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DELBERT A. FISHER, MD

Interview conducted by
Michael Chappelle
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INTRODUCTION

Delbert A. Fisher is Professor of Pediatrics and Medicine Emeritus at the UCLA David Geffen School of Medicine and Vice President of Science Innovation Emeritus at Quest Diagnostics. Dr. Fisher's career has spanned the spectrum of pioneering research in immunoassay development, hormone kinetics, receptor characterization, biochemical studies, and cellular and molecular methods. He has made crucial contributions to the development of a simple, highly effective newborn screening test for congenital hypothyroidism, a condition that when treated within the first month of life completely prevents mental retardation from this disorder. Today, every state in the United States tests newborns for congenital hypothyroidism.

BIOGRAPHICAL SKETCH

Dr. Fisher, a fourth-generation Californian, was born in Placerville, California, in 1928. He graduated Phi Beta Kappa from the University of California at Berkeley in 1950 and earned his MD degree from the University of California San Francisco in 1953. As a medical student he began working with Donald Pickering, studying thyroid function in newborn Macaque monkeys. During his pediatric residency at the Medical Center of the University of California San Francisco, he co-authored six papers describing the effects of radioiodine thyroid ablation on growth and development and iodine metabolism, and he pioneered the use of sodium L-thyroxine for replacement treatment of the athyroid monkey and the human infant with congenital hypothyroidism. After two years as an Air Force physician, Dr. Fisher rejoined Dr. Pickering at the University of Oregon in 1957, where he completed pediatric and endocrine training and continued metabolic studies in infant monkeys. In 1960, he was recruited to the University of Arkansas by Theodore Panos, where, in collaboration with Thomas Oddie, a nuclear physicist, he used whole body isotopic counting technology to study iodine and thyroid metabolism and thyroid hormone kinetics in human subjects. In 1968, Dr. Fisher was recruited by Joseph St. Geme Jr. to the Harbor-UCLA Medical Center as head of the Division of Pediatric Endocrinology and Metabolism. At Harbor-UCLA, he joined a unique group of talented people including William Odell, Inder Chopra, Albert Parlow and Jean Dussault. Together they developed a number of immunoassays for thyroid-related hormones and applied them to the study of the ontogeny of the mammalian fetal thyroid system. Improved understanding of neonatal thyroid physiology coupled with the new immunoassays for thyroxine and TSH were critical events for the development of screening for congenital hypothyroidism. Although thyroid function has been a major focus of his laboratory, Dr. Fisher, his collaborators, and his fellows explored other areas of mammalian endocrine system ontogenesis, characterizing maturation of the neurohypophysial hormone systems; the atrial natruiretic hormone system; the renin-angiotensin system; perinatal aspects of insulin, glucagon, and carbohydrate metabolism; fetal catecholamine metabolism and vascular homeostasis; and maturation and hormone regulation of epidermal and transforming growth factors in mammalian fetal-neonatal growth and development. By 1974 he was vice chair of the Department of Pediatrics, and in 1984 he was named chair of the department. In 1991, Dr. Fisher was recruited by Albert Nichols to become president of the reference laboratories for the Nichols Institute. After several corporate mergers, he was named vice president of science and innovation at Quest Diagnostics, the largest laboratory testing organization in the world. Dr. Fisher is the author or co-author of over five hundred scientific papers, book chapters, reviews, and nine books; and he was editor-in-chief of the *Journal of Clinical Metabolism and Endocrinology* as well as *Pediatric Research*. Dr. Fisher has served as president of numerous medical societies including the Lawson Wilkins Pediatric Endocrine Society, the American Pediatric Society, the American Thyroid Association, and the Endocrine Society.

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I. FAMILY BACKGROUND AND EARLY YEARS

Chappelle: Dr. Fisher, would you tell me a little bit about your family background? When did your ancestors first come to California?

Fisher: I'm a fourth-generation Californian. My great-grandmother came to California in the mid-1800s and settled in Placerville. She had been married before and married once more in Placerville to Jacob Fisher. My father was the second of their children, and he married Thelma Johnson, also from the Placerville area. They had four children, myself, two brothers, and a sister. And so we are the fourth generation of the family. The family members first came to Philadelphia and settled in Western Pennsylvania in the late 1700s. My mother's family were second-generation Californians and were from Switzerland. My great-grandmother was German, my grandmother was Swiss, and Jacob Fisher, I think, was English. So we are mostly German, English and Swiss.

Chappelle: Did you discuss your family background with your parents as you were growing up? Was that a big part of your family?

Fisher: No, that's all information that we have been accruing--my siblings and I--over the past four or five years. One of my uncles decided to collect some family history, and so it's been recently interesting.

Chappelle: What did your mother and father do for a living when you were growing up?

Fisher: They were high school graduates. My father initially worked for the Pacific Gas and Electric Company, running one of the power houses on the American River and that was my first home, actually, when we came from Placerville after I was born. Then the siblings were born there. My father moved from there back to Diamond Springs, which is three miles--it's a suburb of Placerville--and started a grocery store. So we had the grocery store experience, and I worked in the grocery store when I was in grammar school.

Chappelle: What type of education did you have?

Fisher: I went to grammar school in Diamond Springs. There was one school. Placerville was about ten thousand people; Diamond Springs, maybe, was twenty-five hundred, maybe more now. Mrs. Fitzgerald was the teacher who I had most of the time or much of the time in grammar school. I suppose there were fifty of us in the school, maybe sixty; so it was small time, small town environment that I grew up in.

Chappelle: What was it like growing up at the end of the Depression and during World War II?

Fisher: We didn't, at the time, think that anything was unusual. I mean, there was a lot of rationing of shoes--and getting tennis shoes--and tires and gasoline and a constant recognition that there was a problem somewhere; but in our day-to-day lives, it made little difference.

Chappelle: What kind of a student were you?

Fisher: I was usually a nerd, even in grammar school. I went on to high school, and I went from Diamond Springs to El Dorado County High School in Placerville by bus until I graduated from high school and headed off to Berkeley. The family then moved to Placerville, and so about the time I left that area of the country and went to Berkeley to college. The family had moved to Placerville, and my communication with them then was based on visitations and summers.

Chappelle: When did you decide that you might like to become a physician?

Fisher: I guess it wasn't something that acutely happened. I knew a young physician in Placerville, Dr. Sorocco, and I initially was impressed because he drove around in a nice convertible, which was certainly unusual in that area. We became acquainted and talked. And I had an episode of appendicitis and went to the hospital and had some other associations. So I was intrigued by medicine and in talking with him, so that was probably my first inkling about that kind of a way forward.

