

LIKE A RIVER

Barbara Kinder

*Rivers hardly ever run in a straight line.
Rivers are willing to take ten thousand meanders
and enjoy every one
and grow from every one.
When they leave a meander,
they are always more
than when they entered it.*
— from “Rivers” by James Dillet Freeman

The opportunity to think about our lives and try to explain them to others (and ourselves!) has proven to be quite meaningful. Also, I was given pause by both the “intellectual” and “trajectory” descriptors in the assignment. My personal definition of the former would perhaps be “challenging and gratifying” and, for the latter, that my course looking backward, rather than a trajectory, has more the sense of a river’s course: moving and changing direction, finding meanders more, in my case, due to opportunities than obstructions, and in retrospect ending up in just the right place.

I was born in Washington DC in December 1944, where my dad, a lieutenant in the Navy, was stationed before being deployed to an aircraft carrier in the South Pacific. I grew up in Cleveland, Ohio in a close family with aunts, uncles, and cousins nearby and a wonderful brother and sister. My dad was a lawyer and my mom a community activist and science enthusiast. She had wanted to be a nurse but her father, a doctor, discouraged her, feeling women shouldn’t work. I am sure my interest in science came from her. She got me an AC Gilbert chemistry set and took me on bird walks (I still have her copy of Peterson’s field guide with her life list) and visits to the Museum of Natural History. When I was thirteen I told my grandfather that I wanted to be a doctor, thinking he would be pleased. His response was “anything but a hen medic” (whatever that was). I chose to go to a women’s college, feeling that my interest in

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science and medicine would be more supported there. Also I loved athletics, had been a figure skater and tennis player, and before Title IX there were few collegiate opportunities for women in sports except in women's colleges. I went to Smith College in Northampton, Massachusetts. It was a perfect choice for me. A new major, biochemistry, had been created by Professor George de Villafranca, a wonderful teacher, and eleven of us in the Class of 1967 chose this major.

During the summer, I worked in a neurochemistry lab led by Howard Sachs at Case Western Reserve. One of my tasks was to do the bioassay for vasopressin, a hormone secreted by the brain which regulates salt and water balance. This involved inserting a canula, a small tube, into a rat's carotid artery (an artery in the neck that supplies the blood to the brain) to measure changes in blood pressure which correlated with vasopressin levels in the animal's blood stream under different experimental conditions. This was my first inkling that I had some manual dexterity. Subsequently under his guidance, I completed a study on vasopressin biosynthesis in the rat brain for my honors thesis at Smith. Howard was my first real professional mentor, teaching me not only about the scientific process but also awakening my sense of social responsibility. I'd like to think the influence went both ways as Howard eventually left research and went to medical school and into primary care medicine!

Pursuing my interest in medicine, I applied to medical school and chose Yale because there was a thesis requirement but no exams, and a family friend, Barry McAllister, was a professor of pathology here and thought it would be a good fit. As I was to find out over the next several years, he was right. There were eight women in a class of one hundred at that time. I entered medical school planning to become a psychiatrist and maybe do research in neurochemistry. The one thing I knew I didn't want to do was surgery, so I left my surgical clerkship for last. I did my clerkship at our VA Hospital (where I later became chief of surgical service) on the Plastic Surgery service. One day I was scrubbed in on a case of a head and neck tumor, quite advanced. The surgeon was Tom Krizek, an amazing and charismatic surgeon. As the hours wore on, I witnessed the removal of a horrible, fungating tumor and the reconstruction of a man's face. Tom asked how long I had been interested in surgery, and I said I really wasn't. He asked why, and I replied that I didn't think it was a good field for a woman. He again asked, "Why?"

I didn't have an answer, plus I had been bitten by the bug after seeing this miraculous procedure. So everything changed after that. Although no women had completed the surgical residency at Yale at that time, Tom and other surgeons at Yale were supportive of my plan and of course that made all the difference.

