CASE REPORT

THE GONZALEZ THERAPY AND CANCER: A COLLECTION OF CASE REPORTS

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Nicholas J. Gonzalez, MD, and Linda L. Isaacs, MD, run a private practice in New York City, where they research treatments for a variety of diseases, particularly advanced cancer, and see patients.

In our practice, we offer an aggressive nutritional program for treatment of advanced cancer and a variety of other serious illnesses, ranging from chronic fatigue to multiple sclerosis. Whatever the underlying problem, our therapy involves 3 basic components: individualized diets, individualized supplement protocols, and intensive detoxification. The diets we prescribe can range from vegetarian raw foods to an Atkins-type red meat approach. The supplement programs are equally as varied, involving vitamins, minerals, and trace elements in various forms and various doses, as well as glandular and enzyme products, each chosen to meet a particular need in each patient. The detoxification routines, often the most misunderstood component of our therapy, consist of coffee enemas and a variety of other techniques that actually have been adapted from the orthodox medical literature. We believe these procedures help the body neutralize and excrete the multitude of waste products produced during routine metabolism and, in the case of our cancer patients, resulting from tumor lysis.

We are perhaps best known for our work with advanced cancer. For patients suffering malignancy, we rely on large doses, spread throughout the day, of orally ingested pancreatic enzymes derived from a pig source. Though we believe the diets, vitamins, minerals, and trace elements help improve tissue and organ efficiency, in our therapy, it is the pancreatic enzymes that target and kill cancer cells.

HISTORY OF ENZYME TREATMENT

The enzyme treatment of cancer has a long history, beginning with the work of Dr John Beard, a professor at the University of Edinburgh who in 1902 first proposed that the pancreatic proteolytic enzyme trypsin might represent a powerful anti-cancer tool. Beard, an embryologist, detoured into cancer research as a result of his studies of the mammalian placenta and its similarity to malignant tumors.

Beard was the first to report that in many respects, the placenta in its early form behaves like a tumor. It begins growing as a very undifferentiated offshoot from the primitive embryo, then quickly invades the mother's uterus, much as a tumor infiltrates host tissue in any organ. Initially, the cells of the placenta proliferate almost without control, as tumors were known to do even in Beard's day, and it quite efficiently produces a dense blood supply—a requirement for any rapidly growing malignancy, as angiogenesis research today has made clear.

As normal development proceeds, however, at some predetermined point, the placenta transforms from a highly invasive, rapidly growing, blood vessel–producing, tumor-like tissue, to the non-invasive, non-proliferating mature organ. The only difference between the placenta and a malignant growth, Beard claimed, is that the placenta knows when to stop growing, and tumors don't.

Beard concluded that the key to the change lay in the embryonic pancreas. As witnessed in every species he studied, the day the placenta stops its cancer-like invasion of the mother is the very day the embryonic pancreas becomes active and begins pouring out enzymes.

Even in Beard's day, more than 100 years ago, the main categories of pancreatic enzymes had already been identified—the proteolytic, or protein-digesting component; the lipases, which hydrolyze triglycerides, and the amylases, responsible for cleaving complex carbohydrates into simple, easily usable sugars. Physiologists of the time thought all 3 groups were active only in the duodenum, where the enzymes continue the breakdown of food arriving from the stomach. But Beard effectively provided the data to illustrate that above and beyond this function, trypsin, the main proteolytic enzyme, served to control placental growth and prevent the tissue from invading beyond the uterus, as a true cancer might.

Beard proposed that because the early placenta behaves much as a tumor does, because under the microscope its cells even look like undifferentiated, primitive neoplastic cells, and because pancreatic enzymes forcefully regulate its growth and development, these very same enzymes could be—in fact, must be—the body's main defense against cancer and would be useful as a cancer *treatment*.

Beard first tested his thesis in the one animal tumor model available at the time, the Jensen's mouse sarcoma. He injected an extract of trypsin into mice growing such cancers, and the tumors regressed.[1] Subsequently, during the first decade of the 20th century, a number of physicians interested in Beard's hypothesis began, under his direction, to use injectable pancreatic enzymes to treat their human cancer patients. The successes were published in

the major medical journals of the day, including *JAMA*[2] and the *British Medical Journal*.[3]

The enzyme thesis, and the supporting animal and laboratory data, provoked an angry backlash against Beard and his followers. He was vilified in editorials in medical journals, mocked in the newspapers, and belittled at scientific conventions. But Beard fought back in articles and letters to the editor, and in 1911, he published *The Enzyme Treatment of Cancer*,[4] a monograph outlining his decades of research and its promising and compelling results. But interest in Beard's thesis gradually petered out, and when he died in 1924, he died frustrated, angry, and ignored, his therapy already considered no more than a historical oddity.

After Beard's death, other physicians and scientists discovered his work and kept the idea alive. During the 1920s and 1930s, a St Louis physician, Dr F. L. Morse, reported that he had successfully treated a number of advanced cancer patients with pancreatic enzymes. When he presented his findings to the St Louis Medical Society in 1934—a proceeding published in the *Weekly Bulletin of the St Louis Medical Society*[5]—his colleagues attacked him.

During the 1960s, the eccentric dentist Dr William Kelley rediscovered Beard's work and developed his own variation of enzyme treatment. In addition to large doses of orally ingested pancreatic enzymes, Kelley's program included individualized diets, supplement protocols, and detoxification routines. Kelley came to fame at a time of great repression organized against alternative medicine. He was at particular risk because as a dentist, he was not legally entitled to treat cancer in the first place. He was attacked in the press, vilified as a "quack," and investigated by numerous government agencies. He was thrown in jail as a public menace, had his dental license revoked for 5 years for practicing medicine, spent his earnings defending himself against government assaults, and saw his family life fall apart. But, like Beard, he never relented, and his successes created an extraordinary word-of-mouth network that brought an endless stream of patients to his practice.

DR GONZALEZ'S RESEARCH BEGINS

I (NJG) met Dr Kelley during the summer after my second year of medical school. I had as a mentor at Cornell Medical College the late Robert A. Good, MD, PhD, who encouraged a review of Kelley's cases. Dr Good, then President of the Sloan-Kettering Research Institute, was the most published author in the history of the biomedical scientists, the "father of immunology," as The *New York Times* described him, and the man who performed the first bone marrow transplant in history.

Under Dr Good's direction, I began a student project evaluating Dr Kelley's patients, his methods, and his successes and failures. I quickly found evidence of what appeared to be patient after patient with appropriately diagnosed, biopsy-proven advanced and sometimes terminal cancer, who were alive 5, even 10 years after first beginning enzyme therapy. What began as a mere student investigation evolved into a full-fledged research project, completed while I was a fellow in Dr Good's practice, which ended up at All Children's Hospital in Florida.

I eventually interviewed and evaluated more than 1,000 of Kelley's patients and concentrated on a group of 455. From this population, I wrote up in detail 50 cases, representing 26 different types of cancer. Even today, 20 years later, when I review the cases, I am impressed by Kelley's achievement. By 1986, I had put the results of my 5-year investigation into monograph form and intended to publish them. To my disappointment and surprise, I could not get the book published either in its entirety as a monograph or as a summary journal article. The responses from editors ran the gamut from disbelief and accusations of fraud to fear that the book would generate so much controversy that publishing careers might be ruined.

Our inability to get the study published had a damaging effect on Dr Kelley. It appeared that his work would never be accepted for what he believed it was—a promising answer to a deadly disease. In 1986, he closed down his office, and eventually disappeared from sight. After 1987, I never spoke to him again. Determined to keep the enzyme therapy alive, I left Dr Good's group when I finished my fellowship and returned to New York in 1987. I began seeing patients, always with the hope of obtaining proper research support from the academic world.

In July of 1993, the National Cancer Institute (NCI) invited me to present case reports from my practice, detailing patients with appropriately diagnosed poor-prognosis cancer who had enjoyed tumor regression or unusual survival while following my therapy. Dr Isaacs and I put together 25 cases for the session, which was attended by a large group of NCI scientists and lasted 3 hours. After the meeting, I was asked to pursue as a next step a pilot study evaluating my approach in 10 patients diagnosed with advanced adenocarcinoma of the pancreas. In such phase II studies, as they are technically called, a promising new therapy is administered to patients with an aggressive cancer for which there is no effective standard treatment. A pilot study involves no control group, but can still give important information about a treatment. Because inoperable pancreatic cancer has such a grim prognosis, with an average survival in the range of 3-6 months, the associate director who chaired the meeting suggested that if I could get 3 patients to live a year, that would be a significant success.

We were fortunate to get funding for the study from Nestle, the giant international food conglomerate. The then-vice president in charge of research at Nestle, Dr Pierre Guesry, who had previously been medical director of the Pasteur Institute in Paris, had learned of my work and become a supporter.

We finished the study and published the results in June 1999, in the peer-reviewed research journal *Nutrition and Cancer*.[6] We had eventually included 11 subjects, adding a patient when one dropped out. Of the 11, all had biopsy-proven, inoperable disease, 8 of the 11 had stage IV, most had been very sick before consulting with us. All of the patients were approved by a consulting oncologist and the late Dr Ernst Wynder, one of the premier cancer epidemiologists of the 20th century. Of the 11, 9 lived more than 1 year, 5 lived more than 2 years, 4 lived more than 3 years, and 2 lived more than 4 years. As a point of reference, in the clinical trial of gemcitabine (Gemzar), the latest drug approved for the disease, of

126 patients treated with chemotherapy, not 1 lived longer than 19 months.[7] Ours were results that previously had not been reported for the disease.

Shortly after the article was published, the NCI approved funding for a large-scale, phase III clinical trial, again testing our enzyme approach in patients with advanced pancreatic cancer, but this time against a control group that would receive the best available chemotherapy. Eventually, the US Food and Drug Administration (FDA) approved the protocol and the National Center for Complementary and Alternative Medicine (NCCAM) offered to provide the required funding. Columbia University, under the chief of oncology at the time, and the chief of surgical oncology, became the supervising institution in New York, where the study would be conducted. Unfortunately, 7 years later, the project remains unfinished, beset by bureaucratic difficulties.

Nonetheless, as the NCI study bogged down, Dr Guesry at Nestle provided funding for studies to test the enzyme treatment in animal models, to provide supportive data to the human clinical trials. A group at the Eppley Cancer Institute of the University of Nebraska known for their investigations into the molecular biology of pancreatic cancer agreed to take on the challenge. Dr Parviz Pour, the supervisor of the animal work at Nebraska, has developed mouse models of pancreatic cancer that are used to test promising new treatments against the disease.

In May 2004, the results of the experiments were published in the peer-reviewed journal *Pancreas*.[8] In these studies, the researchers evaluated the effect of our enzymes in nude mice injected with human pancreatic cancer cells of a particularly virulent strain. These mice lack a functional immune system, so normally the tumors grow very rapidly and kill quickly. In the first study, which measured survival, the mice were divided into 2 groups, 1 receiving our enzymes, the other given no therapy. The animals treated with our enzymes survived significantly longer than the untreated control group and appeared to be healthy and happy well into the study, in sharp contrast to the controls, which were listless, inactive, bloated, and quite ill.

In a second experiment, again the mice were divided into 2 groups, 1 administered our enzymes, the other untreated. This time, animals were periodically sacrificed and evaluated for tumor growth. The enzymes clearly reduced the proliferation of the tumors, which in the treated mice remained small and very localized. In the controls, tumors were considerably larger and more invasive.

These results are particularly significant because we have never used the enzymes to treat animals before and decided to start at the dose per kilogram that we would normally use in humans. Inbred laboratory mice, however, metabolize most drugs far differently than we do, and normally doses much higher than what would be given humans must be administered to get an effect. Furthermore, the experiments evaluated only the enzyme component of the treatment, not the additional vitamins, minerals, trace elements, and nutritious food we prescribe for our human patients. The animal chow also contained a fair amount of soy, which, however aggressively it may be pushed as a beneficial food, contains one of the most potent natural trypsin inhibitors.

MEASURES OF SUCCESS

Conventional medical journals often publish case reports—descriptions of individual patients whose disease might have taken an unusual course in response to some new treatment. Such "anecdotal evidence" contrasts with a controlled clinical trial, in which different treatments are given to large groups of patients with a particular illness, and the results compared. Some scientists contend that only such rigorous exercises, ideally pursued under the most stringent rules and regulations, can "prove" that a new treatment for a disease has any value. They often argue that case reports, though perhaps interesting or entertaining, have little scientific merit.

Dr Good always insisted that case reports, if properly written and carefully documented, can teach us much about the potential of a new approach. When I first began to evaluate Kelley's records, Good said that if I could find even 1 patient with appropriately diagnosed, biopsy-proven metastatic pancreatic adenocarcinoma who had lived 5 years under Kelley's care he would be impressed, as no one else in medicine to his knowledge had such a case.

In terms of cancer, a case report, to have value, must meet certain basic criteria. First, the diagnosis must be confirmed by biopsy, and the stage by appropriate radiographic studies or surgical procedures. Then, the unusual response to treatment must be carefully defined, explained, and documented. The endpoints of most importance for cancer case reports include objective evidence of improvement in the underlying disease, or unusual prolonged survival.

For patients with the typical solid epithelial tumors, disease regression can be verified by serial radiographic studies, such as positron emission tomography (PET) or computed tomography (CT) scans. For blood cell malignancies such as leukemia or myeloma, normalization of blood parameters, such as white count or blood protein, might be the marker followed over time.

Survival, if particularly unusual, can be a valid endpoint with or without evidence of disease regression. If this is the chosen criterion, the patient in question must have lived far beyond the accepted medians and means for the disease. Such information on expected survival can be culled from a number of sources, both governmental and private, so comparisons can be made. The Surveillance, Epidemiology, and End Results (SEER) Program and American Cancer Society websites, for example, provide survival statistics, including medians and means, for many cancers. However, no precise definition of "significantly" prolonged survival really exists, so it becomes more of a judgment call in each case. When I first presented at the NCI in July 1993, Dr Freidman said that if a patient of mine who had been diagnosed with inoperable pancreatic cancer lived 3 months beyond the reported mean of 6 months, he wouldn't be impressed, whereas survival 6 months in excess of the standard averages would be meaningful. Of course, absolute values for "significance" will vary from cancer to cancer: 6 months of extra life might be unusual for a patient with a pancreatic neoplasm, but not so for a woman with metastatic breast cancer.

In this case, 2 years beyond the mean would, to me, indicate a compelling response to treatment.

Traditionally, the NCI, which sets the standards for oncology worldwide, has not considered survival as a valid endpoint, only objective response as documented by radiographic or other tests. When I presented to the NCI in 1993, for epithelial cancers, the NCI experts defined "response to treatment" as a 50% or greater reduction in tumor size that lasted at least 4 weeks. Unfortunately, as it has turned out, many chemotherapy drugs easily shrink tumors to this degree and within this time span, but the patients live no longer than if they had received no therapy. Tumor reduction, in chemotherapy studies, generally does not translate into longer life for the patient. Though the phenomenon has long perplexed the research establishment—logically, one expects if tumors shrink, patients should live longer—scientists now recognize that chemotherapy may kill the less aggressive population of cells and shrink tumors nicely, but then leave a small, drug-resistant clone that quickly takes over and proliferates explosively. So, the selection for more virulent cells cancels out the initial benefit. In any event, I have long believed the definition of response of a 50% reduction lasting 4 weeks to be rather meaningless, as patients care more about their length of life, not necessarily the size of their tumors.

Dr Isaacs and I learned early on that with our treatment, at times, tumors will reduce significantly or blood parameters will improve, but at other times, the disease does not objectively regress but instead stabilizes. We find that patients in the "stabilized" group often survive as long as those enjoying radiographic or laboratory evidence of benefit, as long as they adhere to their nutritional regimen.

During my 1993 NCI presentation, though I discussed a number of patients from my practice with documented disease reduction on standard testing, I also described several cases with long-term stabilization without proof of regression. I argued that in such instances, the unusual survival should be considered as a response, regardless of what the radiographic or blood tests showed. Today the scientists at the NCI have reworked their definition of response to include not only radiographic or laboratory evidence of regression, but significantly enhanced survival with or without correlating "objective" documentation.

Over the years, I have repeatedly heard the claim that Dr Isaacs and I must be processing and treating thousands and thousands of new cancer patients each year to obtain the results illustrated by these case reports. In fact, a good friend of mine recently remarked that I must be seeing "350-450" new cases of pancreatic cancer yearly, because we are well known for our success with this particular illness. This is simply not the case. In reality, we see no more than 3-5 new cases a year.

Following are 31 case reports that have been culled from our files.

BREAST CANCER

According to *Harrison's Principles of Internal Medicine*, 216,000 women developed breast cancer in the United States in 2004, and 40,000 died, making the disease the most common malignancy (other than skin) among women.[9(p516)] Though surgery can cure approximately 50% of those diagnosed initially with localized disease, for patients with evidence of distant spread, breast cancer remains ultimately incurable despite advances in chemotherapy, hormonal intervention, and blockade and targeted therapies such as Herceptin. The 5-year survival for women with evidence of metastatic disease at the time of diagnosis is around 14%, and in the conventional medical world, even the group of survivors eventually will die of their cancer. In the DeVita textbook, *Cancer: Principles and Practice of Oncology*, the authors report an average lifespan for women diagnosed with metastatic breast cancer of 2-3 years, with some variability.[10(p1700-1701)] Poor prognostic indicators include an incomplete or short-lived response to prior therapy, negative hormone receptor status, involvement of a major organ like the liver or brain, and multiple sites of involvement.

Editor's note: The 6 cases that were presented in the abridged, print version of this article were renumbered consecutively. The first of these is Patient #1, who was also identified as Patient #1 in the print version.

Patient #1: A 16-year Survivor

Patient #1 is a 64-year-old woman with a strong family history of breast cancer. She had been in good health when in the fall of 1986, routine mammography revealed a suspicious mass in the left breast, confirmed by biopsy as ductal carcinoma in situ. Although her surgeon suggested a modified radical mastectomy, the patient insisted a lumpectomy be done. The surgeon agreed, and removed the cancerous tumor. Since she had no evidence of metastatic disease, her doctors did not recommend additional adjuvant treatment.

She subsequently did well until July of 1989, when her physician detected a mass in the right breast. She underwent lumpectomy with excision of a 3-cm right axillary mass that proved to be a poorly differentiated adenocarcinoma, estrogen and progesterone receptor–negative, invading and largely replacing the adjacent lymph node. After surgery, an abdominal ultrasound revealed a density on the right lobe of the liver consistent with metastatic disease. A needle biopsy of the hepatic lesion confirmed metastatic carcinoma, and a bone scan showed "multiple focal areas of increased activity in the spine consistent with metastatic carcinoma."

Patient #1 then began chemotherapy with cyclophosphamide, adriamycin, and 5-fluorouracil (CAF), a very aggressive protocol, which she tolerated poorly. In late 1989, after completing 3 cycles, she refused further treatment and for several months, she did nothing before visiting Stanford in the spring of 1990 for a second opinion. There, after reviewing the previous biopsies and scans, the physicians concurred with the diagnosis of metastatic disease to the liver. The Stanford note reports, "The diagnosis is confirmed and the liver involvement has been documented by the Stanford Pathology Laboratory."

Her doctor at Stanford recommended she immediately

resume chemotherapy with CAF, but once again, Patient #1 refused to consider further orthodox therapy. Instead, after learning of my work, she decided to pursue my program and was first seen in my office in April of 1990.

She was quite ill at the time, suffering chronic pain in her liver. After returning home and beginning her regimen, the liver pain was so severe she required morphine sulfate (MS Contin) for comfort. She also suffered fatigue and malaise lasting many months, before she finally began to improve. When I saw her for a return evaluation in May 1991—a year after she had begun her nutritional protocol—she felt much stronger, and her abdominal pains had largely resolved. Unfortunately, she began to feel so well that without my knowledge, she subsequently discontinued her protocol, assuming she was "cured." In early July 1991, she called me very distraught, having just suffered a grand mal seizure, and admitted she had been off her protocol for several months. A CT scan of the brain revealed a high-density epidural mass in the left sphenoidal ridge and a small, low-density area in the right temporoparietal region. The radiology report reads, "Both areas were heterogeneously enhanced with contrast medium and appear to be metastatic brain lesions."

Her doctors immediately recommended radiation to the brain, which Patient #1 refused. Instead, she resumed her full nutritional program with renewed dedication, quickly improved, and never had another seizure. Follow-up CT scans of both the head and abdomen in April 1992, less than a year after her recurrence, were completely normal—the previously noted liver and brain tumors were gone. The report of the head CT reads, "There is no mass or mass effect. . . . There is no evidence of metastatic disease. . . . Normal CT scan of the head." The summary of the abdominal scan states, "Normal CT scan of the abdomen."

Since that time, Patient #1 has had an up-and-down history on my program, with periods of good compliance and periods of less than good compliance. I haven't seen her in my office in some years, but I've heard from friends that she is still doing well and still taking enzymes. Our last formal contact with her was in October of 2005, when she appeared to be doing fine, 15 years after her diagnosis of terminal metastatic breast cancer.

