



From Both Ends of the
STETHOSCOPE

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DEDICATION

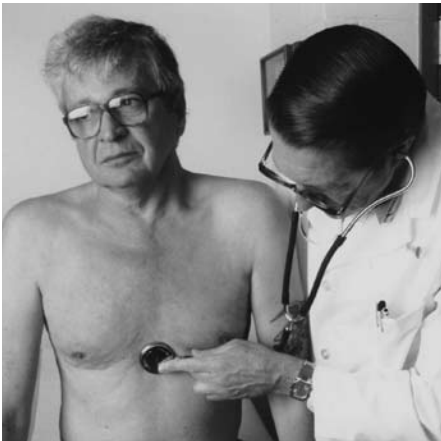
This book is dedicated to the patients I have cared for. They taught me about humanity, humility, and hope. It is also dedicated to Louise Nett who gave me strength.

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FOREWORD

The stethoscope was invented by Laennec from Quimper, France. Laennec was a master clinician and pathologist. He made careful clinical observations of his patients, most of who had tuberculosis. When his patients died, he did autopsies to find out the anatomical basis of his physical examinations. Physical examinations included observation, palpation, percussion and direct auscultation of the chest by placing the ear over the regions of interest. On one occasion, he encountered an obese and buxom woman, with pendulous breasts. He felt uncomfortable doing direct auscultation, and struck upon the brilliant idea of coiling a piece of writing paper, placing one end on her chest, and placing the other end of the coiled paper on his ear for transmission of chest sounds. He was immediately delighted to be able to hear the “murmur of respiration,” as he termed it, and the sounds of the heart valves opening and closing. Later he devel-



oped various wooden rudimentary stethoscopes, which today are featured in medical museums. A few old physicians treasure these keepsakes. Soon flexible rubber tubes were used for binaural stethoscopes which are similar to those doctors use today. Laennec first reported his invention to the Parisian Academy of Science in 1818.

Laennec thus introduced the first technology, which allowed physicians to indirectly perceive the anatomical reasons for the patients suffering. Much later, came the other common instruments used in today's medical practice, the electrocardiogram, which records the electrical activity of the heart, invented by Einthoven in 1882, the x-ray in 1896 by Roentgen, and the sphygmomanometer to measure blood pressure, by Rico Rossi, in 1904. Many other technologies followed and today we have a burgeoning array of CT scans, MRI machines, and ultrasonic devices, just to mention the most commonly used non-invasive diagnostic technologies.

As technology advanced, the human element has sadly become less important in the minds of many physicians. Now new technology often dominates the thinking and therapeutic decision making by physicians, and new barriers to the patient-physician relationships have resulted.

This book cites selected experiences of the author beginning in medical school, often using the stethoscope to help diagnose rare disease states. Several patient vignettes and events are included where the art of medicine became more important than medical technology. Profound events where the author became the patient are included. Some of the experiences were wonderful and satisfying, and others disappointing and frustrating. These experiences cover more than a half century of medical advances and technology and, in some ways, the decline of the doctor-patient relationship.

The stethoscope became the first new technology to aid physicians in their service of patients, and many of the new technologies have amplified this ability immensely. But nothing can replace the experience and kindness of the dedicated physician today, interacting with a patient who suffers from acute illness or chronically failing health.

P R E F A C E

FROM BOTH ENDS
OF THE STETHOSCOPE

I entered medical school the fall of 1954, full of enthusiasm for my professional training which was about to begin. I walked down the corridors to my first class, along with my eager classmates, wearing a long starched white coat, symbolic of the physician. I can remember my first use of the stethoscope in 1955 in a series of tutorial and physical diagnosis sessions given to the sophomore students. I well recall touching my first patient as I tried to hear and understand the sounds that were transmitted to my naïve ears. From the beginning of my career, I found use of the stethoscope to be a critical part of physical examination. It also provided a spiritual bonding with my patients as my hand placed the instrument over the heart, lungs, and abdomen, as I was touching or holding the patient with my other arm.