I made an appointment to meet with Phil Bondy, then chair of the Department of Medicine and someone of whom I was very fond, to tell him that I was applying for a surgical rather than a medical internship. I knew he would be disappointed and indeed, his response was "Barbara, you can teach a chimpanzee to operate!" Phil was right about a lot of things (and possibly that too) but I think he did not understand

much about surgery. It is true that the performance of a surgical operation requires a lot of training just to do the technical maneuvers (even these days with laparoscopic and robotic procedures), but the decision of whether or when to do surgery and what specifically needs to be done requires judgment and experience and an appreciation of the variations in anatomic and clinical presentations that one may confront and which may require changes in the planned approach. Much of this is true of all areas of medicine of course, but the decision process in surgery must take place not only in real time but also often urgently. To some extent the thinking process becomes almost associative in nature; methods used for other circumstances may find utility in a novel situation. One experience I had illustrates this. As a young attending surgeon on trauma call (in those days there were no subspecialties in surgery like trauma), I was called to the operating room one night for a stab wound to the chest. The cardiothoracic surgeons had stabilized the patient and opened the chest but discovered that the bleeding was coming from below the diaphragm. Because of the curvature of the diaphragm, a wound below the nipples can enter either the chest or abdomen. It turned out that the knife had lacerated the veins draining the liver in the abdomen just before they pass through the diaphragm to enter the superior vena cava and the heart. This is a very difficult area to reach and massive hemorrhage continued despite several maneuvers to control the bleeding. It was a desperate situation. Suddenly, and I don't know why, I asked for a pediatric foley catheter. A foley catheter is a rubber tube that goes into the bladder to empty it of urine. An inflatable balloon holds it in place. I threaded the catheter into the bleeding vessel, inflated the balloon and the bleeding stopped. It was then a simple issue to sew up the vein and finish closing the abdomen. I had used foley catheters but had never seen one used for bleeding control. But some associative thought process brought this solution to mind and just in time! The patient, a Yale graduate student, survived, did well, married, and had children; and I get a Christmas card from him every year.

To return to the main stream of my trajectory, years before I got to this point, I had to find a surgical residency. I interviewed at various places and there was a sense at that time that some institutions were moving in the direction of including women in their programs, probably because other specialties like pediatrics and medicine had done so, and I got a lot of encouragement. But I decided to stay at Yale where I felt I had an identity and would not be regarded so much as "The Woman." This was a good decision for me, given the challenges of a surgical residency. Another mentor at this time was Bill Collins, chief of neurosurgery and later department chair. Interestingly he had been at Case Western while I was living in Cleveland, and I was assigned to him as a partner when he was learning ice dancing, one of those odd coincidences in life. He was a strong proponent of women in surgery and had trained Joan Venes, the first woman in the neurosurgery program and later a faculty member—the only woman faculty member in any of the surgical specialties until I joined the faculty after residency. In my residency year, Mary Alice Helikson (now a retired pediatric surgeon) and I were

the first women to make it through the “16-4 pyramid program,” meaning sixteen interns start the program but the cohort is cut to four for the final years. Between years two and three, I secured a fellowship in pharmacology in the lab of Malcolm Mitchell, another supportive mentor, and worked on tumor immunology. I was trying to figure out a way to engage in basic research as a surgeon, and in those days there were no programmatic physician-scientist tracks in surgery. I was told if I took a year off there was no guarantee a position would be available for me coming back, but I took my chances and did get back in. As residency drew to a close, I was offered an opportunity to go back to Haiti, to the Hopital Albert Schweitzer, where our residency had an amazing rotation and where I had done two tours, to replace the American-trained surgeon who was returning to the States. This was *the* major decision point for me. I loved Haiti and the diversity of the practice, but in the end, the lure of an academic practice won out, and I elected to stay on at Yale as an assistant professor.

At this time, Howard Rasmussen was recruited to Yale from Penn to head the section of endocrinology. Howard had purified parathyroid hormone which permitted identification of its structure and understanding how it functioned in the body. At Yale, he developed a focus on calcium metabolism. And in a life-changing opportunity for me, he brought with him lots of patients with hyperparathyroidism, a disease in which the parathyroids enlarge and make too much hormone and cause the blood calcium level to rise. The treatment of this condition was, and is still, primarily surgical in nature. This began a major intellectual meander in my life: all things calcium!

In a general surgical practice, endocrine surgical procedures, that is, operations on hormone-producing tissues such as the thyroid, parathyroid, adrenals, and endocrine pancreas, are relatively uncommon compared with appendix, gall bladder, breast, colon, etc. procedures. My residency experience reflected this. Coming out of residency in 1976 I had done few thyroid procedures, and in the one parathyroid case I had scrubbed in on as a resident, we in fact had not found the offending overactive gland. Nonetheless, Howard decided I was to be his endocrine surgeon. He (I think he secretly wanted to be a surgeon) would come up to the OR and observe and make suggestions, and Virginia LiVolsi (a great pathologist now at Penn) would cheerfully examine tissues I removed to determine their identity. These colleagues helped shape my development as an endocrine surgeon. At the American College of Surgeons annual meeting in 1979, there was an announcement that a few surgeons were getting together to talk about starting a new organization focusing on endocrine surgery. I went to the meeting (the only woman in the room as I recall) and screwing up my courage, introduced myself to Sam Wells, an eminent surgeon at Duke. He welcomed me into the group, now identified as the American Association of Endocrine Surgeons (AAES) and was a consistent supporter of mine over the years.

