrated on those?

DR. STARZL:  Yes.

DR. RICKETTS:  And with respect to surgery, I’ve spoken to a few people who have operated with you, and they said that the experience was magical—

DR. STARZL:  [Laughs]

DR. RICKETTS:  —to see you operate.

DR. STARZL:  That’s one description. [Laughs]

DR. RICKETTS:  So I wonder—how did you prepare for an operation, mentally or whatever?

DR. STARZL:  I had all these books. I’d go back and read up on them.

DR. RICKETTS:  Even though you’d done—

DR. STARZL:  Even though I had written the definitive article, trying to make sure I didn’t do something wrong, or forget something.

DR. RICKETTS:  Did you think about the operation post-op or write yourself notes for the future?

DR. STARZL:  Well, yes, both. And then I had a very good memory, which is slipping now, particularly in the last few years, for distant things. And it wasn’t just about surgery, or cases. If I read an article 25 years ago, I could remember the volume and the page and what it said. Or I could see a
movie 30 years previously and really remember pretty much the dialogue verbatim. So I developed this memory bank. But the important memory bank was about observations that were made, and errors that had been committed, or misconceptions, if not errors, that turned out to [be] convictions that I had at the time that turned out to be wrong. So a big storehouse was developed, and that was quite useful.

When I was working on *The Puzzle People*, I wrote the book, but then I did a fact check, and there weren’t very many things that weren’t pretty much the way they were described in the book—

DR. RICKETTS: What advice would you have to young surgeons now preparing for a career in surgery?

DR. STARZL: I don’t know what I would say now because the [professional] climate has changed so much. I had the great fortune of living in a very free-wheeling era, where the idea that you would do something unethical in transplantation never seemed to be a consideration, never even came up. Perhaps that was because nothing was driven by the economic considerations that tend to tarnish everything. Medical education and science-based improvement of patient care were the driving forces of so-called university hospitals. Now, what the hospitals expect from faculty members is to keep the cash register running, to maintain caseload, things like that. Transplant surgery, when I was practicing it, as far as I was concerned, was a crusade. Surgery now is a business, and so is medicine.

DR. RICKETTS: So—

DR. STARZL: So what to tell them [young surgeons]? I don’t know. I try to teach them old ways. Some listen and some don’t. Generally speaking, the ones that listen do pretty well. [Laughter]

DR. RICKETTS: So after Miami you went to Northwestern for a while and then to [the University of] Colorado [School of Medicine]. I think what I’m going to do is—this tape is going to be almost over, so why don’t we stop at this point, at the beginning of your Colorado career, change tapes, and then we’ll carry on from there.

DR. STARZL: All right, okay.

DR. RICKETTS: Great.

END OF TAPE 1, SIDE B

DR. RICKETTS: —a break. We’ve been talking about various people who we’ve interacted with, one of whom was Dr. [W. Dean] Warren. And maybe I’ll
just go back to that. I started a little bit—you were talking about another field that you were very interested in and instrumental in, [which] is liver regeneration and growth factors. Can you say a little bit about that?

DR. STARZL: I got into that field as a direct result of my interest in liver transplantation, or maybe it was the other way around. You could argue that I got into liver transplantation because of my interest in the metabolic interrelationship of the liver with the other abdominal organs or, to be specific, the functional relationship of the liver and pancreas. Because of that question, I set up a little lab in an abandoned garage in Miami to see what happened to the liver and what happened to sugar metabolism and to insulin metabolism if you did a portacaval shunt in dogs.

At the beginning, one of the hypotheses that I was looking at was that diabetes mellitus was, in fact, a liver disease, and that the reason that you [diabetic patients] were short of insulin was that, in some way or another, the insulin was being delivered to the liver and the liver was gobbling it up, metabolizing it in some unuseful way, and therefore placing an unusual demand on the pancreas to overproduce insulin. That idea actually had been nurtured by an observation at Hopkins of a patient operated upon by Dr. Blalock, with a splenorenal shunt. The guy was a florid diabetic before he had his splenorenal shunt; suddenly he didn’t need insulin anymore afterwards. So why? That was one of those questions that came up, presented itself to someone, myself in this case, who had no background in metabolism. So I suddenly became a student of metabolism and set up a dog lab, did portacaval shunts, reverse portacaval shunts, and so-called transposition operations—and studied what happened to insulin and sugar metabolism.

I published papers on that. This was a question of what did the liver have to do with pancreatic function. But the other side of the coin was: What happened to the liver? What were the consequences of this [metabolic] axis for the liver? And we knew, because it had been described many years before, that after portacaval shunt, the liver shrinks dramatically. It atrophies. That was a vital piece of information in connection with liver transplantation. [C.] Stuart Welch, when he had come down and visited us in Miami, described how he put liver grafts—auxiliary livers—in the flank (the paravertebral gutter of dogs), and was giving those liver grafts a portal blood supply with inferior vena caval blood coming back from the legs or the kidneys.

DR. RICKETTS: There was only a systemic supply.

DR. STARZL: Yes, and so he [Welch] was replacing a splanchnic venous supply [of his liver grafts] with a systemic supply. Was this a conceptual error? That was a very tough question to get your hands around.
I did some experiments, and discussed them at the ASA [American Surgical Association] in 1964, that suggested that the liver could not be normal if the portal inflow did not consist of blood from the pancreas, intestine, and other splanchnic organs [Starzl TE, Marchioro TL, Rowlands DT Jr, Kirkpatrick CH, Wilson WEC, Rifkind D, Waddell WR: Immunosuppression after experimental and clinical homotransplantation of the liver. *Ann Surg* 160:411-439, 1964]. I had much earlier evidence supporting this conclusion in experiments done in 1958 and 1959 at Northwestern, showing that the results were lousy with liver replacement [the orthotopic operation] if you gave the liver graft anything but splanchnic venous blood. Such results dictated physiologically what the optimal operation of liver transplantation should consist of anatomically. It almost had to be an orthotopic procedure: that is, placement of the graft in the normal position, and with a normal portal venous inflow.

Over the next 15 years, I published many papers showing this, but I never actually pinned down what was in portal venous blood that made it so special until we developed a portacaval shunt model, in which you could deprive the liver of all portal venous inflow and prevent it from shrinking if you just put a little catheter in the tied off portal vein stump at the hilum and gave the liver a slow infusion of insulin. Insulin infusion in amounts so small that they did not even affect the blood sugar level completely prevented the typical liver shrinkage of portacaval shunt. So the crucial ingredient of portal blood was insulin [Starzl TE, Watanabe K, Porter KA, Putnam CW: Effects of insulin, glucagon, and insulin/glucagon infusions on liver morphology and cell division after complete portacaval shunt in dogs. *Lancet* 1(No.7964):821-825, 1976].

DR. RICKETTS: That was the growth factor.

DR. STARZL: Those experiments identified insulin as a liver growth factor—the first one known. But they also revealed circumstantial evidence that there were other growth factors. So I went looking for those and eventually identified six of them that had the same effects as insulin. These were some of the first studies of hepatic regeneration in the modern era. There had been an earlier interest in hepatic regeneration, particularly after the description of a rat model of hepatectomy in the early 1930s. But the modern age of liver regeneration dated from the discovery in the 1960s and 1970s that insulin and other “hepatotrophic” molecules were growth factors that were crucial for the maintenance of hepatic size, structure, function, and the capacity for regeneration.

So I continued actively working in this field until 1994 and then decided that we’d sort of milked that dry. So I dropped it [hepatotrophic physiology] for a while. But now we got a new lead,—
DR. RICKETTS: [Laughs]

DR. STARZL: —and we’re back into it now. But I don’t want to get into that. It’s too arcane.

DR. RICKETTS: Let’s go back to your initial work in Colorado. You developed a tremendously successful renal transplant program, and I don’t want to diminish the importance of that, but I would like to start into the liver transplantation. Could you tell me about the first human being to have a liver transplant?

DR. STARZL: Yes, he was this little Hispanic child, a Mexican kid, Bennie Solis who had biliary atresia. That was a terrible case. Of course, there was no such thing as brain death on the day Bennie Solis was dying on a ventilator. He was all blown up, full of ascites, and he had been operated on several times for his biliary atresia. There was another kid who was having an open heart operation at Colorado General Hospital and had a cardiac arrest. That child was on cardiopulmonary bypass, and it was impossible to restore a heartbeat. We had been looking for this kind of circumstance. We took Bennie’s liver out, and sewed in the liver from the child with heart disease. Bennie bled to death on the operating table. We couldn’t—we never even got close—control the hemorrhage. It was a devastating loss in both operating rooms.

Up to this time, of course, nobody had ever seriously considered doing either a liver or a heart transplantation in humans. But if you could have fast forwarded even a few months, you could have asked, “Why didn’t you use Bennie’s heart for the other one, for the other kid?”

DR. RICKETTS: Nobody would have thought of that.