Now, some half century later, I have many memories of the fascinating and challenging patients I have examined and treated. Also, during this 50-year period I have had four open heart operations, five cancers (prostate, three skin, and bladder cancer). Besides these major events I have had several cardiac catheterizations, a cardiac arrest outside the hospital with resuscitation followed by an implanted defibrillator (so far it has already resuscitated me once with a troubling arrhythmia disorder). Most recently I had a hip replacement followed by seemingly endless complications. And even after I was nearly recovered from this series of life-threatening and painful situations, I most recently developed bladder cancer!

Some of these experiences as both a physician and a patient, both good and bad, are captured in this brief book that I have written for

both doctors and the patients they serve. To do this, I have had to use a moderate amount of medical terminology, but my messages have been and will be understood by physician and patients alike.

Today medicine is in a crisis. We spend too much money on so-called health care and little on prevention. Care has become technically oriented, impersonal, and not accessible or affordable for many. Far too many errors occur in hospitals and doctors' offices, sometimes leading to death!

Although the future of medicine is clouded, there are bright rays of hope shining through the clouds. There will always be caring doctors to serve patients in need. Medical knowledge and technology will continue to advance, but the stethoscope will always remain a symbol of technology as it is applied to the doctor-patient relationship.

PART I

LEARNING AND LOVING MEDICINE



C H A P T E R 1

MY LIFETIME LEARNING COPD



I got really high after my freshman year in medical school at the University of Colorado. To be specific, I spent a summer in research in acclimatization to altitude in 1955. During this summer I lived for one month on Mt. Evans, located 14,260 feet above sea level. Our team was made up of four medical students and two faculty members from the Department of Physiology.

Oxygen saturation, which refers to the amount of oxygen in the arterial blood which is oxygenated by the lungs is 96-98% at sea level. Oxygen saturation in normal people at 14,000 feet is reduced to 75-80%. Here I experienced the symptoms of acute oxygen starvation, medically termed hypoxia. At first I got “high altitude sickness.” After unloading all the heavy equipment in the research “shack,” I became suddenly very short of breath and had a terrible headache. I began coughing up frothing sputum. I breathed oxygen from a weather balloon source and was then taken back to Denver. My chest x-ray showed “patchy shadows,” which we now know represent pulmonary edema, a leakage of fluid into the alveolar spaces of the lungs. By this time I felt well and returned to the summit the next day to continue our studies.

Later I acclimatized to this chronic lack of oxygen as we lived and did our experiments and could function there normally with comfort.

During this period, I learned to make measurements of the oxygen and carbon dioxide in the blood and air, which served me well later in my student career. In fact, this experience as a young medical student followed me for my whole professional career, beginning with my first clinical experience.

I can still remember my trepidation as I was assigned my first patient to “work-up,” as a junior medical student on the wards of the old Colorado General Hospital. This was in 1957, over half a century ago. I introduced myself to a large man, who said he had come to the hospital because he was short of breath, fatigued, and had headaches. I don’t remember all the details of his history, but I recall that he was a heavy smoker. He had just put out a cigarette when I approached him. On examination I was impressed with the blue color around his mouth and lips and his swollen ankles. In those days, we were not allowed to review the medical record to get information about the diagnosis before examining the patient, as part of our training.

I listened to his chest with my stethoscope and could hear only scattered and distant breath sounds. The chest was nearly quiet to my ears. Part of our evaluation of patients included doing blood counts and urinalysis. We were not taught to do simple lung functions. I noticed that he had excess red blood cells, but no excess in other elements of the blood such as white cells and clotting cells when I looked at his blood under the microscope, which we learned to do as students. I concluded that the man had “heart failure” and “polycythemia,” the medical term for excess red cell formation.

My professor was a striking woman, in fact, the person who interviewed me to enter medical school in 1952. She said, “Well, Tom, you are not quite right. What this man has is emphysema.” She explained that emphysema often results in low blood oxygen, the body responds by producing more red cells to help compensate for the lack of oxygen. This is known medically as “secondary polycythemia.” I had never

heard much about emphysema in my studies and began to read about it in textbooks. The problem of low oxygen and high carbon dioxide causing a strain on the right side of the heart was the reason for his edema. But in those days it was rare to measure oxygen in the arterial blood. The procedure took 2-3 hours and was mostly used in research such as we were doing on Mt. Evans. Modern methods of blood gas analysis and oximeters were not available for many decades to come. Even though I was disappointed that I couldn't come up with the right diagnosis, that wasn't the point. I had my first patient "under my belt." I will never forget him. In fact, I began to understand this "new" disease process. This encounter, coupled with my own experience with altitude-related hypoxia and the difficulties of measuring arterial blood oxygen, would influence my career for the next half century!