My membership in the AAES was a tremendous help. Colleagues in the organization served generously as long-distance mentors, giving me advice on cases I encountered. I enjoyed serving on various committees, hosted a wonderful meeting of the

group in New Haven, complete with formal dinner at the Peabody Museum, and in 2001, I was given the great honor of being elected president, the first woman so honored, but not the last! And this past year the AAES named the annual resident basic science prize in my name.

What are the Parathyroid Glands?

152

The first description of the parathyroid gland we owe to Sir Richard Owen (1804–1892), Hunterian Professor and Conservator of the Museum in the Royal College of Surgeons of England. In 1834 the Zoological Society of London purchased its first Indian rhinoceros (*Rhinoceros unicornis*). When the animal died on the evening of November 19, 1849, its carcass was offered to Sir Richard. His dissection of this two-ton animal took at least a year during which time decomposition continued apace. In his detailed description of the anatomy, Owen refers to “a small compact yellow glandular body attached to the thyroid” – a structure we now know as the parathyroid gland. The original preparation in which Owen made the observation can still be seen in the Hunterian Museum at the College. I have had the privilege of seeing it there, fortunately by this time well preserved!

Though first identified in terrestrial vertebrates presumably to regulate calcium balance in nonmarine environments, tissues producing parathyroid hormone (PTH) and related molecules have been found in many species, including fish, where they arise in relation to gill development, and insects – except Diptera (flies, mosquitoes) and Lepidoptera (butterflies and moths) – and appear to relate to the development of elements of the exoskeleton, the external skeleton conferring structure in these species. Though the study of human anatomy and physiology of the parathyroids proceeded slowly, clinically the symptoms of parathyroid disease, particularly underactivity of the glands, were known quite early. Low blood calcium, hypocalcemia, causes neuromuscular hyperexcitability (tetany) similar to the effects of tetanus neurotoxin resulting in widespread muscle spasms (including those of the larynx), seizures, and cardiac arrhythmias. When it occurs acutely, it is a medical emergency. On the other hand, the symptoms of hypercalcemia range from mild to severe depending on the level of calcium. They may include increased thirst and urination, abdominal pain, nausea, bone pain due to bone loss, muscle weakness, mental status changes, and fatigue. Hypercalcemia is usually caused by overactivity of one or more of the parathyroid glands. Reflecting the critical role of calcium in homeostasis (physiologic balance in an organism), like hypocalcemia, hypercalcemia can also be a medical emergency

Besides its Structural Use in Bone, Why is Calcium so Important?

Calcium, the third most abundant metal in nature, was amply available to cells from the beginning of life on earth. Calcium, because of several of its chemical features, can bind reversibly to a variety of cellular organic molecules, influencing their function. Many of these also contained phosphates (phosphorus and oxygen compounds)

and were used in energy transfer and genetic materials like nucleic acids such as DNA, etc. Calcium phosphate salts have limited solubility in water, so intracellular calcium concentration had to be carefully controlled. Nature evolved membranes largely impermeable to calcium, pumps to remove it, calcium-binding proteins, and intracellular calcium banks which maintain the intracellular calcium concentration at a level 10,000 times lower than outside the cell. This steep gradient sets the stage for calcium to serve as a messenger that can influence intracellular processes. A controlled increase in intracellular calcium through regulated cell membrane channels or by release from intracellular stores acts as a second messenger affecting virtually all cellular functions. You could think of calcium as providing the same kinds of information and energy transfer in living tissues that electrons perform in electronic circuits. However, calcium is an ambivalent messenger. Although essential to the correct functioning of cell processes, if not carefully controlled spatially and temporally within cells, calcium causes severe cell dysfunction, and even cell death. Having decided to use calcium as a cellular switch, cells have effectively chosen to live in a permanent state of controlled risk. In charge of controlling the risk at the organismal level are the parathyroid glands

How Do the Parathyroid Glands Control Calcium?