Her course with such terrible disease is certainly unusual. Patient #1 also served as her own "control"; when she followed the program she did well, and when she didn't comply, the cancer came back with a vengeance. The disease then completely regressed when adherence to therapy improved.

We usually tell new patients who come to us with a history of metastatic cancer that they need to follow their nutritional regimens indefinitely, and must never assume they are completely free and clear. Dr Isaacs and I think of cancer as a chronic degenerative disease, akin to diabetes, that can be managed successfully for years as long as patients follow their diet and take their enzymes. When a patient fails to do that, as in this case, cancer can return and cause havoc. Renewed dedication to the treatment can usually get the situation back under control.

Especially given her compliance lapses, Patient #1's survival is extraordinary. As the medical literature documents, breast cancer, when metastatic to either the brain or liver, is a deadly disease. In a series of patients with brain metastases specifically, Lentzsch et al report a median survival of 23 weeks for those with more than 1 lesion, despite aggressive conventional treatment.[11] In a group of patients with at least 1 lesion receiving supportive care only, the authors describe a median survival of 5 weeks.

Eichbaum et al studied a group of 350 women with breast cancer that had metastasized to the liver.[12] The authors describe a median survival, regardless of the conventional treatment given, of 14 months, somewhat better than the numbers for brain metastases, but still dismal.

In this case, Patient #1 had evidence of liver, brain, and bone metastases, as deadly a combination as can be imagined.

Patient #2: A 16-year Survivor

Patient #2 is a 72-year-old woman who had generally been in good health when in July 1990, she detected a left breast mass. Mammography revealed, as the official report states, "several areas of increased density with the upper outer aspect of the left breast which appear markedly asymmetric as compared to the right breast and which have the appearance of mass densities with irregular margins." After an ultrasound confirmed a 1.8-cm density in the left breast, the patient was scheduled for a lumpectomy.

A routine preoperative chest x-ray showed nothing, but a chemistry blood screen demonstrated markedly elevated liver functions tests with an alkaline phosphatase of 154 (normal less than 140), AST of 89 (normal less than 50) and a ALT of 138 (normal less than 55). But an abdominal ultrasound revealed a normal liver with "no metastases." Then in September 1990, Patient #2 underwent excisional biopsy (lumpectomy) for what proved to be a much larger tumor than had been expected based on the mammography and ultrasound findings, measuring 4 x 3 x 3.2 cm. The mass, which could not be completely removed, was found consistent with a well-differentiated mucinous adenocarcinoma of the breast, estrogen and progesterone receptor–positive. The pathology report states, "The lesion extends to the margins of the specimen submitted on the lateral and undersurface."

After a bone scan revealed only arthritic changes, Patient #2 met with her surgeon, who insisted a mastectomy was now necessary since residual cancer remained in the breast. He suggested that after the procedure, she undergo a course of intensive multiagent chemotherapy. Patient #2 also met with a radiation oncologist and a medical oncologist, who both agreed that because of the size of the tumor, she required, after surgery, radiation followed by chemotherapy.

However, Patient #2, as she later was to tell me, had seen "too many people cut to pieces and poisoned only to die," for her to agree to any further conventional treatment. She refused additional surgery, chemotherapy, and radiation, and instead investigated alternative approaches. After learning of my work through a friend, she first consulted with me in October 1990 and thereafter followed her program with great determination. Within weeks her liver function tests normalized.

Patient #2 followed her program diligently for some 8 years,

until 1998, when I last saw her in my office. During this time, she refused all testing other than routine blood analysis, saying she wouldn't change her therapy regardless of what the studies showed. Sixteen years out from her original diagnosis, she is in excellent health, active with various activities and hobbies. She follows components of the program, as much as her finances allow, and still refers patients to me regularly.

Patient #2, though lacking evidence of metastatic spread at the time of her original diagnosis, certainly represents a remarkable success. The size of the original tumor, coupled with the fact that residual cancer remained after the original lumpectomy, portended a troubling prognosis, even had she agreed to the proposed chemotherapy and radiation. On my program, however, she has enjoyed a healthy and cancer-free life.

I included her because we have in our practice a number of women who after the original biopsy, despite evidence of substantial residual cancer in the breast, refused any further conventional intervention, instead choosing only our program for treatment. Though these women generally have done very well for very long periods of time—16 years in the case of Patient #2—we no longer accept patients with localized breast cancer who do not proceed with recommended surgical procedures. Our decision has not been dictated by a negative clinical experience, but rather the extraordinarily hostile legal environment that exists for alternative practitioners such as ourselves. Standard-of-care criteria require a woman like Patient #2 to undergo further aggressive surgery, and the world would need a consciousness shift before we would consider taking on such patients again.

Patient #3: A 14.5-year Survivor

Patient #3 is a 62-year-old woman with a long history of fibrocystic breast disease, first diagnosed when she was 19 years old. Thereafter, her doctors followed her closely with frequent mammography, and 2 biopsies showing benign changes.

In 1991, mammography again indicated dense fibrocystic breasts as well as a new "1 cm nodular density in the upper and axillary portions of the right breast. . . . This contains internal microcalcifications in a diffuse pattern, and represents a new finding." Her doctors recommended biopsy, which Patient #3, already interested in alternative approaches, refused, instead choosing to follow a nutritional program under the supervision of a local practitioner. However, repeated mammography in March 1992 showed a worsening picture: "Once again, I note small nodule in the upper outer right breast, in association with many microcalcifications. Number of microcalcifications has increased slightly during the interval."

At that point, in the spring of 1992, Patient #3 underwent needle biopsies of 8 lesions, 4 of which proved positive for ductal carcinoma. Since she had diffuse disease throughout the breast, her surgeon insisted she needed mastectomy. However, the patient decided to refuse all further surgery and any other conventional treatment, instead opting for our regimen.

When Patient #3 first consulted me in 1992, she had, on exam, very dense nodular breasts but seemed otherwise in good health. During our lengthy initial interview, I encouraged her to reconsider surgery, which for early-stage breast cancer often can be curative. In a calm and determined way, she explained her decision to refuse disfiguring surgery or toxic conventional treatment, whether I chose to be her doctor or not, so I agreed to treat her.

She subsequently followed her regimen diligently, and over the years has done extremely well, though declining all further testing. Today, she adheres to a maintenance protocol and appears to be in excellent health, now 14.5 years from her biopsy diagnosis.

As in the case #2, Dr Isaacs and I most likely would not agree to treat a patient like this today. The legal climate for alternative medicine remains repressive, the power and authority of conventional medicine, despite its well-documented and rather glaring limitations, is formidable. However, I am gratified by the success of Patients #2 and 3, and the others like them in our practice, who were able to avoid all aggressive surgery as well as toxic drug and radiation treatments. They still have their breasts, their lives, and their health.

Patient #4: A 15-year Survivor

Patient #4 is a 67-year-old woman who had been in good health when routine mammography in October of 1991 revealed a suspicious breast mass. In late 1991, she underwent biopsy and lumpectomy, with removal of a 2.1-cm tumor confirmed as in situ and infiltrating ductal carcinoma. Though no nodes were sampled, a bone scan in December 1991 as part of routine follow-up testing demonstrated increased uptake in the right proximal femur. An MRI in January 1992 documented a lesion on the right greater trochanter consistent with metastatic disease. The official report reads "A solitary lesion is noted distal to the right greater trochanter . . . most likely representing a metastatic lesion."

The patient did meet with an orthopedic surgeon, who suggested a course of aggressive surgery with hip replacement and radiation to the hip. Her breast surgeon insisted Patient #4 proceed with mastectomy followed by radiation to the chest wall. However, after learning of our approach, Patient #4 refused all further conventional interventions, instead choosing to proceed with my treatment.

When I first saw Patient #4 in early 1992, she reported severe fatigue and chronic right hip pain, severe enough that she had gone on disability from her job. After beginning her nutritional regimen, she proved to be a very determined, compliant patient. During her first months on therapy, she suffered migratory aches and pains, particularly severe in the right shoulder and hip, but these gradually resolved. In fact, after a period of some months, she felt so well she returned to work full-time.

Repeat bone scans in May 1992—5 months after Patient #4 began her treatment with me—showed, according to the report, "No definite evidence of metastatic disease." A follow-up scan in June 1993 was again clear, and today, nearly 15 years after she first consulted me, Patient #4 remains compliant with her full regimen, and disease-free.

Her case is very straightforward. At the time of diagnosis, a bone scan and MRI documented a large tumor in her hip that

regressed while she followed only her nutritional program.

Elder et al report a median survival of 2.4 years for women with breast cancer metastatic to bone,[13] somewhat better statistics than for those diagnosed with brain or liver metastases. However, these numbers reference patients aggressively treated with conventional modalities such as surgery, chemotherapy and radiation, all of which Patient #4 refused.

Patient #5: A 9-year Survivor

Patient #5 is a 70+-year-old woman with a family history pertinent for both colon and breast cancer. She had been in good health when in 1986, after a suspicious mammography, a biopsy confirmed infiltrating ductal carcinoma. She underwent right mastectomy, and 3 nodes were found infiltrated with metastatic cancer. She subsequently completed a 6-week course of radiation to the chest wall, but received no chemotherapy. She did begin tamoxifen.

In early 1989, after she developed rectal bleeding, sigmoidoscopy revealed a 2 cm lesion in the sigmoid colon that was biopsied and found consistent with moderately differentiated adenocarcinoma. Prior to the planned colon surgery, CT scans showed no abnormalities in the abdomen, but a lesion in the lower right lung not evident on prior x-rays. The following day, the patient underwent exploratory laparotomy and resection of the lower sigmoid colon for what proved to be Dukes' C disease, meaning the cancer had spread into regional lymph nodes. At that time, the lung finding was discounted as insignificant.

Patient #5 then completed 6 weeks of chemotherapy with 5-fluorouracil (5-FU), followed by 6 weeks of radiation to the lower abdomen, then another 6 weeks of 5-FU. In addition, she continued on tamoxifen for her previously diagnosed breast cancer. Certainly, at this point, Patient #5 faced potential disaster, with 2 different cancers, each metastatic to local lymph nodes—a poor prognostic indicator for either. But she actually did fairly well, with subsequent CT scans confirming that the solitary pulmonary nodule had stabilized. In 1996, after she had been on tamoxifen for 10 years, her doctors suggested the drug be discontinued; however a year later, in March 1997, a routine chest X-ray showed several new lesions. The radiology report describes "Suspicion of right lung nodules as above . . . a CT scan is recommended."

A CT scan in April 1997 revealed "several 1-cm or smaller non-calcified pleural based lung nodules are noted on today's examination in the region of the right upper and lower lobes."

Her surgeon, a longtime friend, told Patient #5 she had metastatic disease that might have originated from either the breast or colon primaries. He did not advocate for biopsy of the lung lesions because he felt the findings were clearly indicative of cancer. Nor did he press the case for additional conventional treatment when Patient #5 made it clear she would never agree to such an approach again. She had already learned of our work, and had chosen to proceed with us.

I first saw Patient #5 in my office in April of 1997, shortly after her diagnosis of recurrent disease. A determined, compliant, and dedicated patient, she hasn't missed a supplement in nine and a half years. And the results have been gratifying: a chest X-ray in April of 1998, a year after she had begun her nutritional program, showed no change in the left nodular density, but resolution of a right lower lung lesion and partial regression of a third right lower lobe nodule. In March of 1999, after Patient #5 had completed 2 full years of treatment, the report of a chest x-ray describes "Clear lungs." All the previously noted lesions were gone.

After those clear scans, Patient #5 continued doing well. In 2004, 7 years after beginning our therapy, mammography revealed calcifications and nodularity in the left breast that on review, had been present on earlier studies dating back to 1993. After biopsy confirmed carcinoma, I agreed that she should proceed with mastectomy since the left breast had been problematic for more than a decade.

The breast contained a very small, .3-cm area of carcinoma, with no lymph node involvement. I don't believe this to have been a new lesion, but suspect her breast was so dense and fibrotic, with multiple long-standing calcifications, that the blood supply to the area probably had been compromised, allowing this small cancer to exist though her metastatic disease resolved. I have made some changes in her protocol, which she continues to follow faithfully. Two years later, now nine and a half years since her diagnosis of metastatic disease, she continues doing well.

I have decided to include Patient #5 among my breast cancer survivors, though ultimately we don't know whether the lung lesions were breast or colon in origin. In either case, such spread invariably proves fatal, usually quickly. This patient's long-term survival, coupled with radiographic evidence of tumor regression while following her nutritional protocol, certainly demonstrates a rather remarkable course for what would normally be a deadly situation.

Patient #6: A 7-year Survivor

In July 1987, after Patient #6 first noticed a left breast mass, she underwent first a needle biopsy confirming carcinoma, then a modified radical mastectomy. The pathology report describes mixed colloid carcinoma and intraductal and infiltrating duct carcinoma, with 1 of 7 nodes positive for malignancy. A metastatic work-up, including a bone scan, was negative. When estrogen-receptor studies came back positive, she started on tamoxifen.

Patient #6 did well until September 1988 when routine blood testing revealed an elevated CEA at 14. A CT scan showed 2 lesions in the liver, and a bone scan demonstrated a right rib lesion, all thought to be consistent with metastatic disease.

In November 1988, Patient #6 began chemotherapy with cyclophosphamide, methotrexate, and fluorouracil (CMF), which she tolerated poorly. After 6 cycles, a repeat abdominal CT scan in April 1989 showed worsening disease. Though the previously noted two hepatic lesions remained unchanged, the radiologist noted a third new lesion, 2 cm in diameter. Since her disease had progressed, her oncologist added vincristine to the regimen, but Patient #6 suffered such severe side effects, including debilitating nerve pain, she decided to discontinue all further chemotherapy.

At that point, she was told to consider calling in hospice.

Instead, Patient #6 began looking into other approaches, learned of our treatment, and first consulted with me in June 1989. After returning home, an abdominal CT scan before she began her nutritional regimen revealed that the liver disease had only worsened despite the addition of vincristine to the chemotherapy mix: "There are several low attenuation lesions about the liver, the largest measuring 3 cm. In the lateral segment of the left lobe of the liver. This lesion appears enlarged since the prior examination. Additionally, a new lesion is noted about the right lobe of the liver. These likely represent metastatic disease."

Subsequently, Patient #6 pursued her program with great dedication. Her local oncologist agreed to follow her since she lived some distance from our office, and after she had completed nearly a year on her protocol, a CT scan of the abdomen in April 1990 revealed significant improvement as documented in the written report: "Comparison is made to the prior examination on 7/12/89. Since then, the metastatic lesions in the liver have decreased slightly in size. The low attenuation lesion in the medial segment of the left lobe now measures 2 cm in diameter as compared to 3 cm on the previous examination. That in the anterior segment of the right lobe now measures 2 cm as compared to previous measurement of 2.5 cm in diameter. No new lesions are identified."

A bone scan in November of 1990 showed resolution of the previously noted rib lesion: "Comparison is made with the patient's last similar examination performed in October 1988. The only substantive interval change is the apparent resolution of an inferior right rib lesion."

Patient #6 thereafter continued on her nutritional protocol and in April 1991, nearly 2 years after beginning her program, a CT scan revealed continued improvement: "Multiple small liver lesions most of which measure less than 5 mm in diameter in the medial segment of left lobe as well as anterior and posterior segments of right lobe."

A CT scan 14 months later, in June 1992—after she had completed 3 years of treatment with us—demonstrated that the largest tumor, which previously had been solid, now appeared to be cystic: "Three hypodense hepatic lesions remaining, the largest of which is located in the posterior segment of the right lobe of the liver, measuring approximately 1 cm in diameter, and has the CT characteristics of a simple cyst. The other hepatic lesions are smaller on the current study compared with the prior study (of 4/5/91)."

However, during the summer of 1993, Patient #6—after enjoying excellent health for four years while pursuing her nutritional program—reported gradually worsening fatigue. An ultrasound of the liver in July 1993 revealed new progression of the liver lesions, with one now measuring 7.5 cm in diameter.

The sudden worsening I find perplexing even today, years later. Over time, as patients improve, in some cases as cancer becomes less frightening, compliance can falter. As best as I could tell, Patient #6 seemed to be compliant. I do know that her doctor, although willing to follow her, never supported her choice of treatment and repeatedly expressed his belief that my therapy couldn't work. Such comments can, we have found, influence a patient's determination to stay with the treatment.

Also, though I did make some adjustments to her program, today I would have pushed the dose of enzymes far more aggressively than I did in 1994. Often, such a change turns the situation around.

In any event, Patient #6 continued on her program until April 1994, when she decided to stop all therapy, nearly 5 years after she had started with me. She wrote me a gracious note, thanking me for the years of generally healthy good life she had never expected based on the terminal prognosis given her in 1989. I didn't hear from her again, until learning of her death more than two years later in August 1996—some 7+ years after she had first consulted me in June of 1989.

Eichbaum et al describe a median survival, regardless of the conventional treatment given, of 14 months for women with evidence of metastatic breast cancer into the liver, despite aggressive conventional treatment.[12] In this case, Patient #6 had documented bone metastases as well as multiple liver lesions. Certainly, with her stage IV condition and the evidence of progressive disease despite aggressive chemotherapy, at the time she began our nutritional therapy, Patient #6 faced a lifespan that would normally be measured in months. Her 7+ years of survival, her generally excellent health during much of that time, and the documented regression of liver and bone lesions over a 4-year period while pursuing only my regimen represents a most unusual course for a most unusual patient.

Patient #7: A 7-year Survivor

Patient #7 received radiation to the chest as a teenager for treatment of keloids but otherwise had been in good health when in late 1986, she developed a left breast mass. After a biopsy confirmed carcinoma, in January 1987 she underwent a modified radical mastectomy for what proved to be adenocarcinoma, estrogen receptor–positive, with metastatic disease in 8 of 23 nodes—a very poor prognostic indicator. However, chest x-ray, bone scan, and abdominal ultrasound showed no evidence of metastatic disease. Postoperatively, Patient #7 completed a 6-month course of adjuvant chemotherapy with CMF, followed by tamoxifen.

Patient #7 did well until late 1990, when she developed pleuritic chest pain, which her local doctor treated with antibiotics. She improved somewhat, but then her symptoms worsened in the spring of 1991. After a chest x-ray in April 1991 revealed a left pleural effusion, she underwent thoracentesis, with cytology positive for the presence of malignant cells. A bone scan was negative. Tamoxifen was discontinued in favor of Megace, a synthetic progesterone analog used to treat breast cancer, but her respiratory symptoms only worsened. A repeat chest x-ray in May 1991 demonstrated a persistent pleural effusion, as a note from her oncologist confirms: "Chest x-ray today reveals significant amount of fluid, certainly reaccumulation since her post-tap film. . . . The patient will stay on Megace 80 mgs b.i.d. . . . She was encouraged not to take unapproved medications for her cancer."

During a follow-up visit in July 1991, her situation seemed to be worsening: "The patient has a significant amount of fluid which would make be (sic) think that the Megace is not working particularly well."

A chest x-ray in August 1991 showed some slight improvement, described as "Moderately large left pleural effusion, smaller than on the previous examination." Since hormonal therapy had failed to control her disease, her doctors suggested aggressive chemotherapy, which Patient #7 refused.

Patient #7 began investigating alternatives, learned of my work, and first came to my office in September 1991. At that time, she continued on Megace and reported severe shortness of breath as well as a persistent cough. After returning home, she discontinued the drug, began her nutritional protocol, and within weeks noted a significant improvement in her breathing and overall well-being. She thereafter followed her program faithfully, and when I saw her in my office for a follow-up visit in April 1993, she reported feeling "wonderful"—better than she had in years. Her respiratory symptoms had resolved, and her pulmonary examination was normal. A repeat chest x-ray in April 1993 showed no evidence of pleural effusion or mass lesion. The report states: "Lungs are slightly hyperinflated compatible with chronic obstructive pulmonary disease. There has been a right mastectomy. . . . No acute pulmonary infiltrates."

For the first 5 years on therapy, Patient #7 enjoyed excellent health. However, she frequently reported severe personal stress, including a very difficult divorce involving aggressive legal actions. Over the years, she admitted the haggling with lawyers had begun to wear her out. By late 1996, she had developed fatigue, pelvic pain, and chronic nausea that impeded her compliance with the regimen. A CT scan in November 1996 revealed bilateral ovarian masses obstructing the ureters, as the radiologist reported: "These are complicated appearing masses and the differential could include tumors, endometrioma or abscesses."

A chest CT showed no distinct masses, but: "Loculated low density fluid-like collection in the lower left thorax pleural space. . . . This could be consistent either with empyema or possibly an area of previously treated pleural metastatic disease with thickened pleura."