DR. STARZL: Yes. But anyway, that [failure] stopped us in our tracks. We went back to the lab and also recruited one of the world’s finest coagulation experts [Kurt von Kaulla]. This guy had all kinds of tricks up his sleeve about how to switch clotting back on. In the paper that we ultimately published about our failed first three cases—Bennie and two more—we described how to control the bleeding, and particularly how to shut down the thrombolysis that frequently occurs during the liver replacement. By the time of the second and third cases, we were geared up in a remarkable way, including the use of anti-thrombolytic agents that are still being used today. Even though our first report was of three failures, that paper describes very much the way liver transplantation is done today [Starzl TE, Marchioro TL, Von Kaulla KN, Hermann G, Brittain RS, Waddell WR: Homotransplantation of the liver in humans. Surg Gynecol Obstet 117:659-676, 1963]. Unfortunately, we then had two more deaths that had to do with, more than any other factor, the use of veno venous bypasses
to decompress the obstructed inferior vena cava and splanchnic venous beds
during the anheptic phase. Clots formed in the plastic bypass tubing and
migrated to the lungs. In essence, we had overcorrected the coagulation
problem encountered with Bennie Solis in all of the next four patients.
Although these four recipients survived for one to three weeks with good
graft function, all had lethal pulmonary complications that probably were
related to the migratory clots. Nearly four years passed before another
attempt at human liver transplantation was made.

DR. RICKETTS: And then you stopped and went back to the lab.
Immunosuppression changes allowed you to reconsider liver transplant again.

DR. STARZL: That was one area. The biggest accomplishment in
immunosuppression was the development from scratch of ALG [anti-
lymphocyte globulin], which is still a big player. For the first five liver cases,
we had used Imuran and prednisone in the same way as we had done in the
kidney [recipients]. It worked. None of those first transplanted livers had
much evidence of rejection. But still, we thought that too much steroid
therapy was required and that a third agent such as ALG would fill the bill.

DR. RICKETTS: That was in the triple-therapy era?

DR. STARZL: Yes. The triple-therapy was first used in 1966 in human
kidney recipients and consisted of Imuran, prednisone, and ALG. During
the four year human liver timeout of 1963-67, we produced a lot of truly
long-surviving dog recipients of livers. Some of these animals eventually
occupied our kennels for more than ten years. Of great interest, we found
that if we had a dog liver recipient that made it out to 100 days under daily
Imuran treatment, we could stop the drug and they [the animals] didn’t
reject. The long term results with Imuran were presented at the Society of
University Surgeons in 1965. And then, as we developed ALG during that
period, we found that we could sometimes give one or two doses of ALG with
no other treatment, and if you gave it at the proper time—

DR. RICKETTS: Giving it prior to the transplant?

DR. STARZL: Prior to or even at the time of transplant, only one or
two doses sometimes allowed you to have long survival—I mean for years—
without any other therapy at all. The ALG was completely different than
Imuran. Here was a lymphoid-specific immunosuppressant that evolved in
later years into monoclonal antibodies, OKT3, and then into other special
antibodies that are directed at specific lymphoid lineages—leading right up
to the present-day best ALG, [the] super-ALG called Campath. But they’re
all progeny of the original ALG and of the ALG strategy. Those papers that
we wrote about ALG in the 1966 and 1967 have been so-called citation
classics in that they’ve generated hundreds of citations and a lot of derivative studies.

The development of ALG was crucial in the big clinical breakthrough that began in July 1967 with our first successful liver transplantations. Beginning in January 1968, and ending in the autumn of 1969, three other kinds of non-renal grafts were successfully transplanted for the first time in humans—all under triple drug immunosuppression. During that time, we manufactured ALG for those who needed it. When [Christiaan] Barnard did those heart transplantations in South Africa, we provided him with ALG until he could arrange for a European source. We also supplied [Norman E.] Shumway with ALG until the Stanford group got their own production units started. ALG and triple drug therapy also were linked with the first successful human lung transplant [Derom F. et al: Ten-month survival after lung homotransplantation in man. *J Thorac Cardiovasc Surg* 61:835-846, 1971] and with the first one-year survival of a pancreas recipient [Lillehei RC. et al: pancreaticoduodenal allotransplantation : experimental and clinical observations. *Ann Surg* 172:405-436, 1970].

[Note: the date of the lung operation was November 14, 1968; the first successful pancreas transplantation was on June 3, 1969. The lung recipient survived for 10 months, and the pancreas recipient for just over one year.]

DR. RICKETTS: Your first report about the successful liver transplantation under triple-therapy was, according to my records, to the American Surgical [Association] in 1968.

DR. STARZL: Yes.

DR. RICKETTS: It only included children.

DR. STARZL: Yes.

DR. RICKETTS: Why was that?

DR. STARZL: Well, the children played a unique role in the development of transplantation. I don’t think it could work that way these days, but at the University of Colorado the chairman of the Department of Pediatrics was a guy named [C.] Henry Kempe, a famous guy. He was a great infectious disease expert. But he also was known as the great child’s protector. He was the guy who’d invented the term or introduced the whole concept of the “battered child syndrome.” So he was running all over the country, all over the world saying, “Hey, these kids who were coming here with these healed fractures, they're signaling you that they’ve been beaten and so forth.” So he was a powerful moral force.
When I first came out there [University of Colorado], he told me that, “I don’t care what the risks are, liver transplantation has to be developed for children with biliary atresia, because,” he said, “what we are doing today is inflicting torture on these children and on their families, and whatever we do, they [the children] live for a year or two and then die. By the time that happens, they [the healthcare systems] have ruined the families and turned those children into pariahs.”

So he wanted liver transplantation to be used for children. And the very same guy was anxious to have kidney transplants done on children. So this pediatric chairman—a world renowned figure of great moral stature—wanted children to have an early crack at this new technology. That meant that [children also were] in that early kidney batch, the cases that were done in 1962 and 1963. There were 46 cases [of live donor kidney transplantation] altogether. Of that group, nine recipients carried their grafts for at least 40 years. Six of those nine [recipients] were children. Seven are still alive, now bearing renal grafts that have functioned for 44-45 years—the longest in the world.

So if you see them—and I have a photograph I show around once in a while—here they are—these children are now parents, or grandparents. And they’ve grown up and lived a normal life. The point is that the renal transplant program at the University of Colorado was always pediatric-heavy.

DR. RICKETTS: They [the department of pediatrics] supported you, but the medical department didn’t support it.

DR. STARZL: The medical department was bitterly opposed to liver transplantation. In fact, that’s one of the reasons that I just bailed out of there and came out here.

DR. RICKETTS: Was John Lilly in Denver?

DR. STARZL: Yes, John Lilly was with me for two years. His greatest contributions were to the Kasai procedure. But, Lilly came to Colorado specifically because he recognized the limitations of the [Kasai procedure] — he realized that almost all of those Kasai children ultimately were going to need—

DR. RICKETTS: So he was happy for you to operate on some of his patients.

DR. STARZL: He wanted that done, yes, yes. Of course, I was chairman. I recruited him out there as the head of the pediatric surgical group. He was my guy.
DR. RICKETTS: He was a wonderful man.

DR. STARZL: Yes.

DR. RICKETTS: What was your role in introducing cyclosporine into clinical use?

DR. STARZL: Cyclosporine was first introduced clinically by [Sir] Roy [Calne] in England. Of course, we introduced cyclosporine into the United States, and you could say we rescued it. He [Roy] was determined not to use steroids, so he combined the cyclosporine with some of the myelotoxic agents, including Imuran. When they [the Cambridge group] reported their first series—the 34 patients were mostly recipients of kidneys but included two or three with a liver or a pancreas. Because they were trying to use cyclosporine alone [without steroids], they had to use high cyclosporine doses, and so almost every case had cyclosporine nephrotoxicity. Also, there was a 15 or 20 percent incidence of B-cell lymphomas. These are now called PTLDs [post-transplant lymphoproliferative disorders], but they’re true B-cell lymphomas. One in every five of their first patients [treated with cyclosporine] had developed these lymphomas.

Because of these side effects, the Sandoz company [then Sandoz Ltd.] had decided to terminate the clinical trials [of cyclosporine] not long after I got them to give us a supply in 1979. They also got a supply at the Brigham, and in Boston they used it in much the same way as Roy had done with results no better than with Imuran.

We tested our supply of cyclosporine during the last year I was in Colorado [between the autumn of 1979 and August 1980]. We did a dozen livers and 66 kidneys and realized that the whole game had changed. The reason for our good results was that we just simply went back to our original formulation of Imuran and prednisone—replaced the Imuran with the cyclosporine, and intervened with steroids only when necessary for rejection. Everything was changed [in outcomes]. That first batch of livers had a one-year survival of, I think it was 80 percent. We reported that in the NEJM [New England Journal of Medicine] in 1981, having already reported the kidney work—and wow! It was—

DR. RICKETTS: You basically saved cyclosporine, it sounds like.