II. UNIVERSITY OF CALIFORNIA (1946-1955)

University of California Berkeley

Chappelle: Why did you choose to attend the University of California at Berkeley?

Fisher: There was never a choice. I mean the assumption was that that was where you would go if you couldn't afford to go to Stanford. UCLA was yet to be developed, and USC was considered athletic and not an academic kind of environment, and that wasn't an option. So I just assumed that I would be going to Berkeley.

University of California School of Medicine

Chappelle: What was your major?

Fisher: Medical science--from day one. That turned out to be a three-year program because I entered the University of California Medical School as a senior student. It was the last year that medical students in the first year spent their time in the Berkeley campus in the basic science departments. So it seemed my life was kind of programmed from the start: I assumed I would go to Berkeley, and medical science turned out to be the right [major], and

I moved quickly to medical school on the same campus--was Phi Beta Kappa.

Chappelle: Which of your mentors in medical school would you say had the greatest impact on you?

Fisher: It would be Donald Pickering; there is no question about that. I met him in the late-second year--we were then in San Francisco. He was looking for somebody to--especially a student--to help him take care of a monkey colony that he had built there, which was intriguing. We visited and talked, and so I went to work taking care of his infant monkeys--small monkey colony. It became a lifetime friendship and a prolonged period of interaction with him through my time at the University of Oregon.

Chappelle: How did you become interested in pediatrics, endocrinology, and the thyroid?

Fisher: Well, Donald Pickering was a pediatrician and an endocrinologist--one of the few endocrinologist-pediatricians evolving in the area in the United States--Lawson Wilkins on the East Coast, of course. Dr. Pickering was one of the few initial academic pediatric endocrinologists in the country on the West Coast, and pediatrics just went with it; I hadn't thought they were separated.

Chappelle: Was it that first experience of working with Dr. Pickering and working with the infant monkeys that caused an immediate interest in becoming a pediatric endocrinologist, or did it grow gradually?

Fisher: Working with Don Pickering and the monkeys was a really exciting time. He was developing this monkey colony because he wanted to use infant monkeys as a surrogate for infants to do endocrine studies. He had been at Yale, and he had met Gertrude van Wagenen, who had a monkey colony. She had agreed to send him infant monkeys, and they came by airplane. One of my jobs was to pick the infant monkeys up at the airport. Sometimes they would come in on the weekends and they would be dehydrated, and I would have to take them back to the apartment and give them fluids. My wife was a little disconcerted with that, but she became a monkey nurse, as well. So I was really pretty involved with what was going on and, gradually, learned about the research that was going on. Initially, it was just a way to make some extra money; it became much more than that. There were four principals involved: Dean Francis Scott Smyth, head of pediatrics, was involved; one of the radiologists in school was involved; and the head of the radioisotope laboratory was involved. They all became mentors as we proceeded--I as a junior partner--in that research project.

Chappelle: What were you doing in that project?

Fisher: Well, the basic program was to eliminate the thyroid gland from the infant monkeys after they were born--and they would become hypothyroid--and then

to follow them to see what happened to them. We measured their chemical changes in the blood; we measured the changes in their growth rate; we measured the changes in their brain weight, looking for the signs and symptoms that you usually see in hypothyroid infants. So the skeletal changes were studied using X-ray, and the changes were all outlined. So it was a rather comprehensive project.

Marriage

Chappelle: You got married around this time?

Fisher: I met my wife, Beverly, in Berkeley. We had a dinner with the Gamma Phi Beta sorority, and each of us had reluctantly attended that and met. It turned out that she was from the Mother Lode area, thirty miles from Placerville, in the next south county--she was from Amador County. We had a very similar background: her mother ran a grocery store, and she was an only child, and her mother was divorced. It turns out we were registering for Berkeley in the same long line at the same time that we went to Berkeley. We hit it off really well and quickly. She got her bachelor's degree in speech and psychology; she got a teaching credential. We were married in January of my sophomore year. She got a job in the San Francisco Unified School District, and we moved back to San Francisco. We were married in January, and she went to work in February. We will have our sixtieth anniversary this January.

Internship and residency

Chappelle: Why did you choose to do your internship and residency at the University of California San Francisco?

Fisher: I didn't consider anything else. Don Pickering was there, and what we were doing--the research with the monkeys and our growing personal relationship--and I stayed and did the first two years of residency there until I was drafted.

III. UNITED STATES AIR FORCE MEDICAL CENTER (1955-1957)

Chappelle: You were drafted in 1955 into the United States Air Corp?

Fisher: Yes.

Chappelle: What were your duties?

Fisher: I was sent to West Texas at Laughlin Air Base; it was a training base, a gunnery-training base. I was the pediatrician for the group. There were seven of us, actually: two internists, a urologist, a couple of surgeons, and a practicing physician--older--from Texas. Most of us had partial training and we weren't certified in our specialties, but we considered ourselves specialists there and

divided the patient requirements and their needs and the clinic visits, so that at least we got to practice our specialty areas.

IV. UNIVERSITY OF OREGON (1957-1960)

Chappelle: How did you come to be an Irwin Memorial fellow and chief resident in pediatrics at the University of Oregon?

Fisher: Don Pickering had decided to leave the University of California in San Francisco, and had an offer he couldn't refuse, I guess, to move the monkeys to Portland. The NIH was interested in setting up a primate research center. I don't know the details of all of that negotiation and how the NIH negotiated with the University. Don was the director though, and it was one of the first primate research centers that the NIH supported in the country. He called one day and asked if I was interested in coming to Portland and continuing our relationship, and I enthusiastically took him on. We had had our first child, Beverly and I, in Del Rio--where we were in the Air Force. David was born in 1956, so there were three of us when we moved back to Oregon. David went on to graduate from UC San Diego and get his MD degree at the UC Irvine and became an orthopedic surgeon, and he was in Santa Barbara. His hobby was racing cars; he was a racecar driver affiliated with the SCCA, the Sports Car Club of America. He had a BMW that he raced--the club of course helped him move around from one place to another. In 2001, the group was racing in Phoenix, and on Saturday afternoon he was in the final preliminary race and tragically died. Something happened and he hit the wall.

[Interruption]

Chappelle: What feature or features marked your time at the University of Oregon?

Fisher: Well, the first important event was the delivery of twins into the family--Thomas and Mary--they weren't identical twins. They were born in 1958. Tom went on to UC San Diego and got his physics degree and went on to get an MBA and is now a systems analyst for Qualcomm, here in San Diego. Mary got her BA from Berkeley and tried a couple of jobs and more recently married and is now managing a blended family with four children. She is now approaching--obviously--age fifty, so she has got her hands full, but doing a terrific job.