After ureteral stents were placed to decompress the kidneys, a biopsy of an ovarian mass confirmed recurrent, metastatic breast cancer. Though her doctors insisted Patient #7 begin chemotherapy at once, she refused, instead choosing to resume, as best as she could, her nutritional program. Within weeks, she began to improve in terms of her energy and well-being. Unfortunately, eventually the stents obstructed again, and the nausea, anorexia, and fatigue returned. By the spring of 1997, Patient #7 could no longer follow the full program and at my suggestion and that of her local doctors, restarted tamoxifen.

Subsequently, as she struggled to continue my regimen, she was seen by a nephrologist at the Mayo Clinic in Arizona, but despite repeated stent changes, her kidney function never returned to normal. Nonetheless, to her doctors' surprise, she survived another year, ultimately dying in April 1998, nearly 7 years after she had first consulted me.

Though this patient did ultimately succumb, it's important to emphasize that breast cancer recurring after aggressive chemotherapy and hormonal blockade, particularly when invading an organ system such as the lung, usually kills within months. In this patient's case, after developing severe pleural effusions in the spring of 1991, she responded only slightly to Megace. However, while being treated solely with our therapy, she had a quick clinical response with resolution of effusions as documented by x-ray studies in April 1993. Her 7 years of life after her recurrence, and her excellent health until the last year, certainly illustrate a remarkable course.

Sometimes it's productive to look for explanations why one patient survives terrible disease and another doesn't. In the case of Patient #7, she herself said repeatedly that the terrible stress in her life "was killing me." Perhaps ultimately it did. Perhaps her body just wore out, after all she had been through, with the disease and the previous toxic treatment. But her family remains to this day grateful for the unexpected years she had with them.

Patient #8: A 7-year Survivor

Patient #8 is a 53-year-old woman who in January 1999 consulted her primary care physician because of persistent exhaustion. Blood work studies were unrevealing, but during a follow-up physical exam in April 1999, her physician detected a lump in the left breast. Mammography revealed a worrisome area, confirmed by ultrasound as 2 distinct suspicious nodules. A biopsy followed, documenting, as the pathology report describes, "At least 3 of 5 five biopsy specimens are involved by infiltrating carcinoma of ductal type."

A surgeon then suggested immediate mastectomy, but Patient #8, with a long interest in alternative healing techniques, decided to delay surgery and instead traveled abroad for a stay at a healing retreat. She admits she hoped that intensive meditation coupled with a wholesome diet might generate a spontaneous remission.

When she returned home she sought a second opinion at a major teaching hospital in the Canadian city in which she lives. After the doctors again discussed surgical options, she agreed to a double lumpectomy in the left breast for excision of the 2 lesions identified on ultrasound, along with axillary dissection. In late December 1999, she underwent surgery as planned. The pathology report describes a 2.2-cm tumor, high grade III, estrogen receptor–positive, with lymphatic vascular invasion. The tumor extended nearly to the surgical margins, and 2 additional areas distant from the main lesion proved to be cancerous. Cancer had also infiltrated 13 of 16 lymph nodes, an indication of a dire prognosis.

At a follow-up visit in mid-January, because of the lymph-node involvement, her surgeon urged her to consent to a course of aggressive chemotherapy. At that point, Patient #8, who had learned of our work, decided to proceed with our therapy. When we met for the first time in mid-January, only several weeks after her surgery, she seemed to have weighed the options carefully and said bluntly she would refuse all conventional treatments.

After returning home to Canada, she began my program, which she followed with diligence. In March 2000, she met with an oncologist, whom she reported "went nuts" when she told him she was refusing chemotherapy. After he calmed down, he admitted that even with aggressive chemotherapy, he could promise perhaps a 5% chance of long-term survival due to the extensive lymph-node involvement at the time of diagnosis.

In April 2000, after she had been on our therapy for 3 months, she detected a new nodularity in her upper left breast. An ultrasound revealed, "Two solid nodules are seen in the left upper outer quadrant. . . . I feel they should be viewed with suspicion, as they may represent involved lymph nodes. Sonographically guided biopsy is recommended."

Patient #8 chose not to proceed with biopsy, but instead concentrated on her nutritional program. Thereafter, she declined further radiographic testing, stayed faithfully on her nutritional regimen, and today, nearly 7 years after our first meeting, enjoys generally excellent health. The left-breast nodularity long ago regressed.

The *Adjuvant! Online* website provides survival statistics for a variety of cancers, broken down by specific stage. On that site, I was able to find numbers that would apply to someone like Patient #8. In women undergoing surgery for breast cancer who have 9 or more positive nodes but no evidence of distant spread, and who receive no adjuvant therapy, only 5.7% will be alive and disease-free at 10 years.[14] So, the numbers are better than what experts report for those with breast cancer that has invaded distant organs such as the liver, brains or bones, but they are still not great.

In this case, the nature of the tumor—grade III/III on the Bloom/Richardson scale—itself portended a potentially poor prognosis, as did the 13 involved nodes. Importantly, during the initial months on therapy, on exam and as confirmed by ultrasound, Patient #8 had evidence of recurrent suspicious nodularity, which subsequently regressed. In any event, in the 7 years that she has been our patient, Patient #8 has successfully avoided chemotherapy and any other conventional treatment.

Patient #9: A 4.5-year Survivor

Patient #9 has a family history pertinent for multiple cases of cancer, including breast cancer. She herself had a long history of fibrocystic breast disease, followed closely by her doctors at the major academic center in the city in which she lives. In 1990, she developed a new left breast mass that was initially not thought to be problematic based on ambiguous mammography findings. When the mass persisted, in May 1991 she underwent aspiration of the nodule, which yielded cells suspicious for malignancy. Because of the worrisome findings, coupled with her strong family history of breast cancer, Patient #9 decided to proceed with prophylactic bilateral mastectomies. So, in May of 1991, she underwent a left modified mastectomy and a right simple mastectomy with lymph nodes left intact.

The right breast appeared to be cancer-free, but a 1.2-cm lesion in the left breast proved to contain both infiltrating and lobular carcinoma, and 4 of 18 axillary nodes were positive, a negative prognostic indicator.

After surgery, an oncologist suggested Patient #9 enter a clinical trial comparing standard chemotherapy for node-positive breast cancer against a new regimen consisting of cyclophosphamide, epirubicin, and 5-FU, for 6 full cycles. After Patient #9 agreed to participate, she was assigned to the epirubicin arm of the study. She tolerated the protocol poorly, experiencing not only chronic nausea and fatigue, but a persistent peripheral neuropathy. Despite the side effects, she completed the regimen on schedule in December 1991.

Thereafter, Patient #9 reports her health deteriorated significantly. She describes an unending series of various infections, including chronic cystitis, sinusitis, and upper respiratory infections. Then in July of 2001, nearly 10 years after she had completed chemotherapy, her oncologist noted enlarged bilateral axillary lymph nodes. Her physicians, for reasons I don't understand, initially suggested neither biopsy nor treatment. When the lymph nodes did not regress, in December 2001 her primary care physician ordered ultrasound studies of axillary regions, which showed 8 enlarged nodes on the right, 2 on the left.

In January 2002, a biopsy of a right axillary node confirmed metastatic carcinoma consistent with a breast primary, estrogen and progesterone receptor–positive. Follow-up studies, including a liver-spleen scan, chest X-ray, and bone scan, were all clear.

Patient #9 then consulted her former surgeon, who suggested that both axillae be "cleaned out," a procedure she declined. When in February 2002 her oncologist recommended not chemotherapy but a trial on tamoxifen, she agreed to the plan. But she also began looking into alternative approaches and learned of our work.

When I first saw Patient #9 in May of 2002, she was still taking tamoxifen, but anxious to quit because of ongoing severe side effects. On physical exam, she had evidence of enlarged bilateral axillary nodes. She thereafter began her nutritional regimen, discontinued the tamoxifen, and noted gradual improvement in her overall health. A variety of chronic symptoms and problems, including fatigue, neck pain, malaise, and severe allergies, resolved. Today, more than four and a half years after starting her nutritional regimen, she remains a determinedly compliant patient and is in good health, with no evidence of enlarged nodes anywhere, including in the axillae. Since stopping tamoxifen, she has received no conventional therapy.

Her case is unusual for a number of reasons. Her bilateral axillary disease developing after aggressive chemotherapy predicted a dismal prognosis. On her nutritional program, the tumors regressed and remain so today.

Patient #10: A 4+-year Survivor

Patient #10 had a family history pertinent for 2 first-degree relatives with breast cancer and a third who died of stomach cancer. She had herself been in good health, with a distant history of localized melanoma, when in early 1989 she first noticed a painful lump in her right breast. Mammography was unrevealing, but after a biopsy in May 1989 confirmed carcinoma of the right breast, she underwent right modified mastectomy. The tumor con-

sisted of infiltrating ductal carcinoma and in situ carcinoma, estrogen and progesterone receptor–negative, but all lymph nodes were cancer-free. After a postoperative bone scan and CT scan of her abdomen were both clear, she began a 9-month course of chemotherapy with methotrexate and 5-FU.

Patient #10 did well until March 1993, when she noticed a nodule on the right upper chest wall that both her oncologist and surgeon thought was insignificant. Her primary care physician, less sanguine about the situation, referred Patient #10 to another surgeon, who in July 1993 biopsied the lesion, which proved to be recurrent moderately differentiated adenocarcinoma. A bone scan showed suspicious activity in the right third rib, but x-ray studies did not confirm the finding. A CT scan of the chest in late July 1993 revealed normal lungs but "Multiple tiny areas of low attenuation in the liver . . . although some of which are intrahepatic vasculature, others are felt to be due to metastasis."

A CT scan of the liver in August documented "occasional areas of low attenuation throughout the liver . . . these most likely represent an early metastatic process."

In August 1993, at the urging of her oncologist, Patient #10 began a course of radiation to the chest wall for "local control," an approach that makes little sense since the disease had already spread into the liver and possibly into the bones. Unfortunately, she suffered such significant side effects from radiation, including severe burns, that the treatment had to be prematurely discontinued. Her oncologist then insisted she resume aggressive chemotherapy, but the patient, realizing her disease was now incurable by conventional standards, refused the drug treatment and began investigating alternatives. After learning of my work, she decided to proceed with our treatment and first consulted with me in October 1993. At the time, she had recovered from her radiation experience and seemed to be feeling quite well despite her liver disease.

Thereafter, for a time she was an extremely dedicated and compliant patient, aware her life was on the line, and initially she did quite well. A CT scan in February 1994, after she had followed her nutritional regimen for only 5 months, showed "overall improvement in the metastatic process in the liver with some residual areas of low attenuation compatible with a metastasis." The patient's oncologist, who had so firmly insisted Patient #10 resume chemotherapy after the positive CT scan findings in August, now claimed she couldn't possibly have had cancer in the liver, since it was inconceivable that my "bizarre" treatment could have provided any benefit. Patient #10 at that point found another physician to monitor her local care.

When I saw her again in New York in June 1994, 8 months after her first visit, she was feeling remarkably well, with excellent energy and well-being. However, I saw the first signs of trouble when she admitted she had gotten careless with the critically important supplements. After I lectured her at length about the need in her case for not good, but perfect, compliance, she returned home with renewed dedication.

A bone scan in October 1994 was interpreted as "essentially unremarkable," indicating the previously noted rib lesion had resolved. I next saw her in the office in July 1995, at which time she reported no problems and said she felt "wonderful."

She had no further testing until October 1995, when she had completed 2 full years on her nutritional protocol. A chest x-ray was normal, and a CT scan of the abdomen with and without contrast showed total resolution of the lesions in her liver. The report reads, "Normal CT of abdomen without and with IV contrast." Her diffuse liver metastases were gone.

During the first several years of therapy, we require that all our out-of-town patients return to New York every 6 months for a lengthy in-office reevaluation. I find I can learn more about what's going on with a patient after 10 minutes face to face then in a 2-hour phone consultation, particularly regarding such life-and-death issues as compliance. In Patient #10's case, though she was next due for a return visit in the spring of 1996, in February she called to say she could not come to New York because of financial considerations. Unfortunately, insurance companies pay only for "standard of care" treatments, and in this case, Patient #10's insurance company paid nothing for her nutritional regimen—despite her several appeals based on the documented response to our regimen. Already, by 1996, her financial constraints—tragically—raised red flags. When strapped, patients tend to cut back on the supplements—an invitation to disaster with advanced deadly cancer.

When we spoke by phone in early March 1996, she admitted she had again been feeling so well she had become sloppy with all aspects of the therapy. She had resumed eating sweets, forbidden food on the therapy, and was consuming far more animal protein than we had allowed on her particular diet. She had cut down the frequency of the coffee enemas, which we find essential for success, and she had been missing doses of supplements, including the enzymes—the main anti-cancer element of the therapy. I lectured at length about the need for vigilant compliance and she promised she would do better.

In early July 1996, a bone scan revealed a new lesion in the right seventh rib, consistent with a metastasis. Shortly after, she returned to New York for a visit in the summer of 1996, nearly 3 years after she had begun my program. Although she reported she felt "great," her compliance was far off track and I could see that she had been lulled into complacency. To make matters worse, not only was she inadequately compliant with my regimen, but a local "holistic" practitioner had suggested, without consulting me, that she begin taking a variety of supplements, including the hormone dehydroepiandrosterone (DHEA), which I never would have prescribed for someone with her history.

After returning home, in September she developed open sores on her chest wall, I believe directly as a result of damage from the earlier radiation therapy. I urged her to be fully compliant, stick with my protocol, and throw away the supplements from her local doctor. For a time, she did seem to be more determined, and by mid-January, the residual chest lesion had regressed somewhat, to the size of a small pea. However, in February, biopsies of a chest wall and right neck nodule confirmed adenocarcinoma.

I was due to see her for a return office visit in February 1997,

but she again said she couldn't afford to come to New York. During early 1997, we talked frequently by phone, and though her energy was generally quite good, she developed a chronic cough. A bone scan in June showed new areas of involvement, and by July 1997, she had been diagnosed with pleural effusions. These were drained with some symptomatic improvement, but the fluid tested positive for malignant cells.

I never saw Patient #10 in my office after the August 1996 visit, though we kept in touch at least on a weekly basis throughout much of 1997. As her situation deteriorated, she required multiple thoracenteses for reaccumulating effusions. Throughout the fall, she had great difficulty sticking to her nutritional program and she finally died in late December of 1997—4 years and 2 months after she had first come to my office, and nearly four and a half years since her diagnosis of recurrent disease in the chest and liver.

Although she ultimately died, Patient #10 far surpassed the usual prognosis for breast cancer recurring in multiple sites (in her case, the liver and bone) after a course of aggressive chemotherapy. After 2 years of good compliance on treatment, CT and bone scans confirmed resolution of her previously widespread disease. Thereafter, for any number of reasons—finances, the influence of local doctors, her overconfidence—her adherence to the regimen fell off considerably. Nonetheless, this patient's significantly improved clinical status on therapy, the radiographic findings of tumor regression in the liver, and the long-term survival indicate a significant response to treatment.

Eichbaum et al studied a group of 350 women with breast cancer that had metastasized to the liver. The authors describe a median survival, regardless of the conventional treatment given, of 14 months.[12]

UTERINE (ENDOMETRIAL) CANCER

In 2004, 40,300 new cases of cancer of the uterine lining were reported, along with 7,000 deaths.[9(p556)] Fortunately, in about 75% of all cases, the disease is diagnosed at an early stage when surgery can be curative. For decades, radiation to the pelvis has been routinely recommended as adjunctive postsurgical treatment for localized endometrial cancer. However, the data from the only 2 controlled clinical trials completed to address the effect of radiation, published in 1980[15] and 2000,[16] respectively, show overall no survival advantage compared to surgery alone. In certain subgroups, the authors report patients receiving radiation actually have shortened survival times.

Once metastatic, uterine cancer resists chemotherapy and usually kills quickly, with a median survival reported in the range of 6-8 months, and a 5-year survival rate at 5% or less. Hormonal blockade with the synthetic progesterone megestrol acetate (Megace) or a similar drug can offer temporary benefit in some 20% of patients with widespread disease, but the responses are usually short-lived.

Patient #11: A 16-year Survivor

(*Editor's note: In the print version of this article, this was Patient #2.*)

Patient #11 is a 62-year-old woman who had been in good health when in the fall of 1990, she required hospitalization for 2 episodes of deep venous thrombosis. She was placed on warfarin sodium (Coumadin), but shortly thereafter suffered an episode of severe vaginal hemorrhage. When the bleeding persisted, in December 1990 she underwent a dilation and curettage (D&C), which revealed endometrial carcinoma. After a CT scan in January 1991 showed extensive abdominal and pelvic lymphadenopathy, she underwent a total abdominal hysterectomy with bilateral salpingo-oophorectomy.

The pathology report describes endometrial adenocarcinoma with areas of squamous differentiation, high nuclear grade (International Federation of Gynecology and Obstetrics [FIGO] grade III), and papillary serous carcinoma, one of the most lethal uterine malignancies. The tumor had spread to the left ovary, obliterating the fimbriated end of the left fallopian tube. Biopsies of the peritoneal cul de sac as well as the rectal serosa confirmed metastatic disease, and due to the extent of metastasis, her doctors warned of a very poor prognosis.

Postoperatively, Patient #11 met with a radiation oncologist who insisted treatment begin at once. Before agreeing to any therapy, Patient #11 decided to consult with a second oncologist in a Southern tertiary care center. Once again, radiation was aggressively pushed as essential to delay spread of her aggressive disease. However, Patient #11 decided to refuse all orthodox treatments, instead choosing to medicate herself with a variety of nutritional supplements, including high-dose vitamin C and red clover tea.

An abdominal MRI in March 1991 showed a "decrease in degree of periaortic lymphadenopathy with persistent evidence of matted lymph nodes." Pelvic MRI documented "decrease in the degree of diffuse pelvic lympadenopathy although there is persistent evidence of pelvic mass lesion most notable in the left hemipelvis. There is evidence of surgical defect presumably from previous hysterectomy." So with surgery, there had been improvement, though extensive disease clearly remained.

About that time, after learning of our work, Patient #11 decided to pursue my therapy. When first evaluated in my office in April 1991, she reported persistent fatigue, a recent weight loss of 15 lbs, "terrible night sweats," and poor sleep.

Patient #11 subsequently followed her regimen with great determination. Seven months later, in December 1991, repeat MRIs showed no change in the periaortic lymphadenopathy as compared with the study of March 1991, but significant regression of the pelvic adenopathy and the pelvic mass in the left hemipelvis. The official report states, "Compared to the study of [March 1991], there is continued improvement with near complete resolution of previously seen pelvic lymphadenopathy. Currently, there is no appreciable residual mass lesion present within the left hemipelvis."

Thereafter, Patient #11 continued her nutritional program diligently, with reported improvement in her general health. MRI studies of the abdomen and pelvis in January 1993, after she had completed 20 months on therapy, indicated that the previously noted extensive disease had completely resolved. The pelvic scan revealed, "There is no identified pelvic lymphadenopathy." The

official report of the abdominal MRI states, "There is no evidence of significant periaortic or periportal lymphadenopathy."

MRI studies completed 14 months later, in March 1994, confirmed "There is no distinct evidence of metastatic or recurrent disease."

Patient #11 followed her regimen faithfully until early 1997, when I last had formal contact with her. At that time, 6 years from her diagnosis of metastatic aggressive histology endometrial cancer, she remained disease-free and generally in good health. She subsequently continued her therapy in a reduced way, and at last report, now nearly 16 years from diagnosis, is alive and apparently doing well.

This case is straightforward: the patient was diagnosed with extensive, aggressive histology uterine cancer, including papillary serous, one of the most deadly subtypes. The surgeon could not excise all the visible cancer, as MRI studies after surgery documented. She then experienced complete regression of her advanced disease while following her nutritional program and remains alive 16 years later.

NON-HODGKIN'S LYMPHOMA

Traditionally, researchers have differentiated Hodgkin's disease from the non-Hodgkin's lymphomas, though both are malignancies of the lymphocyte cells of the immune system. For 2006, the American Cancer Society predicted 58,870 new cases of non-Hodgkin's lymphoma and 18,840 deaths.[17] This umbrella term actually includes well over a dozen different types that range from the very indolent to very aggressive, potentially deadly disease.

Patient #12: A 15-year Survivor

(*Editor's note: In the print version of this article, this was Patient #3*.)

Patient #12 is a 64-year-old woman from the Southwest who in the fall of 1987 first developed vague abdominal discomfort. When the pain persisted, in January 1988, her physician referred her for a CT scan, which revealed several large abdominal tumors. In January 1988, she underwent exploratory surgery, hysterectomy, and bilateral salpingo-oophorectomy, with resection of 2 large masses attached at the mesentery together measuring 9 cm x 8 cm x 8 cm in diameter. The pathology report describes the lesions as consistent with diffuse mixed lymphoma, mixed small and large cleaved cell type, a very aggressive form of the disease.