In response to the detection of a decreased extracellular calcium level, parathyroid hormone (PTH) is released from the gland. The detector of extracellular calcium levels is a cell-surface protein receptor known as the calcium sensor. The parathyroid calcium sensor was identified in 1993 by Edward M. Brown and colleagues at Harvard in calf parathyroid glands and has been found to be highly expressed in tissues involved in calcium homeostasis or balance, such as parathyroid, kidneys, and bone. Once secreted, PTH exerts three distinct effects on calcium homeostasis: to enhance bone resorption, liberating calcium from the skeletal “bone bank” where 98 percent of body calcium is stored; increase calcium reabsorption from the urine; and increase the synthesis in the kidney of the active form of vitamin D₃, which promotes intestinal calcium absorption. As a result serum calcium returns to the normal range. Further PTH release is inhibited by interaction of this extracellular calcium with the calcium sensor and subsequently other intracellular events, still not fully understood, but which eventually became the focus of my research efforts.

What Causes Problems in the PTH Regulatory System?

The most common syndrome related to parathyroid dysfunction in humans is primary hyperparathyroidism (PHPT), that is, continued parathyroid hormone release even when the serum calcium is already high.

PHPT occurs across all age groups and genders but disproportionately affects women and is increasingly prevalent with age. PHPT is nearly universally caused by tumors of the parathyroid glands, which are classified as solitary adenoma (85 percent), multi-gland disease (15 percent), or carcinoma (less than 1 percent). PHPT is mostly

sporadic, that is, not genetically inherited. A variety of spontaneous mutations have been identified in human parathyroid tumors but interestingly never in the calcium sensor itself.

The clinical diagnosis of hyperparathyroidism is straightforward. A simple blood test reveals PTH levels inappropriately high for the simultaneous serum calcium level, and surgical removal of the abnormal tissue is curative.

154

Typically one of the four parathyroid glands is enlarged and overactive. It is then a (relatively) simple matter to remove that gland, and the calcium returns to normal. In the old days before imaging studies were developed that could identify the parathyroid glands, this meant doing a neck operation under general anesthesia and identifying the parathyroids to see which one or ones were abnormally large. Confounding circumstances can arise. In the first case, unhappily, sometimes the glands are not where they are supposed to be. Here an understanding of embryology can help. Developmentally, the parathyroid glands arise from embryologic elements of the developing pharynx or upper gastrointestinal tract in the fetus. The superior glands arise from one area and inferior glands from another in common with the thymus, important in the development of the immune system. Thus if one is unable to find a superior gland, it may have not fully descended in development and should be sought higher in the neck. A missing inferior gland would most likely have continued to travel with the thymus anteriorly into the chest and can usually be pulled upward from the neck incision.

As time went on, efforts were made to determine preoperatively where the overactive gland might be in the neck in order to minimize the extent of the surgical dissection. A variety of imaging techniques were developed including CT scanning, MRI, ultrasound and nuclear scanning. Nuclear scanning with technetium 99m sestamibi is now commonly used to identify abnormal parathyroids to permit a minimally invasive approach with local anesthesia. This radionuclide was originally developed as a heart imaging agent but serendipitously was found to visualize abnormal parathyroid glands.

Most of the imaging techniques have found their utility in reoperative cases, where the gland was not identified at the initial surgery. Reoperative neck surgery is no fun and knowing exactly where to go is critical to avoid complications including damage to the recurrent laryngeal nerve which innervates the vocal apparatus and which, if damaged on both sides, would require a tracheostomy for breathing. Also remaining normal parathyroids, difficult to see in the midst of post-surgical scarring, can be damaged resulting in hypocalcemia, a major problem. A second confounding issue is that although finding an enlarged gland would likely mean it was the overactive one, it does not follow that it is the only culprit. A friend, George Irwin, at the University of Miami, operated on his OR nurse and having removed one enlarged gland, found postoperatively that the hypercalcemia had not abated. He was so horrified that he developed a technique of measuring PTH quickly in the OR. PTH has a half-life of five minutes so that a blood sample taken in that time frame should reflect a corrected level of PTH. Working with our colleagues in clinical chemistry we adapted

this procedure at Yale, allowing us to assure the patient at the end of the procedure that we had solved the problem.

