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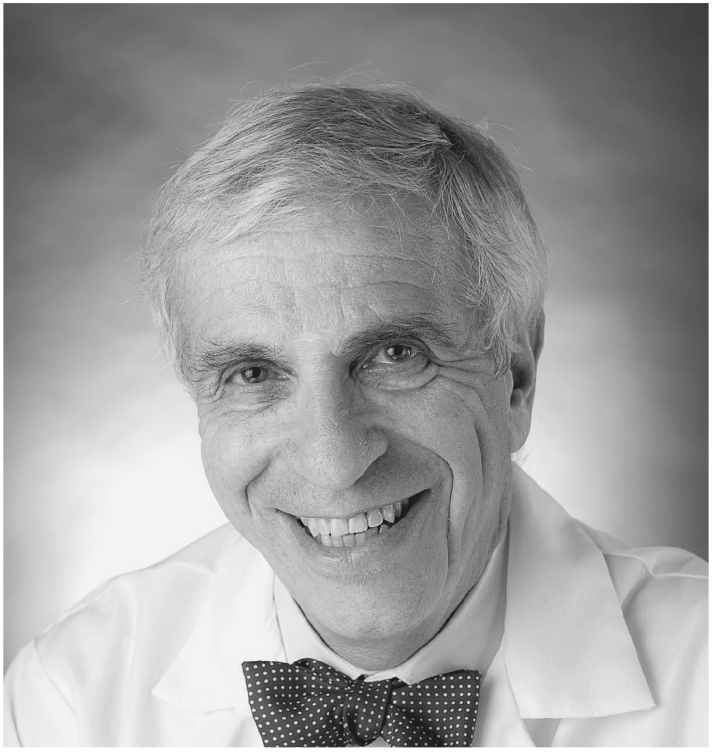

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Special Lecture, Society for Neuroscience Annual Meeting (2001)
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Society for Neuroscience, Treasurer (2006) and President (2010)
Louis P. Rowland Teaching Award, Columbia University Department of Neurology (2006)
Fellow of the American Academy of Arts and Sciences (2006)
Fellow of the American Association for the Advancement of Science (2008)
David Robinson Lecturer, Department of Biomedical Engineering, Johns Hopkins (2010)
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Michael E. (Mickey) Goldberg is a pioneer in the study of the visual and oculomotor systems in the awake, behaving monkey. As a postdoctoral fellow with Bob Wurtz, he discovered attentional enhancement of visual neurons and presaccadic neurons in the monkey superior colliculus. He went on in his own lab to make salient contributions to our understanding of the visual and oculomotor system, attention, and spatial perception He is currently studying how oculomotor proprioception is necessary for spatial memory, the role of the cholinergic system in facilitating activity in the parietal cortex, and the role of the cerebellum in visuomotor learning.

Michael E. Goldberg

Early Days

I was born in a dark time. My parents were the children of immigrants who came to America to escape the perils of Eastern Europe for Jews. My mother's father was an assimilated Rumanian Jew, trained as an architect. The family legend is that he designed a church in Bucharest. When people discovered that the church had been designed by a Jew, they burned it down and threatened his life. He, his wife, and their three children left Bucharest in the middle of night and escaped to New York. He never practiced architecture again but got work as a draughtsman. My mother was his youngest child, born in New York.

My father's parents were religious Jews, living in small shtetls in the Russian Pale of Settlement for Jews, now mostly in the Ukraine. My grandmother's brother had emigrated to America, and when he could afford it, he sent for his sister. At the age of 13, she traveled to London by herself, spent some months with the family of friends who had left the shtetl earlier, and went to New York. She was my only grandparent whom I ever met. I asked her what she remembered about her life in Russia—she said that she remembered the mud, the frequent religious processions of her Polish neighbors, and the worries that the procession might turn into a pogrom. She met my grandfather in New York. My grandfather was a peddler. My father fondly remembered his horse.

My grandfather died early. Somehow my grandmother was able to open a small delicatessen in a Polish neighborhood in the Bronx, and the family survived. My father and his twin brother were excellent students and were admitted to Stuyvesant (then as now one of the best and most selective high schools in New York). From the age of 13 onward, he worked evenings selling souvenirs and trinkets outside Broadway theaters. Simultaneously, he finished high school and then went to the City College of New York (CCNY), where he studied chemistry. CCNY then had no tuition payments. After graduating from college, he earned a master's degree from Columbia in chemistry. His Columbia diploma, signed by Nicholas Murray Butler, is the only diploma on my office wall. He was advised that academic chemistry was not a good place for Jews, so he applied to medical and dental schools and was accepted at New York University (NYU) dental school. By this time, he was working inside the theaters, ultimately becoming head of concessions for the Schubert theaters. One night a producer asked him if he would rather go on the road to be the stage manager of a production of *Oh, Kaye!* (a Gershwin musical).

He thought long and hard about it, but he was already halfway through dental school and had met my mother. He often wondered what his life would have been like had he entered show business rather than tooth business. After he graduated from dental school, he worked for another dentist for a year or so and then started his own practice in Inwood, the neighborhood at the top of Manhattan. Now a Dominican neighborhood, then it was an Irish-Jewish neighborhood, with a Kosher butcher and deli, Irish bars, and two Yiddish newspapers in the candy store. The dental office was a large apartment at the corner of Vermilyea Avenue and 207th Street, near the top of the A train. His waiting room and office were the livingroom and dining room of the apartment, and the family lived in the back five rooms. His lab was the walk-in pantry in the kitchen. My parents asked my grandmother to close her deli and invited her and my father's twin sisters to move in with us. My father often said that the proudest day in his life was when he realized he could support my grandmother, and she no longer had to work in her deli.

I was born in August 1941. Germany had swept through Western Europe, threatening to overwhelm Britain. After Pearl Harbor, my father volunteered to join the army as a dentist. He was commissioned a second lieutenant and spent the war in various places in the south, practicing dentistry on American GIs and German prisoners of war. He later told me that he felt, as a Jew, that he had no choice other than volunteering. My earliest memory was taking the train to South Carolina to visit my father. My next earliest memory was his marching in the memorial parade for President Roosevelt.

I enrolled in kindergarten in PS 98, on 211th Street. I thought I was going to learn to read. Instead, they gave me crayons and told me to draw. I moped. The kindergarten teacher thought I was a bit dull. Kindergarten was voluntary in those days, so after a few months my mother took me out of the class. First grade was much better—I learned to read. In 1947, my parents moved to Eastchester, in Westchester County, a bit north of the Bronx. My mother took me to Waverley School, the local elementary school, to enroll me. We met Mr. Merchant, the principal. I moped. Mr. Merchant said that they had an accelerated first grade, but he would put me into a regular first grade for the few weeks remaining. Two weeks later, he asked my parents if they would allow him to skip me to the third grade for the next year. She thought it would be a bad idea, given that I was the smallest kid in the class and socially quiet. I think she made a brilliant decision. She also turned down a subsequent offer for me to skip the third grade.

From the time that I knew what science was, I wanted to become a physician and a scientist. My father encouraged me to learn about chemistry. I remember a *Child's Book of Atoms and Molecules* as an early gift from him. In those days, you could get a kid's chemistry set that had some oomph, and I loved to play with it with my father. I was entranced by the wonders of phenolphthalein changing color and amused that the chemical was also the active ingredient in Ex-Lax (no longer, because of its mild carcinogenicity).

We made borax beads with an alcohol lamp. I loved looking at and naming stars and planets, learning trees and birds. Science wasn't everything. My parents introduced me to classical music, history, novels, poetry, and the theater. My Aunt May took me to the Metropolitan Opera. I took piano lessons.

I was a star in high school—best student, editor of the newspaper, president of the dramatic club, all-county chorus in the glee club. I finished first in the state on the New York State Regents scholarship exam. I was an enthusiastic Boy Scout: hiking and camping (foreign to my family), winning the county Scout nature contest, and tying the bowline on our prize-winning knot-tying relay team. I accumulated enough merit badges to become an Eagle Scout. In that scoundrel time, darkened by the shadow of Joseph McCarthy, every potential Eagle Scout had to appear before a special board of review. They asked me if I were drafted into the army could there be any order I would not obey. I said, of course, that was what the Nuremberg trials were about. I reserved the right to evaluate the morality of my orders. The board of review decided to make a home visit rather than passing me at once. I suspect they were looking for pictures of Lenin and Karl Marx, and copies of *Das Kapital*. My father calmed them down, as did the picture on the wall of Captain Goldberg in his army uniform. I can still tie knots and identify birds.

I got a summer job at the local drug company, Burroughs-Wellcome. Sir Henry Wellcome divorced his wife who was 27 years younger than he was because of her affair with the novelist W. Somerset Maugham. Wellcome's daughter Syrie was probably Maugham's child, and he disinherited her. He left his fortune to the Wellcome Trust and specified that the profits from his company be used exclusively for the research in the company and at other institutions. I was assigned to a lab that was working on purine and pyrimidine antimetabolites. The head of the lab, Dr. George Hitchings, was a remote, rather austere man who had gotten the idea that compounds that interfered with DNA synthesis and function might slow the growth of cancers. The person who organized the day-to-day work in the lab was Gertrude Elion. She didn't have a PhD. After she graduated from Hunter College (the free woman's college in New York City), she couldn't afford to go to graduate school and worked at various chemical and nonchemical jobs. She started working as a technician for Dr. Hitchings and ultimately became his scientific partner. Trudy was one of the warmest, most welcoming people I ever met as well as a great scientist and an especially talented synthetic chemist. They first developed 6-mercaptopurine and thioguanine, which worked against certain forms of leukemia. Those were also the early years of allogeneic kidney transplants, and they realized that their antileukemia drugs might work as immunosuppressors. Their drug azathioprine, a conjugated form of thioguanine, was the first successful nonsteroid immunosuppressive for kidney transplants. It is still used today as an immunosuppressive for various autoimmune diseases. AZT, a drug that had not worked as an antitumor drug or an immunosuppressive was the first successful drug

for AIDS. Pyrimethamine, an antifolate drug, is used to treat toxoplasmosis. Trimethoprim is an antibacterial drug. Allopurinol slows the degradation of purines to uric acid and prevents gouty attacks. Trudy and Dr. Hitchings shared the Nobel Prize in 1988.

My job was to work on the tumor screen. On Monday, we would implant chunks of a mouse sarcoma into the peritoneum of C57 black mice. For the next four days, we would inject a particular drug intraperitoneally into each mouse. On Friday, we would kill the mice, take out the tumor, weigh it, and compare it to growth in mice treated with 6-mercaptopurine, and others treated with normal saline. The work was boring, but the ideas floating around the lab were exciting. The lab played bridge at lunch. One of my jobs was to substitute for people on vacation. Trudy was a great bridge player. She also loved opera and had front row center seats in the balcony tier at the Met. We talked as much opera as science.

College and Molecular Biology

When I arrived at Harvard College, I discovered that everyone had been a star in high school, and I was way behind. There were two other Goldbergs in my class—Fred (recently elected to the National Academy of Sciences for discovering proteosomes) and George. I started by taking math and science. I took the calculus class for math majors and realized that I was never going to be a mathematician. My ineptitude in quantitative analysis made me think twice about doing science for real, so I decided after my first year to continue premed but concentrate in English. Like most premeds, I took organic chemistry as a sophomore. There was a Cliffie (a student at Radcliffe, the women's college of Harvard University) in my organic chemistry lab section whistling the Franck D Major symphony. Her name was Debbie Baron. We said hello frequently but never dated.

I wrote a tutorial paper on *The Miller's Tale* from Chaucer's *Canterbury Tales*, which my tutor loved and that I thought was trivial. I realized that English scholars comment on what other people had created, but scientists create. I switched my concentration to biochemical sciences to try again. This was the beginning of the great era of molecular biology. Watson and Crick had deciphered the structure of DNA, and Jacob and Monod were unraveling the mechanisms that controlled protein synthesis. I learned about messenger RNA, transfer RNA, ribosomes, and Nirenberg's breaking the genetic code. The most exciting course I took was William Sistrom's course on growth and biosynthesis in microorganisms. I took quantum mechanics, statistical mechanics, Shakespeare, metaphysical poetry, and Middle English poetry. Harvard College had no neuroscience of which I was aware. Psychology was B. F. Skinner's behavior, with the brain as a black box. I spent some time working in my tutor William S. Beck's lab, but I didn't accomplish much. I didn't write a thesis. I did a lot of theater

tech, mostly lighting design, and a lot of music. I still remember the thrill of singing the Verdi *Te Deum* with the Harvard Glee Club and the Boston Symphony under Carlo Maria Giulini.

I applied to medical school and graduate school. Dr. Perry Culver, dean of admissions at Harvard Medical School said that I wouldn't know for the first two years whether medical school were the right choice, but I would know much sooner in graduate school. He told me to defer medical school and go to graduate school. I took his advice and went to the new graduate program at the Rockefeller Institute for Medical Research, now Rockefeller University, where I wanted to work on mammalian molecular biology. I thought it had to be as exciting as *E. coli*, but no one had found that excitement.