Patient #12 then completed 6 months of chemotherapy with methotrexate, doxorubicin, cyclophosphamide, vincristine, prednisolone, and bleomycin (MACOP-B), an intensive regimen consisting of 5 different chemotherapy drugs and the steroid prednisone. Repeat CT scans in August 1988, at the completion of treatment, were negative and her doctors assumed her to be in remission. Subsequent scans were clear until May of 1991, when a CT scan picked up 2 nodules in the lungs, the largest in the lingual, measuring 1.6 cm, the smaller in the left lower lobe, measuring 0.6 cm. In addition, the report describes "small periaortic lymphadenopathy at the level of the kidneys," which had been noted on prior scans. A chest CT in July 1991 revealed a 2.5 cm x 2 cm mass in the left hilar area, an abnormality of the lingula, and a left lower lobe mass: "(1) Left hilar mass and posterior left lower lobe nodule. (2) Progressing mass and associated atelectasis or infiltrate in the lingula."

Although her doctors discussed resuming chemotherapy, Patient #12 had "had enough." After learning of our work, she decided to pursue our program.

When I saw Patient #12 in my office in September 1991, she generally felt well and thereafter proved to be a very compliant patient. Six months after beginning her regimen, in March 1992, a repeat CT scan of the chest demonstrated a small pleural-based density associated with the anterior left cardiac margin, approximately 1 cm x 1.5 cm in size, that had significantly regressed since the scans of 1991. And the additional lesions that had previously been described were not evident. An abdominal CT scan revealed "slightly prominent nodes on the para-aortic area measuring up to 1 cm in diameter" but no other worrisome lesions.

In September 1992, after she had been on her program a full year, CT studies of the abdomen and pelvis were clear, but the chest CT showed a "3.5 cm x 2 cm density in the left mid lung and lower lung field which, according to the previous dictation, has increased in size significantly and, therefore, must be considered an active lesion."

When I discussed the findings with Patient #12, she seemed determined to continue with her nutritional program only, expressing no interest in pursuing any other treatment. After I made some adjustments to her protocol, she decided to forgo future CT scan studies. She said they created enormous anxiety, and she had no intention of changing treatment, whatever the tests showed.

Over the next decade, Patient #12 continued her regimen, with excellent compliance. She generally enjoyed good health, despite some ongoing problems I attribute to her earlier chemotherapy, such as a persistent irregular heart rhythm and episodic respiratory symptoms, including shortness of breath with exertion. One of the drugs in the MACOP-B regimen, daunorubicin, has been associated with heart damage in a significant number of patients, and bleomycin often provokes pulmonary fibrosis, sometimes years after treatment. In January 2004, she underwent cardiac and pulmonary evaluations, which revealed no significant underlying disease. A chest x-ray at that time—her first radiographic study since the CT scan of 1992—showed a "small left apical pneumothorax. Chest x-ray is otherwise radiographically normal." The previously described masses seen on CT were gone, and I attribute the area of collapse to bleomycin use years earlier.

Patient #12, now on her nutritional regimen for more than 15 years, continues to be in good health with apparent total resolution of her once aggressive disease. She enjoys her life, is grateful that she has lived to see her children grow, marry, and raise their own children.

The diffuse and diffuse mixed types represent particularly aggressive forms of lymphoma that frequently come back after even the most aggressive of chemotherapy regimens. *Harrison's* reports that the disease recurs in nearly 50% of treated patients with this diagnosis, and of these, fewer than 10% will respond to

additional chemotherapy.[9(p650-651)] Certainly this patient faced a grim future when the CT scan studies in 1991 confirmed new disease.

Patient #13: An 11-year Survivor

Patient #13 is a 54-year-old man who previously had been in good health when in July 1995 he developed severe chronic indigestion, abdominal pain, and constipation. His symptoms did not improve despite a variety of medications and dietary changes. After he developed swelling of the left testicle in September 1995, he was referred to a urologist who ordered a CT scan of the abdomen and pelvis. The tests, done in October 1995, revealed "extensive retroperitoneal adenopathy including retrocrural, periaortic, mesenteric and paracaval adenopathy. The nodes measure up to 5 cm in diameter individually and in conglomerate measure nearly 15 cm in transverse diameter and 8-10 cm AP."

An excisional biopsy of an enlarged cervical lymph node revealed nodular non-Hodgkin's lymphoma (mixed lymphocytic/histiocytic type). After the diagnosis, Patient #13 received no orthodox treatment, instead choosing to follow our regimen. He was first seen by Dr Isaacs in November 1995, and as he subsequently followed his nutritional regimen, he experienced a gradual improvement in his overall health. For a number of years, he avoided all testing until May 2001, when a CT of the abdomen and pelvis showed "resolution of previously noted adenopathy. The study at this time is essentially unremarkable."

This patient's course has been very simple and straightforward. He was diagnosed initially with extensive stage-IV moderately aggressive histology disease, refused all standard treatments, followed his nutritional program appropriately, and enjoyed complete regression of his cancer and long term survival. He is now 11 years from diagnosis, still in good health.

Patient #14: A 7-year Survivor

Patient #14 is a 48-year-old women who before she developed cancer had a long history of lower back pain treated conservatively with acupuncture, massage, yoga, and swimming, modalities which offered some relief. In 1993, when her pain worsened, she underwent laminectomy of the L2-L3-disc. Postoperatively her back pain, although reduced, did not resolve completely. In November 1993, she underwent an MRI of the lumbar spine which showed L5-S1 disc bulging, and some degeneration in several other lumbar discs. In addition, the radiologist noted left para-aortic adenopathy. The patient then consulted with an oncologist at New York Hospital-Cornell, who recommended a CT scan, which, in December 1993, confirmed enlarged left para-aortic lymph nodes, though the patient was not informed of the findings.

When she didn't hear from her oncologist, Patient #14 assumed "everything must be fine." Thereafter, she did well until mid 1998, when she developed gradually worsening fatigue, associated with recurrent upper-respiratory infections. In the fall of 1998, she consulted her primary care physician, who detected a right parotid mass as well as cervical lymphadenopathy. Initially, her internist was not concerned, assuming the enlarged nodes related to her most recent bout of the "flu." But when the adenopathy failed to regress, Patient #14 consulted the oncologist she had seen years earlier at New York Hospital. The physician referred her for an MRI of the neck in March 1999, which revealed 2, 1-cm lesions in the right parotid gland as well as enlarged upper cervical nodes. A CT scan of the chest in April 1999 demonstrated abnormal hilar nodes, the largest measuring 17x12 mm. CT scan of the abdomen revealed "a chain of enlarged nodes (2-3 cm) in the left paraaortic region from the level of left renal hilar vessels . . . extending into the proximal left common iliac chain. Largest node at L3 level measures 3x2 cm."

A biopsy of the parotid lesion then confirmed malignancy "consistent with a B cell (non-Hodgkin's) lymphoma." A bone marrow biopsy was clear.

With the diagnosis established, the oncologist recommended a "watch and wait" approach, holding off chemotherapy for a time when the disease worsened. Patient #14 sought a second opinion at Memorial Sloan-Kettering, where the slides were reviewed and the diagnosis confirmed. The Memorial oncologist suggested 2 options, the conservative, no immediate treatment approach, or a course of aggressive chemotherapy.

Patient #14 then met with a third oncologist, a lymphoma specialist at New York Hospital, who recommended no treatment initially, but that the scans be repeated in October 1999 to assess disease status.

Patient #14, with a long interest in alternative medicine, knew about my work and decided to consult with me. When we first met in June 1999, she had obvious cervical adenopathy. Thereafter, she followed her nutritional regimen initially with great determination and good compliance. Follow-up CT scans in March 2000, when she had been on her therapy only 9 months, showed substantial improvement. The report for the CT scan of the neck states, "Appearance of regression in intraparotid nodes on the right." The CT of the chest showed "Interval complete regression in adenopathy. There is no evidence for active lymphoma." The CT scan of the abdomen indicated "Interval virtually complete regression in adenopathy. There is no evidence of active lymphoma." The CT scan of the pelvis revealed "Interval complete regression in adenopathy. There is no evidence for active lymphoma."

As Patient #14 continued her nutritional therapy, she experienced a gradual improvement in her overall energy and well-being. When in mid 2001 she went through a period of severe personal and professional stress, her compliance with therapy fell off somewhat. On exam, I could see clearly that the neck disease had worsened. CT scans in October 2001, 19 months after the documented disease regression, showed little change in the chest, abdomen and pelvis, but increased "pathological adenopathy in the right neck." After I lectured her about the need for diligent compliance, for a time she seemed more determined, but the stress continued unabated and her compliance varied. At times, she might have been doing 50% of the therapy, and a CT of the neck scan in January 2002 revealed continued progression in the adenopathy. The report of the abdominal and pelvic CT scans describes "mixed behavior of nodes with periaortic nodes slightly less prominent

and hyperplastic nodes in the small bowel mesentery more prominent . . . Interval appearance of focal splenic lesions."

This time, we talked about the need for complete compliance with all aspects of the regimen, regardless of the difficulties in her life. Fortunately, her oncologist did not insist that chemotherapy begin at once, since she had previously responded so well to my treatment. Patient #14 renewed her dedication to the regimen, with repeat CT scans in January 2003 confirming the benefit. The neck CT showed "substantial decrease in the extensive adenopathy in the right neck." The abdominal CT scan indicated "interval disappearance of small splenic lesions and slight decrease in sight of spleen. . . . No pathologic adenopathy is seen in the abdomen or pelvis."

Thereafter, Patient #14 followed the therapy as prescribed and continued doing well. A neck CT in March 2004 revealed "complete regression in pathologic and borderline sized neck nodes."

The CT scans of the abdomen and pelvis were completely clear, as the official report describes: "There is no other interval change and no evidence for active lymphoma in abdomen and pelvis."

Unfortunately, her stress level subsequently increased markedly, and after a long-term relationship dissolved, for a number of months she went off her program completely. Her energy worsened, her sleep became disturbed. Predictably, a CT scan of the neck in February 2006 showed "new and progressive adenopathy along the right jugular chain and posterior triangle." CT scans of the chest, abdomen, and pelvis showed recurrent disease.

She is now again on her program, determined once again to get well, and clinically the enlarged neck nodes are regressing. She feels stronger, more energetic, and more positive.

Though Patient #14 has not followed a straight and narrow path, her course does say much about our treatment. When she complied fully, her extensive disease regressed completely. When her compliance fell off, the disease recurred, then again regressed when she resumed her full protocol. Over the years, her disease status has correlated precisely with her compliance.

Patients, including mine, do not lead perfect lives. Often, they must deal with many life stresses above and beyond their cancer, stresses that can influence mood, motivation, and dedication to treatment. But Patient #14, despite her lapses, has generally done very well over the past 7.5 years on her nutritional program, has successfully avoided all chemotherapy and radiation, and currently feels strong and healthy.

Patient #15: An 11.5-year Survivor

Patient #15 is a 60-year old woman with a history of an insulinoma, diagnosed in 1977, treated effectively with partial pancreatectomy. Her doctors recommended neither chemotherapy nor radiation after surgery, and thereafter she did well until December 1993, when she first noticed swollen lymph nodes under her chin. When the swelling did not regress, in January 1994 she consulted her internist, who suspected the problem was related to infected gums. She was referred to a periodontist who performed gum debridement, but when the lymph nodes enlarged further in February 1994, she returned to her internist who prescribed penicillin, without effect. About that time, she first developed significant night sweats that persisted for a week, as well as abdominal pain. Her physician referred her for an ultrasound, which revealed a large, 7-cm cystic mass in the tail of the pancreas, which a CT scan confirmed. The radiologist thought the lesion consistent with a benign pseudocyst, and when a needle biopsy proved inconclusive, her doctors recommended no further testing.

Because of the persistent enlarged lymph nodes in her jaw, in April 1994 Patient #15 consulted an ENT specialist who did not initially suggest biopsy, but in June, Patient #15 noted new inguinal adenopathy. At this point, the patient's internist prescribed a course of ciprofloxacin for what was now thought to be cat scratch fever, which antigen testing confirmed. Although the nodularity persisted even after she completed a course of rifampin, her primary physician remained unconcerned. When the adenopathy progressed throughout September, Patient #15 returned to her doctor, who again told her "not to worry." In one of the physician's notes from the time, he described her as "borderline hysterical."

Finally, Patient #15 decided to consult the surgeon who years earlier had resected the insulinoma. In October 1994, this physician—somewhat more concerned about the adenopathy—removed a nodal mass from the posterior neck-right shoulder junction that proved to be "follicular lymphoma, predominantly small cleaved cell type (nodular poorly differentiated lymphoma)." Experts at the Pathology Laboratory of the National Institutes of Health reviewed the slides and confirmed the diagnosis.

In late October, a CT scan of the chest revealed "marked lymphadenopathy in multiple mediastinal, left hila (sic), retrocrural and axillary areas . . . consistent with the clinical diagnosis of lymphoma."

An abdominal CT scan showed: "There is extensive adenopathy in the abdomen and pelvis, with lymph nodes ranging up to 3 x 4.4 cm and 4.6 x 6 cm." A gallium scan documented extensive uptake in the mediastinum and abdomen.

Shortly thereafter, in November 1994, Patient #15 began chemotherapy with CHOP (cyclophosphamide, adriamycin, vincristine sulfate, prednisone), a standard lymphoma protocol, at a local academic medical center. In late December, after she had completed 3 cycles of the proposed course, CT scans demonstrated improvement, but not resolution, in both the chest and abdomen, reported as "interval decrease in size of adenopathy within the right paratracheal group, subcarina, left axillary and retrocrural nodes." About the abdomen the radiologist noted, "lymphadenopathy has decreased by more than 50% since exam of 10/__/94 consistent with partial response to chemotherapy."

In March 1995, after Patient #15 had completed the full 6 cycles of the regimen, CT scans indicated some continued response to therapy, but definitely not complete remission. The chest CT showed "Slight continued improvement in right paratracheal lymph node disease with stability of disease elsewhere . . . " The abdominal revealed, "When compared to previous examination, the lymphadenopathy appears stable except for an apparent worsening in the region of the root of the mesentery."

With chemotherapy completed but her disease not in remis-

sion, Patient #15 began investigating alternative approaches, learned of my work, and first consulted with me in April 1995. Her exam was unrevealing, except for multiple palpable right cervical lymph nodes.

She thereafter began her program with great determination. In November 1995, restaging with CT scans of the chest, abdomen and pelvis confirmed significant improvement: "No significant mediastinal or hilar adenopathy is identified. The lungs are clear without evidence for masses . . . Retrocrural adenopathy seen on the previous examination is now not identified. Small periaortic and mesenteric lymph nodes are identified which have decreased in size since the previous examination."

CT studies in February 1996 showed no evidence of recurrent disease, as did subsequent scans over a period of 2 years. Throughout this time, she was noted to have, on exam, several small right cervical nodes. In May 1998, the oncologist who followed her along with me suggested a biopsy of one of the neck nodes, which revealed residual lymphoma described as "follicular, mixed small cleaved and large cell type." At that point, the oncologist recommended, along with my therapy, a course of rituximab, a monoclonal antibody treatment designed specifically to attack lymphoma cells. I felt the treatment unnecessary since she had already responded so well to my regimen, but the oncologist was persuasive and I did not push the case. So, in the spring and early summer, she completed 4 cycles of the drug, which she tolerated with minimal difficulties. On exam, her cervical nodes regressed completely. CT scan studies of the chest, abdomen, and pelvis in September 1998 reported "No evidence of recurrence." CT scan of the neck showed "Multiple small subcentimeter lymph nodes bilaterally which have decreased in size and number since the previous study."

Since 1998, Patient #15 has done exceptionally well, now 12 years after her original diagnosis of stage-IV lymphoma, and 11.5 years from her first visit with me. She has enjoyed generally excellent health with no recurrence of her once widespread disease. The most recent PET/CT scan in April 2006 documented, "There is no PET/CT scan evidence of recurrent or metastatic disease."

Her course is unusual, both in terms of the long-term survival and the near total resolution of the disease as documented by CT scans after only 6 months on her nutritional therapy. In 1998, before she completed a course of rituximab, scans of the chest, abdomen, and pelvis had been clear. I suspect her neck nodes eventually would have resolved without rituximab. Studies do show that 35%-50% of patients with follicular lymphoma that relapse after chemotherapy will have some response to the drug, though the duration of effect is variable, with few long-term remissions.[9(p651)] In any event, in this case, the disease had nearly completely resolved before her oncologist urged her in 1998 to proceed with rituximab, at the time a fairly new, and highly promoted, drug.

RENAL (KIDNEY) CANCER

In the United States, 36,000 new cases of kidney cancer and 12,500 deaths were reported in 2004.[9(p541)] Cigarette smoking predisposes to the disease, with up to 20% to 30% of cases being linked to the habit. Researchers have suggested associations with obesity, polycystic kidney disease, von Hippel Lindau Disease, and certain genetic aberrations. In recent years, though the incidence has been increasing steadily, no clear-cut environmental risk other than cigarette smoking has been confirmed.

Renal cell carcinoma, the most common form of kidney cancer, accounts for 90% to 95% of all cases. In this type, the disease begins in the epithelial lining cells of the proximal tubules and, if localized, can be cured in well over 50% of patients with surgery alone.[10(p1364)] Once the disease metastasizes, it usually spreads quickly, with deadly results. Conventional therapies such as chemotherapy and immune modulation offer little benefit. As *Harrison's* reports,[9(p542)] "Investigational therapy is first-line treatment for metastatic disease as no immune approach or chemotherapeutic agent has shown significant antitumor activity." Interleukin-II, heralded as a miracle cure in the mid 1980s based on anecdotal evidence, in controlled clinical trials worked no better than placebo.

Patient #16: A 15-year Survivor of Renal Cell Carcinoma

(Editor's note: In the print version of this article, this was Patient #4.)

Patient #16 is an 82-year-old man who had a history pertinent for celiac disease, gout, and chronic borderline anemia. In October of 1990, his primary physician noted an abdominal mass during a routine yearly physical examination. Subsequent MRI and CT scan studies revealed a 14-cm tumor in the left kidney, with no evidence of metastases. Chest x-ray and bone scan were both clear, and in late October 1990, Patient #16 underwent exploratory laparotomy and left nephrectomy. Pathology studies confirmed renal cell carcinoma, with 1/1 adjacent nodes positive for invasive cancer.

Patient #16 was then referred to a major New York medical center for additional evaluation and treatment. There, in December 1990, he agreed to enter a clinical trial testing alpha-interferon, an immune stimulant, against kidney cancer. After repeat chest and abdominal CT scans showed no evidence of residual or recurrent disease, Patient #16 began an 8-cycle course of intensive interferon, which he completed in August of 1991.

Thereafter, Patient #16 did well until November 1991, when he noticed a lump in the left parietal-occipital region of the skull that rapidly enlarged over a period of several days. In early December, needle aspiration of the mass confirmed "adenocarcinoma, consistent with metastatic renal tubular carcinoma."

A subsequent CT of the head indicated that the tumor had penetrated through the skull into the cranium, as the report states: "There is a lytic lesion within the left parietal bone with an associated enhancing soft tissue mass, consistent with a metastasis. There is intracranial extension of the enhancing soft tissue, as well as extension into the subcutaneous tissues of the left parietal scalp."

A bone scan revealed "a large focal area of increased radiopharmaceutical uptake with a photopenic center consistent with metastatic disease in the left occipital region of the skull." A CT scan of the chest indicated "Small nodule at the left lung base . . . which may be an area of fibrosis as described. Two other smaller

densities in the middle lobe and the left lower lobe as described of questionable significance." However, these lung findings had not been reported on the chest CT of December 1990.

Patient #16 then began a 1-month course of radiation to the skull mass, totaling 4,000 rads and completed in January 1992. Despite the treatment, the tumor regressed only marginally. Patient #16, having been told he had incurable disease, decided to pursue my protocol. When we first met in January 1992, only a week after he had finished radiation, Patient #16 reported significantly diminished energy, along with a 20-lb weight loss during the previous 6 weeks. On exam, I immediately noticed a lemon-sized mass sticking out of his skull in the left parietal area.

Shortly thereafter, Patient #16 began his nutritional protocol, complied well, and within weeks reported a significant improvement in his energy and well being, as well as a 20-lb weight gain. After he was on his nutritional protocol for 3 months, the large skull mass completely resolved. A repeat bone scan in June 1993, after Patient #16 had completed 16 months of treatment, revealed "no evidence of bony metastatic disease." Not only had the lesion disappeared, but the underlying skull had healed. Today, nearly 15 years since he first consulted me, Patient #16 remains completely adherent to his treatment and is in excellent health and cancer-free.

Several points bear mentioning. Renal cell carcinoma, once metastatic, is a very deadly disease: DeVita et al report a median survival of only 50 days for patients with stage IV kidney cancer, despite treatment.[10(p1369)] This neoplasm resists not only chemotherapy and immunotherapy, but radiation as well. In this case, Patient #16's doctors suggested radiation not as a potential cure but as palliation, hoping to slow the spread of the tumor into the brain. In any event, the response was negligible. While some radiation oncologists report that at times, the benefit of radiation therapy might continue for up to two months, Patient #16 showed significant response only after his third month on his nutritional program. Furthermore, although his radiologists initially downplayed the new findings on the chest CT in late 1991, in retrospect these lesions may have indicated the beginnings of explosive spread.