DR. STARZL: Well, perhaps we did save cyclosporine. A crisis had been reached at about the time of the international Transplantation Society meeting in [July] 1980, just when I was preparing to move from Denver to Pittsburgh. Two or three months earlier, I had contacted Loyal Davis, who was editor of SG&O [Surgery, Gynecology, and Obstetrics], and got him to do a rush publication of our results with the kidneys in the July 1980 issue of
SG&O. I wanted to have this paper out before I gave a formal talk at the Transplantation Society meeting on the state of the art of liver transplantation.

This was expected to be the usual dreary story of a few successes and many failures. Instead, I went up there [Boston] and gave a talk exclusively about cyclosporine. Although I emphasized how important it was for livers, I also gave the kidney results. The message was reinforced by Loyal Davis’ urgent publication. When I sent the manuscript to him—I think it was as late as May [1980]—I told him, “It’s absolutely necessary that we have a couple of hundred copies of the July issue of SG&O that we can hand out at the meeting.” And he came through. I gave the talk, and at the door we were able to pass out several boxes of the July issue of the journal.

DR. RICKETTS: So you said one reason you left Colorado was the medical department wasn’t supporting you. Were there any other reasons that you decided to leave Colorado?

DR. STARZL: Yes, there were other reasons. Some of them were highly personal, having to do with Joy [my wife], but basically my thought was that I was in a climate in which, for one thing, the chairman of the department of medicine had bitterly opposed the cyclosporine trials. As I wrote in The Puzzle People, this guy was a nephrologist. He had sent a letter out to all members of the Colorado Nephrology Society condemning the use of cyclosporine. He also was bitterly opposed to liver transplantation. Another factor was that the governor of the state of Colorado [Richard Lamm] had come out condemning the University of Colorado for emphasizing tertiary care. In fact, he thought that the university should be producing general practitioners—family practitioners—and that it [the medical school] was going in the wrong direction. Transplantation was Governor Lamm’s bad boy poster child.

I had the sense that if I stayed there, I would be involved in a grueling siege warfare that would go on for the rest of my life. Above all, [I felt that] if we didn’t now take advantage of the breakthrough that had been made possible with cyclosporine, liver transplantation was not going to get into common medical practice for 25 to 50 years. Avoidance of that was not going to be possible in the parochial local environment. I had to do something.

DR. RICKETTS: So you started here in Pittsburgh in 1981, January 1st.

DR. STARZL: Yes. Well, actually, we came in December 1980. [Laughs]

DR. RICKETTS: And you’ve described Pittsburgh as being a perfect place for you and Joy. Is that on a professional basis or a personal basis?
DR. STARZL: Both.

DR. RICKETTS: This has worked out well. Now, when you—well, obviously—

DR. STARZL: I was really concerned about coming east into what I feared would be a highbrow, eastern, bigoted environment. None of that was true.

DR. RICKETTS: Dr. Marc [I.] Rowe was here.

DR. STARZL: Soon to be, yes.

DR. RICKETTS: How did you and Dr. Rowe get along?

DR. STARZL: We got along fine. I greatly admired Marc. Marc wanted to be involved in what proved to be one of the other big transplant accomplishments here, which was putting intestinal transplantation on the map. We worked that out.

DR. RICKETTS: What about with livers? I presume he was doing Kasai procedures at the time.

DR. STARZL: Well, he was. But he was strongly influenced by Mark [M.] Ravitch, and he knew that Mark Ravitch was transfixed [chuckles] by what he had already seen firsthand with liver transplantation. When Marc Rowe arrived [he was recruited from Ohio State in 1981], the liver program was off and running, and had succeeded. Consequently, both Marc Rowe and Mark Ravitch recognized that the role of Kasai procedure would not be a dominant one. In essence, we had powerful support from the Children’s Hospital [of Pittsburgh] for development of liver transplantation.

DR. RICKETTS: So you and Dr. Ravitch got along well?

DR. STARZL: Very well, yes.

DR. RICKETTS: I heard he cut you during an operation one day.

DR. STARZL: [Laughs] He did. He did.

DR. RICKETTS: What was that all about?

DR. STARZL: Well, that was an accident. That was back at Hopkins [30 years earlier].
DR. RICKETTS: I hope it was an accident! Oh, it was at Hopkins.

DR. STARZL: Yes. I was a surgical resident, and he cut one of my extensor tendons. I don’t know that I could easily identify the scar, but he ordered me to take my glove off, he sewed up the tendon and closed the wound—

DR. RICKETTS: [Laughs]

DR. STARZL: —and then accused me of having slashed his scalpel with my hand. [Laughs]

DR. RICKETTS: Accused you of getting in his way, probably. What about Dr. Bill [William B.] Kiesewetter. Was he here at all when you first came?

DR. STARZL: I knew him. My impression was that he was a very warm, kind man. I’d only been here a very short time when he had a fatal heart attack.

DR. RICKETTS: I remember Dr. Kiesewetter. I met him when [I] interviewed here for a fellowship, and I thought he was the most warm and gentle man I’d ever met, and very generous. He gave me tickets to the hockey game that night, and I went with his son to see the hockey game. That was pretty good.

DR. STARZL: Well, he was—people were shattered when he died.

There’s an interesting little side story. It said something about the basic integrity of at least the surgical department. Kiesewetter had no idea he was sick, and a short time earlier, only a few weeks before he died, he had made a decision about his contributions to our [departmental] practice plan. He knew he was retiring. He was just on his way out, and he made some arrangement by which he was to receive a lump sum of $250,000 as he went out the door. But by so doing, he was giving up a conventional retirement program that would have provided a steady income for him and his wife. I can’t remember what the facts were exactly.

When he unexpectedly died, the arrangements that he had made for a lump sum were greatly disadvantageous for his widow. He had signed the document and from a legal point of view, everybody was off the hook. But the department of surgery—Hank [Henry T.] [Bahnson] actually—made the decision that this was unfair and created a different kind of outcome. This was only one of many examples of Bahnson’s integrity.

DR. RICKETTS: So, Dr. Rowe was the chief, I guess, here or at Children’s [Hospital of Pittsburgh] most of the time.
DR. STARZL: Yes, he was. He arrived in Pittsburgh in mid-1981, only a few months after I came.

DR. RICKETTS: And you mentioned that he wanted to get his hands in the small bowel transplantation.

DR. STARZL: Yes. [Laughs] But there wasn’t much room because the liver and intestinal groups were the same. The team was pretty well formed by the time Marc arrived.

DR. RICKETTS: How about liver transplantation? Was he involved?

DR. STARZL: Not directly. I believe that he had wanted when he first arrived to take over the complete intensive care of these patients. But we had a team that had been trained and built up for years to do that. So the prospect of Rowe’s team taking care of our patients could have caused a confrontation. But he gave up on the idea, and I never, ever, from that moment onward, had any conflict with him. He considered it a fait accompli, and that was that. Publicly and privately, I considered him to be one of our greatest supporters.

DR. RICKETTS: So you did the transplants in the Children’s Hospital [of Pittsburgh]?

DR. STARZL: Yes, all of them from the start. That was interesting, because the Children’s Hospital [of Pittsburgh] had a small ICU. It had only six beds. This was part of the vacuum that existed here. They were having trouble filling their [ICU] beds at both the Children’s and at Presbyterian [Presbyterian University Hospital; now UPMC Presbyterian]. They had maybe eight ICU beds at Presby, and there were only four, maybe six at Children’s, but they were never fully occupied. So they were talking at both hospitals about reducing the number. And all of a sudden—

DR. RICKETTS: Changed that!

DR. STARZL: —there was a huge demand.

DR. RICKETTS: Yes, I’ll bet.

DR. STARZL: And, in fact, I was over there the other day just checking. We have 72 ICU beds just for the transplant service today.

DR. RICKETTS: Is that right?

DR. STARZL: Yes, but that, of course, would be all of the transplant services. Seventy-two ICU beds. Overall, this is really a huge tertiary
complex. There are several hundred ICU beds here. They’re all over the place. So rather than shrinking, I tell them to stop [reducing beds]. [Laughs] Instead, we had better expand. So the ICUs came to be dominated by the transplant patients. The Children's Hospital was where the first successful Pittsburgh liver case was done. It’s amazing how many of those kids that we did in the spring and summer of 1981 are alive, have grown up. I just got a message on Friday from a lady named Gail, who had a daughter named Pam, who had been a kid at the time. She called down here and left a message asking me to call back. I called and got a voice box and left her a message. In return I learned that she’s a grandmother now, and Pam had delivered a baby. [Laughs]

DR. RICKETTS: Makes you feel good, doesn’t it?

DR. STARZL: Yes, it does.

DR. RICKETTS: So, then, it sounds like your team took care of the patients postoperatively, and you had some medical, pediatric residents on your service, I guess.