At the Rockefeller Institute, on the Upper East Side on Manhattan, I worked in the Allfrey-Mirsky lab on calf liver nucleus histones. I made two important discoveries: I pipetted with a Poisson distribution (my mean was equal to my variance), and I could kill cell cultures from an adjacent room. Perry Culver was right. I decided to go to medical school because doctors make critical decisions on inadequate data and that seemed like fun. I would never go near a lab again. I reapplied and was accepted again to Harvard Medical School. That year, I spent a lot of time standing at the Metropolitan Opera and practicing the piano. I could actually play Beethoven's Opus 78. I read all of *Strangers and Brothers*, C. P. Snow's multivolume series of novels about British society and British science in the 1930s and 1940s.

Medical School and Neuroscience

I arrived at Harvard Medical School along with the other two Goldbergs, and Debbie (and 9 other women in a class of 100). With the exception of gross anatomy, which I actually enjoyed, most of the first year at medical school was a rerun of college, until the last six weeks, which was all neuroscience, all the time. Steven Kuffler had created the world's first neuroscience department, and he and its young members—Torsten Wiesel, Dave Hubel, Dave Potter, Ed Furshpan, and Ed Kravitz—dedicated their time during those six weeks to teaching. It was a revelation, an awakening like the great chorus *Et Expecto Resurrectionem* from Bach's B minor Mass, with the wonderful stretto fugue of ascending fourths.[1] I asked Dave Hubel to teach me more neuroscience, and he organized a tutorial during the next year at which we read a bunch of papers about systems neuroscience, not only by Hubel and Wiesel but also by Evarts, Mountcastle, Amassian, Kuffler, and Barlow.

The second year of medical school was dreary, with a few exceptions. I had moved off campus, sharing an apartment in Cambridge's Central Square with two economists and another medical student. One day, I asked Debbie to come

[1] See https://www.youtube.com/watch?v=cU2i3gx8880, starting around 22 seconds from the beginning.

over to my apartment for brunch. A few weeks later, we had a date (the Cornell game and dinner), and some time that day, having known each other for five years, we fell in love. It was as if we had we swallowed Brangaene's love potion from *Tristan und Isolde*. She moved in the next week, we got married that summer, and 52 years later, we are still living happily ever after. Another exception was the traditional, raunchy Harvard Medical School Second-Year Show. I organized the tech and lighting and wrote the lyrics ("Nothing remains but the tomb. No ray of hope can come through to us. We cannot return to the womb. Now that we know it is the uterus"). The only courses I liked were neuropathology and physical diagnosis, in which we actually got to examine real patients for the first time. I felt the thrill of clinical medicine.

Entranced by the tutorial in systems neuroscience (columns, hypercomplex cells, and the dream that neurophysiology might render perception understandable), and despite my better judgment, I asked Dave Hubel if I could work in his and Torsten's lab the following summer. He asked me what I wanted to do. I said that there was all this feature specificity in the visual cortex, but no one had reported anything similar in the somatosensory cortex. He told me to look for it. Mark Hallett, another medical student (ultimately to head the clinical neurology branch at the National Institute of Neurological Disorders and Stroke in Bethesda) and I built an electrode advancer, hooked up an amplifier and an oscilloscope, and learned to drill away the skull and peel away the layers of dura until there was only a transparent membrane left. We found lots of touch-sensitive neurons and ultimately found one directionally selective motion sensitive neuron. I was hooked.

In 1967, there was a war in Vietnam, and every physician with a Y chromosome owed the government two years, unless you were 6'6" or taller. There was no exception for those of us on the other side of the Gaussian distribution, but a lucky few could join the Public Health Service and work at the National Institutes of Health (NIH) or the U.S. Food and Drug Administration instead of going to Vietnam. I asked Dave to write Ed Evarts at the NIH to see whether he had space in his lab for still another Harvard Medical student (Tom Thach, Mahlon Delong, and Harris Funkenstein were already there). Ed didn't have space, but passed the letter on to Bob Wurtz, who had just joined the Laboratory of Neurobiology at the National Institute of Mental Health (NIMH) and was looking for his first postdoc. Bob interviewed me and told me about his still unpublished work on visual cortex in the awake monkey. He had trained monkeys to fixate, so the position of a stimulus on the screen corresponded to its retinal position. He could then work out the receptive field properties of neurons in awake monkeys, which for these evanescent few seconds were like anesthetized monkeys. He also was able to study the effects of eye movements on cortical neurons. I was entranced.

I spent the next year as a medical intern in the old Peter Bent Brigham Hospital in Boston. A few weeks into my internship, the NIH offered me a position as a staff associate with Bob, starting July 1969. A few days after

that, I got an invitation from the Army to go to Fort Sam Houston in Texas, and thence to Vietnam, which I delightedly declined. Those were the days of the iron men (and a few iron women). We were on every other night for almost the entire year. Monday, Wednesday, the weekend, Tuesday, Thursday, and Friday. I loved my internship, the clinical challenges, and the human interactions, but it was exhausting—Debbie took care of a choreographer at the Cambridge Hospital who gave us tickets for a Friday night performance of his show. Every other Friday, was the only day that we went to work in the morning and came home the same day, like ordinary humans. We slept through the intermission. July 1, 1969, Debbie and I moved to Washington, DC: I to work at the NIH, and Debbie to do a medical residency at Georgetown.

NIH and the Superior Colliculus

When I arrived at the NIH, I didn't know much. I had never even heard the word "saccade," the rapid eye movement that drives the center of gaze from point to point in the visual world. Bob had done his PhD in psychology at Michigan, studying self-stimulation under James Olds. When he offered me the job, I don't think he realized my level of ignorance. He introduced me to concepts that I had never heard of, like visual attention, saccadic suppression, corollary discharge, and operant conditioning. I read Teuber on corollary discharge (Teuber 1960) and William James on attention, as well as a lot of neurophysiology. Bob taught me the mysteries of awake monkey neurophysiology.

This was the Paleolithic era of neurophysiology. Ed Evarts had developed the equipment for recording the activity of single neurons in awake, behaviorally trained monkeys. We controlled the monkey's behavior by hooking together individual logic modules (or-gates, and-gates, one-shots, flip-flops, digital inputs, and output relays) that would sense the monkey's pressing a bar, open a shutter to present a visual stimulus or turn on an LED fixation point, and reward the monkey with a drop of water. The logic modules, DigiBits, were mounted in rows on a rack and connected in the back by a welter of wires. The tool that connected and disconnected the wires was the digi-bitter, and we referred to the frequent times of trouble as Bitter Digi. We measured eye movements by using electroencephalography electrodes pasted to the monkey's skin to give us a rather noisy direct current (DC) electrooculogram (EOG). We used glass-coated platinum iridium electrodes made by our technician Mary Fran Roark to pierce the dura and then study one cell at a time. We had a wonderful Rube Goldberg (no relation) device to build data analysis raster plots online. We used a Tektronix storage oscilloscope, which didn't erase the screen until you told it to. The electrode signal was hooked up to a voltage-crossing detector, which emitted a pulse when the signal went above a threshold. Jim Bryan, the head of the NIMH technical development section, had built a digital delay line for Bob. Pulses from the voltage-crossing detector went into the delay line and emerged 300 ms

later. We used a raster circuit to feed a DC level into the y input of the oscilloscope, set the timebase for two seconds, and triggered the x sweep of the oscilloscope on an event, like the pulse that opened the shutter. The pulses from the delay line went into the z input, which intensified the beam, so there would be a dot on the screen when a pulse occurred, with the screen otherwise dark. The delay line allowed us to record activity 300 ms before the triggering event, because the pulses that the oscilloscope saw at time τ had actually occurred at time $\tau - 300$. After the trial, the raster circuit would step the DC level down to the next line. When the screen was filled up, we would take a Polaroid picture of the screen, and paste it into the notebook, and then erase the screen. If we could hold the cell long enough, we could pass the EOG trace through a voltage-crossing detector and see the relationship of the activity to the eye movement. We couldn't perform both analyses at once, so Bob convinced the NIH to buy him an 11-track FM Hewlett-Packard tape recorder to record the experiments, and then we could play the tape again, and reanalyze the data on the missing trigger.

It was also a time when one could hope to keep up with all of neuroscience. We had a journal club that included Bob and me, Mahlon Delong and Noriichi Mano from Ed Evarts's lab, Mark Hallett who was working on squid axons with Ichiji Tasaki, and David Carpenter (now professor of environmental health sciences at SUNY Albany) who was working on aplysia. We read Kandel, Mountcastle, and Grillner. I miss the broad extent of that journal club in our much more focused world.

This was a time in which you could close your eyes, throw an electrode into the brain of an awake, behaving monkey, and discover something. Bob suggested that we work on the superior colliculus. The superior colliculus is a multilayered structure, with the superficial layers receiving a strong visual input from the retina and V1. We began by studying the visual properties of neurons in the two superficial layers. Peter Sterling and Barbara Wickelgren (now Gordon-Lickey; Sterling and Wickelgren 1969) had recorded from the superior colliculus in the anesthetized cat. They found that almost all of the cells were motion sensitive and selective for the direction of movement. Very few cells in the superficial layers of the awake monkey were motion selective. I named these cells "pandirectional," to emphasize that they were not motion sensitive or selective, clumsily combining a Greek (pan) and a Latin (directional) word. They should have been called "omnidirectional." Receptive field size increased as the electrode got deeper into the colliculus, but their centers remained in register (Goldberg and Wurtz 1972). One day, we drove the electrode too deep and heard neural hash going "whoosh whoosh" even when the monkey was in total darkness and making spontaneous eye movements. We realized that these whooshes preceded saccadic eye movements (Wurtz and Goldberg 1971; Wurtz and Goldberg 1972). We mapped out the area of the retina to which, when the monkey made a saccade, the cell would fire, and we called that area the movement field of the cell.

Movement fields in the intermediate layers were in register with the visual receptive fields in the superficial layers above. Some intermediate-layer cells had both visual and presaccadic activity. Others had only presaccadic activity. We put an electromyography electrode on the lateral rectus muscle to ensure that the superior colliculus signal preceded the muscle activation as well as the actual eye movement. The movement fields were very large.

We then wondered whether we had missed saccadic activity in the superficial layers. At that time, the only saccade task we used was a simple reflexive visually guided saccade. The fixation point went out, the saccade target appeared, and the monkey made a saccade. Because the reaction time of the saccade varied from trial to trial, the activity of visual cells was better synchronized to the target appearance than to the movement. The activity of movement cells in the intermediate layers was poorly synchronized to the target appearance but well synchronized to the beginning of the saccade. The cells in the superficial layers were all synchronized to the appearance of the target. Half of the neurons, however, gave an enhanced response when the monkey was going to make a saccade to the stimulus in its receptive field, as compared with its responses in the fixation task. There was no enhanced response to a stimulus in the receptive field if the monkey made a saccade to a target outside the receptive field. We thought that the enhanced response was a physiological correlate of attention (Goldberg and Wurtz 1972). We then made electrolytic lesions in the colliculus to see whether there was a behavioral correlate of the neuronal activity that we had discovered. After the lesion, the monkeys made contralateral visually guided saccades, but with very long reaction times. Over a couple of months, the reaction times shortened, until they were only about 25 ms longer than ipsilaterally directed saccades. We concluded that this was an attentional deficit and that because the movement fields of the colliculus were so large, it was unlikely that the colliculus was driving saccades. We argued that the role of the colliculus was to effect a shift of spatial attention and not to drive saccades (Wurtz and Goldberg 1972). Sometimes you write something that you wish you hadn't. It became more and more obvious that the colliculus *was* driving saccades. David A. Robinson (1972) had shown that you could evoke saccades by stimulating the superior colliculus, and the map of evoked saccades resembled the visual map. Peter Schiller and Mike Stryker (1972) had shown that electrical stimulation of the colliculus evoked saccades to the center of the cells' movement fields, and they concluded that the colliculus was driving saccades, not shifting attention. Years later, at a symposium to celebrate the 25th anniversary of the Laboratory of Sensorimotor Research (LSR) of the National Eye Institute (NEI), Mike Stryker told how Peter said, "Those guys not only missed the boat, they missed the ocean." Luckily, Rich Krauzlis showed, many years later, that inactivation of the colliculus did cause an attentional deficit (Krauzlis et al. 2013). Often the answer to a heated scientific argument is "both."

Doing the superior colliculus experiments taught us that our methods were so cumbersome that more progress was going to be quite difficult. Behavioral programming was awkward. Data analysis was primitive. Bob had a worked a bit with a Laboratory Instrument Computer (LINC) and had some experience programming. He said it was time to buy a computer. Buying a computer at the NIH was rather bureaucratic. The Digital Equipment Company, DEC, had just marketed a laboratory computer, the PDP12, which fit our needs. It had analog-to-digital inputs through which we could record eye movements; digital inputs with which we could measure unit pulses; and digital outputs that could open and close shutters and the water solenoid, or turn on, dim, and turn off the fixation point. It had a 5-inch CRT that we could program, pixel by pixel. It had a clock speed of 100 kHz, 12 K 12 bit words of core memory. It was actually two computers sharing a box, the LINC and DEC's workhorse PDP 8 joined together. A software settable mode switch would determine which computer it was. Data storage was on two rewritable three-quarter-inch mini tape drives. It did not have a hard disk. I had to convince the NIH that this was the best computer for us—a "sole source" justification that included a market survey documenting why GE, Data General, Varian, and other minicomputers were not as good for our purposes. It cost $35,000 1970 dollars and occupied two full relay racks. It changed our lives.