Patient #17: A 6.5+-year Survivor of Renal Pelvis Cancer

In July of 1989, Patient #17, at the time a 66-year-old Caribbean woman, first developed hematuria. Cystoscopy revealed only a benign urethrocoele, and a right retrograde pyelogram showed no abnormalities. Subsequent urine cytology in January 1990 was negative, but in May 1991, the patient consulted her urologist again after noticing blood in her urine. According to the physician's notes, this time, "urine cytology showed atypical cells on 2 occasions and malignant cells in one specimen. Repeat IVP showed a defect in the right renal pelvis."

When repeat cystoscopy in June 1991 revealed a normal bladder mucosa, but significant blood in the right ureter, her urologist suspected she "most likely has a right renal pelvis tumor and have advised her family that she will most likely need nephro-ureterectomy." The patient then agreed to a needle biopsy of the right renal tumor, which showed, according to the patient and her family, renal pelvic cancer—though we do not have the actual pathology report of this test in our possession.

When Patient # 17 learned of our approach from her daughter, who lives in the United States, she cancelled surgery despite the urgings of her urologist and decided to proceed with our treatment. During our first session in July 1991, she reported intermittent right flank pain and urethral burning on urination, but no other symptoms. I urged her to reconsider surgery, which I explained could be curative if the disease proved localized. She adamantly held her course, stating that she had had enough surgery in her life—she had undergone hysterectomy years before—and would not allow any more, whether I would accept her as a patient or not. So, with her point well made, we agreed to proceed.

She proved to be a very compliant patient and did well clinically, with rapid resolution of her flank pain and no further episodes of hematuria. On her home island, she studiously avoided contact with all other doctors, despite my suggestion that she consult with them at least on occasion. She had no insurance, so frequent testing to monitor her progress was simply out of the question—not that she would have agreed to it anyway. But in October 1995, after she completed 4 years on our treatment, she did allow an abdominal ultrasound, which revealed a normal right kidney except for a 2.3-cm simple cyst in the pole. Otherwise, the report states, "No solid tumor mass seen. The left kidney and the remainder of the abdominal organs were normal in appearance."

During the first 4 years on therapy, Patient #17 periodically returned to New York for re-evaluation. After 1995, she could not afford the expense of the trips, so I agreed to follow her by phone. My last contact with her was in 1998, after she had been on the program for 6.5 years. At that time she was feeling well, with no complaints.

In this patient, the resolution of signs and symptoms, the lack of disease spread and her long survival all indicate a good response to treatment, particularly since she refused all orthodox interventions, including surgery. Unfortunately, we never received the actual pathology report of the needle biopsy, so her records are in that sense incomplete. But the patient and family members carefully described the procedure and the results that had been reported to her. And we do have the urologist's discussion of the positive cytology and IVP findings to confirm the diagnosis of cancer. Despite the one missing document, I included her case here because she did so well following only our nutritional regimen.

MELANOMA

Melanoma originates in melanin-synthesizing cells located in various pigmented areas of the body. Melanin gives color to the skin and provides protection against sun damage, and though we generally associate melanoma with the body surfaces, the disease can begin in the retina of the eye and even, rarely, in the nasal sinuses.

Excessive sun exposure, especially a history of blistering sunburn in childhood, predisposes to the disease, particularly in those with light skin, red hair, and blue eyes. A large number of moles also increases the risk, with 30% of melanomas developing in pre-

existing nevi. Any change in a mole's size, shape, or color (particularly to blue or purple), or bleeding from a nevus should alert the patient and physician to a possible problem.

Harrison's states that in 2004, approximately 54,200 new cases were reported in the US, with 8,200 deaths.[9(p496)] Melanoma has attracted much attention in the research community because of its rapidly increasing incidence in the United States, with a 300% rise in the number of cases over the past 40 years. Scientists speculate that the dramatic change may correlate with increased recreational sun exposure, perhaps coupled with the shrinking of the ozone layer, which in times past may have more effectively reduced penetration of mutagenic ultraviolent light rays.

If diagnosed early, melanoma can be cured with surgery in most cases. Once metastatic, the disease has a dismal prognosis, as *Harrison's* reports: "Melanoma can metastasize to any organ, the brain being a particularly common site. Metastatic melanoma is generally incurable, with survival in patients with visceral metastases generally <1 year. Thus, the goal of treatment is usually palliative."[9(p903)]

Chemotherapy, immunotherapy, and vaccine therapy have been heralded to some degree in recent years, but none has proven effective to date once the disease has recurred and spread.

Patient #18: A 16-year Survivor

Patient #18, a research scientist, had been in good health when in the spring of 1983, he first developed persistent left-sided sinus congestion. Over the next year, his doctors prescribed a variety of medications, including steroids, with little effect. An ear, nose, and throat (ENT) physician diagnosed a deviated septum, so when his symptoms persisted, in September 1984, Patient #18 underwent surgery for septal repair. Incidental biopsy of a large nasal polyp revealed, unexpectedly, malignant melanoma of the sinuses. A CT scan after surgery documented a residual soft tissue mass in the right anterior ethmoid sinus, with destruction of the intrasinus wall.

The patient was referred to Memorial Sloan-Kettering for further evaluation. In November 1984, at Memorial, Patient #18 returned to surgery for a left medial maxillectomy, with wide resection of the cribriform plate, resection of both ethmoids, frontal sinus, scraping of the mucosa of the sphenoid sinuses, and resection of the contents of the left maxillary antrum. The nasal septum and right superior turbinate were removed en bloc, and the floor of the anterior cranial fossa was reconstructed with a pericranial flap. The pathology report documents, "Residual malignant melanoma of the left ethmoid sinus mucosa with involvement of superior nasal septum. Tumor erodes underlying bone. . . . All margins of resection are free of tumor residual disease, with apparently clean margins."

Postoperatively, the Memorial surgeon did not recommend radiation, which he felt would only cause tissue damage and interfere with healing of the reconstruction.

Patient #18 subsequently did well for a time. In late 1986, routine blood chemistries revealed an elevated lactate dehydrogenase (LDH), a possible harbinger of recurrent cancer, but his local doctors pursued no additional investigations at that point. But in the late spring of 1987, Patient #18 developed persistent abdominal pain associated with bloating and indigestion. When his symptoms worsened, in July 1987 he returned to Memorial for a full metastatic work-up: a biopsy of the ethmoid sinus was negative for cancer, as was a CT scan of the head. However, an abdominal CT in July 1987 revealed a large abdominal mass, consistent with metastatic disease. In September 1987, he underwent exploratory laparotomy and was found to have massive adenopathy that collectively measured 12-14 cm in diameter and was positioned in the distal small bowel mesentery and invading several loops of small bowel. Tumor seeding was identified throughout the pelvis, and the large tumor mass had ruptured, forming a contained cavity adjacent to the terminal ileum. The surgeon resected the involved small bowel with primary anastomosis and debulked as much as cancer as possible, but much remained.

The pathology report describes "metastatic melanoma involving mucosa, submucosa and muscularis of a segment of small bowel. Melanoma also involves three mesenteric lymph nodes."

In a note to the patient's local oncologist, the Memorial surgeon discussed the extensive abdominal cancer he had encountered and his prediction of a poor prognosis: "As you know, a percutaneously guided aspiration revealed cells compatible with malignant melanoma, and at surgery, it was clear that the patients' problem was due to massive adenopathy in the distal small bowel mesentery invading several adjacent loops of bowel and rupturing. . . . The involved loops of bowel were resected with primary anastomosis, but the pelvis had seedlings of tumor adjacent to the major mass. . . . For that reason and the fact that the tumor had ruptured and subsequently become contained by the adjacent mesentery, it was felt appropriate only to 'debulk' the mass. . . . There is minimal gross disease left in the patient's abdomen, but since seedlings had occurred and tumor had ruptured prior to surgery, the likelihood of diffuse melanomatosis is high. . . .

"While in hospital the patient was seen by Dr _____, who is in charge of our various melanoma research protocols including Interleukin-2, immunotherapy, etc. Dr _____ reviewed what is available at our Institution and the results of standard and experimental therapy here and elsewhere. . . . I think he needs to digest what has been told to him, share it with his wife and discuss these options with his internists at home. He has a very poor prognosis, a tragedy in someone so young, courageous and knowledgeable. I only wish we had more concrete options to present to him. His training as a scientist allows him to understand our investigative protocols but also to realize that they are, indeed, investigation only. . . . "

With his options dismal, after recovering from his surgery, Patient #18 began to consider alternative approaches. He learned of the late Dr Robert Atkins, who in the late 1980s sought to branch out from his diet work and began offering his own nutritional approach to diseases such as cancer (he eventually would abandon the effort to concentrate again on obesity).

In November 1987, Patient #18 began therapy at the Atkins' Center in New York City. Initially, his disease seemed stable, with a

CT scan in January 1988 showing no overt evidence of recurrence. However, over the following months, Patient #18 developed an enlarging mass in his lower abdomen, visible on repeat CT scan in May 1988, and described as a "recurrent 4.5 x 3.5 cm soft tissue mass in the region of the aortic bifurcation consistent with recurrent melanoma." At that point, Patient #18 consulted with his Memorial surgeon, who, after reviewing the CT scan, argued against further surgery, which he said would be very debilitating and non-curative. He told Patient #18 that he might, if he were lucky, live 6 months.

Patient #18 had learned of my research study of Kelley and heard I had recently begun seeing patients in New York. After discontinuing treatment with Dr Atkins, he consulted with me in May 1988. On exam, he had obvious inguinal adenopathy as well as a hard, easily palpable large mid-pelvic mass. Thereafter, Patient #18 began his nutritional regimen, which he followed faithfully.

A baseline abdominal CT study in July 1988—shortly after our first meeting—documented worsening disease since the prior scan. Not only had the mass grown slightly, to 5 x 3.5 cm, but now the radiologist noted new adenopathy: "There has been a definite change since the study of 5/__/88 with left retroperitoneal and probably left lower mesenteric adenopathy now being present. Additionally, the mass described previously in the low left retroperitoneum has undergone slight further enlargement."

The tumors described in May and July were solid tumors, through and through, with no areas of necrosis. A follow-up CT scan in September 1988, when Patient #18 had completed 4 months on his regimen, showed a slight increase in the size of the main tumor (4.5 x 6 cm) but improvement in the adenopathy, as the official report states: "The previously described left periaortic adenopathy and mesenteric adenopathy is not as evident on this current study." A CT scan 3 months later in December 1988—6 months after Patient #18 had begun his protocol—showed considerable improvement, with stabilization of the large mass and resolution of previously described adenopathy: "A lower left retroperitoneal prominent mass, described on earlier scan is again identified . . . it measures roughly 4.5 cm in AP diameter and roughly 5 cm in width. This indicates little change in the size of this mass since the previous study. Left retroperitoneal adenopathy below the level of the renal hila, appreciated on the study of 7/__/88, is not clearly seen at this time. . . . No mesenteric adenopathy is indicated on the current study."

During this time, Patient #18 felt well, in fact so well that he was able to resume his executive and scientific work full time. The next CT scan in June 1989 indicated, "Mass in the left periaortic retroperitoneal soft tissue is unchanged in size and appearance . . . mass unchanged since 12/22/88."

In late 1989, after he had completed some 19 months on treatment, Patient #18 wrote his surgeon at Memorial to inform him of his good health and apparent progress. The physician wrote back, saying, "Your letter of December ___, 1989 arrived during the Christmas season and carried with it much good cheer! I was thrilled to hear that your disease is stable and has bothered you no further since we last spoke in September of 1988. . . . it is wonderful to know that you have done so well despite a rather frightening situation which we encountered during your operation in September of 1987. . . . "

In November 1990, Patient #18's internist ordered an MRI of the pelvis, which revealed that the previously solid mass had evolved into a more necrotic lesion: "This study is suspicious for a LEFT SIDED PELVIC MASS which may be necrotic. . . . "

Over the next 2 years, Patient #18 remained extremely compliant with his nutritional therapy, enjoyed excellent health, and actually won an award for perfect attendance at his workplace. However, in the summer of 1992, after 4 years on his nutritional regimen, he became non-compliant with the prescribed diet, though he followed the supplement and detoxification protocols diligently. We have found over the years that for ultimate success, adherence to all aspects of the therapy, including the diet, is absolutely essential. A patient who disregards the dietary recommendations is, in our experience, asking for trouble.

For our melanoma patients, we always prescribe a diet that emphasizes red meat, with the fat, preferably more than once a day. We forbid certain commonly enjoyed vegetables, such as leafy greens, and allow fruit only once a day, and never citrus. Such recommendations countered most expert recommendations emphasizing "low fat" and "no meat" that dominated the orthodox and alternative world during the 1990s, particularly in regard to cancer. In this case, after Patient #18's daughter adopted a completely vegetarian way of eating, Patient #18 decided, without telling me, to switch himself to a similar diet in the summer of 1992, contrary to what I had prescribed.

By late fall 1992, his local oncologist felt, on physical exam, that the pelvic lesion had grown for the first time in years. An abdominal CT scan in December 1992 revealed a 7.0-cm soft tissue mass in the pelvis, containing areas of necrosis and calcification. The radiologist also noted a second, 2.0-cm nodule, also showing areas of calcification, in the right abdomen at the umbilical level.

After a number of conversations with me, Patient #18 and I decided he should return to Memorial for surgery, as the mass was beginning to cause symptoms. His former physician, astonished Patient #18 was still alive nearly 5 years after his previous recurrence, agreed, after CT scans of the brain and chest were clear, to operate. In late January 1993 at Memorial, Patient #18 underwent "exploratory laparotomy, resection of tumor from mesentery/pelvis and right iliac vein and artery." The tumors, the patient was later told, came out very easily, as if they had been encapsulated.

The pathology report from Memorial describes mostly dead tumor, with the main large main pelvic lesion described as "an 8 x 6 x 5.5 cm mass of predominantly necrotic tumor tissue. The tumor is grossly present at the surgical margin." The pathologist identified some residual viable cancer, described as "high grade malignant neoplasm consistent with metastatic malignant melanoma. . . . Tumor is present at surgical margin."

In an additional resected nodule, no viable cancer cells were found: "Mesenteric nodule excision: Necrotic tissue suggestive of a metastatic neoplasm largely replacing a fibrotic lymph node; can-

not identify viable tumor cells."

A second node examined also appeared cancer-free. Overall, although some cancer remained, much of the original tumor evident in the spring of 1988 had died, now replaced by scar tissue.

According to the patient, while he was still recovering from the procedure, his surgeon met with him and discouraged him from consulting with any of the Memorial melanoma "experts." He suggested that he only continue his nutritional program—advice given, Patient #18 said, "off the record."

After recovering from his ordeal, and after several more lectures from me about the need for total compliance, Patient #18 resumed his full program—including the high red meat diet. He said he had learned his lesson. During the following 8 years, he remained faithful to the treatment, enjoyed great health, retired from his job, and began a consulting firm. He also repeatedly expressed gratitude for the program, gratitude for the years of life the therapy had given him.

Unfortunately, beginning in 2001, after he had been on therapy for 13 years, I noticed a distinct change in attitude. He began to grouse about the program, about the "expense," though money didn't seem to be a problem for him; he complained about the supplement protocol, which required he take enzymes throughout the day. He repeatedly urged me to cut down the number of pills on his regimen, to make his life "easier." I was reluctant to do so with his melanoma history and in view of the fact that residual tumor remained after his 1993 surgery. Regardless of how well he had done, he was always at risk for recurrent disease. Eventually I relented and reduced the number of enzyme capsules to what I considered a minimal dose. I later learned that Patient #18 decided without telling me to lower the dose still further, mistakenly thinking he was cured, that cancer could never be a problem again.

Despite the compliance lapses, Patient #18 did very well until late November 2003, when he developed chronic digestive problems, diminished appetite, and a 7-lb weight loss. In mid-January 2004, he consulted his local physician, who on exam detected new inguinal adenopathy. When I saw Patient #18 2 days later in my office, in addition to the enlarged groin nodes, I could now feel a new small mass in the mid abdomen. A CT scan the next day—his first scan in 12 years—showed "interval development of extensive upper abdominal portal hepatis, and superior retroperitoneal adenopathy. Multiple splenic masses. Several tiny low density hepatic lesions are also seen, not identified on previous examination."

The disease had taken off. I immediately raised the dose of pancreas significantly, and Patient #18 agreed to do whatever he needed to do to fight back against the disease. Unfortunately, his abdominal disease had progressed so far he had trouble eating, and in February, his local oncologist and I agreed that surgical debulking might be helpful. Patient #18 called Memorial, only to learn his former surgeon—who had done the abdominal procedures in 1987 and 1993—had retired. He consulted with the younger replacement, who felt the main abdominal mass was inoperable but strongly suggested he meet with a Memorial oncologist to discuss chemotherapy. Patient #18, who knew chemotherapy offered little benefit for his disease, declined the invitation. In early March, he did consult with an abdominal surgeon at Columbia, who concurred that surgery would not be feasible. But at Columbia the patient was aggressively encouraged to consider an interleukin II clinical trial, though the drug had proven to be a consistent failure for over a 15-year period.

Patient #18 decided, despite my warnings, to consult with the interleukin II expert, who helped convince him to enter the study, to "shrink the tumors." In a later conversation with me, Patient #18 told me that, feeling somewhat desperate at the time, he had agreed to proceed with interleukin just "temporarily," to get him in better shape so he could follow my program more religiously. Ethically, I could not tell him to refuse the treatment.

So in mid-March, Patient #18 went into the hospital for his first series of 8 interleukin II treatments, and during that time he could not follow my program at all. To my surprise, his doctors never expressed any interest in his 16-year survival with metastatic disease under my care, refused to speak to me about what they were doing, and when Patient #18 began to crash on the drugs, didn't seem anxious even to talk with him.

After finishing the first course of treatment, Patient #18 went home to bed. After regaining some strength over a period of several weeks, he chose to re-enter the hospital for another round of interleukin. This time, the drug left Patient #18 far more debilitated, with severe anemia and weakness, and once home, he was unable to leave his bed for days. Not only was he exhausted and anorectic, but the bill for 2 weeks of treatment, he said, exceeded $200,000. By that point, Patient #18 decided to refuse all further conventional treatment, but he was so debilitated he could not resume my therapy.

A CT scan done in early May 2004 showed not only an increase in the size of the previously noted tumors despite interleukin, but new lesions in the liver as well. The treatment had done nothing but make the situation worse. A local oncologist suggested chemotherapy; his friends began suggesting a variety of odd treatments, including a special immunotherapy available only in Argentina. I urged him to rest, to regain his strength, and try to restart his nutritional program, which had beaten back his disease in the past. Instead, Patient #18 flew to California to consult with a well-known melanoma expert and surgeon at the John Wayne Cancer Center, hoping this physician might be able to resect the tumor. But after several meetings, Patient #18 was told surgery would not be possible.

Patient #18 returned home, only further tired from the trip. He remained anemic, exhausted, and debilitated. He was angry he had ever allowed himself to be talked into the course of interleukin. In mid-June, we had a long conversation about the situation, reported in my office notes: "He is very upset about the interleukin experience. . . . He said he wrote to Dr _____but never heard a word. No one has followed up. He said it is as if they do not care after spending a couple hundred thousand dollars on 2 sessions. . . . They just do not care he feels."

A week later, not having resumed his nutritional treatment, Patient #18 died at age 73, 16 years after he had begun treatment

with me in 1988, when his predicted life expectancy was only months. Certainly, his long survival is extraordinary, as is the significant reduction of disease during his first years on therapy, when he followed his prescribed regimen diligently. In the fall of 1992, when he decided to adopt a diet completely unsuited for his metabolism (by our standards), his disease progressed. When his compliance improved after surgical debulking, he remained disease-free for 11 years, despite the aggressive nature of his cancer. After his compliance flagged, he ultimately suffered an explosive recurrence in early 2004 and was unable to resume his full program. Well-meaning friends, his own fears, and the power of orthodoxy ultimately led him to an ineffective course of interleukin that left him considerably weakened and with his disease worsened. Nonetheless, though he ultimately died, he had many productive and very happy years. We do miss him.

Patient #19: A 15-year Survivor

Patient #19 is a 72-year-old Englishman who had been in excellent health when in July 1990, a pre-existing mole on his left ear suddenly enlarged and turned black. When the lesion continued to grow, in January 1991 Patient #19 went to his local physician who immediately referred him to a surgeon. He then underwent excision of the lesion, which proved to be melanoma, Clark's level IV, with a 1.9-mm depth.

In late January 1991, Patient #19 returned to surgery for a neck dissection, superficial parotidectomy, and excision of the left ear in toto. The pathology report describes residual melanoma in the original site to a depth of 1.2 mm, but the lymph nodes and parotid were free of cancer. His doctors warned the disease might recur but recommended no additional treatment.