I learned that emphysema was an obstructive lung disease. The lungs become overinflated and the alveolar spaces are "blown out." They lose their elasticity and don't empty normally. This fact interested me and caused me to accept the opportunity to do student research with a surgeon I admired, Dr. Ben Eiseman, who is still on the faculty of the University of Colorado at age 91! Together we proposed that I try to develop a method to produce emphysema in large animals; i.e., dogs. Some very old experiments had suggested that placing a one-way valve in the upper airway (trachea) could cause emphysema by trapping air in the lungs. These were all short experiments, because the moving parts of the valve got stuck with mucus and the animals didn't live long. For my experiments, I used a device with no moving parts known as a Venturi tube, which made breathing in easier than breathing out. My mentor and I thought this obstructive device would cause dogs to overinflate. This way they would trap more air in the lungs than they could exhale. This would stretch the lungs and damage the alveoli, we thought. I spent the summer putting these devices in dogs' tracheas and measuring their lung volumes by analyzing expired air by the laborious technique which I had learned on Mt. Evans. This

occupied most of my summer that year and this proved very exciting. I was working alone in the laboratory, including doing all of the surgery and air volume tests myself. Much to my frustration, I did not produce emphysema. In fact, the dogs learned to breathe at a lower than normal volume and the lungs were actually under inflated at the end of three months' experience. But I learned a lot about animal experimentation and read incessantly about emphysema during that summer.

At the beginning of my senior year, I presented a paper to my colleagues at the Student Honor Society on emphysema because I was so fascinated by it. I asked the head of pulmonary medicine at the University of Colorado, Roger S. Mitchell, to be my discussant. He congratulated me on my interest and knowledge of emphysema. I was also stimulated to write a scientific paper on attempts to produce experimental emphysema. This was presented at the first Aspen Emphysema Conference (now known as the Aspen Lung Conferences) in the Colorado resort in 1958 by my mentor, Dr. Ben Eiseman.¹ I could not present the paper because, following graduation, I was on my way to intern at Philadelphia General Hospital, to continue my medical training.

I don't remember seeing any emphysema patients in Philadelphia. This was a rotating internship with emphasis on surgery, pediatrics, obstetrics, emergency room care, and psychiatry, and it included only two months of internal medicine. Most of my experience was with little supervision and I learned a lot "on the job." I did surgery, delivered a lot of babies and worked in the emergency room.

At this point I was unsure about my future career and was vacillating between becoming a surgeon or an internist. Since I had no deferment during the Korean War, I applied for a one-year post as a resident at the University of Michigan in Ann Arbor, which was highly sought after. Fortunately, there was a vacancy (probably due to the draft which somehow eluded me) and I was accepted. Michigan was quite a differ-

ent experience from Philadelphia General Hospital. Here I had many levels of supervision. I was not free to function as a first-year resident in training on my own, even though I thought that I was pretty skilled in some areas. I had to be supervised in everything I did by a senior resident or fellow. This was actually good fortune, because I needed the “sanding off of my rough edges.” The attending physician often did not appreciate my energies and adventures as I pursued my training. I was too sure of my self-perceived abilities.

One of the most striking and career-changing events occurred in the fall of this first residency year at Ann Arbor when I was taking a rotation on the hematology (blood) service. There I was assigned to evaluate a man, who looked very much like my first patient whom I evaluated in Colorado! He could have been his twin! However, his admitting diagnosis was primary polycythemia; that is a disease related to an uncontrolled growth of the marrow where all blood cells proliferate in a nearly malignant fashion, resulting in excess red cells, white cells, and platelets. I thought this man must have emphysema because the tests I did on his blood did not show excessive white cells or platelets, but just red cells. Primary polycythemia is not associated with low oxygen. Since I knew that excessive red blood cells alone is a response to a deficit of oxygen, I decided to measure his blood oxygen by sticking a small needle attached to a syringe directly in an artery (which I had never done before), obtaining blood for the same kind of measuring device used on Mt. Evans. In those days it took two hours to determine the oxygen and carbon dioxide. I talked a cute young respiratory technician into doing the blood gas and the next day reported to the attending that the patient could not have primary polycythemia with normal blood oxygenation because I had measured the oxygen and it was low; i.e., 82% of normal (similar to low levels of oxygenation while on Mt. Evans). It is an understatement to say the attending physician was not pleased by my diagnosis or by my aggressiveness in proving this. I was suspended on the spot because I had not “gone through channels” by