I had no computer experience at all. On nights when Debbie was on call at the hospital, we would have dinner (I usually brought her dinner that I had cooked, hot food kept warm during the two-block walk from our house to Georgetown University Hospital) and then drove back to Bethesda to learn to program. The LINC had a language called FOCAL that could do math, but it was far too slow to be usable in real time. I had to learn LINC assembler. I wrote the behavioral control and data analysis sections. Bob wrote the eye movement recognition section. DEC had inserted a new feature into the computer, vectored interrupts, which had not been on either the PDP8 or the LINC. Instead of polling input devices, when an event (like a clock tick) occurred, it would interrupt the current program. In our case, the program running the display and the computer would switch to the interrupt service routine, and when it was done, it would restore the background program. It didn't work. The program would run for a while and then blow up. I called DEC, and it turned out that we were the first customers to use vectored interrupts. An engineer from DEC came down, sat in the lab, and after a week or so discovered that there was an occasional glitch in the postinterrupt restoring hardware, so it would restore the program counter to the wrong memory block.

The fixed computer worked. It controlled the behavior, sampled eye movements and unit pulses, and calculated rasters. It displayed a running line of the experiment—behavior, units, eye movements, or a data raster for which we could specify triggers and time bases. We hooked up a Grass

oscilloscope camera to a slave oscilloscope. When we hit a hardware switch, the camera would take pictures of the rasters synchronized on the beginning of the saccade, the end of the saccade, the appearance of the stimulus or saccade target, and example eye movements. We used the Grass camera negatives to make figures.

The first experiment using the new computer system was a study of the monkey frontal eye field (FEF). Emilio Bizzi (1968), using an awake but untrained monkey, had recorded FEF neurons while monkeys made saccades in total darkness. He was unable to find neurons that responded before these saccades, but he did find neurons that fired during and after eye movements. He suggested that this was a corollary discharge signal, telling the rest of the brain that an eye movement had taken place. Bob, I, and then Chuck Mohler, who replaced me as Bob's staff associate after I had left the NIH, found that neurons in the monkey FEF had visual receptive fields, which were a plausible contribution to saccade generation. At the 1973 Society for Neuroscience (SfN) meeting in San Diego, Luke Teuber, chair of psychology at the Massachusetts Institute of Technology (MIT), gave a summary of what he thought were the most interesting 10-minute talks at the meeting. It is hard to imagine anyone doing that today. Luke mentioned our description of visual receptive fields in the FEFs. He said that he wished they had been discovered in another place—and that he was not referring to another place in the brain.

Debbie got pregnant in 1969. She was the first woman resident in medicine in 10 years at Georgetown and was determined not to let her pregnancy get in the way of her performance as a resident. One day late in her pregnancy, she noticed a rapid pulse. She took a quick EKG on herself. It was a supraventricular tachycardia. She asked the resident about it—he looked at it and said, "I don't know, admit it." She never said that it was her EKG. The tachycardia went away by itself. She was on call every third night until two days before our son Josh was born. She organized her vacation period and elective rotations to start with her delivery, and never missed a day of serious call. We lived two blocks from Georgetown University Hospital. When Josh was hungry our full-time live-in childcare person would beep Debbie and take him to the Georgetown emergency room, where Debbie would breastfeed him. We had some day-care problems, solved by Debbie's Aunt Esther, who had just retired and offered to help us. She lived with us for more than 20 years.

That was the era of the great anti–Vietnam War marches on Washington. We signed on as physicians to serve as a medical presence. I remember a man with Huntington's chorea in a wheelchair, who came by the medical unit to rest, but said he would not have missed the march for anything. After the first march, the Yippies charged the justice department building. Our medical van followed them. They fought with the police who fought back with tear gas. Josh was gassed in utero and born on the day Nixon began bombing

Cambodia, April 30, 1970. Jon, our second son, was born while Debbie was chief resident in medicine on the Georgetown Service at DC General Hospital. Debbie made rounds four days after Jon was born, carrying him to the hospital in a baby attaché case so she could breastfeed him.

Boston and Neurology

I was committed to being a neuroscientist, but I also wanted to be a neurologist, so I entered the Harvard Longwood Program in 1972, after three years in Bob's lab. The program included the Peter Bent Brigham Hospital, Boston Children's Hospital, and the Beth Israel Hospital (B.I.). The chief was Dr. Charles Barlow, a great clinical neurologist. He was famous for aphorisms: "In neurology you are lucky if you know it is above or below the foramen magnum," but "you are in trouble if you don't have an idea of what it is by the time you cross the room to the bedside." He had incredibly accurate intuition, which complemented his traditional neurological compulsiveness.

Debbie took a job practicing internal medicine in a B.I.-related practice in Chelsea, Massachusetts, with Alan Kaitz, an older internist who needed an associate. They took care of most of their patients in the little Chelsea Memorial Hospital, but shipped their sickest patients to the B.I. where they were called "Chelsea Specials." I was the only resident in the hospital whose wife was an attending.

During the second year of my residency, Nixon impounded the funds that paid for neurology residents, and the program found itself strapped for money. Charlie Barlow offered me a position in the Harvard Neurology Department, but with inadequate startup funds. In the meantime, David Carpenter had been given the opportunity to build a neuroscience department at the Armed Forces Radiobiology Research Institute (AFRRI) on the grounds of the Bethesda Naval Hospital. He made me a fabulous offer, with money for a technician and postdocs; computer facilities, including our own lab computer; faculty-equivalent staff positions for me and for David Lee Robinson, who was doing a postdoc with Bob; and access to a fully staffed primate vivarium. I asked Charlie Barlow what to do—he said that the AFRRI offer was much better, but he could give me a lot of prestige. I went to AFRRI. Charlie asked me if I would not mind saving money for the program by going to Bethesda nine months before my actual three years of residency ended. He said I could do a research rotation with myself, and he would certify me for three years of residency so I could pass neurology boards. In my last rotation in neurology I was chief resident at the B.I. My neurology team was Marc Dichter and Barry Richmond, both of whom have gone on to illustrious careers in neuroscience. My intern was Steve Bergman, a rather hostile, depressed kid who was the poster child for the Vietnam Era counterculture. We worked hard on teaching Steve some neurology and on calming him down. Steve went on to write *The House of God*, under the alias Samuel

Shem. It is a scathing, funny indictment of academic medicine, still read today by almost every medical student and resident. He never mentioned the neurology department in the book. We took good care of our patients and our intern.

Washington and AFRRI

Nine months before the official the end of my residency we moved to Washington, DC. We bought a large house for two kids, Esther, Debbie, and me, in Spring Valley, a suburban area of the District, which originally had a restrictive covenant. The house could not be sold without the express permission of the W. C. and A. N. Miller Company and all of the neighbors on the block—effectively no Jews, African Americans, or Catholics. It was around the block from Lyndon Johnson's mansion The Elms. It had been originally owned by a conservative Republican senator, Peter Dominick, and then by a liberal Dutch economist from the World Bank. One next-door neighbor, Hillie Paige, was a pioneer in rocket engineering, a member of the National Academy of Engineering, a former president of General Dynamics, and at that time a broker of nuclear reactors. He was a friend of the Shah of Iran. Our other neighbor, John Peabody, was a diplomat, a foreign service officer working in the Agency for International Development, specializing in spreading the good news of contraception. His program's particular champion in Congress was a Texas congressman named George H. W. Bush. Allegedly Bush was so supportive of the program that his congressional colleagues called him "Rubbers." Debbie got a job with Group Health and ultimately went into private practice, founding the first all-woman internal medicine practice in Montgomery County, Maryland. We commuted antidromically, in the opposite direction of the usual traffic flow, I to AFRRI and she to Silver Spring. I also arranged to do a bit of neurology at Georgetown—first in the clinic, and then when I passed my boards, as an attending on the ward and consult services.

David Lee Robinson joined me from the NIH, and we started to build our lab. We planned to look for attention-related activity in the pulvinar. We had a PDP11-10 computer, and I set about to recreate Monkrule, the program Bob Wurtz and I had written for the PDP12, for the PDP11. I called the new program Monk11. The PDP11 also had vectored interrupts—it treated hardware devices as actual memory locations. I still think it was humankind's best computer design. It had a 2.2-megabyte hard disk drive with 18-inch white plastic disk cartridges, which looked like the Millennium Falcon, Han Solo's ship from Star Wars. We had to make sure that we had multiple copies of our programs stored on different disks—despite our attempts to keep them clean, the disks got dirty and the heads crashed and destroyed the data not infrequently. The computer could only be booted by a paper-tape routine, not from a disk. AFRRI was right across Rockville

Pike from the NIH, and we maintained a close relation with Bob. His people made our electrodes. He switched to the PDP11 and used our software.

A funny thing happened on the way to the pulvinar. Vernon Mountcastle had published two papers claiming that the posterior parietal cortex had command neurons that drove arm and eye movements that were not sensitive to light (Mountcastle et al. 1975; Lynch et al. 1977). Dave and I thought that unlikely, and that the command neurons that Mountcastle had described were actually visual neurons with enhanced responses. We decided to record from the posterior parietal cortex. We found lots of neurons that discharged before arm and hand movements, but they all had enhanced visual responses. Greg Stanton, an anatomist at Howard University Medical School, helped us with the histology (Robinson et al. 1978). We described command activity as "epiphenomenal visual responses," something else I wish I had never written.

Bill Keys (now a practicing neurologist) from the University of Connecticut, and Cathie Bushnell (a star in the pain world, and now director of the Intramural Program of the National Institute for Alternative Medicine) from American University joined us as our first postdocs. Dave and Bill Keys went on to study the pulvinar. Cathie (then, Catherine now) and I decided to study Area 7 and the FEF Area 8 in parallel experiments, using two tasks, a saccade task and a peripheral attention task. In that task, first used by Bob in study of the superior colliculus and V1 (Mohler and Wurtz 1977), the monkey had to make a hand movement, releasing a bar, when a peripheral stimulus dimmed. Neurons in the colliculus only gave enhanced responses in the saccade task. In our experiments, neurons in the FEF also only gave enhanced responses in the saccade task, but neurons in the parietal cortex gave enhanced responses to both the saccade task and the peripheral attention task. This was further evidence that the parietal cortex had more of an attentional role than an oculomotor role. It is now clear that the FEF also has an attentional role (Kodaka et al. 1997; Thompson et al. 2005; Zhou and Thompson 2009), and I often wonder why we failed to see it. I think that it is because Catherine and I did our experiments in blocks—a peripheral attention block, and then a saccade block, or vice versa, and the more recent experiments used randomly interleaved trials. I suspect that comparing randomly interleaved trials to blocks in the FEF is something on my bucket list that I will never get around to.

I wondered why the AFRRI wanted two guys studying cognitive physiology in the monkey cortex, or for that matter a behavioral science division at all. Unhappily, I found out. This was the height of the cold war, and one of the problems faced by the Department of Defense (DOD) was that there were five times as many tanks on the east side of the West German border than there were on the west side of the East German border. It is the job of a defense department to assume that one day, after a sunbath or a dance (as Vladimir Nabokov wrote in *Ada*, before a catastrophic event) those tanks would cross that border. An ordinary toxic dose of radiation will not stop a tank—the driver will die in Paris, not in Fulda. The solution was the

neutron bomb, which had more radioactive effect than blast effect. A much higher dose of neutron radiation will dump the histamine from all of the mast cells in the body, render the driver unconscious, and stop the tank at once. The question was how high? The DOD set up a committee to work on this problem, with representatives from both sides of the military-industrial complex, and me. The first meeting was in Fort Knox, the epicenter of the American tank corps. I got to drive an American tank, just like Michael Dukakis. It was fun. The second meeting was at Edgewood Arsenal. The Syrians had given the Israelis a number of Russian tanks during the Yom Kippur war, and the Israelis gave the Americans a few, in gratitude for their help. Because it is important that a tank have as small a profile as possible, the Russians designed their tanks for short people. I was the only member of the committee who could fit inside a Russian tank. I decided it was time to leave the DOD.