In June of 1991, just 5 months after his major surgery, Patient #19 developed 2 nodules in the left mastoid area adjacent to the previous surgical incision, as well as a nodule in the skin of the right axilla. In late June, he consulted his surgeon, who removed the lesions: ". . . at a recent follow up visit in June I noted that he had a couple of little nodules under the skin of the mastoid area on the left adjacent to the ear resection and he also showed me another little clump of nodules in and under the skin of the right upper arm. I excised all these under local anaesthetic and histology of that has confirmed that all three are malignant melanoma. . . .

"I have put Patient #19 and his wife fully in the picture about the fact that his melanoma appears to have spread by the bloodstream and may turn up at other distant sites in the future."

After the surgery, Patient #19 underwent a full metastatic workup. A chest x-ray was clear, as were CT scans of the head, chest, and abdomen. However, Patient #19's doctors advised him that his disease would recur and prove terminal, most likely within a year. No further treatment with either chemotherapy or radiation was thought warranted, due to its ineffectiveness.

Patient #19 began investigating alternatives, learned of my work, and first came to my office in September 1991. At that time, he felt well and had a normal physical examination except for evidence of his extensive head and neck surgery. He thereafter proved to be a very compliant patient, and on treatment, he felt well and continued his demanding career. When seen in March 1993, after completing 18 months on his nutritional regimen, he felt fatigued from overwork and frequent air travel, but otherwise appeared well.

When I saw him 19 months later, in October 1994, after 3 years on the regimen, he reported increasing stiffness in his neck and symptoms consistent with optical migraines occurring 1-2 times a month. When his headaches worsened upon returning home to England, he consulted with a London neurosurgeon. A CT scan of the brain revealed a 4.5 x 2 x 2 cm mass in the right occipital area of the brain. Since the tumor appeared easily accessible, the surgeon strongly recommended resection, and after discussing the situation by phone with me, I agreed he should proceed with surgery, the sooner the better. So, in mid-December 1994, Patient #19 underwent craniotomy and excision for what proved to be an encapsulated melanoma tumor. The pathology report states, "Sections show a discrete tumor mass bounded by gliotic brain. Tumor extends to margin of excision in some sites. There are conspicuous lymphocytic collections around the tumor. The tumor consists of sheets of poorly differentiated cells. A few contain granules of melanin. There are areas of necrosis."

He had no further conventional treatment and resumed his program as soon as he returned home from the hospital. Initially, he felt quite well, with his neurological symptoms resolved, but by February 1995, just 2 months later, he once again developed persistent headaches. A CT scan and MRI of the brain confirmed that a tumor had regrown in exactly the same location as the prior lesion. His surgeon felt that once again, the tumor could be easily resected, so after multiple phone consultations with me, in early March 1995, he underwent repeat craniotomy and excision of the mass. The pathology confirmed "recurrent metastatic amelanotic melanoma." The report elaborates: "The appearance resemble those of the previous biopsy, but now inflammation is less obvious and there is much more necrosis."

His local doctors suggested 2 doses of localized stereotactic radiation to eliminate any lurking malignant cells in the tumor bed, and I concurred with the recommended treatment. Patient #19 tolerated the radiation well and subsequently continued on his nutritional program, which I adjusted to take into account the recent series of events. After that, he experienced no further recurrence, and today, nearly 12 years from his last surgery, he remains compliant with his full regimen, now 15 years since he first consulted with me. He is in excellent health, and continues his productive professional life.

As I put this case together, I realized that although a CT scan of the brain in July 1991 showed no tumor, he didn't start his nutritional program until early October, a full 3 months later. Given the nature of his disease and its tendency to spread and kill quickly, it is possible the brain lesion first grew in the interim before he started treatment with me. During that 3-month period, he was on no therapy whatsoever, and it is also possible that once he began treatment, tumor growth slowed. In my experience, it would be unusual to see a new tumor forming in a fully compliant patient.

In late 1994, Patient #19 traveled considerably, and perhaps

this physical stress, not an inconsequential variable, weakened him enough to allow the tumor to grow despite his good compliance. But then, within 2 months of the first brain surgery, the tumor recurred in the same exact location. We do find that in areas of prior surgery, blood circulation, and with it the enzyme supply, can be compromised due to fibrosis and scar formation. In such protected areas, sometimes tumors can reform, though rarely do they spread beyond the scarified boundaries. The fact that in the nearly 12 years since his second surgery Patient #19 has remained completely cancer-free indicates something unusual was going on in that particular area of the brain in 1994. I admit what I propose above falls into the realm of conjecture, but it's important to keep in mind that metastatic melanoma usually kills within months, regardless of the conventional therapy, such as radiation, that might be employed.

DeVita reports, "Metastatic melanoma has a median survival of only 6 to 9 months and current systemic therapy has been shown to induce complete durable responses in only a small minority of patients." The DeVita chapter on melanoma has a section devoted to brain metastases specifically. In this case, the author writes of the dismal prognosis even when the disease is treated aggressively: "A series of patients with symptomatic solitary intracranial lesions showed a median survival after craniotomy of only 10 months."[10(p2048-2050)] Radiation offers little additional survival benefit to surgery.

Finally, I want to remark about this patient's attitude toward me and toward the program. When he developed evidence of recurrence in December 1994, he didn't immediately assume the program had "failed" and that I didn't know what I was doing. Quite the contrary: he understood he had terrible disease, and he knew his survival even at that time was unusual. Though perplexed by the recurrence, he listened carefully to my hypothesis that perhaps this tumor was not new. When the disease recurred 2 months later, he again assumed that the therapy would eventually gain control of the situation, as it apparently has over the past 12 years. At no point did ever lose faith in the treatment, or in me, and within days of each of his 2 brain surgeries, he resumed his full program with only greater devotion.

Patient #20: A 17-year Survivor

Patient #20 is a 68-year-old man with a history of significant sun exposure when he was younger, including summer stints as a lifeguard. He first developed skin cancer in 1987, thought to be secondary to excessive sun damage. Over the following year, his dermatologist removed 12 basal and squamous cell carcinomas from his chest, back, and face. Then, during a routine follow-up exam in 1988, Patient #20 was found to have a suspicious lesion on his scalp, above his left ear. This was removed in September 1988 and described as "malignant melanoma, near left ear, measuring at least 1.3 mm in greatest thickness." A chest CT at the time showed no evidence of metastatic disease.

Shortly after surgery, in the fall of 1988, Patient #20 detected a new lesion anterior to his left ear. Initially, his surgeon and dermatologist were unconcerned, but when the lesion continued to grow, he was admitted for evaluation to a New York City hospital in August 1989. After CT scan studies of the brain, chest, and abdomen were negative for metastasis, Patient #20 underwent left superficial parotidectomy and left radical neck dissection. The pathology report describes "metastatic malignant melanoma to intraparotid gland lymph node," but no other areas appeared to be infiltrated with cancer.

However, a postoperative CT of the neck showed a new subcutaneous lesion in the back of his head, in the occipital area. At this point, Patient #20, aware of his dismal prognosis with recurrent melanoma, began investigating alternative approaches to cancer, learned of our work, and first consulted with me in November 1989. During my initial examination, I detected a small, 0.5-cm nodule in the scalp of the right occipital area, as well as many areas of sun damage on his chest and back.

Subsequently, Patient #20 proved to be a very determined, compliant patient. By March of 1990, when he came to the office for a routine visit, the occipital lesion had completely regressed. Thereafter, as he continued his program diligently, he reported an overall improvement in his general health and returned to work after a medical leave to resume leadership of a successful business.

Over the next 2 years, his dermatologist removed several small pre-existing superficial basal and squamous cell carcinomas in sun-damaged areas, but his melanoma did not recur. After 1992, he developed no more skin cancers while following with his nutritional regimen.

Patient #20 continued on therapy fully for some 5+ years, before his compliance fell off during mid 1994. In July 1996, after an 18-month absence, he returned to the office and reported he had adhered to the prescribed diet and continued the detox procedures including the coffee enemas, but had gradually dropped off the supplement protocol. He felt "great" and admitted he had gotten careless since his diagnosis of recurrent melanoma seemed so far in the past. After that visit, I periodically heard from him by phone but I didn't see him in the office again until May 2001. He told me that after following the full regimen for another year or 2 after the 1996 visit, he had gradually drifted away from the supplements once again. In early 2001, he had developed a nodule on his left shoulder, which had been excised in March 2001 and found to be not melanoma, but a cutaneous leiomyosarcoma. After experts at the Armed Forces Institute of Pathology confirmed the diagnosis, Patient #20 had undergone a wide excision of the original area, but no additional cancer was detected.

He also reported that after stopping the supplements, he had once again developed a number of squamous cell carcinomas of the skin after being cancer-free for years. But after our May 2001 visit, Patient #20 resumed his full nutritional program, which he followed faithfully for more than a year, before again slacking off the supplement regimen. However, neither his melanoma nor his sarcoma has recurred; he recently reported to my office staff that he was in "great shape," cancer-free since his last bout in 2001.

Clearly, this patient's course has been unusual. He had a history of poor prognosis, recurrent melanoma, with—at the time he

first consulted me—evidence of a suspicious new lesion in his skull. This nodule regressed quickly and completely on our therapy. During his first 2 years on the regimen, a number of basal and squamous cell carcinomas were removed, but these I suspect had been festering for years. Eventually, as long as he followed his program fully, no new malignant skin lesions developed.

Patient #20 ran into trouble after his compliance fell off in the late 1990s, when once again squamous cell cancers, but no melanoma lesions, began forming. After he developed cutaneous leiomyosarcoma in 2001, he resumed his protocol and remains, 17 years from our first meeting, cancer-free.

SARCOMA

Experts recognize some 20 varieties of sarcoma, all of which originate in the connective tissue or muscle. Such tumors thus differ from the common solid tumors of the lung, colon, breast, or pancreas, that form in the epithelial lining of organs, or the immunological malignancies such as leukemia or lymphoma that affect the white blood cells. Sarcomas are rare, accounting for only 8,800 cases in 2004.[9(p559)] Sixty percent appear first in the extremities, and when localized, surgery can be curative. Once metastatic, this cancer type—notoriously resistant to chemotherapy and radiation—usually proves fatal within a year.

Patient #21: A 12+-year Survivor

Patient #21 is a 58-year-old woman, who in the summer of 1993, first noticed a mass above her right ear. After the lesion became chronically irritated by her eyeglass frames, in August 1993 she opted to have it removed. The nodule, measuring about 1 cm in diameter, was found to be consistent with "malignant neoplasm, probably metastatic." The slides were sent for review at the Mayo Clinic, where the pathologist classified the cancer as an epithelioid sarcoma. A subsequent third review of the slides confirmed the diagnosis of epithelioid sarcoma.

The patient then underwent a metastatic work-up. A bone scan in September 1993 revealed "single abnormal focus of uptake in the left occipital-parietal region, worrisome for metastatic neoplasm."

A skull series the same day showed a "9 mm geographic lucency in the left occipital bone, possibly representing a calvarial metastasis." The report of a CT scan of the head a week later stated, "Images of the skull demonstrated one small lytic area . . . in the left occipital bone. . . . It measures under a centimeter in size. It is in the medullary space of the bone but appears to affect the cortex also. No soft tissue component is noted."

A CT scan of the neck and chest showed a probable right thyroid cyst and 2 areas of decreased attenuation in the liver compatible with either cysts or metastatic disease.

Patient #21 then met with a head and neck surgeon, who proposed wide excision with removal of much of her jaw, followed by reconstruction. But when she was told she most likely would die of her disease anyway, she refused surgery. After investigating alternative approaches to cancer, she learned of our therapy and consulted with Dr Isaacs in late September 1993. She thereafter followed her program diligently.

In June 1994, 9 months after she began her nutritional regimen, she noticed a lump above her right ear in the same location as the original tumor. The nodule stabilized for 2 years before it was resected in August 1996. The pathology report describes once again an epithelioid sarcoma. After Dr Isaacs made some adjustments in the protocol, Patient #21 continued her therapy faithfully as before.

Over the years, Patient #21 has been very compliant with her regimen and has enjoyed improvement in her overall energy and sense of well-being. Since the surgery of 1996, the disease has not recurred. When last seen by Dr Isaacs in August 2006, this patient was in good health, with no visible evidence of cancer.

Epithelioid sarcomas tend to be fairly aggressive. If localized, as with most sarcomas, surgery can be curative, but once metastatic, survival is usually measured in months. A review of epitheloid sarcomas reported that "median post-distant metastasis survival was 8 months."[18]

We don't think the lesion that appeared after this patient began her therapy indicates global treatment failure. As mentioned previously, we find at times that tumors will recur in areas of prior surgery, though nowhere else. We suspect that in areas of such tissue disruption, the resulting fibrosis and scarification compromise blood supply to the area and create a protected area where residual cancer cells can grow unhindered. We suspect such a scenario in this patient's case. Regardless, today, 13 years after her original diagnosis of metastatic cancer, Patient #21 is in excellent health, with no clinical evidence of her disease.

OVARIAN CANCER

In 2004, 25,580 women in the United States developed ovarian cancer, and 6,000 died from the disease, making it the leading cause of gynecological cancer deaths in women.[9(p553)] Ovarian cancer tends to occur in family clusters, with some 5% of all cases linked to inherited genetic aberrations, particularly mutations in the BRCA1 and BRCA 2 genes—mutations long associated with breast cancer as well. The protein products of 2 these alleles normally serve as tumor suppressors, so irregularities in the DNA encourage carcinogenic transformation.

The disease has also been linked to infertility, use of fertility-enhancing drugs such as Clomid, and nulliparity. Each pregnancy reduces the risk, as does breast feeding. Regular use of oral contraceptives actually reduces the risk of ovarian malignancy, while hormone replacement therapy doesn't influence incidence either way, despite earlier concerns.

Ninety percent of women diagnosed with strictly localized disease survive 5 years, many of them cured by surgery alone. Once the disease spreads, ovarian cancer can be very aggressive, with fewer than 5% of stage IV patients living 5 years despite aggressive treatment.[10(p1604)] Chemotherapy regimens that include one of the taxane derivatives, given along with platinum agents such as carboplatin, cut the recurrence rate for localized tumors and marginally improve survival for patients

with advanced disease.

Patient #22: A 10-year Survivor

(Editor's note: In the print version of this article, this was Patient #5.)

Before developing cancer, Patient #22 had a long history of neuro-muscular symptoms dating to 1979, when she first developed a mass in her left calf that was associated with muscle pain, atrophy, and numbness. As the symptoms worsened, she consulted numerous physicians at numerous centers. Though multiple muscle biopsies had all been unrevealing, she was nonetheless treated empirically and unsuccessfully with a variety of drugs, including prednisone. In 1985, she sought another evaluation at the Mayo Clinic, where another muscle biopsy confirmed polymyositis. After she was diagnosed with motor and sensory neuropathy, type II, Patient #22 began another course of prednisone but with little improvement, followed by 6 months on Imuran. The latter drug did nothing for her disease, but did lead to weight gain, insomnia, and anxiety.

As her symptoms worsened, Patient #22 decided to seek treatment with me for her neuromuscular problems. When she first came to my office in 1989, she had been off all medications for 3 years, during which time her symptoms of weakness, nerve pain, and numbness continued to progress. When I first saw her, she had no gynecological problems other than the history of a hysterectomy for uterine fibroids.

I designed a protocol to treat this patient's muscle and neurological problems without the high doses of enzymes we use against cancer. Subsequently, Patient #22 complied well with her program, and when I saw her for a return visit in August 1989, she reported that her condition, which had worsened without respite over the previous 10 years, had improved significantly. She described a "20%" overall gain in motor strength and calf thickness, a marker her previous doctors had used to track her decline. The proximal muscle weakness in both legs had reversed to the point that she could stand from a sitting position for the first time in years. However, on exam I detected a small pelvic mass and told her she needed to follow up with a gynecological evaluation upon returning home.

Some weeks later, in early fall, an ultrasound revealed a 7 cm x 8 cm cystic lesion posterior to the bladder. In early November 1989, at the Moffitt Cancer Center in Tampa, Fla, she underwent exploratory laparotomy and was found to have extensive malignant disease throughout her pelvis and abdomen. Her surgeon proceeded with bilateral oophorectomy, omentectomy, and extensive lymphadenectomy of pelvic, periaortic and precaval lymph nodes. The pathology report describes "Omentum diffusely infiltrated by papillary serous carcinoma" of ovarian origin, as well as tumor in both ovaries that involved both fallopian tubes. Cancer had infiltrated into all 21 of 21 nodes evaluated, and peritoneal washings were positive for "metastatic adenocarcinoma consistent with ovarian primary."

After surgery, Patient #22 met with an oncologist who strongly recommended intensive chemotherapy, but she decided to refuse all conventional treatment, instead choosing to begin the cancer version of my therapy. At that point, I redesigned her regimen to include high doses of pancreatic enzymes throughout the day.

In December 1989, her oncologist wrote a summary note to me, which accompanied the records of her recent hospitalization. In his letter, he said, "She is diagnosed as having a Stage IIIC Grade I papillary serous cystadenocarcinoma of the ovary. I have recommended that she receive chemotherapy. She would be a candidate for GOG [Gynecologic Oncology Group] Protocol 104 intravenous cisplatinum and cyclophosphamide versus intraperitoneal cisplatinum and cyclophosphamide. Mrs _____ unfortunately did not wish to pursue the idea of chemotherapy."

She thereafter followed her program diligently for 6 years. By the mid 1990s, her muscle weakness began to progress once again, making return trips to New York difficult, though she continued on the regimen and we worked together by phone. We last spoke in August of 1999, when she wrote after hearing me on the radio. She was 78 at the time, able to walk with a leg brace, and otherwise doing fine, apparently cancer-free nearly 10 years after her diagnosis of extensive ovarian malignancy.

Regarding ovarian cancer patients such as this, DeVita et al report, "Patients with state III disease have a 5-year survival rate of approximately 15%-20% that is dependent in large part on the volume of disease present in the upper abdomen."[10(p1604)]

In this patient's case, the disease did extend into the upper abdomen at the time of diagnosis. Furthermore, these survival statistics refer to patients treated with aggressive chemotherapy, which Patient #22 refused, choosing to follow only my regimen. Her prolonged disease-free survival can be attributed only to her nutritional program.

LUNG CANCER

Harrison's reports that in 2004, approximately 173,000 new cases of lung cancer of all types were diagnosed in the US, 93,000 in men, 80,000 in women. Fully 90% of all cases occur in current or former smokers, so it remains a largely preventable disease.[9(p506)]

Though rates in males have declined in recent years largely due to aggressive anti-smoking campaigns, incidence in women has increased rapidly. Today lung cancer is the leading cancer killer in both sexes, surpassing even breast cancer in women. Despite widely promoted early detection campaigns, public awareness of the disease, and advances in treatment approaches, only 14% of patients survive 5 years. As Minna writes in *Harrison's*, "Thus, primary carcinoma of the lung is a major health problem with a generally grim prognosis."[9]

Pathologists divide lung cancer into 2 major categories, small cell carcinoma and the non-small cell variants, which include squamous carcinoma, adenocarcinoma, large cell carcinoma, and bronchoalveolar carcinoma. Small cell and squamous cell most clearly relate to cigarette smoking, large cell less so. Adenocarcinoma, the most common of the lung cancers, accounts for approximately 40% of all cases, large cell, the rarest, affects only 15% of patients.[10(p928)] Small cell carcinoma responds best to chemotherapy and/or radiation, the non-small cell carcinomas far less so—though few in either group survive 5 years. In the conventional medical world, surgical resection of localized

disease remains the best chance for long-term survival, whatever the subtype.

Patient #23: A 3-year Survivor

Patient #23 is a 63-year old man with a history of myasthenia gravis diagnosed in 1993, which forced him to retire from his high-stress profession. Since then, his myasthenia has waxed and waned, with exacerbations treated with edrophonium chloride (Tensilon).

In June 2003, while playing tennis, Patient #23 developed significant shortness of breath. At a local emergency room, a chest x-ray showed several pleural-based densities in the left lung, and a CT scan revealed several nodular lesions in the left chest pleura up to 2 cm in diameter. Posterior pleural thickening was also noted, thought consistent with mesothelioma. When his symptoms worsened, a second chest x-ray documented a left pleural effusion, subsequently treated with chest tube placement and drainage. After recovering from the acute episode, a second CT scan in July demonstrated a collapsed left lung, a persistent left pleural effusion and numerous large tumors. The official report states, "The largest pleura bases (sic) mass in the left upper lobe laterally measures 2.976 cm. . . . The largest mass in the left lower lobe posteriorly measures 5.39 cm. . . . There are at least 18 pleural based masses present on the left."

Patient #23 then underwent bronchoscopy, left video assisted thoracotomy with pleural biopsies, and pleurodesis. The initial pathology report of the biopsy specimen suggested most likely mesothelioma, but a review at The Armed Forces Institute of Pathology confirmed not mesothelioma, but, as the note describes, "Pleura, left, biopsy: Metastatic papillary adenocarcinoma, of pulmonary origin."