obtaining the arterial blood gas study. The protocol for this procedure was the placement of a special large needle into an artery. This required the approval of the cardiologists who did the procedure in the operating room! I was called on the carpet for not consulting with them for the procedure to be done. I was later reinstated the next day with an admonition by my department chairman, Dr. William “Bill” Robinson, not to be so aggressive and to follow established protocols. He was a very understanding chairman and we remained friends years later. He presented an award to me commenting on this life-changing experience at Grand Rounds at the University of Michigan some 15 years later.

In those days, oxygen was never given to correct these oxygen deficits in that case for fear of eliminating the “drive to breathe.” I never believed this concept and was later able to prove that it was wrong! The attending at Michigan actually did me a favor. “What’s the big deal about an arterial puncture?” I thought. I later used a simple



Author with his Chairman William Robinson in 1976, commemorating event of “the first arterial blood gas” done by a resident at the University of Michigan (author)

technique to measure oxygen quite readily once modern methods of blood gas analysis became available. I later wrote a scientific paper for the *Journal of the American Medical Association* on this simple procedure in 1966.² This was the dawning of a new era of care for patients with respiratory disorders.

During my further training at Michigan, and later upon return to the University of Colorado to complete my training in internal medicine and pulmonary disease, I became very interested in emphysema once again. I was again impressed with the opportunities in training in pulmonary disease with Roger Mitchell, who monitored my student research paper and encouraged me. Dr. Mitchell, one of the world's experts in tuberculosis, was beginning to study emphysema and chronic bronchitis, today known as chronic obstructive pulmonary disease (COPD). He was a dynamic and charming leader. I became interested in tuberculosis, but later shifted my emphasis into failure of the lung to provide adequate oxygenation or carbon dioxide removal known as "acute respiratory failure" due to emphysema and related disorders.

In 1962, I saw a most unusual patient at the Veterans Hospital during my pulmonary fellowship. He was a Marine veteran, who had suffered from rapidly progressive shortness of breath. He began smoking at age 17. He was only 27, but had marked hyperinflation of his chest and very poor lung function due to emphysema. Along with my attending, we hypothesized that he must have some unique susceptibility to smoking. We could measure immune globulins in the serum in those days. He showed an absence of the normal alpha one globulin. This was one year before Laurel and Erickson first reported on the alpha one antitrypsins deficiency in emphysema in 1963.^{3,4} We did not make the connection ourselves. Later, when he died at age 29, I was able to get permission to study both his lungs in the artificial thorax I was using to measure postmortem lung function (see below). His huge lungs with massive areas of destruction known as bullae,

nearly filled the artificial thorax. It showed a dramatic example of destructive emphysema at an early age.

In 1972, I was called for a consultation at the Children's Asthma Center in Denver, by one of the icons in asthma treatment, Elliot Middleton. I saw a young girl with marked hyperinflation and clear clinical evidence of emphysema, only age 14! Her illness was believed to be asthma, begun at age 10. She had never smoked, but came from a very polluted area of Los Angeles. She had a rare genotype of emphysema (SZ). Her brother, age 13, had the same abnormality, but did not have respiratory symptoms. Later Dr. Middleton reported this unusual patient, who remains the youngest emphysema patient I ever saw. Another was a young man age 29 and a nonsmoker. He did not have the alpha one deficiency, but had lifelong asthma. I became fascinated by this risk factor for emphysema at the early stage of my career, as well as other patients with precocious emphysema.

Following training, the Chairman of Medicine, Gordon Meiklejohn, bought me a modern blood gas analyzer as my inducement to join the faculty. I quickly learned to measure blood gases myself. I sampled blood by the simple technique I first used at Michigan. I could now make these measurements quickly using the new technology in a matter of a few minutes. I established the first service blood gas laboratory at the Colorado General Hospital, which later became a reference quality control laboratory for the United States. Here we learned for the first time that controlled low flow oxygen could be given to patients with emphysema without carbon dioxide retention.⁵ What a thrill it was when I learned that I could study oxygen in patients with emphysema and related disorders.