I went out on the job market, applying to a number of neurology departments. I also wrote a grant, using our first paper as preliminary results, and proposing to do the dual frontal-parietal attention experiments. I submitted the grant to the NIH and the National Science Foundation (NSF), delivering 1.5 cubic feet of grant applications to each place. The grant received a fundable score from the NSF, but it went to a behavioral sciences study section rather than to VisB at the NIH. The study section accused me of lèse majesté for arguing against the command neuron hypothesis, and disapproved the grant. That was the 1970s version of triage—if the NIH had all the money in the world, it would be better to drop it into the Potomac than fund Goldberg. Dr. Carl Kupfer was the first director of the NEI. He invited Bob Wurtz to become the chief of a new laboratory, the Laboratory of Sensorimotor Research. One of Bob's first acts was to offer positions to Dave and me. I turned down very nice offers from the University of California, Irvine and the University of Michigan to join the LSR. Two pieces of paper crossed Carl's desk the same day—one was a request from Bob to hire Goldberg. The other was a notice from a study section to disapprove Goldberg's grant. Carl said he couldn't hire me. Bob said that the grant went to the wrong study section and that Carl should send the grant to a few members of VisB—he sent it to Gerald Westheimer and Torsten Wiesel, both of whom loved the grant. Carl relented. I called the NSF and said that I was not going to use my grant. You don't often hear real joy over the telephone. We rented space at AFRRI for a few years, but at least our SfN nametags said "LSR-NEI-NIH" and not "AFRRI."

The LSR

Bob recruited Fred Miles from Ed Evarts's lab, and Fred Miles's postdoc Lance Optican as the next members of the LSR. Fred was recording from the cerebellum at that time and discovered that the Purkinje cell signal

was unlikely to be permanently responsible for the gain changes of adaptation of the vestibuloocular reflex (VOR). His battle with Masao Ito rivaled mine with Vernon Mountcastle. Lance was an engineer who had gotten his PhD with David A. Robinson at Hopkins. Lance brought a quantitative and modeling sophistication to the LSR and true computer expertise. We hired Al Ziminsky as our first electronics technician, Art Hays as our computer engineer, George Creswell as our histologist, Chuck Crist as our machinist (later to found the Crist Instrument Company, purveyor of equipment for awake monkey neurophysiology to the world), and Nita Hite as our administrator. We were going into new space the NIH was creating in Building 10, the Clinical Center. The new wing was called the Ambulatory Care Research Facility, and the LSR was adjacent to the eye clinic. Someone in the NIH bureaucracy got worried about the proximity of monkeys and patients, and I had to create a list of hospitals that had monkey facilities in them, to assuage their fears. The great thing about the LSR was the community on the floor. We would brutally criticize each other's manuscripts and ideas and stay friends. A few years later we moved again, into Building 49, the Silvio O. Conte building, dedicated specifically to neuroscience, with a large part dedicated to research on nonhuman primates.

The Frontal Eye Field

Charlie Bruce was my first postdoc at the LSR. He was unhappy that we had never found a saccade-related neuron in the FEF that didn't have a visual response, so we developed the learned-saccade task. The monkey first did a number of trials in which the fixation point disappeared and a visual stimulus appeared at the same location. The monkey had to make a saccade to the stimulus. Then the fixation point went out and no visual stimulus appeared. The monkey had to make the same saccade, relying on its memory either of the stimulus location or of the prior movement, in total darkness. We found three different classes of neurons in the FEF—visual neurons, which had no presaccadic response; visuomovement cells, which responded to the visual stimulus in the fixation task and also had a presaccadic response; and movement cells, which had no response to the stimulus in the fixation task, but responded before the saccade in the learned-saccade task. The movement neurons did not discharge when the monkeys made spontaneous, non-task-related saccades of the same amplitude in total darkness. We also found cells that fired before learned saccades in one direction, usually contralateral, and after all saccades in the opposite direction (Bruce and Goldberg 1985). Finally, throughout the FEF, we found cells that discharged during fixation, but only after the monkey had acquired the target. We also found a few cells selective for smooth pursuit, a few cells that responded to auditory signals, and many postsaccadic cells that confirmed Emilio's discovery. We also found evidence for a corollary discharge effect. A subclass of cells

gave tonic responses to briefly flashed visual stimuli. The disappearance of the visual stimulus did not change the response of the cell. If the monkey made a saccade that brought the spatial location of the vanished stimulus out of the receptive field, the cells would stop firing. Most cells were tuned for direction, with a Gaussian tuning, but for distance with a log Gaussian.

David A. Robinson and Al Fuchs (1969) had discovered that electrical stimulation of the FEFs evoked saccades. Because we had discovered a number of different types of cells, we wanted to see whether the nature of the cells at the tip of the stimulating electrode affected the results of electrical stimulation. We discovered that we could evoke saccades at a threshold lower than 50 μA, even while the monkey fixated, for the most part only if the cells at the stimulating electrode were movement cells. When the cells at the recording electrode were visual or postsaccadic cells, the threshold for evoking saccades by electrical stimulation was much higher. The saccades evoked by electrical stimulation had the same direction and amplitude as the peak of the directional tuning curve of the neurons. Electrical stimulation at the site of pre-post cells evoked saccades in the presaccadic direction. There was an unusual map of saccades in the FEF. Saccade direction went from small saccades in the most posterolateral part of the medial bank of the arcuate sulcus, to large saccades in the most anteromedial parts. The direction of the first evoked saccade in a penetration at any given point was never predictable. The second electrically evoked saccade in a penetration rotated away from the first saccade in either an upward or downward direction. As the penetration grew deeper, the direction of the evoked saccades continued to rotate until they came close to the vertical meridian, at which point the rotation changed direction. We often found a cycle and a half of rotation. This point-to-line representation had previously been described in the cat suprasylvian cortex by Larry Palmer, Alan Rosenquist, and Ron Tusa (1978). The low-threshold area, which we always considered to be the real FEF, lay in the anterior bank of the arcuate sulcus. Pure movement cells were found only in this region, and the layer V pyramidal cells were particularly large here (Stanton et al. 1989). Now that we had a good idea of the location of the FEFs we were in a position to study their anatomical projections. Charlie Bruce and I made a number of anterograde tracer injections, and Greg Stanton worked out the cortical and subcortical projections of the FEF (Stanton et al. 1988a, 1988b; Stanton et al. 1989, 1993, 1995). The FEF projected to the superior colliculus, and to LIP, but not to the paramedian pontine and mesencephalic reticular formations that drive saccades.

Camp Goldberg

Debbie and I made an significant social discovery. Dave and I couldn't pay Bill Keys's salary for a while, and Debbie and I invited him to live in a spare bedroom. It turned out that we enjoyed having young people unrelated to

us by blood living with us. We called them "house kids." Over the years, we probably had more than a dozen house kids who lived with us for more than a year, most of whom are still important people in our lives. The house kids found us in various ways. One day one of our son Josh's ex-girlfriends called to ask me if her friends, who were coming to DC, could stay with us while they found a place to stay. I said, "Sure." She said that there was a problem, that they had a baby. I said that it was not a problem (Debbie was dubious). Holly and Michael arrived, red-headed 8-month-old baby in tow. They couldn't find an inexpensive place to stay, but Debbie and I had fallen in love with the baby, named Canaan because her parents wanted a gender-nonspecific name. We asked them if they wanted to stay with us. They said there was a problem, that they had a very large dog. We had a pretty large dog ourselves, so it wasn't a problem. They lived with us for two years. The baby, now 26, is like a daughter to us. I had a Howard Hughes Cloister Fellow, Alan Wu (now an associate professor of neurology at University of California, Los Angeles [UCLA]), working in my lab. Alan's girlfriend Joyce was a medical student at NYU who had a friend who was coming to DC for a weekend. Alan asked us if he could stay with us for the weekend. Alan said that if Joyce's friend stayed with us he would cook us a great Chinese meal. He did cook us a great Chinese meal. Three weeks later he wrote me, asking if he could come to DC and live with us. He wanted to learn how to live with his emotions on the surface. What he really wanted was an emotionally supportive place where he could come out of the closet. He lived for us for more than two years and became comfortable with his homosexuality. Having house kids was ultimately useful because my mother, who couldn't live by herself, was moving in with us. We built an extra floor to the house. We had a shift of day care to take care of the two elderly women, whose ages by now were greater than their IQs, and having young people in the house to help us was good.

Camp Goldberg was a great party house. We had a swimming pool and hot tub, a great kitchen with a professional stove. We had all of the LSR parties, including the triennial party for friends who came from all over the world to the DC meeting of the SfN. There was a fishmonger who came to the NIH with great fish, and we would have an informal Friday night fish dinner almost every Friday. Years later we realized this was a Jewish Shabbat, and we still do it. We had a monster New Year's Eve party every year, frequently with a pianist playing some classical music before midnight.

Spatial Accuracy

One of the great problems in psychology is to understand how the brain can generate a spatially accurate, stable representation of the visual world for action and perception, despite the fact that visual information only enters the world via the retina, and the retina is moving all the time. Herman von Helmholtz, inventor of the direct ophthalmoscope and Helmholtz Free

Energy saw a patient who awoke with a paralysis of his lateral rectus muscle. He also had a macular hemorrhage in the other eye, so he relied on the now-paralyzed eye for vision. Every time he tried to move his eye laterally, he perceived the world to jump in the opposite direction, and slowly drift back. Helmholtz postulated that the brain achieved spatial stability by feeding back the plan of an intended movement to the visual system, to compensate for the shift of the visual world on the retina. Sir Charles Sherrington, having discovered the importance of muscle spindles in proprioception, suggested that the way the brain solved the problem of spatial accuracy was to measure where the eyes were in the orbit, and where the head was on the body, and to use those data to calculate where an object was in space.

The early models of the saccadic system used the position of the saccade target on the retina to calculate the saccade necessary to acquire the target (Young 1963). In a brilliant experiment, Hallett and Lightstone (1976) showed that the retinal location was inadequate for driving saccades. They found that human subjects made accurate saccades to targets that appeared and disappeared before an intervening saccade. This was a double-step task: If the subjects made the second saccade to the retinal location of the target, the saccade would be wrong, but they made their saccades to the spatial location of the vanished target. Hallett and Lightstone concluded, "This paper presents more evidence that saccades are towards the physical positions of targets—which is only possible if retinal image position and eye position information are correlated." Larry Mays and Dave Sparks (1980) showed that if they asked a monkey to plan a saccade to a flashed target and then deranged the eye position by stimulating the superior colliculus, the saccade was accurate despite the dissonance between the retinal location of the target and the vector of the saccade need to acquire it. They, too, concluded that the brain constructed a model of target position in space. Finally, they showed that tonic visuomovement neurons in the colliculus, with sluggish visual and movement responses, discharged when the monkey made a saccade to a target that never appeared in the movement field or visual receptive field of the neuron (Mays and Sparks 1980). They called these neurons "quasi visual." The target position in space seemed pretty clearly established as the targeting information for saccades. There was a plausible mechanism for establishing a representation of the target position in space. Richard Andersen and Vernon Mountcastle (1983) had demonstrated that the position of the eye in the orbit modulated the intensity of retinotopic visual responses in Area 7, the "gain fields," and it was easy to calculate target position in head-centered coordinates from gain fields—using a three-layer neural net, in which a back-propagating network developed gain fields in its hidden layer (Zipser and Andersen 1988), or using the gain fields as basis functions (Pouget and Sejnowski 1997).

Charlie Bruce and I wondered how purely visual cells in the FEF would discharge in the double-step task. We found a type of cell that had

absolutely no activity in the learned saccade task but that would discharge in the double-step task before the beginning of the first saccade, which was going to bring the spatial location of the vanished stimulus into the neuron's receptive field. We realized that this was an example of Helmholtz's theory—the motor system fed back a copy of the motor command to adjust the visual response, subtracting the vector of the first saccade from every point in the visual map and creating a coordinate transformation from a map centered on the original fovea to one whose origin was the first saccade target. This mechanism did not require an explicit computation of target position in space. The brain did not need to know where something was in absolute coordinates, it only had to know how to get there. We sent the paper off to the *Journal of Neurophysiology*: "Primate Frontal Eye Fields. III. Maintenance of a Spatially Accurate Saccade Signal by the Coordinate Transformation of the Visual Map" (1985). I have never had a more hostile review. One of the comments was "Target position in space is absolutely necessary!" (exclamation point the reviewer's). The paper, submitted in 1985, was ultimately published in 1990 (Goldberg and Bruce 1990). The reviewers made us take the phrase "by the coordinate transformation of the visual map" out of the title. We at least were allowed to use the phrase in the discussion.

In 1986, Michelle Brouchon invited me to be a visiting professor in Marseille, at the Ecole des Hautes Etudes en Sciences Sociales, for six weeks. We were going to do some experiments on a patient of hers who was able to reach for objects close, but couldn't throw a ball at an object far away. I was working with a superb graduate student, Jean-René Duhamel (now director of the Institute for Cognitive Neuroscience in Bron, France). I asked him if he wanted to do a postdoc in my lab. He was worried that he didn't know any physiology. I reassured him that physiology is easy; creativity is hard. Carol Colby (now professor of neuroscience at the University of Pittsburgh and the Center for the Neural Basis of Behavior) arrived in the lab at around the same time from Charlie Gross's lab at Princeton.