DR. STARZL: Yes, we did, but we took care of—I’ve always taken care of my own patients.

DR. RICKETTS: I also understand that you preferred to eat lunch at Children’s rather than at the university hospital because the food was better.

DR. STARZL: Yes, well, that’s true.

DR. RICKETTS: [Laughs]

DR. STARZL: That’s true. They had a great dining room. I haven’t been there lately, but—

DR. RICKETTS: Okay. Now, during the time that you were starting here in Pittsburgh, randomized, controlled trials came in vogue. I understand you had some problems with those. Can you explain that?

DR. STARZL: Because by the time I arrived here in Pittsburgh, we had already proven, as everyone else eventually did also, that cyclosporine was a superior drug compared to anything previously available. I thought it was a great injustice to inflict a randomized trial on an unsuspecting population just to establish what we already knew—or just to publish a paper. So we fought that. But we had to make a small compromise and did a randomized trial in a limited number of kidney cases, and only adults. We never allowed that to happen to the children. And the Children's Hospital didn’t want that, either. But that's an interesting situation because when the
next advance came along, which was tacrolimus a decade later, the powers that be [at the Children’s Hospital] again supported me in resisting a randomized trial of tacrolimus. Because tacrolimus could rescue almost all cyclosporine failures, I did not want its use randomized in the children. A pitched battle followed, and eventually we were forced into a limited randomized trial with livers, only with adult recipients. We never allowed the children to have to bear that burden.

DR. RICKETTS: How do you feel about it now? Do you feel that there are some ethical issues related to randomized controls?

DR. STARZL: Oh, for sure there are. I have called it “randomized trialomania.” [Laughter] In fact, all the great advances in transplantation, I think, have come just from careful clinical studies of a relatively small number of cases. In this way, you can get valid answers. What sometimes happens with randomized trials is that a new drug comes along and people apply it improperly in some kind of a rigid formulation for the randomized trial and the value of a drug or a procedure is underestimated because it hasn’t even been standardized yet [before randomization]. I’m not against randomized trials. We’re starting one now to test the effect of prostacyclin or prostanoids on reperfusion injury. That’s been so controversial that I think we have to do a randomized trial. It isn’t going to hurt anyone, because the ones that don’t get the prostacyclin are going to be no worse off than they always have been, and the ones that get it, I believe, may do better. But we have to prove that.

But I have heard ridiculous proposals [for randomized trials] such as one made at a consensus development meeting in Washington in 1983 [Consensus Development Conference on Liver Transplantation]. One of the persons who was on the panel that was going to render a decision about whether liver transplantation should go forward wanted to do a randomized trial in which half of the patients with truly terminal end stage liver disease would be randomized to “no transplantation.” At the time, the patients that we considered to be candidates and for whom we couldn’t find livers had all died within 90 days. The randomized trial would be in this kind of patient. [Laughs] The lucky ones could get a liver transplant, and the others would be controls.

DR. RICKETTS: [Laughs]

DR. STARZL: I mean, really! [Laughs]

DR. RICKETTS: I was going to bring up that meeting, that panel. Dr. [C. Everett] Koop, another famous pediatric surgeon, was—

DR. STARZL: Yes, he was our greatest ally.
DR. RICKETTS:  Tell me about your relationship with Dr. Koop, and what you think about him.

DR. STARZL:  I had a very high opinion already of Koop just when he was in practice [pre-Washington].  He was a revered figure.  Although I didn’t really know him, I went to see him, because I had heard that he was determined that children with biliary atresia have access to liver transplantation.  He was right inside the White House [as Surgeon General], so he and I were the ones who engineered that [consensus development] conference.  I went over to see [President Ronald] Reagan—and, by the way, Reagan personally got behind this—and I was scooted down to Reagan’s personal physician [Daniel Ruge] and within an hour or so I was in Koop’s office.  Koop was always behind this.  He was indispensable.  In fact, Koop was the guy who told me, “The way to do this [get liver transplantation established] is to have this consensus development conference.  Do it quickly, all the steps."

DR. RICKETTS:  He was quite instrumental in saving it for the whole world.

DR. STARZL:  Yes, no doubt about it.

DR. RICKETTS:  So with respect to research, I think of you as doing the most clinically relevant research of anyone I’ve ever read about.  How would you suggest that young people prepare for the type of career that you’ve had?

DR. STARZL:  [Laughs]  I would say:  Avoid it all costs.  [Laughter] But if you have to do it, it would be good, somewhere along the line, to get some background in [basic] research.

DR. RICKETTS:  Does the best research go from the bedside to the bench, or from the bench to the bedside, or what?

DR. STARZL:  An argument can be made that all of the great advances that have occurred in transplantation have come from bedside to the bench, because they [patients] presented us with very clear problems that required some kind of definitive resolution if we were to improve their care.  [That is] what happened in transplantation — [Pause as he retrieves a document.] Here’s a paper that’s in press.  This paper is the account of the great advance—along with a horrendous error—that was made way back when transplantation started.  Here, I have clipped out bits and pieces of this thing [document] that will be coming out in a few months [Starzl TE:  Acquired immunologic tolerance: with particular reference to transplantation.  Immunol Res 38(1-3):6-41, 2007].  And, in fact, just between you and me, we will soon be sending a paper to the New England Journal of Medicine, which is pretty much the tail end of this talk [Starzl TE: Immunosuppressive

The great accomplishment was the demonstration in humans that kidneys, and then other organs like the liver could be successfully transplanted under drug immunosuppression. This occurred over a nine-year period (1959-1968). The breakthrough was led predominantly by surgeons. Improvements in results in following years were mostly dependent on better drugs than the original Imuran and prednisone—ALG, cyclosporine, and tacrolimus.

The horrendous error that I referred to a few moments ago was acceptance of the false premise that organ engraftment—the great accomplishment—was unrelated to tolerance and involved different mechanisms than the donor leukocyte chimerism-associated ones of tolerant bone marrow cell recipients. Once this error was accepted as dogma, it became impossible to understand what the mechanisms were of organ engraftment. The false premise was overthrown in 1992 when we discovered small numbers of donor leukocytes (microchimerism) in the tissues and blood of our long-surviving kidney and liver recipients. That discovery and its ramifications are described in this paper [points to manuscript] as they relate to the ultimate objective of creating tolerant children or adults after organ transplantation. And, by the way, we’ve gotten a whole bunch of them [tolerant patients] over there [at Children’s Hospital]. [Laughs]

DR. RICKETTS: Good!

DR. STARZL: I think we now know how to do that [produce tolerance]—as described in this article in press in Immunologic Research [Note: the paper was published later in 2007] and also explained in the manuscript that I will be sending to the NEJM. As soon as we finish [talking], I’m going to go back to work on it. When it’s accepted, I’ll slip you a pre-print. [Note: this paper eventually was published in the NEJM in January 2008]:

DR. RICKETTS: Okay. I was going to pick your brain about that a little bit later when we go on here.

DR. STARZL: Anyway, to get back to the question you asked about the driving force of transplantation, what I often have said is that there was a bidirectional flow between the bench and bedside. In my opinion, the most powerful current consisted of discoveries and observations that went from the bedside to the bench. To be fair, however, almost all progress from the beginning, depended on better drugs—Imuran, cyclosporine, tacrolimus, ALG—and these came from the bench to the bedside. But nothing much would have been done with these drugs had it not been for—
DR. RICKETTS: Clinical trials.

DR. STARZL: Yes

DR. RICKETTS: So getting on to that, then, what was your role in bringing tacrolimus to prominence?

DR. STARZL: Well, that involved much more than a clinical trial. We worked with that drug from the test tube to the clinic—beginning before there had been any publications about its discovery by scientists at the Fujisawa Pharmaceutical Company in Japan. I first learned of the drug through our [Pittsburgh] cancer people, who told me that tacrolimus has been found [by the Japanese] while searching for drugs that might be of some value for cancer treatment. The rest of the story is told in The Puzzle People. Knowing about the drug, I picked up and went to Japan and got a small supply. We did the early pre-clinical stuff here, and eventually it turned out to be an enormous clinical success. That’s the No. 1 immunosuppressive drug today.

DR. RICKETTS: Yes. In looking over your career, I was struck with the fact that here you are an amazing surgeon, [who] developed a very complicated and amazing technique, but yet the real impact, not to take away from technical, was bringing along the ALG and the cyclosporine and the tacrolimus.

DR. STARZL: I think that’s true. In fact, when I was in practice—and was being given the role of, you know, a clever monkey with quick hands [chuckles]—it wasn’t like that at all. From the beginning, I think that my career was much more about the development of concepts. Of course, some of those concepts required skill to be brought forward, but I think it [the main factor] was the ideas rather than the hands.

DR. RICKETTS: How did your team get involved with small bowel transplant work? Was that because of all the short gut patients that we, as pediatric surgeons, create?