As serendipity dictated, my boss had the first portable liquid oxygen prototype device given him by the Linde Corporation of New York in 1965. My assignment was to see how it worked and to learn if patients could benefit from long term oxygen. I well remember the hissing

sound of filling the liquid canister from its reservoir source on the floor way back in 1965, just 43 years ago. It was easy to carry this oxygen around (it weighed only 9.5 pounds). Two fellows and I, along with nurse Louise Nett, designed a study to evaluate the effectiveness of oxygen in improving patients' outcomes in advanced stages of COPD where excess blood formation and right heart failure were present. These were the same factors that were present in my first patient and the man at Michigan. In all, we studied six patients in the research unit of the hospital. They were hospitalized for two months, i.e., one month for stabilization and the second month for oxygen. We measured the pressures in the heart and red cell formation and found a marked improvement in these physiological tests.⁶ But the most impressive thing was how the patients were transformed from being house-bound because of shortness of breath and unable to cope with their existence



Louise Nett, using first liquid oxygen cannister adapted to the trachostomy of a ventilator dependent patient, using a portable ventilation assist device.

to enjoying independent living and a full life. Thus began our interest in pulmonary rehabilitation, which is now the standard of care for patients with advanced COPD.

I tested the Linde device, known as the “oxygen walker” myself in trips to high altitude for fishing. Here I could climb easily as if I were at sea level. This helped me understand the relief the patients get, and I did not have a lung disease or heart disease at the time, as many patients did.⁷

After other investigations confirmed our original work, a major randomized, prospective clinical

trial of ambulatory oxygen compared with nocturnal oxygen therapy was done. This was known as the Nocturnal Oxygen Therapy Trial.⁸ This established oxygen as the standard of care for selected COPD patients who have chronic, stable hypoxemia.

These are just a few of the several chapters of my lifetime learning of emphysema and COPD, which led to the establishment of ambulatory oxygen therapy and pulmonary rehabilitation.⁹

During my fellowship and early faculty career I developed a method of measuring lung function in whole fresh human lungs, immediately following deaths from all causes, including COPD. In this research I could learn what smoking damage did to lung function by damage and loss of alveolar walls and obstruction of air passages. Thus I could finally explain what causes obstruction (and abnormal spirometry) in COPD.¹⁰

Today 1.4 million patients with COPD and related disorders receive oxygen. Newer ambulatory methods are enabling new dimensions to this therapy. Pulmonary rehabilitation has become the standard of care. I can look back and appreciate how powerful my first patient encounter really was. Ultimately we must find new therapeutic targets that will reverse or arrest this progressive disease.

Today we recognize that emphysema is a broad spectrum of diseases ranging from emphysema to chronic bronchitis, with chronic asthma in some patients. We call this spectrum chronic obstructive pulmonary disease (COPD). We know that COPD clusters in families, even beyond the alpha-one-antitrypsin deficiency state. We have improved the outcome tremendously of the patients with these disorders over the past many years. But now we must focus on the early diagnosis of COPD so that treatment can prevent or forestall the advancement of disease that results in premature morbidity and mortality. COPD can be readily diagnosed by a simple breathing test known as spirometry.

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C H A P T E R 2

IN THE CARDS WAS ARDS



My interest in COPD has dominated my career from the beginning of medical school. My COPD patients develop an oxygenation deficit due to lung damage. Another major theme in my career has been the clinical and pathological entity – the Acute Respiratory Distress Syndrome (ARDS). ARDS is a sudden flooding of the lungs with fluids, often from the lungs' own circulation (pulmonary circulation). Key to the diagnosis of ARDS was the ability to measure arterial blood oxygen CO₂ (see Chapter 1).