Carol had done a very nice anatomical study with Riccardo Gattass and Carl Olson, showing that a number of areas in the intraparietal sulcus project to area PO in the occipital-parietal sulcus (Colby et al. 1988). She was interested in exploring differences among the different areas in the lateral intraparietal sulcus. She and Jean-René showed that there a number of representations of the visual field in the intraparietal sulcus, each describing a different motor workspace, with its attending specific somatosensory input—the medial intraparietal area (MIP) described reaching space, the space explored with the arm. The lateral intraperietal (LIP) area described far space, the space explored with the eye (Colby et al. 1996). The ventral intraparietal (VIP) area at first didn't make sense. The neurons in VIP were all motion selective—very much like middle temporal (MT) neurons (Colby et al. 1993). Then Jean-René and Carol discovered that many of the neurons in the VIP also had receptive fields on the face and were

topographically mapped to the face, if you consider the mouth to be the fovea of the face (Duhamel 1998). Many VIP neurons were selective for objects near the monkey. These "nearness" neurons did not depend on retinal disparity for their estimation of nearness—they were near-selective even when one eye was occluded. Many would also respond to stimuli coming toward their tactile receptive fields on the face. We decided that VIP described the motor workspace of the mouth.

We then went back to the problem of spatial accuracy in LIP. Visual neurons in LIP gave the same sort of remapping responses in the double-step task as the ones in the FEF (Goldberg et al. 1990). The remapping seen in the double-step task might be a feature only of that task, however, a laboratory curiosity. We asked what happened when a monkey made a saccade that brought a visual stimulus outside the spatial location of its receptive field (the current receptive field) into its receptive field, without the monkey's ever having to make a second saccade to the stimulus. Delightfully, about a third of the neurons responded to the stimulus in the spatial location that would enter the cell's receptive field by virtue of the saccade (the future receptive field) before the saccade began. Remapping was a general feature of parietal function (Duhamel et al. 1992). The response required that the saccade bring the remapped target into the receptive field. If the monkey made the saccade without the target being present, or if we flashed the target when the monkey was fixating and not planning a saccade, the cell did not fire. We then asked whether visual memory remapped as well as constant visual stimulation. We flashed a target briefly outside the neuron's receptive field and asked the monkey to make a saccade that brought the spatial location of the vanished stimulus into the receptive field. Neurons that did not show presaccadic remapping often fired after the saccade, showing that a memory trace of the spatial location persisted. Makoto Kusunoki (now a research scientist at the Medical Research Council Cognition and Brain Sciences Unit at the University of Cambridge), came from Hideo Sakata's lab in Nihon University in Tokyo. He discovered that neurons became less responsive to stimuli in the current receptive field as they became responsive to stimuli in the future receptive field (Kusunoki and Goldberg 2003).

Jean-René and Ed Fitzgibbon (now chief of the Acuity Section at the LSR), a fully trained retinologist who had joined my lab after his ophthalmology residency and fellowship at the Cleveland Clinic, found a patient with a right frontoparietal lesion who was willing to participate in eye movement studies using the limbic ring eye coil. In this technique, invented by David A. Robinson (1963), the subject wears a contact device that fits on the sclera outside the cornea, with an embedded eye coil whose wire connects to an outside plug. The subject sits inside a device with two high-frequency orthogonal magnetic fields oscillating at different phases. An electric signal is evoked in the eye coil, from which the position of the eye in the space between the fields can be decoded. We now use the same system to record eye position

in monkeys, implanting the coil subconjunctivally. I wore a limbic ring eye coil once or twice, and after a few minutes, I was willing to confess to heresy to get the thing removed, but our brave patient persisted. She had a field cut in the left visual field periphery, but her central vision was preserved. She had a bit of neglect for double simultaneous stimulus presentation. Her leftward saccades were a bit less accurate and had a 100 ms or so longer latency than her rightward saccades. This was true for 5° saccades across a 40° range of orbital positions, regardless of the spatial position of the target. We then asked her to do the double-step task, flashing two targets, one in the normal field and one in the affected field before the reaction time of the first saccade. When the target flashed in the right (normal) field and then in the left (affected) field, her first saccades were perfectly accurate, and her second saccades, into the left (affected) field were reasonably accurate, but not as good as single visually guided saccades into the affected field. When the target appeared first in the affected field, the patient made the usual slightly inaccurate, longer latency saccades, but then she was devastated. She could not make saccades to a target that appeared in the good field, in the direction of her normal saccades. She knew where the target was—about 30 percent of the time, she made saccades directly to the second target in the good field because of her neglect. We postulated that the deficit was a deficit of corollary discharge—she was unable to remap the location of the stimulus to the new fixation point. We published a single case report (Duhamel et al. 1992). I always thought that the single case report was the last refuge of the scoundrel, but Wolfgang Heide, in Lübeck, Germany, found the same deficit in a large number of parietal patients (Heide et al. 1995).

Mark Walker, a Howard Hughes Cloister Fellow in my lab (now associate professor of neurology at Case Western Reserve) and Ed Fitzgibbon showed that neurons in the intermediate layers, but not superficial layers, of the superior colliculus remapped, with both predictive remapping and memory-trace remapping (Walker et al. 1995). Marc Umeno (now the project manager for the VMware Cloud on Amazon Web Services) was a graduate student in experimental physics at American University. He convinced the Department of Physics that we did quantitative neurobiology, which could be considered biophysics. I suggested that he see whether the FEF had remapping as well. I showed him how to do the control experiments first (no response when the remapped target flashes while the monkey fixates; no response when the monkey makes the saccade without the remapping target being present) and then to look for remapping. The FEF exhibited remapping like LIP and the superior colliculus (Umeno and Goldberg 1997). Marc was extremely efficient, and unlike most of the other people at the LSR, he had a life. He was a nationally ranked bridge player and a member of the best ultimate Frisbee team in DC. He decided that doing the control experiments first was a waste of monkey time and water, so he ran the remapping experiments first and then did the controls only when he had found a cell that remapped. The

saccade control stopped working. After a stimulus had appeared at a specific spatial location for a number of trials designed to demonstrate remapping, neurons that had not responded when the monkey made the remapping saccade without the target now responded, as if the brain had established a memory that lasted across trials. This intertrial memory was different from remapping. Some cells with intertrial memory didn't remap. Some cells with remapping didn't exhibit intertrial memory (Umeno and Goldberg 2001).

The strange thing about remapping was that it seemed to require that a remapping neuron have access to every part of the retina and that the corollary discharge from every possible saccade. Lance Optican, Christian Quaia, and I developed a model that explained how that could happen, with a lot of developmental pruning and Hebbian synapse strengthening. I was always unhappy about that model (Quaia et al. 1998).

Years later, I would return to modeling remapping. After I moved to Columbia, Mingsha Zhang (now professor of neuroscience at Beijing Normal University in China) came to the lab. He (and his wife Xiaolan Wang) were Chinese physicians who earned their PhDs at the Weizmann Institute of Science in Israel with Shabtai Barash. Xaiolan then went to work with Patricia Goldman-Rakic and Charlie Bruce at Yale, and Mingsha spent a few months with David Sparks in Houston, where he ran up against terrible bureaucratic problems and wasn't allowed to touch a monkey. He asked to join my lab. He wondered whether during the remapping process, the receptive field expanded or merely went down at the current receptive field and up at the future receptive field, as Bob and Marc Sommer had shown for a few neurons in the FEF (Sommer and Wurtz 2006). He discovered that the receptive field expanded to encompass the entire part of the retina through which the saccade sweeps the receptive field, but not elsewhere. A student of his lab in Beijing, Shaobo Guan, tried to model this receptive field expansion and, again, I didn't like the model. Mingsha, I, and his colleague at Beijing Normal University, Si Wu, struggled with this model in Beijing and New York. We finally came up with a model that evoked a cortical wave traveling from the cell whose receptive field included the future receptive field to the cell with the current receptive field. The model required the retinal signal only to drive the future receptive field cell and required the corollary discharge signal only to enable lateral connections among cortical neurons to create the cortical wave. We took advantage of the moving wave in the colliculus to provide the corollary discharge (Munoz et al. 1991). We wrote a paper that included the data and a model that included a one-dimensional retina, and one direction of saccade, and sent it off to *Neuron*. Reviewer 2 said that the model suggested that the latency of the intermediate location should be shorter, and the response intensity greater, than those of the ultimate remapping response. We analyzed the data and both of these suggestions turned out to be true. He also said that a one-dimensional, one-directional model was worthless. We needed a two-dimensional model that worked for

all directions of saccades. Si, Mingsha, and I struggled with the two-dimensional, multisaccade-directional model, and we finally figured it out (Wang et al. 2016). This was the best review I had ever had in my life and should be the model for all reviews, constructive rather than destructive. A few years later, Roozbeh Kiani, assistant professor of neuroscience at NYU, told me that he was Reviewer 2. He should have been an author on the paper.

John Leigh and Ari Zivotofsky at Case Western (1996) had discovered a significant exception to the idea that corollary discharge and eye position are sufficient to determine target position in space. When you look at the moon on a cloudy night, and clouds pass over the moon, you think that the moon is moving in the opposite direction as the clouds. This is called the Duncker illusion. When human subjects pursue a single dot moving at right angles to a moving background, they think that the dot is moving diagonally away from the direction of the background motion and that their eyes move along the same trajectory as the dot.[2]

In fact, their eyes follow the actual trajectory of the dot. If they try to make a memory-guided saccade to a target that flashes early during the pursuit, the saccade is inaccurate, as if the subjects adjusted their eye position for the pursuit trajectory that they thought they followed, rather than the one they actually followed. They would make this mistake even though they presumably had an accurate corollary discharge of the pursuit movement, and an accurate eye position signal. Ari came to the LSR to work with Fred Miles and asked to join my group to solve the puzzle of how the Duncker illusion fools the brain. Ari and Keith Powell (now working for Teva Pharmaceuticals), from Barry Peterson's lab at Northwestern, showed that monkeys report the Duncker illusion (Zivotofsky et al. 2005). Years later, Naoko Inaba (now an assistant professor at Sapporo University) came to my lab from Kenji Kawano's lab in Kyoto to study the cortical responses during Duncker pursuit. She showed that pursuit neurons in the middle superior temporal (MST-l) area but not the MT area are tuned for perceived rather than actual pursuit. We presented this at the SfN and are writing it up.

Music and Theater

We did a lot of music. I was still playing the piano, and Debbie the flute. We mangled sonatas by Bach and Poulenc, and our kids grew up listening to classical music. Jon, age five, with a 60 db flat hearing loss said he wanted to play the violin. We got him an eighth-sized violin, and it soon became apparent that he was very good at it. I installed a smoke detector and tested it. Josh asked "What's that?" Jon said, "a G." We ran down to the piano, and he was right. One night a friend came over and we hacked our way through a

[2] Dunker Motion Illusion, YouTube, October 29, 2014, https://www.youtube.com/watch?v=QUbJKakfmZw.

Beethoven cello sonata. Jon sat at the foot of the cellist, entranced. He asked if we had the records and the book. I said that we did. For the next three weeks Jon listened to the Beethoven cello sonatas full time and kept asking us where it was in the score. Soon he stopped asking. He had decoded bass, tenor, and treble clefs. He then went on to the Beethoven violin sonatas and never asked where he was. I loved going to concerts with him. He always brought the scores. Public television did a broadcast of *Don Giovanni*. He was outraged that Joan Sutherland sang "Or sai chi l'onore" transposed down a full tone. We bought a fairly new Steinway L because we couldn't tune our old piano adequately. We played a lot of Baroque trios with Jon. Josh, not a musician himself, had an acute ear for music, and dragged me into 1970s minimalism. I learned not to be bored by Phillip Glass, Steve Reich, and Terry Riley. Jon stopped playing the violin in college, but was a rock dj at WHRB, the Harvard radio station. After college, he did odd computer jobs and ultimately became an Orthodox Jew. After a few years in Israel, he decided to go to law school at Penn, where he made the Law Review, and clerked for a federal district court judge. He was, however, not cut out for the corporate law life.

Josh inherited the theater gene. He was the star of his high school plays, and we had Josh's high school cast parties in our house. He put on a production of David Mamet's *Sexual Perversity in Chicago* in our livingroom. At Hampshire College, he studied directing and lighting design. After a time in New York doing off-off Broadway lighting and directing plays on the Fringe, he became disillusioned with the political world of New York theater. He got a master's degree at the NYU's School of Interactive Telecommunications and became a computer-based light artist, specializing in motion-driven environmental lighting and museum displays. He does lights and video for an occasional off-off Broadway play and is a video jockey at an occasional club gig.

Visual Attention

All of our experiments had been done looking at the effect of flashed or moving stimuli on neurons in various parts of the brain. It struck me that in the real world most objects are stable, and they enter the receptive fields of neurons because of eye movements. The assumption had been that flashing a target in the receptive field was not terribly different from the target's entering the receptive field by way of a saccade. Jackie Gottlieb (now a professor of neuroscience at Columbia) joined the lab. She had worked on the FEF's role in smooth pursuit as a graduate student at Yale with Charlie Bruce. She joined Makoto, who had to return soon to Japan, to work on the project. We designed the stable target task to test the assumption.