DR. STARZL: Only in part. By the time that we did our first really successful [intestinal] transplantation (1990), we had begun to understand what [were] the mechanisms of engraftment. In 1991, I published a paper in SG&O [Surgery, Gynecology & Obstetrics] about the migration of cells from intestine-containing multivisceral grafts into the recipient. The title of the paper is “The many faces of multivisceral transplantation [172:335-344, 1991].” Now, where did that start? Multivisceral transplantation was an operation that I had developed in Chicago, at Northwestern, in 1958. And why? I was using that dog operation as a model to determine—to get at the primary question of—what the metabolic relationship was of the liver to the
pancreas and to the other splanchnic organs. The multivisceral operation was published in 1960 in the *Surgical Forum* [of the American College of Surgeons], and then described in a big paper in the *American Journal of Surgery* two years later [Homotransplantation of multiple visceral organs. 103:219-229, 1962].

The primary objective was to look at what happened to the liver when it was transplanted with all the other abdominal organs [stomach, pancreas, small and large bowel, spleen] versus [the fate of] livers that were transplanted by themselves. And, of course, the flip side of that was: What happened to all those other organs, including the bowel, if they were transplanted alone versus if they were accompanied by a companion liver? So that question [of organ interrelationships] was always there, and so was the multivisceral operation.

In 1987, we did that [operation] on a child, Tabatha Foster, who lived for a half year or so and then died of B-cell lymphomas, the PTLDs [post-transplant lymphoproliferative disorders]. During most of her post-transplant life, she maintained her nutrition by mouth, proving for the first time in humans that an engrafted intestine could function. By the time that we tried again, it was in 1990, and we had tacrolimus as our baseline immunosuppressant instead of cyclosporine. No one else did. [Chuckles] Then we successfully transplanted the small bowel alone or as part of a multivisceral graft. So the intestinal transplant program was all part of this big concept that put all kinds of transplantation, no matter what the organ, on common ground. In other words, the mechanisms of engraftment, whether of the kidney, liver, heart, or bowel, were the same. The specific roots of the intestinal transplant were in the paper on canine multivisceral transplantation from a third of a century before. It [the operation] was always there, lingering around, saying, “Hey, why don’t you use me?” [Laughs] We finally got around to it.

DR. RICKETTS: Speaking of the small bowel transplant now. Is it the condition of the liver—that is, the TPN [total parenteral nutrition] cholestasis—that determines when you should do it?

DR. STARZL: Yes, and it also determines what operation should be done: intestine alone or multivisceral. Kareem Abu-Elmagd has done a tremendous job with these procedures. Now the one-year survival in Pittsburgh with multivisceral and intestinal transplantation is right around 90 percent. Those procedures are available in many centers now. About six months ago, there was an international meeting here on intestinal transplantation and I saw one of the last recipients that I had anything to do with before retiring. [She] was an infant named Tracey Gonzales from New York, who now is a premedical student whose life is sustained with somebody else's liver, stomach, whole intestine and so forth. So she’s a real forerunner.
Another reason why she came up recently is that I nominated her for a nice prize given to heroes, pioneers of transplantation [the Burl Osborne Pioneer Organ Replacement Hero Award]. It is given by the International Federation for Artificial Organs. She gets to go to Tokyo or someplace to get a nice check.

DR. RICKETTS: So in general, what do you think the quality of life is for small bowel transplant recipients?

DR. STARZL: It was pretty punk until recently, but all of these advances that we have been discussing, including those contingent on understanding how the immune system works [pointing to manuscript draft], have been invested in and have improved small bowel transplantation. At a practical level, it boils down to this: You can in many or most cases of intestinal transplantation end up with somebody who has good bowel function, is being treated with a single drug (tacrolimus, no steroids) and is getting a dose two or three times a week instead of daily. If you can achieve that, then your quality of life is very good—close to normal. There’s only one final step, and that is to get completely off of immunosuppression. I don’t think that will be possible for every patient. But it should be possible to at least reduce the burden of immunosuppression for just about every patient.

DR. RICKETTS: And does the same hold for liver transplantation?

DR. STARZL: Yes, absolutely. And if you take a group of liver recipients who have gotten completely off drugs, they actually have close to a normal life expectancy.

DR. RICKETTS: Tell me about the Thomas E. Starzl Transplantation Institute here at the University of Pittsburgh.

DR. STARZL: I didn’t have anything to do with that. [Laughs]

DR. RICKETTS: Are you the director?

DR. STARZL: No, I have a sphere of influence, that’s all. I don’t want more. One of the promises made to me by Hank [Bahnson] after I decided that I didn’t want to be a chairman anymore [University of Colorado] was that I wouldn’t have administrative duties, and that I would not have to go to these meetings where they divvy up money or space or anything like that. I was off that treadmill. So I’ve not been to a meeting like that in the last 25 years.

DR. RICKETTS: You’re lucky. [Laughter]
DR. STARZL: They formed this thing [the Institute], and they named it in 1996. I did direct it for a day or so, but [laughs] then I pronounced myself as “emeritus.” After I retired from active clinical surgery [1991], I took a year off to write the book, *The Puzzle People*. And then I wanted to get back into research. During all of the time from 1992 onward, I worked on the research that led us to where that paper goes [points], which is tolerance. Essentially all of the many clinical and experimental accomplishments of the Transplantation Institute were made possible by the leadership up to 2004 of Dr. John Fung, a world class surgeon and PhD immunologist.

DR. RICKETTS: So how many, more or less, research staff are in the Institute? I mean, you must have hundreds of PhDs.

DR. STARZL: The number of people who show up at the Institute’s annual Christmas party probably is in the range of about 1,000. I don’t know how many of those belonged.

DR. RICKETTS: And it [the Institute] involves all organs that are transplanted.

DR. STARZL: All organs and all people on the clinical services. The clinical services are really the crucial components of the Institute.

[END TAPE 2, SIDE B]

DR. STARZL: I influence the management of a few cases, but try to do this at arm’s length. I have a meeting every Monday night, and we go over the tolerance protocol cases. They [the clinicians] usually do what I suggest about medications and strategy and what not, but the final decisions are theirs. I don’t want any formal title except professor of surgery. That is how I sign all my papers and letters.

DR. RICKETTS: So you had trainees from all over the world train with you. Did you do that purposely to try to disseminate knowledge to the rest of the world or did you just take the brightest people that came your way?

DR. STARZL: Well, both. Certainly, we ended up with the brightest people whenever there was a major breakthrough. As soon as the word got out about those early kidney cases in Colorado [1962-63], we were flooded with people from all over—from different countries, as well as the [United] States—wanting to learn kidney transplantation management. Because we had no funding for this work, we had to ask the fellows’ sponsoring university or their country to pay their way. We never paid a salary for at least the first year. Some stayed for short periods, others remained for as long as 15 years before going back home, and some stayed in the United
States permanently. A flood of departmental chairmen and division chiefs around the world emerged from the talent pool. This was a highly refined group of truly skilled and wonderful people.

Many people who worked with me throughout the 1960s and 1970s on the Colorado kidney service also were interested in being on the ground floor of liver transplantation. But enthusiasm for the liver was hard to maintain because of the high mortality, the small number of cases (about one per month), and opposition from some of the non-surgical faculty. Then in 1980 during my last year in Colorado, and from 1981 onward here in Pittsburgh, liver transplantation went over the top. What happened as visitors and trainees came to Pittsburgh was probably on an even larger scale that had occurred in Denver with kidney transplantation in the 1960s. The people who came to Pittsburgh in the 1980s and early 1990s are now well known, and can be found in almost every country. There is hardly a liver transplant surgeon in the world who wasn’t either a fellow here during that time, who wasn’t trained by someone who was, or who wasn’t a student of my friend, Sir Roy Calne (Cambridge, England). Departmental chairmen, division chiefs, and section chiefs too numerous to keep track of came out of that group.  

[Telephone rings]

DR. RICKETTS: I’ll stop this.

[Recording interruption.]

DR. RICKETTS: So we were talking about all the people who have come through here, many, many, many trainees, many of whom, as you’ve said, arrived and formed departments and are division chiefs. Can you name a few of whom you are most proud?

DR. STARZL: Oh, I’m proud of them all and would hesitate to single anyone out. At your own school (Emory University School of Medicine) do you know Andy [Andrei C.] Stieber, or Tom [Thomas] Heffron? Andy trained here in the 1980’s and Tom was trained at the University of Nebraska by Byers (Bud) Shaw [Jr.] who also was part of the Pittsburgh 1980s crop (fellow and faculty 1981-1985).

DR. RICKETTS: Andrei? Sure.

DR. RICKETTS: Who else of your trainees stands out in your mind?

DR. STARZL: If you focus on an individual, you’re looking always at multiple factors: the conceptual part of their professional lives which may or may not involve research, their technical abilities, their ability to manage patients, and then the element of true grit. There were people who, no matter how tough the case, would stay at the operating table for 24 hours
and never leave it until every last bleeding capillary was brought under control.