Chappelle: What about research that you were doing?

Fisher: We continued, Don and I, to do some monkey work. The most important change for me was the first year as a chief resident--that was my third year of pediatric training, and I was a chief resident there, and that was pretty time consuming--coordinating the activities of the resident staff. I was involved with teaching and patient care as well. Raising the new family--we lived in a two-story house with the only bathroom downstairs and the bedrooms upstairs--taking care of three

small children [David and the twins] was, for my wife, a bit difficult. It was a rather heavy schedule, trying to do the work with the research group as well, but I had exposure to the laboratory. I had a good friend who I worked with and learned to measure thyroid hormones and do a variety of laboratory kinds of testing. In addition, I became interested in fluid and electrolyte metabolism--in San Francisco as well as at the University of Oregon--treating the children. So Dr. Pickering and I wrote our first book, *Fluid and Electrolyte Therapy: A Unified Approach*, published by the University of Oregon Press. That was great fun, working with him. That was my first rather extensive writing activity, and I really enjoyed that. Actually, we wrote two other papers: one, "Therapeutic Concepts: Relating to Hypothyroidism in Childhood" and that was published in the *Journal of Chronic Diseases;* that was an exciting event for me at the time. The other one was "Infantile Hypothyroidism: Diagnosis and Treatment," and that was published in the *Pediatric Clinics of North America*. From my work in the laboratory, I got involved with the Department of Obstetrics. Dr. Ralph Benson was the chair of obstetrics, and he and Don and I were doing a study on thyroid function in pregnancy that we finally published. That turned out to be one of the earlier papers that was published using the butanol extractable method to measure thyroxine. There was limited information when we did that, but otherwise it wasn't a terribly impressive publication.

V. UNIVERSITY OF ARKANSAS (1960-1968)

Chappelle: How were you recruited to the medical school faculty of the University of Arkansas as an assistant professor and director of pediatric endocrinology and metabolism?

Fisher: Well, my time at Oregon--in terms of salary--was coming to an end. I had salary from the university for the residency and I had an Irwin Memorial Fellowship that Don had acquired from outside sources, and that was a two-year support. So at the end of that I had to find a job. Don and I were looking at options around the country for an academic job in pediatric endocrinology. There were two that came up--one at Yale and one at the University of Arkansas--and I visited both. The Yale option didn't turn out to be very interesting, and the University of Arkansas *did*. Dr. Theodore Panos had come from the University of Texas to head the department. Dr. Robert Ebert was chairman of medicine. James Melby was heading endocrinology and medicine. Dr. William Flanigan was heading nephrology and medicine and heading a clinical study center. Dr. Panos was interested in getting a research program going in pediatrics. I also met Dr. Thomas Oddie, who was a radiation physicist heading the radioisotope department, and he was interested in thyroid research. We rapidly got together--I was introduced to him on my visits. Dr. Panos, in addition, offered me research space in a brand new research building that had been constructed. So it was almost a no-brainer to decide to go down there. I have never regretted that decision. It was an interesting time in Arkansas, however. Of course, in 1964 the civil rights legislation was passed. There was a lot of civil rights activity

going on in Arkansas at the time. My wife got involved in a lot of that. There were some dramatic changes between 1964 and 1968--when we left. So it was an environment--in terms of civil rights--that we had never been exposed to before, and we learned a lot and [I] hope we accomplished something.

Chappelle: What studies did you work on with Dr. Oddie?

Fisher: It was early in the radioisotope era; there wasn't much in the way of isotopes available before World War II. Of course, radioiodine became available for the treatment of hyperthyroidism. We used it to ablate the thyroids in the infant monkeys we had, but hadn't had much other experience. Dr. Oddie had built a whole body counter, which was created by and shielded by battleship steel so that no radiation from outside could penetrate to the radiation detectors. We were looking at iodine kinetics. We would give a dose of radioiodine to the patient and follow them over a period of time to measure the disappearance and the accumulation of thyroid and the excretion in urine, and [we] were able to characterize pretty clearly iodine metabolism in adults. The radiation dose was minimal, and all those patients were consenting to what they were doing. We also decided then to look at thyroid hormone kinetics and disappearance and metabolism, so it was radioiodine-labeled thyroid hormone. Thyroxine has four iodine atoms on the molecule, so make one of them radioactive and you can follow it just as you would radioactive iodine. We would give a dose of labeled thyroid hormone, either thyroxine or triiodothyronine--the two active hormones--and could measure the disappearance rate over time, correcting for some vagaries that are associated with it. These were the first studies of thyroxine metabolism in human adults that we were aware of, and [we] were able to publish a paper about production rates for thyroxine. Also, we became interested in the interaction of iodine intake and thyroid hormone function, of course. The iodine is ingested, it's picked up by the thyroid gland, and if you are iodine deficient, it picks up more iodine. So we showed a nice correlation of average iodine intake and radioiodine uptake. Dr. Oddie then recruited information from his friends around the country about the radioiodine uptake and the average uptake in various areas of the country. He was also a pretty expert mathematician and had a mathematician who he worked with. So we published a paper on average iodine intake in the country--the first that was created to look at it in that way--and reported that the iodine intake was--in selected parts of the country--really quite high, presumably because they had put iodine in bread, of course, but that had the people concerned about iodine and thyroid metabolism looking around the country at whether we were getting too much non-radioiodine. We studied, then, thyroid function in infants, and that was quite a surprise. I had three technicians in the laboratory and a grant from the NIH and a career development award from the NIH; we spent the time in the laboratory developing improved methods to measures thyroxine a little more carefully. Dr. Lester Van Middlesworth in Memphis had done some studies in infants to show--and he had done some radioiodine uptake studies in the thyroid. So we created a radioiodine detection device that could detect

radioiodine in the thyroid gland of very small quantities, and [we] did a few radioiodine uptake studies with permission of the parents to confirm Dr. Van Middlesworth's work. And, yes, they had high radioiodine uptake, which we had no reason to suspect. They also had high levels of thyroxine, which we didn't suspect, and we tried to figure out why they were hyperthyroid. These were normal infants. We, by accident, looked at some of the infants that were in incubators, and they had lower levels of thyroid hormone than those that were outside the incubators. So we did a study to see if it was [the] cooling in the extrauterine environment that caused this hyperthyroidism, and it turned out they were quite different: if they were incubated their thyroid hormone levels were lower than if they were outside and cooler. So we thought maybe it was TSH being stimulated because of exposure to the extrauterine environment, but we couldn't measure TSH. But Dr. William Odell at the NIH had developed a method to measure TSH.