By 1989, Bill Collins had become chair of the Department of Surgery, and he asked me to become the chief of surgery at the West Haven VA. I was pretty sure I was not interested in administrative advancement or eventually becoming chair of an academic department. However, as a good soldier (military metaphors are the rule in surgery), I agreed and negotiated for lab space and a “7/8ths” assignment so that I could continue my clinical practice at Yale but hopefully pursue research efforts on parathyroid function. I was particularly interested in understanding how calcium regulates PTH secretion and whether the dysfunction seen in parathyroid disease is due to abnormalities in the secretory process or simply to the increased mass of secreting cells (spoiler alert: it’s likely both and is still not fully understood). In virtually every secretory system known, calcium as a second messenger stimulates secretion, but in parathyroid cells and in the renin-secreting cells of the kidney, it inhibits secretion. Fred Gorelick, who had been a GI fellow while I was a surgical resident, was working with Jim Jamieson in cell biology on calcium regulation of pancreatic secretion. They took me in scientifically and helped me get started. Lisa Matovcik, a recent PhD in cell biology, and I undertook to dissect out intracellular mechanisms involved in this reverse regulatory situation. For our experimental model we used a well-characterized (by the Brown lab at Harvard) system of isolated dispersed parathyroid cells which had been shown to exhibit the inverse regulation of PTH secretion by calcium seen in humans. We obtained tissue from calf parathyroid glands at a local abattoir and processed them for same-day experiments.

155

In general, secretory products are released from cells (exocytosis) in two ways: continuously, a process that is unregulated and called constitutive secretion, or in response to a specific trigger, a regulated process. The process itself has many steps from the synthesis of the product, to its packaging in membrane-bound granules, to its transport to the cell membrane and its ultimate release in response to the appropriate signal. Each of these key steps consists of multiple molecular interactions, many of which are regulated by phosphorylation, the transfer of a phosphate group to a protein. The addition of the phosphate group is catalyzed by an enzyme called a protein kinase and results in changes in the activity of the protein and its ability to interact with other proteins in the cell. This is a reversible process, and the removal of the phosphate group is carried out by another enzyme, a phosphatase which then returns the function of the protein to its baseline.

Of interest to us was that in other secretory systems phosphatases, particularly protein phosphatase 1 (PP-1), appeared to be involved in several key aspects of the life cycle of secretory granules. As we have seen, in the parathyroid, an acute drop in extracellular calcium is transduced by the calcium sensor and immediately stimulates release of preformed hormone waiting at the cell membrane in secretory granules. Continued release of hormone requires new synthesis and transport to the cell

membrane. Inhibition of hormone secretion could occur by interfering with any of the stages mentioned above. In intact cells, individual steps of the pathways that lead to exocytosis can be difficult to resolve. The bacterial toxin streptolysin-O causes the formation of stable pores in the plasma membrane, permitting manipulation of the intracellular environment and facilitating the analysis of cellular signal transduction mechanisms. We showed that bovine parathyroid cells permeabilized with SLO exhibited relatively normal characteristics on electron microscopic evaluation and retained typical functions including the ability to undergo inverse, calcium-dependent PTH secretion. This suggested that this system could be helpful in defining control mechanisms in PTH secretion. We identified parathyroid PP-1 and demonstrated that it is concentrated at the cell periphery in the vicinity of the plasma membrane, consistent with a role in the modulation of PTH secretion, and remains in place in permeabilized cells. Inhibition of PP-1 by adding a specific inhibitor to both dispersed and permeabilized cells resulted in blocking low but not high calcium stimulated PTH release, suggesting that the role of PP-1 is on regulated secretory pathways, perhaps involving transport processes. The research life, framing questions, investigating and learning new techniques, collaborating with colleagues, devising and conducting experiments, and analyzing and interpreting data, was deeply meaningful to me. The scientific process has informed not only my clinical practice but also the way I see the world. But I found myself torn between the conflicting demands of the lab and the clinic. I think it is very hard to be both an active clinician, especially a surgeon, and a basic researcher. The work environment and support systems have to be structured intentionally to promote optimal productivity. Understanding the critical developmental needs of physician scientists has improved but is not fully realized even now in surgery.

Meanwhile, like a river, medicine is always changing. In the 1980s a major event occurred in surgical practice: the revolution of laparoscopic surgery.

For any major change or progress to take place, many factors must fall into place. In the case of laparoscopy, dramatic technical innovations were required. Equally important is a favorable and supportive philosophical environment.