His local doctors also sent the pathology slides to Brigham and Woman's Hospital in Boston, a research center for mesothelioma, where, in July, the tumor was thought most likely a papillary adenocarcinoma of the lung, staged at IIIB.

In late July 2003, Patient #23 decided to consult with Dr David Sugarbaker, a thoracic surgeon and expert in pleural lesions at Brigham and Women's. At Brigham, CT scans of the abdomen and pelvis were clear. A total body PET scan confirmed the extensive left pleural lesions but showed no evidence of distant metastatic disease. Because the disease seemed localized to the chest, Dr Sugarbaker proposed the tumor be treated as if it were a pleural lesion like a mesothelioma with extensive surgery, including removal of the entire left lung, the pericardium, and the left side of the diaphragm.

This debilitating approach seemed excessive, so Patient #23, upon returning home, consulted with an oncologist in the Washington, DC, area who believed the situation should be approached initially not with surgery but instead with an aggressive chemotherapy regimen. If the tumors regressed significantly, a less aggressive procedure might be feasible. The oncologist also consulted with 3 additional thoracic surgeons, including one within the National Institutes of Health system, who felt the surgical approach suggested in Boston was overly aggressive and that the tumor should be treated as a primary lung cancer, not as a pleural tumor like mesothelioma. All believed chemotherapy should be the initial therapy of choice.

Patient #23 then traveled to New York for a consultation with the chief of thoracic surgery at Memorial Sloan-Kettering, who concurred that the disease appeared to be lung cancer that had spread to the pleura, not the other way around. She recommended chemotherapy as the first line treatment, perhaps followed by surgery.

With the debate resolved, in September 2002, Patient #23 began a 4-cycle course of Gemzar and carboplatin. After he completed his last treatment in November 2002, a CT scan revealed some slight worsening in the largest tumor, despite the chemotherapy: "The cystic structure in the posterior left upper lung . . . measuring 4.8 x 6 cm, compared to prior measurements of 4.5 and 5.9 cm. The pleural-based lateral left upper lung lesions are also essentially unchanged, measuring 2.6 and 2.9 cm, compared to prior measurements of 2.8 and 2.9. The rest of the pleural-based masses and left basilar pulmonary nodules are unchanged."

Because the disease had progressed, even slightly, Patient #23 began investigating alternative approaches, learned of our work, and consulted with me in mid-December 2003. At the time, he generally felt well and seemed to have recovered from chemotherapy quickly. Thereafter, he began his nutritional regimen with great dedication and superb compliance. When I saw him for a return office visit 3 months later, in April 2004, he reported feeling "great." Two months later, in June 2004, PET/CT scan testing confirmed improvement in his disease, as he followed only his nutritional regimen. The CT describes: "*CT-CHEST*: Numerous pleural-based masses, and small ones adjacent to the pericardial surface are present. . . . Most of these lesions appear marginally smaller than they previously did (note: compared to the November 2003 CT scan), by a few millimeters. The largest lesion, located posteromedially in the mid-chest, again appears largely necrotic. . . .

"Soft tissue abnormality in the left upper quadrant of the abdomen, anterior to the splenic flexure, appear slightly smaller in overall bulk as compared to the prior study."

Note that the prior radiology reports had not described the lesion in the abdomen, a metastatic focus which would confirm stage IV, not stage III disease. Apparently the lesion had been evident on prior scans, but not described in the official report.

The overall summary of the June PET/CT states, "Impression: PET scans shows numerous pleural-based pathologic foci in the left hemithorax, consistent with numerous foci of metastatic neoplasm. A lesion at the anterior aspect of the left upper quadrant of the abdomen, or immediately adjacent diaphragmatic surface is present. . . .

"CT examination of the chest shows minimal decrease in the overall size of the numerous pleural-based masses in the left hemithorax, and in the region located either in the left upper quadrant of the abdomen."

So, while the PET confirmed residual active cancer, the CT scan indicated universal, though slight, reduction in the many tumors with advancing necrosis in the largest remaining tumor.

Since that time, Patient #23 has continued his nutrition regimen vigorously, and has done extremely well. He has declined all invitations for follow-up CT and PET/CT scanning, stating he wouldn't change his treatment regardless of what the tests show. So, while we don't have clear evidence of additional tumor regression, his continued survival, now at 3 years since he began his nutritional regimen, and his excellent general health speak for themselves.

His course has had only 1 complication. In the spring of 2006, Patient #23 felt well enough to take a trip abroad with his wife. Upon arriving in Europe, he developed severe headaches requiring hospitalization. After CT scans and MRIs of the head showed nothing, he was eventually diagnosed with a cerebrospinal fluid leak. He returned to the United States, the problem eventually resolved, and once again, Patient #23 is back to his usual state of well being.

In analyzing this case, it's important to keep in mind that although the disease was originally classified as stage IIIB lung cancer, the PET/CT scans in June 2004 clearly showed an abdominal lesion that would indicate stage IV metastatic disease. Though evident on prior scans, this lesion was not mentioned in the formal reports. Also, a CT scan done weeks after Patient #23 completed his 4 cycles of aggressive chemotherapy showed no reduction in any of the tumors, and some enlargement. Only after he had followed the nutritional program some 6 months did the PET/CT scans document regression in all tumors and the appearance of significant necrosis in the largest.

For patients with stage IV non-small cell lung cancer, studies show chemotherapy improves average survival by about 1 month over supportive care only. Even with the newest, most aggressive chemotherapy regimens, median survival is still only 9-10 months, with, depending on the regimen, a mere 25%-40% of patients living one year.[10(p969)] Virtually none survive 5 years. Patient #23's 3-year survival and excellent health are even at this point extraordinary.

Patient #24: A 36+-week Survivor

Patient #24 was one of the first I treated with a diagnosis of metastatic lung cancer after I opened my practice in late 1987. He had smoked cigarettes heavily for 28 years, before quitting some 15 years before developing cancer. Otherwise his health had generally been good when in early 1987, he first developed persistent chest pain and cough. When his symptoms did not resolve, he consulted his local physician. After an x-ray revealed a right lung mass, in March 1987 he underwent bronchoscopy with biopsy confirming adenocarcinoma of the lung. A CT showed 2 tumors, one in the right apex, the second in the right hilum, though the left lung appeared clear. Since the disease appeared limited to the right lung, surgery was immediately suggested. Patient #24 initially refused all conventional intervention, but when his symptoms worsened, he agreed to proceed with surgery. In July 1987 he underwent a right pneumonectomy, with the pathology report describing a 2.5-cm lesion, consistent with poorly differentiated adenocarcinoma, extensively invading the hilar lymph nodes. He was staged at III, and proceeded postoperatively with a course of radiation to the chest totaling 4500 rads.

Patient #24 subsequently did well until September 1988, when he developed persistent headaches and olfactory hallucinations described as putrid foul smells. A CT scan of the head in October 1988 revealed multiple tumors located in the temporal, right frontal, and left occipital areas with associated edema.

He was begun on the steroid dexamethasone (Decadron) to reduce the cerebral swelling but his symptoms did not improve. In early November 1988, he proceeded with a 10-day course of radiation to the head, ultimately receiving a total of 3000 rads, with some improvement in his symptoms. In December 1988, a month after completing radiation, a CT scan of the head revealed the situation had worsened despite treatment: "Multiple, bilateral intracerebral ring-enhancing lesions, consistent with metastases. In addition there appears to be an early left cerebellar hemisphere lesion. Many of these were noted on Oct __, 1988. However, several new small areas of abnormality are identified on the present exam, not previously seen."

At this point, with his disease progressing, Patient #24, who already had been investigating alternative approaches to cancer, came to New York for a consultation with me. He reported severe neurological symptoms, including headaches, which had recently recurred despite the use of dexamethasone. He thereafter began his nutritional program with initial great enthusiasm, and in January 1989, after he had completed but a month on his nutritional program, a CT scan showed significant improvement: "When compared to the last previous exam of 12/__/88, there has been diminution both in the size and number of the visualized intracranial lesions. No new areas of abnormality are seen."

According to his oncologist's notes, a bone scan in March 1989 showed clearing of previous noted bone lesions, though I do not have the actual radiology report. At that point, Patient #24 was symptom-free and strong enough to return to his stressful job. Unfortunately, he felt so well he became careless with his supplement regimen and diet, and by April 1989 was by his own admission less than 50% compliant with his overall protocol. Not surprisingly, after his neurological symptoms returned with a vengeance, a CT scan in May 1989 revealed worsening disease: "Increased intracranial edema and size of previously reported intracranial metastases when compared to 3/__/89."

After a discussion with me about the need for perfect compliance, Patient #24 resumed his full program as prescribed. His symptoms rapidly improved and a CT scan in July 1989 demonstrated reduction in all his brain tumors: "The three metastatic lesions on the 5/__/89 CT have decreased in size. No new metastatic lesions are seen."

With the return of his good health, Patient #24 again became careless with his program. I last saw him in September 1989, 9 months after our first session, when after several weeks of poor compliance, his neurological symptoms had returned. Thereafter, he was lost to follow-up. He had no family that I knew of, and despite my efforts, I could never learn what happened to him

In this case, the patient's disease, before he had consulted with me, had progressed despite intensive radiation to the brain.

After he began his nutritional program the brain (and apparently the bone) lesions regressed, only to worsen when compliance fell off. When Patient #24 became more adherent to the prescribed regimen, the brain tumors again improved. Ultimately, he lacked the dedication and discipline to stick to the program as required.

DeVita reports a median survival of 15-18 weeks for patients with multiple metastatic brain lesions from non-small cell lung cancer treated with intensive radiation.[10(p973)] So, despite his compliance problems, this patient's 36+ weeks of survival beat the odds.

And, again despite his lapses, I thought this patient of interest since he remains one of the few I have ever treated with brain metastases from a primary lung neoplasm. Though in recent years occasional patients in this situation have contacted our office seeking information about our therapy, most are so far into the terminal stages of their illness we can't justify trying to treat them. For better or worse, in this age of aggressive oncology, patients facing this diagnosis invariably get shunted frantically and immediately into multi-agent chemotherapy and radiation. Only after months of futile treatment, when the disease explodes and the patient weakens, do they begin looking into alternative options. By then, it is too late. I believe we could help many diagnosed with this terrible condition if, like Patient #24, they came to us earlier in their course, but over the last decade this has simply not been the case. And we do not accept patients for treatment whom we believe we can't help.

COLON CANCER

In 2004, 146,940 new cases of colon cancer were reported in the United States, and 56,730 deaths, making the disease the second leading cancer killer.[9(p527)] Only tumors of the lung claim more lives. The overall incidence has remained fairly steady over the past 30 years, but the mortality rate has dropped, perhaps due to public awareness campaigns emphasizing early diagnosis and regular colonoscopy in those over age 50, the population most vulnerable to the disease.

Over the years, scientists have proposed a number of causative factors, including inherited genetic abnormalities that may play a role in some 25% of all cases. Familial syndromes such as polyposis coli, in which afflicted family members can develop literally thousands of colonic polyps, significantly increase the risk for colon malignancies, as does inflammatory bowel disease, particularly ulcerative colitis. Colon cancer develops in up to 30% of patients with a history of colitis for more than 25 years.

Much if not most colon cancer has been linked to environmental factors, particularly diet. A number of studies support an association with a high intake of animal fat, presumably due to conversion of saturated fatty acids to carcinogenic compounds in the gut. A correlation between high serum cholesterol, obesity, and colon cancer also has been proposed. However, recent studies suggest that fiber in the diet has little influence on incidence rates despite early positive reports claiming a protective effect.

Clinicians traditionally divide colon cancer into a 4-tiered "Dukes" staging system based on the depth of tumor penetration in the bowel wall and named after the researcher who in the 1930s first proposed the schemata. In this hierarchy, Dukes A identifies cancer limited to the superficial layers of the colon with no invasion of underlying tissues. Dukes B indicates the tumor has invaded through the bowel wall but not into regional lymph nodes. Dukes C signifies the disease has spread into local lymph nodes, and Dukes D, the worst, means the disease has metastasized to distant organs such as the liver or lungs. Survival correlates directly with the Dukes stage at the time of diagnosis; more than 90% of patients with Dukes A live 5 years, whereas only 5%, if that many, of those classified as Dukes D live that long.

Studies going back 15 years confirm that chemotherapy with 5-fluorouracil and leucovorin administered after surgical resection of Dukes C tumors improves 5-year survival by about 10% compared to those undergoing surgery alone. However, aggressive chemotherapy offers little long-term benefit once the disease has spread to distant sites.

Patient #25: A 4.5-year Survivor

Patient #25 is a 57-year-old man with a family history pertinent for brain cancer in his mother, colon cancer in an uncle, and lung cancer in a second uncle. He himself had generally been in very good health when, beginning in 2000, he noticed a change in his bowel habits, including increased mucus in his stools, chronic indigestion, bloating, and what he described as gas pains. He adopted a whole-foods, vegetarian way of eating hoping for some relief, but over time his symptoms only worsened.

In mid 2001, he first noticed intermittent bright red blood in his stools. Some months later, in October 2001, he developed symptoms consistent with a bowel obstruction, including severe pain, bloating, abdominal distension, and an inability to move his bowels. When the symptoms resolved after several hours, he chose not to seek medical attention.

Several weeks later, in November 2001, the symptoms returned with a vengeance. He hoped once again to ride out the crisis, but over a 3-day period, the pain, bloating, and distension worsened to the point that he finally went to the local emergency room. A barium enema revealed an "apple core" lesion in the sigmoid colon indicating a tumor. When a subsequent sigmoidoscopy revealed a complete obstruction, the patient went for emergency laparotomy, resection of the sigmoid colon along with the tumor, and placement of a temporary colostomy. The surgeon also discovered, as his operative note reports, "palpable nodules in the liver, which I felt to be more cystic than solid, but there were a couple studs that were solid." He removed one of the liver lesions for evaluation.

The pathologist's summary describes a large colon tumor, but doesn't give exact dimensions, though it states, "The mass locally grossly appears to extend to the underlying adipose tissue," and defines the tumor as "moderately differentiated adenocarcinoma, extending through the bowel wall, and present on the serosal surface." Though cancer had infiltrated 2 of 9 lymph nodes examined, the liver tissue seemed most consistent with a benign hemangioma.

Postoperatively, a carcinoembryonic antigen (CEA) test, a tumor marker for colon cancer, came back elevated at 5.1 (with normal less than 3), an indication of remaining malignant activity. No CEA had been done before surgery, so there were no results for comparison.

Patient #25 did subsequently meet with an oncologist who suspected the tumor had invaded the liver, despite the negative biopsy. He insisted chemotherapy needed to begin quickly, but upon questioning admitted if the cancer had indeed spread, treatment would do little. Patient #25, who already had a strong interest in alternative medicine, decided to refuse conventional treatment and instead began self-medicating with a variety of nutritional supplements. After learning about our work from a local chiropractor, he chose to proceed with our treatment. He contacted our office in early January 2002, but we suggested he come in only after reversal of his colostomy.

Since the patient had been rushed into surgery in crisis from an obstruction, no preoperative CT scan had been done. Finally, in mid-January, his doctors pushed for a scan, which revealed evidence of multiple metastatic lesions in the liver, as the official report describes: "Unfortunately, within the liver there are numerous small hypo-enhancing lesions, some of these are very hypo enhancing to the point where one might consider cysts, but others are more intermediate density. Five-millimeter-thick slices were obtained to increase the sensitivity. The largest of these lesions is only about 1 x 1.5 cm. These are suspicious for metastatic disease."

The radiologist also noted "very minimal subpleural densities seen at the mid left lung field" which he felt "should be rechecked within several months." In his summary, he reports that "I suppose the liver findings increase suspicions of the left lower lobe findings however my feeling is that the lung changes will prove to be benign."

Quite likely, based on the CT findings, cancer had spread into the liver and possibly to the lungs. The negative liver biopsy, the patient was told, might only indicate that the liver contained both benign and malignant nodules, as the CT scan seemed to show.

In late January 2002, the patient returned to surgery for reversal of the colostomy and lysis of adhesions that had formed since the first operation. During the procedure, unfortunately, none of the liver lesions were biopsied.

When Patient #25 was first seen in my office in mid March 2002, he seemed enthusiastic about the therapy and subsequently followed the regimen faithfully. Today, more than 4.5 years on treatment and 5 years from his original diagnosis, he remains fully compliant and enjoys excellent health.

Over the years that he has been my patient, Patient #25 has chosen not to undergo any further CT scans, a decision I have respected. He says no matter what the scans show, he wouldn't agree to chemotherapy nor would he change his treatment. He doesn't want the radiation exposure, which is significant, the worry, or the expense. So, I have no idea what has happened to the liver, or its lesions, I only know the patient is alive and well.

Even if we disregard the CT liver findings for a moment, a number of salient signs point toward a dismal prognosis. The literature reports that patients who initially present with an obstructing lesion have a far worse prognosis than those who don't, even if the disease is otherwise localized. DeVita states, "The presence of obstruction has been found to reduce the 5-year survival rate to 31%, as compared with 72% for patients without obstruction."[10(p1227)]

Furthermore, in this patient's case, the fact that the tumor had already invaded through the bowel wall and infiltrated into 2 lymph nodes signaled future trouble. The CEA level after surgery, though only mildly elevated, nonetheless also warned of a future recurrence—regardless of what may have been going on in the liver. *Harrison's* reports that a high CEA *before* surgery, whatever the stage, suggests a poor prognosis: "Regardless of the clinicopathological stage, a preoperative elevation of the plasma carcinoembryonic antigen (CEA) level predicts eventual tumor recurrence."[9(p530)]

Patient #25's elevated postoperative CEA served as an even more worrisome prognostic indicator. But finally, if we accept the expert radiologist's conclusion that cancer had infiltrated the liver, the prognosis turns dire. DeVita reports median survivals in the range of 4.2 to 8.7 for patients diagnosed with metastatic colon cancer receiving aggressive chemotherapy.[10(p1227)] In a large-scale study. Manfredi et al report "1- and 5-year survival rates were 34.8% and 3.3% for synchronous liver metastases (meaning liver metastases occurring at the time of the original diagnosis of colon cancer).[19] These statistics include patients with solitary liver lesions, which can at times be resected along with the primary colon tumor, allowing for long-term survival. In this case, Patient #25, with multiple malignant appearing tumors on CT scan, not only has far outlived the predicted lifespan but has successfully avoided the toxic treatments his oncologist insisted 5 years ago needed to be done.

PANCREATIC CANCER

According to *Harrison's*, in 2004, pancreatic cancer killed 31,270 Americans, making it the fourth major cancer killer.[9(p537)] It is particularly virulent, killing 98% of all patients within a year of diagnosis. The cause still eludes orthodox thinkers, though over the years they have uncovered some clues. Cigarette smoking increases the risk 3 times, with up to 30% of cases linked to the habit. Chronic pancreatitis and obesity predispose to the illness, as does diabetes mellitus. Experts argue for a genetic component in some families, with approximately 3% to 9% of all cases thought due to such an inherited predisposition.[10(p1127)]

Ninety percent of all cases begin in the enzyme-producing (exocrine) cells of the pancreas, only 5% to 10% in the endocrine, hormone-secreting tissue. For the most common form, adenocarcinoma of the exocrine pancreas, the conventional medical literature reports an average survival for those with metastatic disease in the range of 3-6 months from the time of diagnosis, while earlier stage patients live 10-14 months on average. The prospects for long-term survival remain dismal whatever the stage.

In the orthodox oncology world, surgical resection of localized adenocarcinoma provides the only prospect for long-term

survival, but at the time of diagnosis, most patients already have evidence of widespread cancer and for them, surgery offers no benefit. Chemotherapy does little; the FDA approved Gemzar (gemcitabine HCL), specifically for the treatment of pancreatic adenocarcinoma after data from clinical trials showed that patients treated with the drug lived an average of 5.6 months, 4 weeks longer than those receiving other forms of chemotherapy.[7] Researchers did claim that in addition to this slight survival advantage, 29% of Gemzar-treated patients enjoyed an improved "quality of life," defined as less pain, increased appetite, and an overall slight enhancement of their general "well being." Though short-lived, such benefits still represented an advance over previous options for the disease. Recently, investigators at a number of academic centers have reported little additional improvement when they added other powerful chemotherapy agents into the Gemzar mix.

Scientists divide the rarer islet cell tumors into many subtypes, depending on the specific hormone released; for example, insulinomas secrete insulin, glucagonomas, glucagon, and gastrinomas, gastrin. These cancers may secrete these hormone products in dangerous amounts—frequently patients with insulinomas first seek medical advice after repeatedly fainting between meals, when excessive insulin drives so much glucose out of the bloodstream that blood sugar drops precipitously. Whatever the particular type, islet cell carcinomas tend to be less aggressive than adenocarcinomas: even patients with metastatic disease at the time of diagnosis can live 5 years due to its inherently slow progression, but it usually does progress, eventually with fatal results.