VI. UNIVERSITY OF CALIFORNIA LOS ANGELES SCHOOL OF MEDICINE (1968-1991)

Fisher: About that time I had a call from Dr. Joseph St. Geme at UCLA asking if I was interested in a pediatric endocrinology job at Harbor-UCLA Medical Center in Los Angeles. I visited and it was kind of like visiting Arkansas: it was a no-brainer. Dr. Odell had been recruited a year before by Dr. David Solomon in the Department of Medicine to [become] head [of] adult endocrinology. He was interested in what we were doing. Joseph St. Geme had just been recruited to head the Department of Pediatrics; it was a small department--it was an LA County public hospital at the time, and ULCA had a contract with Harbor to provide the medical faculty. UCLA was interested because it was an excellent place for junior medical students to do their medical training, and it was a desirable internship and residency hospital site for new physicians because they had a chance to be really involved in patient care. That was before supervising university physicians had to put notes in the chart and document everything that had been done. There were also several research buildings available that were old barracks from a World War II embarkation hospital, and there were forty buildings on the site of the Harbor Hospital. The county of Los Angeles had taken over the hospital after the war--paid one dollar for it--and built a seven-story hospital to go with the barracks, so we had a huge area of potential research space. Dr. David Solomon had been recruited from UCLA to head the department down there. Dr. Odell had brought Stan Korenman with him from the NIH.

Bill Odell, Stan Korenman, and Dr. Solomon--who was also an endocrinologist interested in the thyroid--and I, and Dr. St. Geme thought it would be a good idea to have a combined medicine/pediatric/endocrine unit. We discussed the advantages and disadvantages; obviously, it would be efficient: an internist could learn about children, and [pediatricians] could learn about adults, and that would provide more effective endocrine training. We could combine efforts for

funding from the NIH and research; we could more efficiently deal with the teaching and the in-patients and the out-patient services; and even, perhaps, provide more time for research than we would have if we were having to run the single departments exclusively. So that was done, and all the research was combined as well. It turned out to be pretty effective.

Chappelle: What was the ethos of the Harbor-UCLA at the time?

Fisher: That was another of the enticing reasons to move there; everybody was interested at that time in collaboration. It was a rather small faculty. At that time there were eight or ten faculty in pediatrics and probably twice that in medicine. Dave Solomon and Dr. St. Geme started recruiting and built rather larger departments. Much of this was with county funding. The South Bay area in Los Angeles--the population was increasing such that clinic space became tight, and that eventually had to be expanded. The patient care needs of the county were growing, and they were willing to grow the departments. That happened pretty quickly, actually. And as we combined the divisions and the department activities, we expanded our NIH funding and got training grants and started recruiting fellows to embellish the endocrine division. The other departments were doing the same thing, building their departments and building their research activities in the barracks. A Research and Education Institute was set up to run the barracks--it was fairly easy with those rather simple barracks to modify the space and create research workspace--and they managed the grants from the NIH. The Research and Education Institute became a pretty large operation. It's now the LA Biomedical Institute [Los Angeles Biomedical Research Institute] that is still functioning and running that area, and there are now three research buildings and some others planned--real research buildings, not barracks; there are a few barracks still there--but it is still evolving as we had tried to put it together.

Exploiting new immunoassay technology

Chappelle: What assays were being developed?

Fisher: Well, Bill Odell was interested in pituitary hormones; and Stan Korenman in estrogen and testosterone and the steroid hormones; and Dr. Solomon was interested in thyroid, as I was. And the plan, at least in my mind, was to exploit the immunoassay technology, which was new; Berson and Yalow had developed this research option, radioimmunoassay, in the late-1950s and early-1960s. Solomon Berson died and Rosalyn got the Nobel Prize for that. It was new technology. The NIH was very willing to fund it. We recruited fellows who were interested, because it was also a tremendous opportunity for them to learn a whole new technology. I thought it was useful to exploit the technology to characterize fetal and newborn thyroid function and endocrine systems' ontogenesis--Bill Odell was interested in some of that as well--and to recruit collaborators and fellows to characterize the various endocrine systems, and that

would increase the options for funding: if we had a program that looked at more than just thyroid function and looked at other areas of endocrine systems' development, we could expand the funding. So over three to four years, we evolved four teams in the pediatric section to look at thyroid--and vasopressin was another, and insulin and glucagon and carbohydrate metabolism was another, and then later looking at growth factors. The four teams were each comprised of five to eight people--and a lot of interaction, and a lot of interaction with medicine fellows as well. This worked, I guess, so well that over twenty-five years we published 350 papers, as well as review publications dealing with endocrine systems' ontogenesis. Bill Odell and the endocrine group in medicine were very productive as well, and we exchanged methodology and information and expertise. It was quite an exciting place. Actually, we presented the first Endocrine Society radioisotope workshop in the early-1960s, and Rosalyn Yalow came as one of the faculty. That was highly successful and that helped advertise our program. So we had plenty of fellowship applications.

Development of a newborn screening program for congenital hypothyroidism

Chappelle: Would you talk a little bit about newborn screening and how you got into that with all the knowledge you were gaining at the time about the thyroid system ontogenesis?

Fisher: Remember the hypothesis was that TSH was stimulating these newborns to be hyperthyroid, and Bill Odell had a TSH assay that he brought with him from the NIH. One of the first publications--he and I--was to look at TSH in the newborn. It turned out that they had a TSH *surge*--we called it--and the levels went up from about 10 in cord blood to 70 or 80 within an hour after birth. It stimulated the thyroid to release thyroid hormones, and over the next two to three days the thyroid hormone levels became very elevated--they were hyperthyroid. The TSH surge subsided over the subsequent week, and the hyperthyroid state subsided progressively over the first month. It was in part due to cooling--we didn't have to repeat those studies. Umbilical cord cutting also helped trigger this response, and there were obviously some chemical changes involved as well. But we went on to characterize the changes in T_4 levels--thyroxine--and triiodothyronine levels, and TSH levels; and we proceeded to develop a newborn research kind of plan where we could study infants in the hospital and study fetal sheep. Dr. Calvin Hobel in the Department of Obstetrics had been interested in the studies in fetal sheep as an obstetrician--they were interested in delivery and those kinds of things. Dr. George Emmanoulides was in the Department of Pediatrics, interested in heart function, and was working a little bit with the sheep. So we got NIH grant funding to build a really significant fetal sheep operation--research wise. The Research and Education Institute initially funded part of that, and then the NIH funded it. The obstetric people were using it; and we introduced studies, then, in fetal sheep. To get access to the newborn infants was difficult in a public hospital. Dr.