DR. RICKETTS: Right.

DR. STARZL: Everyone is different, but I consider them all to be equal. I can’t think of anyone whom I’m ashamed of. What they had in common was integrity, honesty, and—considering their unique qualifications—the absence of pretentiousness.

DR. RICKETTS: You reminded me. I was going to ask you what your role was in the ultimate development of UNOS [United Network for Organ Sharing].

DR. STARZL: I also described that in The Puzzle People. UNOS was aspiring to be the agency for organ allocation when that [National] Organ Transplant Act was passed [in 1984]. Earlier, I described Chick [C. Everett] Koop’s indispensable role in empowering transplantation. However, he did not think the government should actually be involved in medical practice, so he was leery of that [National] Organ Transplant Act. The act called for a contract to be signed between the government and a private agency that would take charge of organ allocation. UNOS, a preexisting regional allocation group in the southeastern states [founded in the 1960s by David Hume in Richmond], was one of the bidders. They and other bidders were given money with which to come up with a contract. There was so much infighting, especially amongst the transplant surgeons, that no one could agree about anything. The problem was that everyone had an understandable interest in controlling organs obtained from cadaveric donors in their own hospitals and surrounding area while also wanting access if possible to organs in other regions.

Although those who were in the bidding for the contract had a year or more to come up with a contract, UNOS and the other contenders had failed to develop a document. They could never get one written because they could not develop a consensus. As I described in The Puzzle People, we already had developed a system of equitable organ allocation in Pittsburgh. It was based on computer technology, and was designed to prevent cheating within our own region—deals cut for any kind of consideration or selection of recipients to whom someone on our team had promised an organ. We also wanted to eliminate racial or gender bias that would cause a candidate recipient to be overlooked. So we set up a system to prevent all that, and had it in place for more than a year.

In fact, I had written a paper with Hank Bahnson describing our system and our experience with it, and had submitted it to JAMA [Journal of the American Medical Association]. The manuscript was sent by the journal for review to a New York surgeon named Felix [T.] Rapaport, who was on the
UNOS committee that was trying in vain to come up with a contract. When he saw the *JAMA* manuscript, he said, “Shit, here’s a contract. It’s right here. It’s being published.” [Laughs] So they contacted me. I was not even involved in UNOS. When they contacted me, they were facing a two-week deadline after which they would be in default. Now they sucked me in, took the *JAMA* paper, used it word for word as the contract, and sprang it on the membership of the two American transplant societies at their annual meeting in June 1987. The members of the American Society of Transplant Surgeons and the American Transplantion Society both said, “Vote it in.” So my *JAMA* paper was the contract that just made the deadline, was accepted by the government, and went into effect in November 1987. [Laughs]

DR. RICKETTS: So it’s based on yours, because you already had a system going.

DR. STARZL: They just used our system. But, as soon as the government awarded UNOS the contract, the possibility was promptly introduced of permitting so-called “variances” from the supposedly “national” system. The local and regional variances undid quite a bit of the value of the original plan. These had to do mainly with giving too much credit for less than perfect levels of HLA [human leukocyte antigen] matching. Our original Pittsburgh system gave major credit points only for a perfect or near perfect HLA match. We had proven that this was the correct and fair way to exploit tissue matching—in research with a guy at UCLA named Paul Terasaki, who was the king of the typers.

In earlier studies with Terasaki, we had shown that if you had a perfect HLA match between donor and a given recipient, it gave the recipient a very real survival advantage. There usually are six antigens that are being matched. Our experience had demonstrated that it was worth looking for a perfect match. Once you got away from a perfect match, however, anything less all the way down to as many as six mismatches didn’t make any difference. So our original Pittsburgh plan called for a scrupulous search for a perfectly matched recipient when a cadaveric kidney became available. But if such a recipient could not be found, no credit or very little credit was given for any lesser level of matching. This was all changed by the variances that immediately crept in. Just to give you an example of a common variance, six credit points toward getting the kidney were given if you had a perfect match, five, four, three, two, one credit points if you had these lesser degrees of matching. This linear credit scale overturned a system that was based on real biology, and it introduced something that institutionalized racial bias.

Why the bias? Simply because it was now hard to match the African-Americans. Their HLA types are distant enough from the Caucasian and the Asian population so that the linear credit system greatly reduced the black patient’s chance for a kidney or any other organ. Once this variance was
installed, it took 15 years for UNOS to accept the fact that an error and injustice had been institutionalized and to get back where we started. This was an uneven 15-year journey, every step of which took two or three years, because the backtracking occurred unevenly with long pauses in between times. In the end, UNOS was forced into their course correction, not by the science of it at all, but as the result of public pressure—by the outrage when it became obvious that the variance amounted to systematic ethnic discrimination.

So we’re back now to the policy of a big search for a perfect match, after which everything else is a wash. We’re right back to square one. [Laughs]

DR. RICKETTS:  What comes around goes around.

DR. STARZL:   Yes.

DR. RICKETTS:    Well, let’s go on now to tolerance. What are your current views regarding tolerance for solid organ transplantation?

DR. STARZL:   Oh, I think it’s well within our grasp, at least partially. Why is fully explained in that pre-print. It’s almost too complicated to get into, but in a nutshell, my view of tolerance began to form as early as 1962 and 1963, but did not come into focus until we carried out a simple research study after I finished [writing] The Puzzle People. As explained in the epilogue of that book, it had become an embarrassment to make the series of advances that permitted the rise of human organ transplantation without knowing what actually was being accomplished. So we contacted those original Colorado kidney patients that had been treated in 1962 and 1963 and also our long-surviving liver recipients. We brought 30 of them to Pittsburgh and studied them. We biopsied the transplanted organs—in kidney or liver recipients—and also got little pieces [biopsies] of recipients’ skin, lymph nodes, and in some cases other organs. We also took blood samples.

DR. RICKETTS:    That’s laid out nicely in your talk on the mystique of organ transplantation, which I read as well.

DR. STARZL:   That was when we found—[he turns from microphone looking for photograph]—maybe that picture isn’t even in here, but you know what it looks like. We found small numbers of donor leukocytes unevenly distributed all over the recipient (so-called microchimerism). This is what I suspected we would find. However, the findings were at first generally considered to be amazing and not credible. Eventually, however, it was possible with the microchimerism discoveries to explain essentially all previously enigmatic observations made in experimental and human organ transplant recipients and also to recognize that the worldwide practice of
giving heavy post-transplant immunosuppression could have the self-defeating consequence of shutting down tolerance mechanisms. In the last few pages of this article [Immunologic Research], it is shown how results improved when we corrected the error of over immunosuppression. The article that we’re preparing for the NEJM explains further how this insight can be exploited [Turns pages]

DR. RICKETTS: So if I can put it in laymen’s terms, I think as I understand it, you felt that we were over-immunosuppressing people so that they couldn’t develop a rejection episode and that the rejection episode is actually important to developed tolerance later.

DR. STARZL: Yes, but this must be explained. You have to have immune activation—call it rejection—but hopefully not so such that it permanently damages the organ. You have to have immune activation because the fundamental mechanism of tolerance is clonal deletion. And, clone deletion depends on clonal activation. If you prevent the first step of clonal activation with heavy immunosuppression, you can’t delete the clone efficiently. You can’t keep giving this much heavy immunosuppression permanently, so you eventually have to come down [reduce treatment], and as you get lower, the clone recovers with the result of chronic or delayed acute rejection. The sequence—ending with chronic rejection—was being seen in essentially every organ transplant clinic in the world.

The relation of rejection to organ engraftment, and to tolerance—and how immunosuppression can be most efficiently given—were described in a paper that I published [in 2001] with a guy named Rolf [M.] Zinkernagel [Starzl TE, Zinkernagel R: Transplantation tolerance from a historical perspective. NATURE Reviews: Immunology 1:233-239, 2001]. This was our second joint review. Our first one, explaining how engraftment occurs, was published in 1998 [Starzl TE, Zinkernagel R: Antigen localization and migration in immunity and tolerance. New Engl J Med 339:1905-1913, 1998]. The 1998 review was based on our Pittsburgh microchimerism studies of 1992, and on independent studies in Zurich by Zinkernagel of infection immunity. Our conclusions about immunity, tolerance, and immune regulation challenged the views of several previous generations of immunologists, who had been trying to explain alloengraftment by mechanisms other than those of donor leukocyte chimerism. Our paradigm was therefore controversial and difficult to explain to persons whose grants or legacy depended on not understanding it [chuckles]. Also, who wants to be put out of a job by finding the answer to an unresolvable question?