Being able to see the inside of the body in circumstances other than surgery or autopsy had long been a goal of medical practitioners. Philipp Bozzini is credited with developing the first cystoscope in 1805, a hollow tube lighted by candles and mirrors to examine canine bladders. In the 1930s, with better lenses and light sources, gynecologists used laparoscopy diagnostically and for simple procedures like tubal ligations. The early slow pace of endoscopic and laparoscopic evolution was in large part related to the limitations of technology. It was further slowed by skepticism of the medical and surgical communities. During the mid-1960s and 70s, gynecologist Kurt Semm in Kiel, Germany, contributed greatly to laparoscopic technology. He perfected many technical refinements, including importantly the recognition that introducing a gas (now carbon dioxide) into the peritoneal cavity created a three-dimensional space in which to work. In 1983, he performed the first laparoscopic appendectomy, bringing him criticism and censure rather than accolades.

Clearly the ability to work through several small incisions rather than a large one, a minimally invasive approach, carried many benefits for patients.

Originally the surgeon would lean over and peer through the scope directly, resulting in back strain for the operating physician and poor visualization (and no visualization for the assistants) of the peritoneal structures due to use of monocular vision (one eye at a time) through a narrow aperture. Camran Nezhat, a gynecology resident in Buffalo, had the vision to couple a video camera to his laparoscopic eyepiece and work from a monitor. He worked through the 1970s and 80s to refine his technique to basically the way it is done today. In 1987, French surgeon Philippe Mouret performed the first videolaparoscopic cholecystectomy (gall bladder removal) and, like the other pioneers, was strongly criticized.

Laparoscopic cholecystectomy was introduced to the general surgery world in the exhibit hall of the American College of Surgeons annual meeting in October 1989. Although the evolution of laparoscopy took many decades, with no scientific evidence to support or justify the change from open to laparoscopic gallbladder surgery, utilization of this technique did, in fact, occur almost overnight. Marketing of the new approach was everywhere, patients were demanding the new surgery, and instrument companies were supporting courses and even paying tuitions for surgeons to be trained to use their products. In this instance, the drive to change treatment approaches came from private practice and the internet, not from the academy. Almost immediately though, fortunately, standards were instituted which required training and supervised experience before surgeons were credentialed to perform these procedures. Subsequent studies confirmed the superiority of the laparoscopic approach in terms of decreased postoperative pain, physiologic derangements, and recovery time. A major challenge remains in that the surgeon is operating in a three-dimensional space with a two-dimensional field of view and without the haptic (touch) information that is part of an open procedure.

In any event, like my colleagues, I had to relearn surgery. It was certainly humbling to find that my hard-won technical skills counted as nothing in the laparoscopic arena and that furthermore the students and residents who had grown up with video games were far more facile from the beginning than was I. Of course not all operations can be done laparoscopically, but adrenal and pancreatic surgery, part of the endocrine practice, definitely are.

The adrenal glands sit atop the kidneys and are about as far away from the anterior abdominal wall as you can get; ditto the pancreas, which lies against the spine. Operating on these organs from either the front or the back requires large incisions and cutting of various muscles, which increases postoperative pain and disability and a whole range of physiologic effects which take time to return to normal. The laparoscopic approach is perfect for these organs. It bears mentioning, though, that although laparoscopy and robotics are a major addition to clinical care and surgical practice, there is now a training deficiency in open surgery, such that many residents don't know how to do procedures open. Many procedures in the abdomen, such as

reoperative surgery; cancers, especially in advanced stages; and trauma may require facility with an open approach. Perhaps some of us may not be so obsolete after all!

158

As gravity powers the flow of a river, a commitment to patient care, a sense of it as a calling, has powered my trajectory. From the first meeting through the last followup visit, the establishment and maintenance of a therapeutic relationship, I have understood as fundamental to an excellent outcome. An abundance of neuropsychiatric data has shown that patients who are optimistic about their outcomes do better than those who are not. Many studies confirm an “optimism bias” in humans and have begun to illuminate how that translates into lived experience. The surgeon has a powerful tool in the therapeutic relationship to bring the optimism bias into play. It is important to note that the relationship is truly reciprocal, benefiting surgeon as well as patient!

For internal medical residents and students, the model of the complete physician has been Tom Duffy, as the mantra goes, “what would Duffy do?” For us in surgery, the exemplar of passionate commitment to patients and superb technical skills was my late husband, Elton Cahow. In addition to providing me a professional model, he also gave me our extraordinary daughter, Caitlin. We miss him every day.

Viewed from my current destination (the waters of the Atlantic Ocean in Penobscot Bay, Maine and the Florida Keys), my river trajectory conveys the sense of inevitability, but many people and opportunities conspired to get me here. I am most grateful to have found a stimulating career, the trust of many wonderful patients, shared learning with remarkable students and residents, and the friendship and support of so many colleagues in and outside of medicine.