George Bray was there in the Department of Medicine and had recruited--he and Dave Solomon--a NIH clinical study center, but it wasn't appropriate to study infants. So Dr. St. Geme and Dr. Solomon and Dr. Bray and the endocrine group and the neonatal group got together again and decided that we could create a separate perinatal research center, if you will, that wasn't geographic, but would be wherever the infants were and wherever the pregnant women were. The NIH agreed to boost the budget a little bit and provide nurses and support people to study the infants and the mothers in the hospital, so we were able to do clinical studies in patients and able to do more complicated studies in the fetal sheep.

In the first three years that we were at Harbor, we developed information about what was really going on during the last weeks of life in the uterus in the sheep, and--in both human and sheep--what was going on in terms of thyroid function in the newborn period. We would characterize the neonatal hyperthyroid state, the TSH peak, the increase in T_4 and T_3. My background interest--since I had been exposed years before to the congenitally hypothyroid infants--was, maybe we could screen for them. Because if they were born on their own--and it was relatively rare, about one in three thousand to four thousand newborns--so nobody was used to looking for them or caring for them, and they didn't have much in the way of signs and symptoms. So they were hypothyroid and treatment was delayed--usually, three to six months--at which time they had lost about 3 to 5 points of IQ a month over that period. So it was an important cause of mental deficiency. Dr. Jean Dussault became interested in this, and he was going back to Quebec in 1971--he was an internist, by the way, not a pediatrician, and had come to Harbor to work with Dr. Solomon, an internist. He found out what we were doing in the newborn period. He was interested in T_3 metabolism, and so he joined our group--that happened several times so that the people who came as fellows got to choose from among several options in terms of research opportunities that they had, and he was the first to do that. The second fellow I had was Dr. Walter Skowsky, and he was an internist as well, and he was interested in water metabolism and vasopressin. Dr. Inder Chopra was a fellow working with Dr. Solomon, he [Chopra] was a fellow that emigrated from India, and he was interested in the thyroid. He was working on developing a thyroxine assay; and Dr. Skowsky, a vasopressin assay; and Dr. Dussault, a triiodothyronine assay, and we had the chemical thyroxine assay that we had used in Arkansas that we used in the interim before we got regular immunoassays running. So that defined those areas of perinatal research at the time. We submitted research grants for those, and every time we had a new opportunity, we would submit a grant to the NIH so that we could expand the activities. That worked out pretty well. So when Dr. Dussault decided to go back to Quebec, he decided--and we decided--since the T_4 assay was clearly going to develop and we had preliminary data--Dr. Chopra finally finalized it--to apply it to newborn screening. Quebec had a newborn screening program.

Chappelle: Following Jean Dussault's return to Quebec, what work were you doing?

Fisher: We had just finishing characterizing fetal and newborn thyroid function in some detail and decided that there were options for newborn screening. Quebec had established a newborn screening program for phenylketonuria--some few years before--and they were in a bit of trouble because that's one in fifteen thousand or so births, and so it wasn't very cost effective. So they agreed--because we thought at that time that the prevalence of congenital hypothyroidism is about one in six thousand--so that it would increase the efficiency of the screening program. So Dr. Dussault took the embryo thyroxine immunoassay back with him to Quebec**.** The screening for phenylketonuria was done using filter paper blood spots from the heel of the newborn infant, and he developed a method for measuring T_4 in the blood spots, published that in 1973, I think, and started to apply it to the screening program, and published the first newborn screening in 1975. By then they had screened forty-seven thousand infants, and they found one in 6,714 births, and so the screening programs were established. We had a Kroc Foundation meeting that Bob Kroc funded for us in California and invited some other thyroidologists to come and talk about newborn thyroid function and Jean's screening results. In Pittsburgh, there had been a study of thyroid screening measuring cord blood, but that was rather clumsy and it didn't work, and they didn't have a program, so that broke up. Jean's publication in 1975 stimulated a lot of interest, and there were newborn screening programs then established in the Northeast United States--in Boston--that screened Rhode Island, Connecticut, Maine, New Hampshire and, of course, Massachusetts. In the Northwest United States, they had a screening program in Portland that was screening Oregon, Alaska, Idaho, Montana and Nebraska. They introduced the newborn thyroid screening there. We set up an American Thyroid Association Screening Committee that I chaired and Dr. Dussault and Dr. Reed Larsen, Dr. Evelyn Man, Dr. Paul Walfish and Dr. Dorothy Hollingsworth [made up the committee of six]. The intent was to review what had gone on in these several programs in Boston, Portland, Quebec, and Pittsburgh. And [the committee] decided that newborn screenings seemed to be an effective way to deal with congenital hypothyroidism. The ATA gave its blessing, I guess you might say. There were other people interested as well, and there was a rather rapid expansion of newborn screening to Europe and Australia, New Zealand, Japan, and Israel. The committee put together a report on screening the first one million North American infants in these five laboratories and reported that it was one in about thirty-five hundred newborns. We all were participating and developing information about how to treat these infants, how soon they should be treated, how they should be studied when they were detected, how to get rid of false positive tests--an enormous series of questions that came up that everybody began to focus on. We set up an international screening committee--myself and Dr. Dussault and Dr. Gerard Burrow from the United States, Dr. François Delange from Belgium--a good friend--Dr. Gabriella Morreale de Escobar from Spain, Dr. Minoru Irie and Dr. Hiroshi Naruse from Japan and Dr. John Connelly from Australia. Over the next few years, we set up screening

meetings in Quebec in 1979, in Tokyo in 1982, and Brussels in 1988, to deal with all of these issues in newborn screening and try to come to some conclusions or proposals about how to deal with them. By that time newborn screening, of course, was salvaged--with the one in three thousand versus the one in fifteen thousand--and it's been supported and expanded in the United States now with government support. They are now screening, of course, for some thirty newborn defects that are benefited by early detection and treatment. It's been a highly successful program.

Later studies on thyroid metabolism

Chappelle: What later studies were you doing on fetal thyroid metabolism?