In this task, an array of circular objects stayed on the screen all the time. When the monkey was fixating the center of the array, one of the objects would be in the cell's receptive field. The monkey made saccades

that brought that stable object into and out of a neuron's receptive field. If the monkey made a saccade that brought a behaviorally irrelevant, stable stimulus into the receptive field of a neuron, its response was very weak. We then took advantage of the fact that the abrupt onset of a visual stimulus grabs attention (Yantis and Jonides 1990). The weak response of the neuron could have been because stable, task-irrelevant objects do not evoke much of a response, or it could have been that making a saccade to bring a stimulus into the receptive field damps down the response. To distinguish between these two possibilities, we made a hole in the array at the spatial location that would be brought into the receptive field of the saccade. We flashed the stimulus in the hole and asked the monkey to make the saccade that would bring the recently flashed stimulus into the receptive field. The neuron fired as if the stimulus had just flashed in its receptive field. The low response evoked by stable targets was due to their intrinsic lack of interest to the monkey, not to the saccade. We also could make the neurons respond intensely to one of the stable objects by cuing the monkey to make a subsequent saccade to the object. In this case, the response became enhanced as soon as the monkey made the first saccade that brought the cued target into the neuron's receptive field, but not when the monkey had been cued to make a saccade to another target (Gottlieb et al. 1998). These experiments showed that most of the visual world is barely represented in LIP. LIP represents only objects that are behaviorally important, by virtue of top-down priority or bottom-up salience.

James Bisley (now associate professor of neuroscience at UCLA), who had gotten his PhD in Melbourne under Tony Goodwin, was working in Tanya Pasternak's lab on memory for motion in monkeys. His fiancée Andrea had gotten a postdoc in Baltimore, so he decided to move to Maryland and joined my lab. The debate about whether LIP was primarily involved in attention or saccade planning was still raging, and James and I decided to return to the fray. I had spent a lot of time studying attention, but I used a rather weak, ad hoc definition of attention: If the monkey responded to a stimulus, then it had, at some time, paid attention to that stimulus. Williams James had pointed out that attention increased perceptual efficiency and shortened reaction time. We knew that the abrupt onset of a task-irrelevant visual stimulus lowered humans' perceptual threshold at the spatial location of the stimulus (Yantis and Jonides 1990). Heiner Deubel (1996) and Eileen Kowler (1995) used perceptual threshold to show that planning a saccade focused attention at the goal of the saccade.

We used perceptual threshold to understand the determinants of a monkey's attention in a very complicated task. Monkeys first had to plan a memory-guided saccade to a point in space. On any given day, the saccade target could appear at one of four places: one in each quadrant of the visual field, symmetrical across the horizontal, or the vertical meridians. On each trial, a go–no go probe would appear for one video frame during the delay

period (800, 1,300, or 1,800 ms) after the saccade target had appeared, while the monkey was planning the saccade. The go–no go probe consisted of three circles and a capital C. Each object appeared at one of the four potential saccade target locations. The monkey had to discriminate the orientation of the C to determine whether actually to make the saccade or to hold fixation after the fixation point disappeared. We varied the contrast of the probe to determine the monkeys' perceptual threshold and found that planning a saccade to a location lowered the contrast threshold for detecting the orientation at that location. This enabled us to assume that the monkey was attending to the spatial location with the selectively lowered perceptual threshold. On half of the trials, we also flashed a distractor, identical to the saccade target, 500 ms after the saccade target appeared. We found that 200 ms after the distractor appeared, the locus of attention left the saccade and moved to the distractor location. By 700 ms after the distractor appeared, the locus of attention returned to the saccade goal. We then recorded the activity of LIP neurons in this task and placed either the distractor or the saccade goal in the neurons' receptive field. The response to the saccade target had a large transient, and then a sustained, lower but significant level of activity during the delay period. The response to the distractor had the same large transient, but it rapidly fell beneath the level of the delay period activity evoked by the saccade plan. For about 90 ms, the "window of neuronal ambiguity," there was no significant difference between the activity evoked by the saccade plan and that evoked by the distractor.

We then studied the monkeys' perception when the go–no go probe appeared at the center of the neuronal window of ambiguity. We had expected both sites to be somewhat attentionally advantaged, but instead, the monkeys had no attentional advantage at either site. This was a paradoxical result. It is generally assumed that attention is divisible and graded. Our result showed that attention as measured by perceptual threshold is binary and indivisible. We wondered whether attention were quantized, with a shuttle that moves the quanta so rapidly that only very short stimulus presentations can unmask it. Different monkeys had different neuronal windows of ambiguity. Each monkey had no attentional advantage during his own window of neuronal ambiguity, but had an attentional advantage during the other monkey's window of neuronal ambiguity (Bisley and Goldberg 2003). The activity of neurons in LIP tracked the monkey's locus of attention, regardless of what evoked that attention.

We then looked at the response of LIP to the probe stimulus. There was no difference between the response to the circle distractor in the receptive field and the response to the go probe. The no-go probe evoked a greater response than either. These results had some interesting implications. The first was that activity during the delayed saccade task can determine the monkey's locus of attention as well as its saccade plan. The second was that we could not determine the monkey's locus of attention by looking at the

activity at one locus on the visual map in LIP. Attention is determined by the emergence of a statistical peak on the map—activity that can sustain attention during the delayed saccade task can no longer do it when the response to a task-irrelevant distractor becomes the peak. The third is that when a probe appears for a single video frame, attention is not determined by the response to the probe but by whether the peak of the map is at the probe location when the probe appears. The fourth is that because LIP responds more intensely to a probe that cancels a saccade plan than to a probe that sustains it, it is unlikely that LIP by itself is driving saccades. We concluded that LIP integrates top-down signals like a saccade plan and bottom-up signals like the transient visual response to an abruptly appearing, task-irrelevant stimulus, to create a priority map of the visual field. The visual system pins attention to the peak of the priority map. The oculomotor system drives saccades to the peak of the priority map when saccades are appropriate.

Although James and I did the recording and psychophysics in the LSR, we wrote the paper at Columbia. The center of the window of neuronal ambiguity for a given neuron was the time at which the decay of the visual transient crossed the delay period level evoked by the saccade plan. For the majority of the neurons, this crossing point occurred within a 40 ms interval. We wanted to know whether this were a membrane phenomenon or a network phenomenon. We took our data to Ken Miller, in the new Columbia Neurotheory Center, and posed the question to him. He and his postdoc Suriya Ganguli came up with a delightful solution: a network model in which generically high-dimensional firing-rate vectors rapidly decay to a single mode. The model predicted that slowly varying activity patterns, like the delay period activity, are proportional to spontaneous activity, which turned out to be true (Ganguli et al. 2008).

Prefrontal Cortex

Rolf Boch came to my lab from Burkhardt Fischer's lab in Freiburg. He and Burkhardt had discovered express saccades (Fischer and Boch 1983). They also studied prestriate area V4 (they called it the "prelunate gyrus") in an overlap task, where the monkey fixates, a saccade target appears; a second or so later, when the fixation point goes out, the monkey makes the saccade. Neurons in V4 show an enhanced response to the saccade target (relative to the response to the same stimulus in a fixation task) and also a presaccadic reactivation (Fischer and Boch 1980). Rolf wanted to know whether we would see the same thing in the prefrontal cortex. We did—some cells showed enhancement of the visual transient, others showed presaccadic reactivation, and some showed both (Boch and Goldberg 1989). I didn't know what this meant. Rolf was a wonderful artist, and when he returned to Germany, he decided to support himself and his family by teaching physics in a technical school and doing art.

I left the prefrontal cortex alone until Ryohei Hasegawa (now group leader of the Neurotechnology Group at the Tsukuba Electrotechnical Institute in Tsukuba, Japan) came to my lab from Aki Mikami's lab in the Primate Research Institute of Kyoto University, in the little town of Inuyama. Michael Petrides and Brenda Milner had developed the self-ordered task as a way to study nonspatial memory (Petrides and Milner 1982), and Michael had shown that monkeys with lesions in middorsolateral prefrontal cortex were impaired on the task (Petrides 1995). Ryo; Ari Blitz, a Howard Hughes Cloister Fellow (now assistant professor of neuroradiology at Johns Hopkins); and I trained monkeys to do a saccadic self-ordered task. In this task, the monkey fixated a central fixation point, and then three of six possible objects appeared equidistant from the fovea and spaced 120° apart. The monkey made a saccade to one, his choice, and got a small reward. The objects reappeared, at shuffled spatial locations, and the monkey had to make a saccade to either of the two objects to which he had not previously made a saccade, for which he got a larger reward. The objects reappeared a third time, again shuffled spatially, and the monkey had to make a saccade to the remaining object to which he previously had not made a saccade, for which he got an even larger reward. We had hoped that we would find neurons that were selective for remembering a specific target. We didn't. Instead the neurons increased their activity from the first to the second to the third saccade. The activity was not an increasing reward signal. If we merely increased the reward in the context of a simple saccade task, we didn't see the responses increase. Most of the monkeys' errors were on the third trial. There was no increase in activity between the second and third trials on third-trial errors (Hasegawa et al. 2004). We also noticed that the baseline activity of the neurons, during the fixation period of the first trial, predicted how the monkey was going to do on the third trial several sets in the future, or how it had done on the third trial several sets in the past—but rarely if ever what was going to happen on the current set of trials (Hasegawa et al. 2000).

Barry Peterson, professor of physiology at Northwestern, came to the lab for a sabbatical. One of the hallmarks of patients with frontal cortex lesions is the inability to suppress stimulus-bound behaviors. They have difficulty performing Dan Guitton's antisaccade task, in which they have to look away from a visual stimulus (Guitton et al. 1985). Barry and Ryo decided to see whether the FEF and its adjacent prefrontal cortex had neurons that responded selectively to targets to which the monkey was not allowed to respond. They designed a spatial nonmatch to a sample task, in which a stimulus flashed and disappeared, and then after a delay, the original stimulus and another stimulus appeared. Depending on the color of the fixation point, the monkey had to make a saccade either to the original stimulus or to the new stimulus. Visually responsive neurons in the FEF and the prefrontal eye field were often selective to the stimulus to which

the monkey did not make the saccade (Hasegawa, Peterson et al. 2004). This experiment shows that the frontal cortex has a direct role in suppressing unwanted stimulus-bound behaviors, rather than only an indirect role through the basal ganglia.

Saccadic Adaptation

S. C. McLaughlin (McLaughlin 1967) invented saccadic adaptation as a tool to look at plasticity. Subjects make a saccade to a visual target, and during the saccade, the stimulus consistently moves to a new location toward or away from the target. Gradually, humans and monkeys change the amplitude of their saccade, so the eye moves to where the target ends up, not to where it begins. Separating the amplitude of the saccade from the location of the stimulus enabled us to see whether a neuronal signal described the stimulus or the movement. Mark Segraves (now professor of neuroscience at Northwestern) came to the lab from Alan Rosenquist's lab at Penn, and after graduation, did a year with Giorgio Innocenti in Lausanne. He and Ed FitzGibbon decided to train monkeys on the task and record from the colliculus. We discovered that visual and movement neurons in the colliculus signaled the target location and not the actual movement (FitzGibbon et al. 1986). We assumed that some downstream center, probably the cerebellum, adjusted the saccadic amplitude to compensate for the adaptive process. However, electrical stimulation of the colliculus failed to produce the adapted saccade, which should have happened if the adaptation occurred downstream. Jay Edelman (now professor of biology at the CCNY–CUNY) came to my lab from Ed Keller's lab to study saccadic adaptation. He showed that electrical stimulation at high currents did indeed produce the unadapted saccade, but stimulation at lower currents produced the adapted saccade (Edelman and Goldberg 2002), confirming that the change in the saccadic amplitude occurred after the colliculus.

After I arrived at Columbia, Matt Phillips (now staff research engineer at Kitware, a computer consulting company) who had been a postdoc with Jay Edelman at CCNY, and Sara Steenrod (now a science editor at Washington University of St. Louis), a graduate student who had spent time as a technician in Barry Richmond's lab at the NIH, studied saccadic adaptation in LIP, using a memory-guided delayed saccade task. They showed that visual transient signals, delay period activity, and the presaccadic burst all described the location of the saccade target, and not the amplitude of the actual saccade (Steenrod et al. 2013).

Columbia

I loved the LSR. I had great colleagues and wonderful support. I didn't have to write grants except occasionally to support a postdoctoral fellow. We had

Jim Raber, probably the world's best primate veterinarian, and Nick Nichols and Tom Ruffner, spectacularly good machinists. John McClurkin and Art Hayes were great computer guys. I had wonderful colleagues not only in the LSR but also in the Laboratory of Neuropsychology of the NIMH, a flight down from the LSR in Building 49: Mort Mishkin, Leslie Ungerleider, Bob Desimone, Barry Richmond, and Betsy Murray. Bob Wurtz, although no longer lab chief, still shielded us from the NIH bureaucracy. There were some downsides. The NIH had little teaching, no formal way to have graduate students. I didn't have a real role in the Neurology Department at Georgetown and making rounds required a lot of commuting time. I missed New York. The Metropolitan Opera is much better than the Washington National Opera.