The following is adapted from: “*IN THE CARDS WAS ARDS*” (*Am J Respir Crit Care Med* 2001;163:602-603) (*How We Discovered the Acute Respiratory Distress Syndrome*)

My offer to join the faculty of the University of Colorado was \$2,500 to buy blood gas equipment and a 250 square foot “laboratory” with a sink and running water. I have a vivid recollection of that Saturday in August of 1964 when I finally mastered the Clark PO₂ and Severinghaus CO₂ electrodes, which came with my new Radiometer blood gas equipment. Earlier I had learned the Astrup tonometric method for calculation of PCO₂, the pH electrode, and used an American Optical oximeter for oxygen saturation in arterial blood gas analysis. This was less than 2 months after I completed my Chief Residency year, following my pulmonary fellowship. Dave G.

Ashbaugh was Chief Surgical Resident at the same time I was Chief Medical Resident. We were both frustrated over our inability to deal with the problems of acute respiratory failure that we occasionally encountered. The inadequate ventilators, including the Drinker tank negative pressure machine and the Bird, and Bennett IPPB machines were all that we had to use in our early attempts at treating acute respiratory failure. At this point in my career, blood gas analysis was limited to research applications. We desperately needed blood gas measurements on our sick patients to be able to monitor what we were doing. I finally was convinced that I could do them. As I walked home that day, I was kicking a stone and singing, "I can do blood gasses, tee-hee, tee hee." What a thrill!

My first assignment after I joined the faculty as a young Assistant Professor was to develop a Respiratory Care Service for the new Colorado General Hospital, which was to open in early 1965. We had no respiratory therapists. Tanks of oxygen for bedside use were delivered by Central Supply by an "Oxygen Orderly." Dave, and two fellows, Bernie E. Levine and D. Boyd Bigelow, joined me as we launched our Respiratory Care Service. On the night that Barry Goldwater accepted the Republican nomination, we received our first call. A patient with emphysema, Earl, was admitted via the Emergency Room to the Medical Intensive Care Unit with advanced COPD and acute respiratory failure. Dave did the tracheostomy while Boyd and Bernie put old Earl on the Bird Mark-VII. I ran the blood gases all night long. Earl recovered, and we were never the same again. We knew we could effectively treat acute respiratory failure caused by COPD.

Later that fall, as we tried to mobilize all available resources, Dave found an old 1954 model of the Engstrom anesthesia/ventilator unit in storage. We both read the instructions and still wondered what the "expiratory retard (control)" on the exhalation manifold

meant. One night, Dave was called to the Surgical Intensive Care Unit to take care of a motor vehicle accident victim, who had bilateral symmetrical pulmonary infiltrates, and stiff lungs. The patient could not be adequately ventilated by either the Bird or the Bennett pressure-cycled machines. Since he had just found and cleaned up the device, Dave tried the Engstrom. It was a volume-controlled respirator with high inflation pressure capability. In desperation, Dave set an end-expiratory pressure of 10 cm H₂O when it became impossible to achieve adequate arterial oxygenation. The blood gases that I did that night showed marked improvement in oxygenation with that maneuver. The patient died 48 hours later of progressive hypoxemia. At autopsy, both lungs had the consistency of liver, and each weighed nearly 1,000 grams (normal is 300 – 400 mg). Both were collapsed and congested with bloody fluid.

We moved from Colorado General Hospital to a new University Hospital on February 23, 1965. By prior agreement with the Department Chairman, the late Gordon Meiklejohn, I was given a generous laboratory space next door to the 12-bed Medical Intensive Care Unit, and immediately above the Surgical Unit. This is where I moved my blood gas equipment and all of our respirators. On this day, we began to make morning rounds at 7:30 in both the Medical and the Surgical Intensive Care Units. Louise M. Nett, R.N., joined the team as the Respiratory Nurse. Susie Tyler was now running blood gases during the daytime, but the fellows did them at night. Dave, the fellows, Louise, Susie, and I discussed the clinical and physiological features of each patient who was receiving mechanical ventilation at the end of rounds each day in “the lab.”

Dave and I had other similar patients that we could not ventilate due to stiff lungs with the pressure-cycled devices that were more available than the Engstrom. Dave liked to use the Engstrom in the post-operative period, following open heart surgery, because of its special

capabilities of high volume cycling to the feature of positive end expiratory pressure (PEEP). Thus, the Engstrom was not often available for other use.