Fisher: Well, we started measuring thyroid hormone production rates in the fetus. We measured thyroxine production rate, and it was about 40 micrograms per kilogram per day. It blew us away because after birth the secretion rate is one-sixth or something of that [order], and we measured triiodothyronine production rate and it was minimal. Triiodothyronine levels were extremely low, and at best the production rate would be 2 micrograms per kilogram per day, so that all of the thyroxine in the fetus was being converted to something else, and not making active thyroid hormone. Dr. Oddie; Dr. Hobel; a new fellow, Dan Polk; Inder Chopra; and a new collaborator, Dr. Jimmy Wu at the University of California Irvine, began to focus on what those other things were, that we weren't measuring. It was a several-year project--work in the laboratory over the seventies and early-eighties. And there was work going on about thyroid metabolism, not necessarily in the fetus, but in general. It was being worked out that there were two methods of degradation. One, deiodination: thyroxine has four iodines on the molecule, and you take off that one critical one and then the thyroid hormone binds to its receptor and does its thing. If you remove any other of the iodines, it inactivates the molecule. It turned out there were three deiodinases--1, 2, and 3--that removed different iodines, and I'll get to that. The other degradation system was sulfation--putting a sulfate in a hydroxyl position at the end of the molecule, and it became inactive. We developed assays for sulfated thyroxine and assays for the reverse T_3, which was removing one iodine atom to inactive the molecule--and found that the levels were pretty high in the fetus. We did production rates, but it turned out that reverse T_3--the deiodinated one--was still only 5 micrograms per kilogram per day, so the deiodination pathway certainly wasn't the most prominent. So we developed an immunoassay for T_4-sulfate, and T_4-sulfate and reverse T_3-sulfate were half of the 40 micrograms per kilogram per day. We went on and developed assays for the rest of the metabolites in the system: the deiodination pathway is reverse T_3 and T_2 and T_1 and T_0--T for thyronine molecule. For the sulfate--thyroxine sulfate, T_4-sulfate, T_3-sulfate, reverse T_3-sulfate, T_2-sulfate, and T_1-sulfate --and as I said the sulfate compounds made up most of the production rate in the fetus. So the serum and the production rate studies showed that the combined sulfation and deiodination pathways amounted to 90 percent of the compounds that were

floating around in the fetus. It turns out that the deiodinase enzymes were doing this as well as sulfate. Type 1 deiodinase converts thyroxine to triiodothyronine in the liver and other tissues, and there was very little of that in the fetus. Type 2 deiodinase is present in brain and converts thyroxine to triiodothyronine, and it is present there. The type 3 is in placenta and other tissues, and it does the rest of the inactivation. So the fetus has lots of type 3 monodeiodinase that went down the deiodination pathway, and the type 2 in the brain was there to convert thyroxine to triiodothyronine because the fetal brain, if it doesn't have thyroid hormone, doesn't develop normally. And so it turns out that the system is set up to inactivate thyroxine, except in the brain, which requires thyroxine for normal development. The inactivation, presumably, is to promote growth in the fetus. Thyroxine is a catabolic hormone that causes increase in metabolic rate, and so it would probably impair growth, but that's just a hypothesis. The whole system in the fetus is vastly different than it is, actually, in the newborn period and the adult; that transition occurs in the last five to eight weeks of pregnancy and in the newborn period.

Carbohydrate metabolism

Chappelle: What lead you to study carbohydrate metabolism?

Fisher: Well, the carbohydrate studies came about because newborns develop hypoglycemia: they all do, and it's just a question of degree. As you cut off the glucose and the other nutrients from the mother when you cut the umbilical cord, they are then on their own--normally--and they have some liver glycogen carbohydrate stores, and epinephrine and glucagon will stimulate release of that. There is plenty of epinephrine release in the fetus--as we will find out. We developed immunoassays--Dr. Robert Fiser, an early fellow from Arkansas--to look at insulin; and Dr. Mark Sperling, whom I had recruited from Pittsburgh as a pediatric endocrinologist was interested in diabetes--and I wasn't so interested in diabetes--he developed the glucagon assay. And they [Drs. Fiser and Sperling] worked together to look at insulin and glucagon in the newborn infant, in the perinatal research unit and in the sheep laboratory. It turns out that insulin and glucagon are minimally secreted in the fetus--the mother provides glucose and the fetus doesn't have to worry about it. If there is hypoglycemia, the mother's system would respond. So that system has to then change in the newborn period, and it takes several days for the glucagon release system and the insulin-glucagon control system for regulating glucose to mature in the newborn period after suppression *in utero*. It turns out that the insulin and glucagon *ratio* is what is most important in regulating glucose levels. So that evolutionary system in the fetus--in terms of endocrine ontogenesis--is much different that the thyroid. It turns out that each of these systems has a function in the fetus that has to change when the baby is born, and this was intriguing from that regard.

Catecholamine metabolism

We were also studying catecholamine metabolism. It turned out it wasn't possible to develop immunoassays effectively for the catecholamines. There were radioenzymatic assays--that we are still using, that we are in the process of changing these days--but we set up those assays for epinephrine, norepinephrine, and dopamine and started looking at responses in the infants and the sheep. We looked at responses to maternal exercise and hypoxia in the fetus--obviously these things in the sheep--and acidosis and hypotension, and looked at the metabolic clearance of the hormones in the fetus. [We] developed a number of insights about this system, as well: norepinephrine secretion, which comes from the ganglia--the autonomic ganglia--and epinephrine, which comes from the autonomic center within the adrenal gland--secretes epinephrine--and epinephrine secretion is significantly less in the fetus than norepinephrine because it's almost a direct neuroresponse to a stimulus. So norepinephrine secretion exceeds epinephrine in the newborn by way of direct secretion from the autonomic ganglia; epinephrine secretion from the adrenal increases more slowly with age. Epinephrine infusion, interestingly--and Dr. Skowsky was involved--stimulates vasopressin secretion. We were looking at both because they were stress response systems. It turns out AVP--arginine vasopressin--from the pituitary acts as a corticosterone-releasing hormone to increase cortisone secretion, as well. So this fetal response system--catecholamines, vasopressin, and cortisol--all of these are activated in concert in the fetus, and that isn't true after birth. All that, presumably, is to potentiate the fetal stress response and provide optimal levels of stress response to get the fetus into the extrauterine environment through the labor process. So it's another pattern of the endocrine system's development--different from insulin, glucagon, and thyroid--that was intriguing.

Water metabolism in the fetus and the newborn

Chappelle: Would you give a little background about water metabolism in the fetus and the newborn?