In addition, it was counter-intuitive to think that immunosuppression could itself be self-defeating and prevent you from getting tolerance. Immunosuppression has been indispensable for the development of transplantation. The critical therapeutic principle in our view was to use as
little immunosuppression as possible [the principle of minimal post-transplant immunosuppression]. Here’s how it works [pointing to an artist’s drawing. This is the immune response. Here, it’s going up [the response]. And if you don’t control it, of course, you get rejection. But if you completely prevent the antidonor response, by piling on immunosuppressive drugs from the time of transplantation—

DR. RICKETTS: That’s the old way.

DR. STARZL: That is the old way. And the new way is [shows different document] is this one. It begins with a second therapeutic principle: that is, recipient pretreatment. We currently pre-treat with an anti-lymphoid drug. What drug? Currently Campath. That’s an ALG.

DR. RICKETTS: Granddaughter of ALG.

DR. STARZL: Yes. It’s a super ALG. We started using [our strategy of tolerogenic immunosuppression] with one of the old-fashioned ALG’s (thymoglobulin), and we published a series in The Lancet in 2003 [Starzl TE, et al. Tolerogenic immunosuppression for organ transplantation. The Lancet 361:1502-1510, 2003]. Then we switched to the Campath. The objective with either old-fashioned ALG or Campath is to weaken the expected antidonor response before arrival of donor antigen, making it easier to delete [the pretreatment principle]. This allows you to give minimal immunosuppression—a single drug. You can almost always accomplish variable deletional tolerance that is roughly quantifiable by the amount of long-term immunosuppression needed to maintain stable graft function.

DR. RICKETTS: In the long term, then, they accept—

DR. STARZL: Yes, and then you sometimes can stop [immunosuppression]. Here is the graphic course of a liver recipient whose follow-up was only six months [when] this paper was written in late September 2006. She was pre-treated with Campath here [point], and then leukocytes [an infusion] from her live liver donor. Then [she] had [the] liver transplant here. Her graft hepatic artery clotted in the first few post-op days, and she had to have a [arterial] graft put on on the 13th day. She was very sick by this time, and all immunosuppression was stopped within about eight weeks. I probably have a slide here of her follow-up out to 13 [months]—she has been off of all treatment for one year, completely off. I was over at the NIH [National Institutes of Health] here a month or so ago and presented the results from this and 14 other cases [10 liver and five kidney recipients]. These were all adults. But it’s pretty obvious that we’re also going to be able to get kids—liver and other kinds of organs recipients—nearly off or in some cases off of drugs. We haven’t used the leukocyte infusion protocol in children, but I am sure this will be done in the future.
And here are the results with five adult kidney recipients. This recipient of an HLA-matched kidney (from his sister) was reduced to one dose a week of tacrolimus by about six weeks. And this man—also with an HLA-matched kidney [brother donor] was on one dose per week by six weeks. This man developed recurrent IgA nephropathy, and we couldn’t wean him. Two other recipients of HLA mismatched kidneys have been weaned more slowly.

DR. RICKETTS: It’s really exciting.

DR. STARZL: It’s very exciting.

DR. RICKETTS: Very exciting. You’ve been a visionary all your life, and people are slow to “catch on,” it seems like.

DR. STARZL: Sometimes.

DR. RICKETTS: Is there a general acceptance of this?

DR. STARZL: It’s been very controversial. Well, maybe not anymore. The original paradigm [of alloengraftment and tolerance] was published in *The Lancet* in 1992, almost 15 years ago. It created a firestorm. The reason for that is that if what we were saying at that time was true, many established dogmas would be overturned—not only clinical dogmas but basic science dogmas. This table consists of a partial list.

DR. RICKETTS: [Chuckles]

DR. STARZL: The most clinically relevant dogmas on the list?—okay—“macrochimerism is the holy grail of organ transplantation.” It’s not true. “Tolerogenesis is tougher in humans than in lower species.” Not true. “Organ engraftment occurs by leukocyte chimerism—-independent mechanisms.” That’s certainly not true. All these others [dogmas] are basic science issues, like “antigen-specific memory cells do not require persistent antigen.” Probably not true, but here it is. And I have suggested in this manuscript that a very large amount of detailed transplantation science should be reassessed and re-interpretated in this new framework [paradigm].

[Our immunologic paradigm] is quite well accepted in some circles. This manuscript that I have been turning to as we talked was the keynote address for the inaugural meeting of a new immunologic society named in honor of Bob [Robert A.] Good. [The Robert A. Good Immunology Society] You probably know his name.

DR. RICKETTS: Yes.
DR. STARZL: Great figure. He’s now dead.

DR. STARZL: When I was invited in 2006 to give this lecture, I was seriously concerned about making this paradigm the theme of my one-hour talk to an audience of some of the world’s most distinguished immunologists. The lecture would have to begin with the concession that the error made nearly a half-century ago [that we discussed earlier] had created a flawed foundation for the then new field of transplantation immunology and particularly clinical organ transplantation. My question was “would I be shot full of holes?” As it turned out, the only comments were along lines of “How could this have been missed?”

DR. RICKETTS: They’re finally catching on.

DR. STARZL: [Laughs] Within the transplant community also there has been a big change. I don’t think that most transplant clinicians are interested in the basic science details of the matter. But what has happened is the realization that “Oh, wow, yeah. We’ve been giving too much immunosuppression.” And there has been a reform that consists of dampening down this excessive treatment. I think that’s not—[insignificant].

DR. RICKETTS: That’s fantastic. I was going to ask you about some of your thoughts about the future, but maybe this is going to be our answer. What would you envision the role of artificial organs to be?

DR. STARZL: I think it’ll be a big one, but that’s down the line awhile. I don’t think artificial organs will ever replace live grafts, but there certainly is going to be—the engineers are very clever—there will be a lot [of progress].

DR. RICKETTS: What about tissue-engineered organs?

DR. STARZL: Well, now you’re talking about stem cell biology.

DR. RICKETTS: Yes.

DR. STARZL: That’s, at the moment, a pie in the sky. If people are envisioning using stem cells to grow a limb or a kidney or something, if that comes down, it’s going to be long after you and I are both dead.

DR. RICKETTS: What about xenotransplantation?

DR. STARZL: Well, that too is problematic. I’ll give you, if I can find it, a very nice paper that we just published in the *Proceedings of the National Academy of Sciences [of the United States of America]* [Koike C, Uddin M, Wildman DE, Gray EA, Trucco M, Starzl TE, Goodman M: Functionally...
important glycosyltransferase gain and loss during catarrhine primate emergence. Proc Natl Acad Sci 104:559-564, 2007]. The paper might suggest that xenotransplantation is going to be tough or impossible. But the paper is really not about that. The paper is about what happened in evolution 25 million years ago that allowed the sudden, rapid ascension of higher primates, including humans. A series of metabolic changes occurred at that time, including the inactivation of a gene, whose product foreclosed any possibility [until recently] of using lower mammalian organs in humans. It’s called the alphaGAL gene.

During the time this gene was inactivated, about 25 or 23 million years ago, multiple other genes underwent mutation. Several of these genes, including alphaGAL, had to do with sugar metabolism. The complex of mutations during this evolutionary “hot” period contributed to the switch from anaerobic to aerobic metabolism that was associated with rapid higher brain development in primates. It’s one of these lines of inquiry initiated by transplantation that is important in and of its own right. I suppose there’ll be a big spit fight that goes on—

DR. RICKETTS: [Laughs]

DR. STARZL: --for a while. But maybe we are wandering too far from tolerance.

DR. RICKETTS: I want to go back to the Ladd Medal. You were the 1993 recipient, and in looking through your CV, you put little asterisk marks on your most important awards out of many awards that you’ve had, and I noticed that the Ladd Medal did have an asterisk, so it was important to you. Why was it so important to you, the Ladd Medal?

DR. STARZL: I don’t know, probably because the children have been so important in the whole story, all the time, from the beginning to the end.

DR. RICKETTS: What do you think that your main contribution to the lives of the children has been?

DR. STARZL: Well, letting them grow up. But the tolerance issue has always been conceptually critical as far as I’m concerned because so many of those kids get off drugs.

DR. RICKETTS: They have a long life ahead of them.

DR. STARZL: We’ve got 40 or 50 children that have been able to come off of drugs without rejecting for at least 10 years. We run a weaning clinic here [that includes such patients from the distant past]. Anyone who’s on chronic immunosuppression has a shortened life expectancy. But if you can
get a child off drugs and they’re stable, they can have a normal life expectancy. Although the number of such patients is small, their existence means that transplantation is potentially curative, not merely palliative. Therefore tolerance as reflected by a low or zero need for immunosuppression should be our objective. If you’re on drugs and you have to go through life drug-immunodepressed, you’ve always got a bat wing on your shoulder ready to bite. And with what we have learned, we can just make it go away, or at least remove the teeth.

DR. RICKETTS: Fantastic.

DR. STARZL: That’s the real issue. I was always fearful 30 or 40 years ago—particularly because I knew the patients so well—of getting a phone call in the middle of the night that some horrible thing had happened to somebody. Once they get off of drugs, if the horrible thing happens it means they’ve had a car accident or they’ve had one of the normal vicissitudes of life, but they haven’t been killed by the immunosuppression.