Shortly after Michael M. Finigan (Rochester, New York) joined us as a first-year fellow in 1965, he was taking care of a patient with acute hemorrhagic pancreatitis complicated by massive pulmonary congestion, located next door to the Respiratory Care Laboratory. This patient could not be adequately oxygenated. Mike came and got me. We had just cleaned the Engstrom tubing, and it was available in "the lab." I helped Mike use the Engstrom for the first time in his training. "What does this knob do?" Mike asked. I said, "I think it is probably useful, since Dave could improve oxygenation in a crushed chest patient a few months ago." I then dialed in a positive end-expiratory pressure of 10 cm (at that time, we called this continuous positive pressure breathing (CPPB)). It was dramatic to see a blue patient lying in Trendelenburg, gradually flush to a healthy pink color; blood pressure improved. We stood there amazed. "We need another gas to see what's going on." I said to Mike who was astonished over the sudden color change in this desperately ill woman. The Po_2 had increased from 44 to 125 with that simple positive end-expiratory pressure maneuver. After standing there for about another thirty minutes, we decided to turn the end-expiratory pressure off to see what would happen. Once again, the patient turned blue, and her Po_2 dropped to 45. With reinstatement of end-expiratory pressure, the Po_2 again rose to 135. After three more days, this patient died of refractory hypoxemia and carbon dioxide retention. Autopsy also revealed heavy lungs and alveolar debris, hyaline membrane formation, and marked edema. Bernie Levine, who had learned the method of measuring surface tension of the foamy fluid expressed from the lungs, found markedly elevated surface tension, indicating a deficiency in surfactant. Later, a second specimen showed the same surfactant abnormalities.

A few days later, one cold Saturday morning, I remember walking across the parking lot between the New Colorado General Hospital and the Veterans Administration Hospital, and talking with Dave. We talked about this new patient and his original patient with traumatic injury when he first used the Engstrom respiratory and positive end-expiratory pressure. "Something" is different with these patients, we agreed. Later over coffee, we concluded that the common denominator was sudden diffuse, and usually symmetrical, pulmonary infiltrates following a variety of seemingly unrelated lung insults, stiff lungs requiring high inflation pressures, and marked difficulties with oxygenation. The pulmonary edema that is associated with heart failure was different from this pattern and a material called hyaline membrane, which represented original proteinaceous fluid, lined the collapsed alveoli.

The first patient with this distinctive clinical picture who survived was a 15-year old adolescent who suffered hemorrhagic shock following a motor vehicle accident. Like the first two patients, his chest X-ray showed diffuse, bilateral symmetrical pulmonary infiltrates. The ventilator's inflation pressure was high (i.e., 40 to 50 cm H₂O). Arterial Po₂ was dramatically increased with positive end-expiratory pressure. In a matter of a few months we had 12 similar patients from a total of 272 adult patients who required mechanical ventilation from all causes of acute respiratory failure since the start of our new Respiratory Care Service. These 12 patients all had massive acute lung injury from trauma, shock, presumed viral pneumonia, or hemorrhagic pancreatitis. High inflation pressures were required to ventilate lungs that were densely consolidated in a symmetrical fashion. End-expiratory pressure usually improved the oxygen transfer across the lungs. All of the patients who died had hyaline membrane formation. The five patients who survived made a full or near-full recovery.

On another Saturday morning, Dave and I sat in the lab with the fellows and asked ourselves again what was different about these patients with acute respiratory failure, compared with the other patients who required mechanical ventilation. Because of the marked resemblance of our patients with the infantile respiratory distress syndrome, which is also characterized by hyaline membrane function and a surfactant abnormality, we wrote up our experience of 12 patients with five survivors. The title was "Acute Respiratory Distress Syndrome in Adults." We proudly submitted the paper to the *New England Journal of Medicine*. Since Dave had made the first observation about the value of end-expiratory pressures in improving oxygenation, it was agreed that he should be first author on the manuscript. Our report came back very quickly with the scathing criticism that end-expiratory pressure had been proven to impair cardiac output by causing a reduction in venous return. "What were the idiots in Denver doing, anyway?" was implied. We then sent the paper to the *Journal of the American Medical Association* and got an identical response. It had to be the same reviewers. Dave then got angry and sent the paper to the *American Journal of Surgery*, being confident that surgeons would appreciate our discovery and the concept of the acute respiratory distress syndrome. It also bounced! Finally, in desperation, we sent the paper to the *Lancet*, and received word within two weeks that our discovery was of such importance, that it would be published as a lead article, without delay (Ashbaugh DG, Bigelow DB, Petty TL, Levine BE: Acute respiratory distress in adults. *Lancet* 1967;2:319-323). It is impossible to exaggerate the thrill we felt when our first paper on ARDS was accepted.