Fisher: Yes. It is much different than it is after birth. In the fetus there are two large water compartments: one, the fetal compartment; and the other, the amniotic fluid. There is fetal lung fluid--there is no air in the lung, but there is water in the lung, and it flows out of the lung into the amniotic fluid. And there is a fetal kidney that is beginning to function, and so the infant can urinate into amniotic fluid, and there is transfer back from the fetus to the mother--it can go either way, depending upon the circumstances. So the fetus is part of the mother. It turns out in the sheep fetus that the amniotic fluid volume is about 650 ml, the fetal lung produces about 250 ml of fluid a day, and the fetal urine is about 600 ml per day. The fetus swallows amniotic fluid, and it swallows about 500 ml a day. So in that exchange, the difference is about 350 ml a day that isn't accounted for. Probably that is the bulk flow from the amniotic fluid into the

mother. So the system is pretty well defined. Of course, we wondered how the hormones that regulate water and electrolyte [metabolism] after birth are involved in this system. So we developed AVP and AVT--arginine vasopressin and arginine vasotocin assays--those two peptide hormones differ just in one amino acid. We did a variety of studies to perturb the system to see how they responded, and we measured AVP and AVT, and urine and amniotic fluid osmolality, and other things. It turns out--there is no AVT, by the way, in mammalian species--in humans--after birth, but it is present in about the same amounts as AVP in the fetus. Lower species have AVT as their vasopressin hormone. But the fetus continues to secrete it, and I guess it's to increase the efficiency or something. It is ontogeny recapitulating phylogeny, or something of the sort. In any case, the levels are higher than we expected. And it turns out that AVP and AVT decrease the fetal to maternal flow across the placenta, [and] that decreases the lung flow and amniotic fluid, and increases urine osmolality--as it does after birth--and decreases urine flow into amniotic fluid. We also looked at atrial natriuretic factor--it is a hormone that's involved in fluid metabolism [and is] secreted by the heart--and it is present in the fetus, and it decreases the lung flow into amniotic fluid. And we looked at prolactin, which is minimally involved, and it decreases amniotic fluid flow into the maternal compartment. [And we looked at] plasma renin activity, which controls sodium electrolyte release, decreases urine flow in response to volume depletion. So it is an entirely different system than it is after birth. The defense of blood volume and cardiovascular function, lung fluid, amniotic fluid, and renal function are controlled in the fetus by the interaction of arginine vasopressin, arginine vasotocin, atrial natriuretic factor, prolactin, and--to some degree--cortisol in the third gestation fetal sheep. So the system kind of resembles the catechol stress pattern, that is, collaborative endocrine systems to meet fetal needs and to meet the problems of neonatal adaptation. That, too, was pretty interesting.

Growth factors

Chappelle: How did your interest in growth factors come about?

Fisher: Well, endocrinology is growth and development in large part because of the growth hormone from the pituitary. Growth hormone has been shown by Dr. William Daughaday, years ago, to have its effect by stimulating insulin-like growth factor from the liver and other tissues that stimulate the growth and development. Interestingly, IGF-1 and IGF-2--the insulin-like growth factors--are present in the fetus, but they are not regulated by growth hormone. It turns out it is mostly nutrition that regulates it. If the fetus is adequately fed, it will grow faster by stimulating IGFs. And if there isn't so much food available, it doesn't grow so fast. The other important ingredient is that thyroid hormone, which with growth hormone is responsible for growth after birth, has little effect on growth in the fetus. It becomes important at birth, and it's all inactivated anyway--as we have discussed. So we were postulating that other growth factors may be involved in the thyroid hormone effect or other hormone effects, and

there is information about a variety of growth factors that are active in the fetus: epidermal growth factor is one and nerve growth factor is another. So we decided to look at these two systems. We developed assays for EGF and NGF, but nobody had purified sheep EGF or NGF, so we couldn't produce an assay. We had mouse hormones, and so we produced immunoassays for the mouse and used the mouse model, that had the added advantage, and it is easier to study tissues, and it's easier to do treatments, and it doesn't cost so much, and you can see the effects sooner. So we looked at EGF in skin, brain, lung, and submandibular salivary gland, and there were high levels--and lower levels in other tissues--but it was widely distributed. Thyroxine treatment increased EGF in these tissues, and thyroxine treatment increased NGF in the brain and the submandibular gland and the kidney. That doesn't imply that it is influencing in the human fetus or the sheep fetus. When we look at the mouse, we are looking at essentially the third trimester of fetus in the extrauterine period, so that the fetal mouse is born as an embryo, still, almost. It has to be warmed and fed until it matures significantly, and it grows. These growth factors and thyroxine--in that model and in that period--seem to be influencing growth by way of EGF, as well as probably other growth factors. So there is support for EGF and NGF in meeting thyroxine effects, and that is probably in the late part of pregnancy in the early neonatal period and later. We did studies of epidermal growth factor mRNA--the gene products--in SMG [submandibular gland] and kidney. Dr. Luciano Barajas in the pathology department was very interested; he's a nephrologist, kind of pathologist--does the kidney pathology. He did studies of *in vitro* hybridization--looking at message and looking at EGF and [used] electron microscopy to see where it was in the kidney and the submandibular gland. To make the story short: EGF and the EGF pro-hormone are present in saliva and urine; EGF responds to thyroxine treatment in both. Testosterone is also important in the salivary gland, so it's a dual control. But all of this work suggested that the submandibular gland and the kidney EGF are there to provide EGF to maintain the surface integrity of your gut on the one hand, and your urinary tract on the other. You know from your own life that if you get a cut inside your mouth or damage it with biting too hard on something, that it rapidly heals. And that's from salivary gland EGF. The EGF that the kidney manufactures in the kidney tubules does the same maintenance for the urinary tract. Now these are both large areas of research, and they are interesting because there are a number of hormones involved in these various times and various tissues regulating these and other growth factors. That, *too*, was interesting.

VII. NICHOLS INSTITUTE/QUEST DIAGNOSTICS (1991-2007)

Chappelle: I would like to ask you a little bit about the Nichols Institute/Quest Diagnostics.

Fisher: Yes.

Chappelle: You became president of the reference laboratories of Nichols Institute/Quest, in 1991. Would tell me a little bit about the institute and about Dr. Albert Nichols?