Patient #26: A 15-year Survivor

Patient #26, like so many of my patients, has an unusual background, with a graduate degree, study abroad, and expertise in art. Before we first met, he had worked successfully in business for many years. His very devoted wife had a PhD and had, before retirement, worked as a college professor.

He had been in good health when in July of 1991, at age 70, a routine chest x-ray at the time of his yearly physical revealed a small right lung nodule suspicious for possible malignancy. A repeat x-ray in August 1991 again demonstrated "a parenchymal nodule in the right mid lung." CT scan studies of the chest in late August 1991 confirmed a "6 millimeter nodule in peripheral lateral aspect of right upper lobe. It is consistent with bronchogenic carcinoma, metastatic lesion or granuloma." In addition, the radiologist noted "an enlarged lymph node posterior to the ascending thoracic aorta."

A CT scan of the brain in early September was clear, but a CT scan of the abdomen revealed extensive disease throughout: "There are about 4 lesions in the upper right lobe of the liver. . . . An ultrasound examination is recommended for further evaluation. . . .

"There is a round enlargement of the right adrenal gland up to 2 cm in diameter. There is also what appears to be diffuse enlargement of the left adrenal. . . . Both these findings are suspicious for metastatic disease. There is a mass in what may be the cephalad portion of the head of the pancreas or it is a mass or adenopathy just adjacent to the head. The mass measures about 4.5 cm in its greater diameter."

A bone scan the same day revealed "abnormal activity of the right hip and right shoulder suggesting metastatic disease."

Though the situation appeared dismal, the patient's doctors still needed a biopsy specimen to confirm not only cancer, but also the most likely primary site. After reviewing the scans, they decided the lung lesion to be most accessible for tissue sampling, so in late September, Patient #26 was admitted to his local hospital for mediastinoscopy and a limited right thoracotomy. In his admission note, the surgeon reports his belief that the situation was most consistent with metastatic pancreatic cancer, not lung cancer that had spread into the abdomen: "At some point, I suspect he will require oncology and radiation medicine consultation for what is most likely a pancreatic carcinoma with multiple metastatic lesions."

The lung nodule proved to be adenocarcinoma, as the pathology report describes: "Right upper lobe lung nodule, biopsy: Infiltrative moderately differentiated adenocarcinoma."

After surgery, an ultrasound revealed the liver lesions most likely represented metastatic cancer: "Areas consistent with metastatic involvement of the liver, the largest of which is approximately 3.4 to 4 cm in maximal dimension near the hilus. The second is just under 2 cm in the right lobe and possibly a third smaller one in the right lobe."

With the testing done, Patient #26 was told he had metastatic pancreatic cancer, perhaps 2 months to live, and that neither chemotherapy nor radiation would be of benefit. But, instead of giving up and getting his affairs in order as the doctors suggested, he and his wife decided to take the situation into their own hands. They both began reading voraciously about cancer, nutrition, and alternatives. He began ingesting large numbers of supplements, including vitamin C, vitamin E, even pancreatic enzymes after reading an article discussing our work. He switched his eating habits to a largely plant based, raw diet, and began juicing intensively, with his devoted wife's help. When he felt sufficiently recovered from surgery, he decided to consult with me.

I first saw Patient #26 in December 1991. Despite his prognosis, he seemed determined to fight his disease, and talked as if he had absolute faith that he could get well on my therapy. He subsequently proved to be a very compliant patient, and the results, though gradual in coming, were gratifying. Within a year, his general health had improved substantially, and a CT scan of the abdomen in February 1993—some 15 months after his initial diagnosis—showed no change in any of the lesions. Technically, the cancer hadn't improved, but it hadn't advanced, and he was still alive.

After that set of scans Patient #26 told me he wanted no more testing. Since he had already long outlived his doctors' dismal predictions, he figured he didn't care what the scans might show and wouldn't change his treatment anyway. So he continued his therapy and enjoyed with his wife the retirement for which they had long planned.

In 1997, after he had followed his nutritional protocol for 5

years, he agreed—with some pleading from me—to allow radiographic studies. A CT of the abdomen in March 1997 showed 2 mildly enlarged adrenal glands and a single, very small, less than 1 cm mass in the dome of the liver. The other large liver lesions were gone. The radiologist in his report described the pancreas as normal—the previously documented large tumor had simply disappeared: "The liver demonstrated a single small hypodense area in the dome of the liver which has the appearance of a cyst, measuring well less than 1 cm. A metastatic lesion is still a possibility especially in view of the patient's history of lung cancer and adrenal mass. . . . The adrenal glands are both abnormal. . . . The pancreas, the spleen and the kidneys are within normal limits. There is no evidence of periaortic lymphadenopathy."

Then, 16 months later, in July of 1998, nearly 7 years after his diagnosis, Patient #26 agreed to undergo repeat scanning. The radiologist reports: "Reading the report from the 1993 study it sounded like the patient had obvious metastatic disease and the largest structure being a large porta hepatis and peripancreatic mass. No such masses are seen today. There is no adenopathy. The adrenals are prominent and there are two very small liver lesions that cannot be characterized because of their small size."

Thereafter, Patient #26 continued his program and continued doing well until he drove his car off the side of a road in 2004. Unfortunately, he required lengthy rehabilitation, followed by life in an assisted care facility. His wife, 3 years older, no longer able to care for herself at 87 years old, also entered an assisted care facility, where she recently died. But Patient #26 at age 85 years old is still alive, now more than 15 years since his diagnosis of terminal metastatic pancreatic adenocarcinoma.

His case does not require much discussion. He was diagnosed appropriately with terminal cancer and given 2 months to live. He did his program, the tumors went away, and he survived.

Patient #27: A 10-year Survivor

(Editor's note: In the print version of this article, this was Patient #6.)

In 1985, Patient #27 had undergone surgery for localized colon cancer but subsequently received no adjuvant radiation or chemotherapy for the disease. He thereafter did very well until he developed a large right neck mass about the size of a golf ball in October 1996 while traveling outside the country. Upon returning to the United States in December 1996 he underwent a biopsy, which confirmed "adenocarcinoma." His doctors assumed the cancer had metastasized from some abdominal organ, though they weren't initially certain which one. Patient #27 then traveled to Memorial Sloan-Kettering in New York, where he was seen in early January 1997. There, after the biopsy slides were reviewed and adenocarcinoma confirmed, the pathologist reported the neck disease most likely represented metastasis from a new primary tumor, not recurrent colon cancer, as the note describes "metastatic poorly differentiated adenocarcinoma with focal signet ring cell features to lymph node. Possible primary sites include lung, stomach and pancreas."

Patient #27 then underwent CT scanning of the chest and abdomen as well as bronchoscopy, all of which were unrevealing. A CT scan of the neck demonstrated "Pathologic appearing adenopathy within the right posterior triangle."

A PET scan a week later revealed "(1) Abnormal FDG Pet scan showing focal FDG uptake in the right posterior neck, consistent with lymph node metastasis. (2) Focal uptake seen in the right upper quadrant, just anterior to the right kidney, may be due to primary tumor. The location could be in the head of the pancreas or the second part of the duodenum."

At this point, after the Memorial doctors concluded the primary to be most likely pancreatic cancer, they suggested a conservative approach, holding off treatment until the disease further advanced. However, Patient #27 had learned of our treatment approach, decided to proceed with us, and first consulted with me in January 1997. Thereafter, he followed his program diligently, with good results. Follow up MRIs of the abdomen and pelvis at Memorial in July and October 1997 revealed no evidence of cancer anywhere. The October report reads, "Since the previous study of 7/__/97: (10 No significant interval change is appreciated. (2) No evidence for neoplasm in the abdomen. (3) No abnormalities are identified in the pelvis."

Patient #27 continued his aggressive protocol for 3 years, before winding down to a maintenance regimen. Today, nearly 10 years after he started his nutritional regimen, he appears to be in excellent health, enjoys retirement, and remains free of his once life-threatening cancer.

This case is very straightforward. Biopsy confirmed metastatic carcinoma, considered by the Memorial experts most likely, based on the PET scan, to be of pancreatic origin. The patient followed his regimen faithfully; subsequent scans showed no evidence of disease, and he remains cancer-free to date.

Patient #28: A 10.5-year Survivor

Patient #28 had been previously very healthy when he first developed chronic heartburn, gradual weight loss, and persistent diarrhea throughout the summer of 1992. In August of that year, he suddenly became very weak and short of breath; when his local doctor found him to be anemic, he was hospitalized for a transfusion. An endoscopy showed multiple stomach ulcers, thought to be the source of the blood loss. Additional testing revealed elevated blood levels of the hormone gastrin, which was assumed to be responsible for the ulcerations. Usually, excess blood gastrin warns of a hormone secreting pancreatic tumor, but despite extensive testing, no such lesion could be found. So, after prescribing omeprazole (Prilosec), his doctors sent Patient #28 home.

On the medication, he actually did fairly well, with no further bouts of severe anemia until October 1994, when his gastrin levels on routine blood testing were again elevated. This time around, a CT scan showed a 6 to 7 cm mass in the retroperitoneal area of the abdomen. After a series of delays, he underwent exploratory abdominal surgery in March of 1995 at a local hospital; unfortunately, his surgeon discovered a very large tumor that because of its size and degree of infiltration throughout the pancreas could not be removed, though it was biopsied. In addition, a metastatic

lesion at the base of the liver was resected. The operative note describes the extent of disease: "There was however a large uncinate process grossly clinically involved with tumor. Also, the whole head of the pancreas clinically was involved with tumor as well. Lateral to the head of the pancreas on the other side of the SMV and the neck and body region was also palpable tumor.

"Palpation and exploration of the porta hepatis revealed approximately a 3 cm mass noted. . . . This was sharply dissected and free (sic) and sent to pathology for quick frozen section."

The pathology report confirms that the pancreatic and portal lymph node biopsies were consistent with "metastatic carcinoid-islet cell tumor."

After recovering from surgery, Patient #28 decided to travel for a second opinion to the Mayo Clinic, where he was seen in May of 1995. At Mayo, the original slides were reviewed, and the diagnosis of islet cell carcinoma verified. At the time, the consulting oncologist recommended no additional therapy, as the official Mayo note reads: "I briefly discussed the case with my surgical colleague, Dr _____. He did not feel that any further surgical intervention was warranted at this time. A Whipple procedure would be entirely palliative at this time. The patient may eventually come to a bypass procedure as there is some bile duct dilatation on CT scan. We discussed the fact that there is no good evidence for benefit from radiotherapy. . . . I discussed with him the role of chemotherapy in patients with islet cell carcinoma . . . there is no evidence that earlier treatment will show improved response and survival. Given his asymptomatic state, I did not recommend any intervention at this time."

Initially, Patient #28 continued on only his Prilosec. By early 1996, he wasn't content to wait until the disease progressed, so he began investigating alternative cancer therapies. After learning of my work, he first came to my office in March of 1996 and subsequently proved to be determined, very diligent, and very disciplined with his nutritional regimen.

In June 1997, a little over a year after he first began treatment, his local doctor sent him for a follow-up CT scan to check his progress. The radiologist reported "no significant change in the appearance of the patient's pancreatic mass since previous examinations." The tumor was still there, but no bigger.

For several years, since he felt so well, he avoided any testing until agreeing to another scan in September 2002. The official report stated: "Findings: Images of the pancreas demonstrate no mass lesions. The liver, spleen adrenal glands and kidneys are unremarkable. Impression: 1. Normal CT scan of the abdomen."

The large tumor in his pancreas had simply gone away. A more recent scan was also completely clear, and today, 10 years after beginning his nutritional therapy, Patient #28 continues on his program and continues doing well.

This is not a complicated case. Patient #28 at surgery was found to have unresectable disease that had metastasized to the porta hepatis lymph nodes. Biopsies of the large pancreatic mass and the metastatic lesion revealed islet cell carcinoma, findings confirmed at the Mayo Clinic. Patient #28 then began my program, followed it faithfully, his tumors went away, and he remains cancer free and in excellent health, 10.5 years from his original diagnosis.

Patient #29: A 6-year Survivor

In November of 2000, Patient #29 first reported a gradual 25-pound weight loss to her local physician. A CT scan of the abdomen in early December 2000 revealed a 3.4 cm mass in the head of the pancreas, but no evidence of metastatic disease. A subsequent CT scan guided needle biopsy in February 2001 confirmed a "poorly differentiated adenocarcinoma, ductal type," the most aggressive form of pancreatic cancer. The slides were also sent to the Mayo Clinic, where the consulting physicians agreed with the histological diagnosis.

Because the disease seemed localized to the pancreas, the patient's physicians thought the tumor might be operable. She was urged to undergo extensive surgery, but the patient decided the risks were too great, the potential benefits too meager, to warrant such an approach. She subsequently learned of our approach and in March 2001, consulted with Dr Isaacs in our office. In April 2001, a month after she began her nutrition treatment, repeat CT scans revealed a 3.2-cm mass in the head of the pancreas, with no evidence of metastatic disease.

A follow-up CT scan performed in January 2002, some 10 months after she began treatment with Dr Isaacs, indicated a 3.0 x 3.0 cm mass in the head of the pancreas, smaller compared to the scan of April 2001. The next CT in July 2003, after Patient #29 had followed her nutritional regimen for more than 2 years, showed a 3.16 x 2.6-cm mass in the head of the pancreas, and a scan not quite a year later revealed a 3 x 2.8-cm mass.

Patient #29, now a 6-year survivor, currently is in good health despite her original poor prognosis. In her case, the CT scans show perhaps some slight shrinkage in her tumor, but no spread. Given the aggressive nature of pancreatic adenocarcinoma in general, and the virulent nature of the poorly differentiated variety diagnosed in this case, its tendency to metastasize and kill within a year even when aggressively treated, this patient's course has truly been remarkable. She has been able to avoid aggressive surgery, chemotherapy, and radiation while enjoying excellent health.

As a side note, we do find in our practice that though tumors often disappear—as in the previously discussed cases of pancreatic cancer—at times they seem to stabilize, sometimes for many years.

Patient #30: A 5.5-year Survivor

Patient #30, with a long history of gastroesophageal reflux disease, decided in January of 2001 to undergo laparoscopic surgery for correction of what was presumed to be a simple hiatal hernia. During the procedure, his doctor discovered "multiple umbilicated, white, firm, and gritty tumors in both the right and left lobes of the liver, apparently occupying approximately 50% of the volume of the liver."

A biopsy of one of the liver lesions confirmed "poorly differentiated metastatic carcinoma," with some "neuroendocrine differentiation." The final diagnosis reads, "Liver needle biopsy positive for malignancy, favor metastatic adenocarcinoma."

After surgery, a CT of the chest, abdomen, and pelvis revealed

a large 6.5 x 3.7-cm mass in the tail of the pancreas, with "diffuse hepatic metastases." The radiologist wrote, "This likely represents primary pancreatic adenocarcinoma."

The patient subsequently met with an oncologist at Barnes Hospital who suggested aggressive chemotherapy with cisplatin and etoposide for 4 cycles, though he admitted that even with chemotherapy, the disease would ultimately progress and prove deadly. Before agreeing to the treatment, in February of 2001, Patient #30 traveled to Memorial Sloan-Kettering in New York for a second opinion. There, the Memorial pathologists reviewed the slides and confirmed a very aggressive pancreatic carcinoma. The consulting oncologist then proposed the same chemotherapy protocol that had been previously recommended but again warned that even with aggressive treatment, Patient #30 might live 2 years at most. Chemotherapy, as he had been told before, might shrink his tumors and prolong his life, but would not provide a long-term solution.

At the time of the Memorial consultation, Patient #30 was not doing well clinically. The official note states, "The patient has significant fatigue, takes naps usually by the end of the afternoon. He does notice recent onset back pain which is alleviated with pain pills. He has significant nausea without vomiting. . . . He does have occasional palpitations but denies flushing. He notes mildly decreased appetite and has had an approximately ten-pound weight loss.

After returning home, Patient #30 began the proposed course of chemotherapy in February 2001 administered by his local oncologist. After his first cycle, a CT scan in February 2001 indicated some response to chemotherapy: "As on the prior examination, there is a low attenuation mass within the tail of the pancreas. The mass is smaller is size, measuring 6.4 cm x 3.0 cm on the current examination . . . on today's study there are innumerable low attenuation lesions throughout the liver, measuring up to 2 cm in diameter, consistent with metastatic disease."

After the second cycle of chemotherapy, a repeat CT scan in March 2001 showed "1. Marked improvement in numerous liver metastases with a decreased (sic) in size as well of the pancreatic tail mass."

Patient #30 completed the first 3 cycles of chemotherapy without much difficulty, but during the fourth round he became so ill the drugs had to be discontinued in April of 2001. Then, after learning about our work, he decided to forgo further chemotherapy and proceed with our treatment.

I first saw Patient #30 in my office in May 2001, a month after his last round of drugs. Thereafter Patient #30 proved to be very compliant with his nutritional regimen and within months he reported significant improvement in his general health. His many symptoms, including persistent debilitating fatigue, had resolved.

A CT of the abdomen in February 2002, 10 months after he had first come to our office, indicated multiple small lesions in the liver, which had been seen on previous scans, as discussed in the official note: "1. Multiple tiny lesions in the liver, all less than 3 mm in size. Some of these lesions have been noted on prior studies which were obtained at slightly larger collination (*calibration*) and have not changed since the previous studies. 2. No pancreatic lesion. 3. No abdomen or pelvic lesion."

At that point, I made several adjustments in his regimen. A repeat CT scan in October 2002, some 17 months after he had first begun his nutritional therapy, confirmed that all the liver tumors were gone. The report states, "1. No pancreatic lesion identified. 2. Multiple tiny lesions in the liver seen on the prior examination are not identified on today's study."

Follow-up scans in March 2003 and June 2004 were also completely clear. His most recent scan in March 2005 revealed, "The liver, gallbladder, pancreas, spleen and both kidneys appear unremarkable."

He has been following his program for 5.5 years and is nearly 6 years from his original diagnosis of very advanced and very terminal pancreatic carcinoma. He remains disease-free.

This case, like the previous 4, is not complicated. Though aggressive chemotherapy did shrink the primary pancreatic as well as the liver tumors, the disease did not completely regress on drug treatment. Futhermore, the experts he consulted at Barnes and Memorial Sloan-Kettering warned him even if he showed some response, the benefit would be short-lived. No one, even the most fanatical oncologist, claims chemotherapy cures pancreatic carcinoma metastatic to the liver. Finally, it was only on his nutritional regimen that the tumors regressed completely and stayed that way.

Patient #31: A 24-year Kelley Survivor

I first learned of Patient #31 while reviewing the records of patients with pancreatic cancer treated by Dr Kelley. I thought I would include her to illustrate the kind of successes uncovered in Dr Kelley's files, as I pursued my 5-year study of his therapy.

In early 1980, Patient #31 first experienced occasional bouts of mid-abdominal pain that gradually worsened over a 2-year period. Despite the symptoms, she did not seek medical assistance until August 1982, when she was admitted to the local emergency room of her Midwest town with excruciating pain. When an ultrasound showed only gallstones, her doctors assumed she might be suffering from gallbladder disease and proposed cholecystectomy.

Several days later, she underwent exploratory surgery and removal of the gallbladder. However, the surgeon also discovered a pancreatic mass that had invaded into the surrounding tissues, as well as a single 1-cm tumor in the liver, which he biopsied. Due to the extent of disease, he made no attempt to excise the pancreatic tumor.

The liver specimen proved consistent with adenocarcinoma that had spread from a pancreatic primary. After recovering from surgery, Patient #31 met with an oncologist, who told her that although chemotherapy might prolong her life slightly, no treatment could cure her disease. He suggested she get her "affairs in order." In the official records, this physician wrote: "The patient's prognosis is judged to be between 9 and 15 months at most."

After recovering from surgery, Patient #31 decided to seek out a second opinion at the Mayo Clinic in Rochester, Minn. When seen at Mayo in mid-September, a CT scan revealed an enlarged pancreas, and blood studies indicated abnormal liver function

tests. At the conclusion of his evaluation, the consulting oncologist wrote, in the official discharge summary: "I had a long discussion with her regarding treatment for her cancer. At the present time I would favor simply observation since we know of no known treatment that will necessarily prolong her life. Since she is feeling well at the present time I did not feel justified in making her symptomatic from the side effects of chemotherapy."

Fortunately, Patient #31 learned of Dr Kelley's work from a local health food store owner, and shortly thereafter began treatment with him in December of 1982. She responded quickly, and within 6 months was back to working long days in the family business.

By the time I completed my study in 1987, Dr Kelley had closed down his office and disappeared from sight. After I started my own practice, I lost touch with Patient #31 until she referred a patient to me in the mid 1990s. At that time she was in excellent health, still following her prescribed diet and still taking pancreatic enzymes. I heard recently that she is still alive, still active, and still enjoying her life, now 24 years from her original Mayo-confirmed diagnosis of metastatic adenocarcinoma of the pancreas.

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