Eric Kandel had convinced David and Hillie Mahoney to donate enough money to build the Mahoney Center for Brain and Behavior Research. Columbia and the New York State Psychiatric Institute (NYSPI) were going to renovate the whole fifth floor of the NYSPI for a center dedicated to systems neuroscience, concentrating on neural recording in awake monkeys. Eric had just recruited Jackie Gottlieb as a tenure-track assistant professor. Vince Ferrera and Ning Qian were already there. Eric realized that he needed a senior scientist to head the center. He asked me to come up and look at the position. I gave a seminar for the Center of Neurobiology Behavior on the parietal cortex and ran a session of the neurology department's morning report. The residents always present the case as a mystery for the professor to solve. Luckily, it was a case of methotrexate-induced white matter disease, a syndrome I had recently seen and could discuss. Eric offered me the job, and Tim Pedley, then chair of the Neurology Department, asked me to join the Neurology Department as well and attend in the clinic. The NEI asked me what would induce me to stay. I said new young colleagues. We hired Bruce Cumming from Oxford (now the lab chief of the LSR) and Kirk Thompson from Vanderbilt. I dithered for a long time and Columbia improved the offer—I would be the David Mahoney Professor of Brain and Behavior Research, and also have a state salary at the NYSPI, an apartment with rent at half the market rate, and even a parking space. Finally, Debbie announced that we were going to New York. After I had agreed to go to Columbia, but while I was still working at the LSR, I recruited Daniel Salzman from Bill Newsome's lab and Aniruddha Das from Charles Gilbert's lab to the Mahoney Center. Columbia found Debbie a job in the Department of Medicine.

We sold Camp Goldberg in DC on my 60th birthday, and because our apartment in New York was not going to be available for a month, we were actually homeless. Josh asked us if we would like to go to Burning Man, a somewhat-notorious alternative libertarian arts festival in the Nevada desert. It is an honor to play with your children, so we decided to do it. Because there are no spectators at Burning Man, only participants, we signed

on as medical volunteers, doing first aid. One of my first patients asked me whether she could take ecstasy even though she was on Coumadin, an anticoagulant, because she had Leiden factor V, a cause of pulmonary emboli in young people. I said that I didn't know; therefore, no. Debbie said someone should do a study. We saw a lot of dehydration in the desert, and caffeine-withdrawal migraine headaches. We got hooked on Burning Man and went back another nine times. We became the unofficial den mother and father for chunks of the New York Burning Man community.

Life in New York

Debbie and I finally arrived in New York at 12:15 A.M. on September 11, 2001. The next morning, we were running north in Riverside Park, and a friend called us and told us to turn around. We could see the smoke of the buildings, which had not yet collapsed. We went over to the nearby Red Cross and asked if we could do anything. They decided that we could be a mental health unit and sent us down in a Red Cross van to the foot of the Manhattan Bridge to help people who might be upset. The subway had shut down, and thousands of people dressed in air-conditioning business clothes were walking from Manhattan to Brooklyn across the bridge. It was a hot, sunny day, and we realized that although some few people might need someone to talk to, our Burning Man desert experience told us that everyone needed water. Luckily, the Red Cross van had a large number of paper cups and water dispensers, and the Popeye's across the street from the bridge had water. We hydrated thousands of people. Around six o'clock the foot traffic had ended, and the cops asked us if we wanted to go to St. Vincent's hospital to see whether we could help. There was nothing to help—people were either in good shape or dead. We walked up Broadway from 14th Street to 73rd Street, where we were staying with friends, because Columbia had not yet finished our apartment. The only cars in Times Square were police cars and press cars. We were the only pedestrians.

In November, we finally moved into our Columbia apartment, in the Columbia professorial ghetto, across the street from Barnard. The apartment is immense, with a large formal space of dining room, livingroom, and library in the front and a warren of small bedrooms in the back, separated by a very large (for New York) kitchen that we had the Columbia Real Estate people make out of two small rooms. Living in an apartment with a great staff made me realize how stressful maintaining a large house is. I will never make another midnight trip to Home Depot. Because most of the people on the street are faculty members, we know more of our neighbors than we did in DC. Seventeen years later, New York is still breathtaking. Josh and his family live two miles away. Tuesday is grandparent night. Debbie works as an internal medicine hospitalist in a rehab facility owned by Mount Sinai. She walks to work.

New York is heaven for classical music and theater. We see half of the productions at the Metropolitan Opera, lots of off-off Broadway theater, and lots of other music. The building has two apartments per floor, and for a year, we shared the landing with Menachem Zur, an Israeli composer. His son Yonah was a freelance musician in New York. Yonah's quartet had a grant to study the Brahms clarinet quintet with the Emerson quartet, and they practiced it in our livingroom. They did a house concert for us and we invited friends. More and more, professional freelance musicians would rehearse in our livingroom. They would give house concerts if they wanted to try out a concert in front of a live audience—to get the adrenaline running for a real concert they would be giving in the following week at a real concert venue. We now have about 20 house concerts a year. A piano concert tried out in our livingroom was listed by the music critic of the *New York Times* among the 10 best concerts of 2014 when the pianist, Benji Hochman, performed it for real a week later. Many of the musicians have become personal friends, coming to dinner on Friday nights, meeting Burners, neurologists, neuroscientists, and old college friends. I grew old and arthritic, and I rarely play the piano myself any more. We maintain the Steinway for our friends.

Visual Search

When we moved to New York, the Mahoney Center labs were not yet finished. I kept my lab at the NIH and commuted to DC for a few days every week. When the labs were finished, I decided to make the atmosphere of the Mahoney Center like the LSR. Every door on the floor has the same key. We shared a single administrator, Latoya Palmer (now very high up in the administration of Cornell Medical School), and a computer hardware and software technician, Lloyd Kim. Columbia had a machine shop, but it took a long time to get anything done. I wrote an R24 grant to provide us with our own machinist, now replaced by an NEI core grant. We destroy each other's manuscripts at pizza and potshots sessions.

When the labs were finished, James Bisley and Anna Ipata came to Columbia with me. Anna had done pediatric neurology in Pisa, and then got a PhD with Leo Chelazzi and Giovanni Berlucchi in Verona. She was an American citizen because she was born while her father, a biochemist, was doing a postdoc at the NIH. Angela Gee, who had been an undergraduate working with Gerald Westheimer at University of California, Berkley joined the lab at her mentor's suggestion. Angela had the makings of a great scientist, but she decided instead to spend her life teaching neuroscience to underprivileged students. She is now a professor at Los Angeles Trade and Technical College, where she gets ecstatic student ratings.

We realized that the great bulk of my experiments had been done in paradigms in which the monkey made eye movements to earn a reward. In the real world, however, eye movements are made to facilitate visual perception,

and there is no such thing as a wrong eye movement. With Jackie Gottlieb, we designed a visual search task in which the monkey had to search for a capital T among an array of lower case t's and then tell us, with a hand movement, whether the capital T was upright or inverted. The monkey was free to move its eyes. On most of the trials, the target and distractor were all black, but on some trials, either a distractor or the target was bright green. The monkeys in the Goldberg lab behaved like the humans in the Treisman lab. There was a clear effect of the number of objects in the array on reaction time when there was no popout, but there was no effect when the target popped out. A single popout distractor lengthened the monkey's reaction time. We recorded from LIP in the search task. We realized that we wanted to study what happened when the monkey made saccades away from the target, so having established the effect of a popout target on reaction time, we never had the target pop out during recording. We found that the monkey was able to make fewer saccades to the popout distractor than to the black distractors and that activity evoked by a popout distractor was less than that evoked by a nonpopout distractor (Ipata et al. 2006).

I had spent a lot of time and energy on showing that parietal activity could be dissociated from saccade planning. In our visual search task, the monkey adopted the strategy of making many rapid saccades, some of which could be to distractors, rather than making sure it was going to make a saccade to the target. These short reaction times were opposed to those in tasks in which the monkey did not get a reward if it made the wrong saccade, like Jeff Schall's popout search task (Schall and Hanes 1993). We found that activity in LIP correlated with the monkey's saccadic reaction time. LIP was not entirely dissociated from saccade planning (Ipata et al. 2006). Luckily, LIP gave an enhanced response to the target as compared to the distractor even when the monkey made a saccade to the visual hemifield opposite the target. We showed that in visual search LIP had three different signals: an undifferentiated ("dumb") visual response to the abrupt appearance of the search array; a presaccadic signal; and a cognitive, feature attention signal. The cognitive signal was present even when the monkey made a saccade to the target.

We used linear summation to model the waveform evoked when the monkey made a saccade to the target using the waveforms evoked when the monkey made saccade to a distractor or a target in the receptive field and when the monkey made a saccade away from a distractor in the receptive field (Ipata et al. 2009). Although spikes might be generated from motor or sensory inputs, they lose their input identity. Recipient areas do not know the source of the spikes. The visual system will pin attention to the peak of the LIP priority map whether that peak arises from delay-period activity during a memory-guided delayed saccade task or from the response to the abrupt onset of a visual target. The oculomotor system will drive saccades to the peak of the map, when appropriate, even when the input to the map was entirely visual. We wrote a review called "On the Agnosticism of Spikes" (Gee et al. 2007).

The question then arose whether LIP demonstrated the nonsaccadic signals when the monkey did a visual search task without moving its eyes. Mingsha trained two monkeys on the task. LIP did demonstrate the dumb visual, presaccadic, and cognitive signals. The fascinating result was in the baseline. The task began with the monkey fixating for 500 ms, after which the search array appeared. If the fixation point went out, the monkey was free to move its eyes and make saccades until it found the target. If the fixation point stayed on, the monkey had to solve the search task without moving its eyes. The fixation task was harder—the monkey got the correct answer around 70 percent of the time, as opposed to nearly 100 percent when it could make a saccade to the target. The baseline activity, while the monkey was fixating, predicted the monkey's likelihood of success on the fixation task as well as the intensity of the visual transient response to the appearance of the array. The baseline, and the monkey's likelihood of success, correlated inversely with a recency-weighted measure of the monkey's past performance.

The problem with the original experiment was that we did not know where the monkey was attending during the fixation period. We added a 50 percent valid cue during the fixation period. The monkeys pinned their attention to the cue location, as measured by manual or saccadic reaction times, but the baseline predicted success even when neither the cue nor the target was in the neuron's receptive field. This was an arousal effect, not an attentional effect (Zhang et al. 2014). A key feature of attention is its spatial or feature selectivity—the baseline effect was not at all spatially selective. We postulated that the baseline effect was due to some ascending modulatory pathway that affected LIP neurons outside the traditional sensorimotor pathways.

Having discovered that the baseline activity in LIP predicted the probability of the monkey's success on a difficult task, as well as its memory of prior reward, we postulated that this nontuned signal resulted from an ascending modulatory system. Our first candidate was the cholinergic system. We decided to see whether iontophoresis of acetylcholine affects the neuronal responses and behavior. Mingsha and Xiaolan got iontophoresis working. To do so, Xiaolan puts a tungsten electrode down the center of a seven-barrel micropipette and fills the surrounding barrels with various chemicals. She has learned not to let me within six feet of a filled pipette. I feel uncomfortable having something done in my lab that I can't do. We are finding that acetylcholine increases the baseline and visual transient responses. Mecamylamine, a nicotinic antagonist, and scopolamine, a muscarinic antagonist, decrease both signals. Iontophoresing acetylcholine actually increases the monkey's performance on the difficult task, but mecamylamine and scopolamine do not (yet).

Oculomotor Proprioception

Although the visual responsiveness of neurons in LIP and many other parts of the brain is modulated by the position of the eye in the orbit, no one had

demonstrated the source of that modulation. Andersen and Mountcastle had postulated that it came from a corollary discharge (Andersen and Mountcastle 1983), but no one had demonstrated a cortical eye position signal that preceded the fixation regardless of the saccade made to the location. I wondered whether the signal came from oculomotor proprioception. For years, I suggested to a number of now-successful postdoc candidates that maybe they would like to work on oculomotor proprioception in somatosensory cortex, and they all took postdocs elsewhere. Finally, Xiaolan Wang agreed to look for it. She was working in Charlie Bruce's lab at Yale, but Charlie lost his funding, and she had to go elsewhere. Her husband was working in my lab, and the mere fact that the proposed project was insane was not a barrier. Xiaolan found the oculomotor proprioceptive area, deep in the central sulcus, in Brodman's Area 3a, where the projection from skeletal muscle spindles lies. The cells had monotonically increasing responses with eccentric eye position starting around the center of gaze. The cells had a phasic and then a tonic response after a saccade. Cells in a given hemisphere were tuned for all directions in the orbit, not merely the contralateral direction. Activity came from the contralateral orbit: If we did a retrobulbar block, the activity disappeared, and it came back when the monkey recovered from the block. Because the other eye continued to move, we assumed that all of the corollary discharges were intact, and therefore this was a proprioceptive signal (Wang et al. 2007).