Fisher: Well, Dr. Nichols is an endocrinologist. He finished his training in Boston with Dr. James Melby, who I knew from Arkansas, and he came back to California. His father and the family were in the heavy construction business--actually his father built the oil islands off of Los Angeles. Al decided to go into medicine, into endocrinology, and he came back to work for Kaiser Hospital, nearby us. He would come over to Wednesday endocrine clinic, and we were by then using our assays to manage the patients there--our hormone assays--and he was very interested in that, and he wanted to use those for his patients at Kaiser. So he arranged to set up a lab, and we arranged to set up the assays for him--the ones applicable to the human: thyroxine and cortisol and estrogen and testosterone, *et cetera*, seven or eight of them--and he began to use them and was very excited about it. The Kaiser physicians have to make a decision about whether they are going to stay on with Kaiser after two years--they have a two-year trial period. And the pathologists there--the laboratory--weren't so happy with his laboratory. I guess he decided he would set up his own laboratory. He thought that these kinds of assays weren't available elsewhere, and that if he created a laboratory--a commercial laboratory--he could develop them for clinical use. His goal was to make the systems available and the research lab available to the clinician for patient care. So he started the Nichols Institute for endocrinology in a garage nearby. Dr. Odell and I helped him out, and Dr. Richard Horton from USC, who did steroid assays--and the growth hormone and pituitary assays, Dr. Odell had helped out with. And the vasopressin and thyroxine--the ones we were dealing with--we helped set up in the laboratory. We were UCLA faculty, and we couldn't invest in a laboratory--and wouldn't have anyway. He decided he would make us what he called academic associates, I guess, consultants. He supported some technicians in our laboratory to help out with all of this. So we got involved as consultants to the laboratory, and it grew rapidly. He was a good businessman, and his father--in the construction business--helped him build a new laboratory and modify one in Long Beach. He outgrew that and then developed a bigger one in San Juan Capistrano. It expanded, and he started then to introduce other tests--with the same arrangement--with academic associates transferring or helping transfer exciting new assays to the laboratory, in genetics and in immunology. Interestingly, the immunoassays were important in immunology. In endocrinology you used an antibody to measure a hormone, and in immunology you use a product to measure an antibody. It may be a hormone--if it's an antibody in the patient to a hormone--but it can be an antibody to anything else. It is the reverse of a routine immunoassay. So the whole radioimmunoassay business rapidly expanded into immunology, and that part of the laboratory grew rapidly. The volumes got so large that he then had to start creating regional laboratories that would set up the commoner assays, and sent the rest to San Juan Capistrano. That worked quite well. It was in early 1991 that Al contacted me; his reference laboratory executive had decided to go

back to Colorado and go into the horse business, and he asked me if I wanted to come and run the reference laboratory. I had just gotten a new NIH grant and a special NIH Merit Award with it, and I said I didn't think I wanted to leave what I was doing. He came back to me a couple of times later, and I went down and visited with him. He had a whole new laboratory in San Juan Capistrano just about completed; it was an impressive edifice, I might say. And the intrigue of expanding into other areas of endocrinology, and understanding same--immunology as well, [and] oncology--was interesting. So I decided I would try it. So in the spring of 1991, I became president of the reference laboratory, but that lasted only two years. As president of the reference laboratory--it is largely HR issues, and that wasn't what I wanted to do. So Al put one of the other medical directors in charge of the laboratory and made me the head of the academic associates and the R&D program. So that's what I started to do, and [I] began to recruit medical directors to head those laboratories, and that was an exciting new option.

Chappelle: And that's what you mean by referring to it as a virtual medical school?

Fisher: Yes. At one time we had forty academic associates from the various academic institutes [or] enterprises in the country, and by then we had created the division of endocrinology, the division of immunology, the division of genetics, the division of oncology, the division of infectious disease, the division of toxicology, a comprehensive reference laboratory--about 60 percent of the assays in the whole operation were radioimmunoassay, so the radioimmunoassay absolutely transformed some of these laboratory operations. Some of the academicians that were involved in various areas of medicine were interested in what was going on in terms of providing assays, commercially, to physicians, and there was a lot of interaction. It seemed like a medical school. I had forty people out there that I communicated with, and we communicated with, and the medical directors and the research staff that were doing the R&D, and then a separate medical director for the laboratory that took care of the operations. We set it up so that the medical directors got half their salary from operations for supervising the day-to-day conduct of testing, and half their salaries from R&D, running the R&D staff to make sure they always had new assays on line with the academic associates behind them. It made for kind of a virtual medical school, it seemed.

VIII. THE ENDOCRINE SOCIETY

Chappelle: I would like to ask you a little bit about the Endocrine Society.

Fisher: Yes.

Chappelle: What has the Endocrine Society meant to you in terms of community?

Fisher: Well, it has been my major contact with the whole of endocrinology. I was involved in the program for the Endocrine Society for many years. I was editor-in-chief of the *Journal of Clinical Endocrinology and Metabolism* for five years. I became president of the Endocrine Society in 1983. I have been involved *academically*, now, for forty-plus years. Initially, when I was president, Nettie Karpin was running the Endocrine Society, and she used to work at the NIH, I think, for and with Griff Ross. The Endocrine Society, initially, was kind of an endocrine club. The need for its survival and growth has become obvious in the fact that it has become the most influential and the largest endocrine enterprise in terms of teaching and providing information about endocrinology to all sorts of clients and audiences, myself included. I have also belonged to the Thyroid Association and the Pediatric Endocrine Society, but that's just thyroid, and that's just pediatrics. There are the other Societies that have split off, most recently the American Association for Clinical Endocrinology--that was an issue when I was president and that was created in 1985 or 1986, something in that era--to link up with and provide more direct support of practicing endocrinologists in terms of business acumen and support for political and other needs that the Endocrine Society hadn't been providing before. It was a more elitist society interested in just research. You know, initially, the Endocrine Society--way back--was pretty much, as I say, a club kind of environment for academic endocrinologists, and now of course it is vastly more than that as the need became clear. I have enjoyed the expansion and been involved with all of it. Over time, I find it the most effective source of information and constant stimulation that is available to me. The thyroid and pediatric I attend, but it's not the same; it is not the comprehensive coverage of endocrinology that is otherwise provided by the Endocrine Society.

Chappelle: Thank you.

Fisher: You are very welcome.

[End of Interview]

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Interview History—Delbert A. Fisher, MD

Dr. Fisher was interviewed by Michael Chappelle on June 20, 2010, during the Endocrine Society's Annual Meeting held at the San Diego Convention Center. The interview lasted one hour and forty-three minutes. The transcript was audit-edited by Mr. Chappelle and reviewed by Dr. Fisher prior to its accession by the Oral History of Endocrinology Collection. The videotape and transcript are in the public domain, by agreement with the oral author. *The original recording, consisting of three (3) 45-minute mini DV cam tapes, is in the Library holdings and is available under the regulations governing the use of permanent noncurrent records*. Records relating to the interview are located in the offices of the Clark Sawin Library's Oral History of Endocrinology Project.