DR. RICKETTS: I noticed that you’re very empathetic in the stories that you tell about your patients who have been children. Did you ever consider a career in pediatric surgery?

DR. STARZL: Well, I was in pediatric surgery in a way, because at the time [early 1940s] I started my adult life, there was only one well-known pediatric surgeon around, and that was [Robert E.] Gross. And, of course, he was that guy up in Boston who was everybody’s hero. And then that next generation came along. There were a few in the early 1960s, but pediatric surgery was underrepresented.

DR. RICKETTS: Just getting started.

DR. STARZL: Yes.

DR. RICKETTS: Looking back over your career, would you have done anything differently?

DR. STARZL: I don’t know. Probably not, because what’s the use? You know that movie, Sliding Doors?

DR. RICKETTS: No, I haven’t seen that one.

DR. STARZL: Well, you change one little thing and there’s a domino effect, you never know what the outcome would be. If I were to change anything, it would be to spend more time with my children. When I went to Colorado in late 1961, my children were three to five years old. Within a few months, gasoline was thrown on the fire with the big breakthrough in kidney
transplantation. And then liver transplantation followed that. In the blink of an eye, 18 years went by and all three children were in the university.

I don’t know if what happened in transplantation could have been brought forward without steely concentration on the professional side. In other words, if I had been a soccer father, and that was the option that existed at that time, it is possible that little if any of my transplantation work would have been done.

DR. RICKETTS: Dr. Starzl, as a representative of the surgical section of the AAP [American Academy of Pediatrics], I want to personally thank you for all the contributions you made to the lives of children with respect to liver disease and renal failure and now short gut syndrome. We owe you a tremendous debt for all you’ve done, and I want to thank you for that. Would you have any final comments that you might want to make before I let you escape?

DR. STARZL: Well, I’m honored, actually, that you came here, and I’m grateful for the trouble you took. It would have been just as reasonable or better for me to come down there. [Laughs]

DR. RICKETTS: It might have been a little bit warmer down there. [Laughs]

DR. STARZL: Yes. So anyway, thank you for coming.

DR. RICKETTS: Well, thank you very much, Dr. Starzl.
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THOMAS EARL STARZL

SUMMARY CURRICULUM VITAE

PERSONAL

Born: March 11, 1926, Le Mars, Iowa
Marital Status: Married to Joy Denise (nee Conger) 3 children

EDUCATION

1950 Northwestern University, Chicago, IL, M.S.
1952 Northwestern University, Chicago, IL, Ph.D.
1952 Northwestern University, Chicago, IL, M.D.

BOARD CERTIFICATION

1958 General Surgery
1959 Thoracic Surgery

POSITIONS

1958 Instructor in Surgery, Northwestern University, Chicago, IL
1959 Associate in Surgery, Northwestern University, Chicago, IL
1961 Assistant Professor in Surgery, Northwestern University
1962 Associate Professor in Surgery, University of Colorado
1964-80 Professor in Surgery, University of Colorado
1972-80 Chairman of Surgery, University of Colorado
1981- present Professor of Surgery, University of Pittsburgh Medical Center
1990 Director of Transplant Institute, University of Pittsburgh
1996 Professor of Surgery, and Director of the Thomas E. Starzl
Transplantation Institute, University of Pittsburgh

PROFESSIONAL SOCIETIES

Total: 57
International Transplantation Society
1970 Vice President
1990 President
American Society of Transplant Surgeons
1975 Founding President
Transplant Recipients International Organization
1987 Founding President
1987 Permanent Honorary Chairman

LEARNED SOCIETIES

1971 American Academy of Arts and Sciences
1991 L'Academie Nationale de Medecine (French Academy of Medicine), Paris, France
1999 American Philosophical Society
1999 National Academy of Science (Institute of Medicine)

HONORS AND AWARDS

1952 Student Borden Award
1959-64 Markle Scholarship
1965 Alumni Achievement Award, Westminster College
*1965 Prix Socite International de Chirurgie
1967 Colorado Man of the Year Award
*1968 William S. Middleton Award, Outstanding Research in Veterans Administration System
1969 Merit Award, Northwestern University
1969 Modern Medicine Distinguished Achievement Award
*1970 Eppinger Prize, Freiburg
1971 Award, Council Creative Research (Highest Award, University of Colorado)
*1974 Brookdale Award (Highest Award of American Medical Association to person under 50 years)
1975 Josiah Macy Scholar
1976 Robert L Stearns Award (Highest Award of Colorado Alumni Association)
*1978 David M. Hume Memorial Award (Highest Award National Kidney Disease Foundation)
1981 Pittsburgh Man of Year
*1982 Sheen Prize, American College of Surgeons (highest award)
1985 Gold Medal, Fondazione Giovanni Lorenzini, Italy
1985 Pennsylvania Medical Society Distinguished Service Award
1986 Alumni Medal Northwestern University
1986 Pittsburgh Man of the Year
1986 Biannual Prize of Italian Hepatology Society
1986 Distinguished Service Professor of Health Sciences of University of Pittsburgh
1987 Jewish National Fund, "Friend of Israel" Award
*1989 Bigelow Medal, Boston Surgical Society
*1989 City of Medicine Award (Durham, NC)
*1990 Medallion for Scientific Achievement, American Surgical Association
1991 American Liver Foundation Distinguished Service Award
*1991 William Beaumont Prize in Gastroenterology
1991 Recipient Hugh R. Butt Award, American Gastroenterological Association
1991 American Association for the Study of Liver Disease Distinguished Achievement Award
"Thomas E. Starzl Surgical Ward," Denver VA Hospital
*1991 Fondazione Basile Prize World Association of Hepato-Pancreateo-Biliary Surgery, Italy
*1992 Medawar Prize, Transplantation Society (Highest Award)
*1993 William Ladd Medal, Pediatric American Surgical Association
1994 Governor's Award for Excellence in the Sciences
*1995 Jacobson Innovation Award of the American College of Surgeons, Chicago, IL
1995 The Order of Saint James of the Sword, Portuguese Orders of Merit (Knighthood)
1995-98 Department of Veterans Affairs Distinguished Physician Award
1996 Lifetime Achievement Award, Indian Transplantation Society
1996 Gold Medal of the Catalan Transplantation Society, Barcelona, Spain
1996 Thomas E. Starzl Chair in Surgery and Designation of Thomas E. Starzl Transplantation Institute
1997 Aaron Bannett Prize, Bannett Transplant Society
*1997 International Chiron Award, Italian Academy of Science
*1998 Lannelongue International Medal (awarded every 5 years) Academie Nationale De Chirurgie, Paris, France
1998 Roche Pioneer Award American Society of Transplant Surgeons
*1998 Thomas E. Starzl Transplantation Ward, University of Colorado
*1998 Ellen Browning Scripps Medal and Prize, Scripps Memorial Hospital LaJolla, CA
1999  First Recipient of The Global Conference Institute’s Healthcare Humanitarian Award.
1999  Chancellor’s Medallion Highest Distinction, University of Pittsburgh
2000  Distinguished Alumnus Award of Northwestern University Medical Center.
*2001  Recipient of the King Faisal International Award in Medicine, Riyadh, Kingdom of Saudi Arabia.
*2003  Prince Mahidol Award for 2002, Mahidol University at Salaya, Bangkok, Thailand.
2004  John Scott Medal Award, City of Philadelphia and the American Philosophical Society, Philadelphia, PA.
2005  Gold Medal of the Mutual Madrilena Foundation, bestowed by Their Majesties the King and Queen of Spain, Madrid, Spain.
*2005  Recipient of one of the 8 Presidential National Medal of Science medals awarded for 2004 (announced November 15, 2005).

*Most Significant

HONORARY FELLOWSHIPS

1970  Deutsche Gesellschaft fur Chirurgie, Munich
1984  La Societe de Chirurgie de Lyon
1991  Asian Surgical Association
1993  John Hopkins Society of Scholars
1995  Japanese Surgical Society
1996  European Surgical Association
1996  Romanian Academy of Medical Sciences
1997  Austrian Surgical Society
1997  Italian Surgical Society
1998  Sociedad Argentina de Trasplantes
1998  Association Francaise de Chirurgie, Paris, France
1999  International Xenotransplantation Association
2000  Philadelphia Academy of Surgery
2000  Spanish Association of Surgeons, Madrid
2005  European Society of Organ Transplantation

HONORARY DOCTORATES (total 24)


Doctor of Law (1): University of Wyoming (1971)

EDITORIAL BOARDS AND EDITORSHIPS

Total: 40

PUBLICATIONS

Scientific Articles: 2219
 Full Books: 4
 Chapters: 302
 Presentations at Major Meetings: 1319

NAMED LECTURESHIPS

Total: 114