Yixing Xu (now assistant professor of ophthalmology at the University of Southern California) joined the lab as an MD-PhD student. He had bachelor's degree and a master of science in biomedical engineering from Yale. Yixing was a wonderful violinist. He could have been a world-class concert violinist but decided to go into academic medicine instead. While he was in my lab, he was also the concertmaster of the Camerata Orchestra, a semipro orchestra in New York. After years of frustration, he decided that Yixing was too difficult a name for Anglophones to pronounce, and so he added the first name "Benjamin." Ben, Xiaolan, and Chris Peck, a graduate student rotator, found that the proprioceptive signal lagged the eye by an average of 60 ms during both smooth pursuit and the VOR. Activity in pursuit and the VOR correlated perfectly, showing that there was no visual input to the signal: The retinal image was stable in the VOR, even though the eyes moved in the head but changed with the pursuit movement (Xu et al. 2011). Carine Karachi, a fully trained neurosurgeon from the Salpetrière in Paris, joined Ben.

The eye position signal was slow to develop, but was it too slow to provide a spatially accurate signal in the double-step task? To answer this question, Ben and Carine asked what happened to eye position modulation after the monkey made a saccade. They trained monkeys to make memory-guided delayed saccades and double-step saccades soon after the monkeys made a first saccade, which we called the conditioning saccade. For 50 ms and 150 ms

after the conditioning saccade, two thirds of the neurons gave visual responses modulated by the position of the starting point of the conditioning saccade, and the other third gave visual responses unpredictable from the steady-state eye-position modulation of the neurons. At 250 ms after the conditioning saccade, the eye-position modulation became accurate. If the monkey solved the double-step saccade using gain fields, a double-step saccade performed 50 ms after a conditioning saccade should result in the second saccade being made to the spatial location of the receptive field before the monkey made the conditioning saccade (which is what the gain fields would calculate), rather than to the spatial location of the receptive field after the conditioning saccade (Xu et al. 2012). At 50 ms after a conditioning saccade, the monkeys made accurate double-step saccades even though the gain fields were wrong. The brain uses corollary discharge, and not gain fields, to solve the short-latency double-step task.

Linus Sun is a Columbia-trained neurologist who earned a PhD at MIT with Matt Wilson before he went to medical school. Linus came to my lab under a resident research R25 grant. He then won a K08 mentored award to work on gain fields. Steve Lomber came down from Canada to put a cooling probe in the oculomotor proprioceptive part of Area 3a. Linus found that cooling the proprioceptive region of Area 3a reduces or eliminates the gain fields. With luck, that story will be published before this memoir. Mary Hayhoe had discovered incontrovertible evidence for the presence of a craniotopic representation: If a human makes a number of saccades and then makes a memory-guided saccade, the error of the memory-guided saccade does not increase as much with the number of saccades as one would expect from multiple remappings. Martina Poletti (Poletti et al. 2013) showed that spatially accurate memory can be modeled as a remapping process dominating early but as a craniotopic process dominating after a number of saccades. We hypothesized that the latter craniotopic process requires gain fields to establish the longer-term memory. Linus and my technician, Zikang Zhang (Mingsha's and Xiaolan's son) trained a monkey to make a memory-guided saccade after a number of intervening visually guided saccades. The monkey had no trouble making the memory-guided saccade after the first few saccades, but failed after five or nine saccades when the target was in the contralateral but not the ipsilateral field. Zikang presented a poster on this at the recent Gordon Conference on Eye Movements. The idea that long-term visuospatial memory is dependent on oculomotor proprioception makes people giggle when I tell them about it.

Further evidence for craniotopic processing in the parietal cortex comes from studying environmental memory in LIP. Mulugeta Semework Abebe came to my lab after a PhD in brain-machine interface studies at SUNY Downstate. He came to America from Ethiopia and studied in Israel and Georgia. Before she left the lab, Sarah Steenrod had discovered intertrial memory in LIP, and in one monkey, she showed that she could evoke that

memory by stimulating a spatial location without ever stimulating the cell's receptive field. This suggested that LIP has access to a craniotopic memory, although we could not find an explicit representation of it. Mulugeta finished the study on second monkey, and we have submitted the manuscript. The question then arose what was the source of this craniotopic memory? We postulated that it might come from the medial temporal lobe. The parahippocampal gyrus projects to LIP. Mulugeta is now exploring the medial temporal lobe. He has found cells exhibiting spatial memory, and an interesting class of cells responding to visual stimuli all over both hemifields except for a small ring around the fovea.

Sharpening the Priority Map

Suresh Krishna (now at the Cognitive Neuroscience Laboratory at the German Primate Center in Goettingen, Germany) came to my lab from Mal Semple's lab at NYU, and Annegret Falkner (now assistant professor of neuroscience at Princeton), a graduate student who had been an undergraduate at Oberlin, decided to work on surround effects in LIP. They first discovered that planning a saccade to the surround of an LIP neuron decreases the response of that neuron to a task-irrelevant object in the center of its receptive field (Falkner 2010). They then showed that planning a saccade to the surround decreases the variability of the response to a task-irrelevant stimulus in the center, in a manner independent from the response-lowering effect (Falkner et al. 2013). Both surround suppression and decreasing variability sharpen the priority map. Annegret then trained monkeys on a Sugrue-Newsome foraging task (Sugrue et al. 2004). She recorded simultaneously from neurons that did not share excitatory centers. She placed a saccade target in the center of each receptive field. In this task, the monkeys choose their saccade goals at rates that match the probability of reward. She found that during the fixation period, the activity in the two neurons was highly correlated. After the targets appeared and the monkey was deciding which saccade target to choose, the correlation fell precipitously and even became negative, which is what should happen if surround suppression were important in shaping the priority map. The correlation during the fixation period was sensitive to the monkey's recent history of reward. This provided further evidence for an untuned, reward-sensitive neural signal that resembled the baseline signal that Mingsha had found. Annegret still owes me a manuscript.

Cognitive Processing in the Cerebellum

Anna was working on a project to understand the relationship between LIP and V4 in visual search. Unbeknownst to her, she made an electrode hemorrhage in V4 and kept driving the electrode until she found cells. The cells

responded with hand movement. She was intrigued. We figured out, after an MRI, that the electrode was probably in Crus II of the cerebellar hemisphere. She wanted to study it, but I was dubious: We had no way of measuring hand movements or separating simple from complex spikes. I did not know much about the cerebellum. There is a large literature on the possibility that the cerebellum might be involved in cognitive, as opposed to motor processes, but no monkey physiology. We devised a simple, cognitive task. The monkey was already trained on the search task, in which it had to make a right- or left-hand movement depending on the shape of a symbol. We decided to see whether the monkey could learn to assign the right hand to one new symbol and the left hand to another while we were holding a cell. We found that the monkey could learn the new association within 80 trials. The exciting thing was that the simple spikes of the cerebellar Purkinje cells tracked the learning.

Naveen Sendhilnathan, a student of Adi Murthy's in Bangalore, joined the lab as an observer and turned to be a superb data analyst and physiologist. He asked to work a year on the cerebellum project as a technician, and then he applied and was accepted to Columbia (and Harvard) as a graduate student. He has officially joined my lab. Some cells gave a greater response when the prior trial was correct; others gave a greater response when the prior trial was wrong. Most cells did so only during a small epoch of the trial. The epochs in which there was a difference in the response after prior reward or failure, however, differed from cell to cell, so the population tiled the entire trial from fixation to after the movement. There was no difference in the kinematics of the movements when the task changed from overlearned to new symbols, although the cerebellar activity changed. There was no change in the activity when the movements changed but the symbol assignment did not. We presented this story at meetings of the SfN and the Neural Control of Movement Society—I hope this too is published before this memoir appears. Anna decided to leave the lab because of family issues, but Naveen is continuing the project.

Columbia Neurology

I hadn't dreamed what pleasure Columbia Neurology would provide. The department is one of the best neurology departments in the country, and it attracts spectacular residents. I started working in the Thursday outpatient resident clinic. I had not done outpatient neurology since my residency and was a bit insecure. Kirk Roberts, a great clinician, shared the Thursday clinic with me. Kirk taught me as much outpatient neurology as he taught the residents, and he tolerated my occasional bouncing around the world. After a few years, I started attending on the neurology ward and consult services in the Columbia campus of the New York Presbyterian Hospital. It is immense fun. The neurology is fascinating, much more so than in the outpatient clinic, and I love the residents. They have both a thirst for

learning and an esprit de corps that makes this incredibly arduous residency worthwhile. The department doesn't have a real neuroophthalmologist, so I teach them a lot about eye movements and vision. In 2006, the residents voted to give me the Lewis P. Rowland Award for outstanding teaching. They still like to round with me.

The Society for Neuroscience

In 1971, Peter Strick came from Ed Evarts's lab with membership forms and asked Bob and me to join the new SfN. I am member number 1,768. All three of the people who made me a neuroscientist, Bob, Torsten Wiesel, and David Hubel, were active in the Society and ultimately were elected president. They taught me to do service to the community by example. My first foray into working for the Society was purely out of self-interest. Physicians need a certain amount of certified continuing medical education (CME) credits to maintain their licenses and hospital appointments, and I found it difficult to accumulate the hours. I asked Torsten if the Society would be interested in offering CME to its members at the Annual Meeting. He suggested that I organize it. The first year, we did this as subcontractors to Georgetown University, and then I decided that the SfN could be a primary CME provider. I filled out the Accreditation Council for CME forms and completed the interview, and SfN became a CME provider. I became a member of the Education Committee. I still get my CME from the SfN.

At that time, abstracts were submitted on paper and had to be perfect and camera-ready. The society staff would then Xerox the abstracts for the program committee and ship them out via overnight delivery. When I was a member of the Program Committee, I sorted my 500 abstracts into poster and platform sessions by making 50 piles on the livingroom floor. Harvey Karten and I brought up the idea of having electronic submission of abstracts and making a CD. The SfN Council thought it would be a good idea and appointed me chair of the Ad-hoc Committee on Electronic Initiatives. We asked several companies to submit bids to provide an electronic interface for the Society that would include abstract submission, abstract sorting, a CD that included all of the abstracts, and an itinerary planner that would help you chart your way through the maze of posters and minimize conflicts with platform sessions. The first year, 1999, electronic submission was voluntary. About half of the members submitted abstracts on paper. I was worried that everyone would submit their abstracts electronically in the last half-hour, and I was right. The computer system crashed. The next year, more people submitted electronically, and ultimately electronic submission and the abstract volume became the norm. I was elected treasurer of the Society in 2004, and president-elect in 2008.

Advocacy is a major activity for the Society. After the crash of 2008, there was outpouring of letters, e-mails, and phone calls from members of Society and other scientific organizations in support of the Spector amendment to

increase the NIH budget by $10 billion as a part of Obama's stimulus package. The amendment passed. I testified in front of the House of Representative Committee that oversaw the NIH, emphasizing that not only will funding for science result in our understanding how the brain works and curing brain disease, but also that NIH funds return twice their amount to the economy. I described myself as the steward of a small business dependent on the government, supporting many people who weren't themselves scientists. "John the Machinist" became a watchword on Capitol Hill. I then realized that the many members of the SfN who lived and worked in other countries were supporting our advocacy. Working with Sten Grillner and Helmut Kettenmann, the presidents of the Federation of European Neuroscientists, we spread the idea of advocacy abroad and provided the funds to support it.

My other major issue was animal research. In 1977, Alex Pacheco, the founder of People for the Ethical Treatment of Animals, volunteered to work in Ed Taub's lab. While Ed was on vacation, Pacheco took pictures of the lab in disarray that he had created and of monkeys seemingly in pain, although in fact they had been sensorially denervated and couldn't feel pain. The story hit the front page of the *Washington Post*, created a huge fuss, and Ed lost his grants without due process. It was apparent that animal research was vulnerable. The SfN at that time provided no support for beleaguered scientists, and usually their institutions did not help at all. Animal researchers and their families were physically threatened. We worked to make institutions more supportive and to provide statements on the importance of animal research that the universities could use immediately to support their attacked scientists without waiting for extensive exonerating investigations. The SfN website has instructions about what to do if you are the subject of animal activist harassments. We are not all the way there, but universities have become more supportive and prepared. I became chair of the Committee on Animal Research after my term as president. There will always be antivivisectionists, and the scientific community must be ever vigilant in the struggle against them. Our job is to convince the great mass of undecided people that not only is animal research likely to provide the answers that will treat diseases like schizophrenia and Alzheimer's disease, but also that basic research seemingly unrelated to clinical problems ultimately will be related to solving those problems.

Coda

Neuroscience has given me a wonderful life. Many of my medical school classmates are retired—I am having so much fun that I still write grants and work in the lab. The stuff in the lab is currently as exciting as it has ever been. There is nothing like the thrill of discovery, understanding something that no one else has ever understood, or proving a hypothesis or stumbling upon something that you never dreamed of. It is worth the trench warfare of writing grants and struggling with journal reviewers. In my own small way, I have felt what Archimedes must have felt when he realized the implication of the wet floor.

But modern science is not one man in a bathtub. I have been lucky to have had a great mentor and a fabulous bunch of collaborators, students, and postdocs who made my life better. They have been my second family. Fred Plum, the great neurologist, once said in a talk, "When I say 'I,' I mean 'we.' When I say, 'we,' I mean 'they.'" That is certainly true for me. I am intensely grateful.

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