The timing of this publication was extremely fortunate, because our article was read by military surgeons in Southeast Asia treating casualties in the Vietnam War. They realized that many of their wounded were suffering from ARDS. Quickly, the surgeons led by Ben Eiseman, my former mentor (a Rear Admiral in the Naval Reserve

organization) planned a conference in acute lung injury. This was held in Washington, D.C., in May 1968. It was sponsored by the National Science Foundation and the National Research Council on "The Pulmonary Effects of Nonthoracic Trauma." Dave, Henning Pontoppiadan from Boston, and I were among the few civilians at the meeting. Here, I presented our further results in the treatment of a series of 21 patients with ARDS and the anecdotal use of PEEP. Ten of 14 had survived with PEEP, compared with only 2 of 7 who had been treated before we had learned about the effectiveness of PEEP in improving arterial oxygenation in patients with acute lung injury. Alas, this was not a proper clinical trial. To date, no controlled clinical trials have ever been done to learn if PEEP improves survival. I doubt if one will ever be proposed as I once did (*Am Rev Respir Dis* 1988;138:475-478).

Of course, we were lucky. We were at the right place at the right time. We had modern blood gas technology right next to the patients. We often did arterial blood gases ourselves. We wondered what was different about these patients compared with the usual causes of acute respiratory failure in COPD, pneumonia, in the post-operative period, asthma, and neurological conditions. We were not bound by conventional thinking in those days. We challenged dogma and flew by the seat of our pants. As physicians and surgeons, we worked together, like in the old tuberculosis era. That's why the cards played out ARDS."

Today there are an estimated 150,000 – 200,000 patients identified with ARDS each year. The survival rate has gradually increased from 40% to 80% in some centers by virtue of controlled low tidal volume ventilation and other forms of supportive care. Thus far, no pharmacological intervention has proven effective. I continue to believe that some method of surfactant preservation or restoration is needed in the early stages of emerging ARDS, but this has never been adequately studied.

C H A P T E R 3

OXYGEN AND PULMONARY REHABILITATION (*HISTORICAL FOUNDATION*)



Oxygen has become a revolution in our ability to provide home care for many patients with respiratory insufficiency. This practice has been dubbed long term home oxygen therapy (LTOT). In Japan it is known simply as home oxygen therapy (HOT). There are over a million known LTOT patients in North America and probably a similar number in Western Europe, and 140,000 HOT patients in Japan.

Our current understanding about the science and technology of LTOT begins in 1965. This is the year that we learned how to fill a liquid portable oxygen system. We immediately sensed its value and quickly conducted a controlled trial of the new unit, known as the Linde Oxygen Walker, in six patients in that year. These were highly selected patients who demonstrated clinical, EKG, and radiologic evidence of pulmonary hypertension and each patient had secondary polycythemia.

Each patient was admitted to the clinical research unit of the hospital and stabilized for a month on room air. They then had cardiac catheterization and careful measures of red cell formation, followed by a second month when oxygen was given. Each patient was exercised twice a day, and breathing retraining was used throughout both the control and oxygen months. A second cardiac catheterization was done at the end of the oxygen month, along with the blood formation studies.

We found a remarkable reversibility in the level of pulmonary hypertension and complete control of the red cell mass after oxygen. The most remarkable improvement was in daily exercise tolerance which improved some during the control month, and exponentially during oxygen. We reported our findings at the annual meeting of the American Thoracic Society along with our new data that oxygen did not cause dangerous CO₂ buildup in patients with advanced COPD. Both papers were well received at the meeting. Indeed they caused quite a stir, since the dogma of the time taught that oxygen could not be given to such patients!

One of the most remarkable events took place in 1967 when I carried the charts of one of our early patients to the Office of the Director of Social Services. It showed how the thickness of this patient's medical record, including numerous hospitalizations for the 18 months prior to receiving home oxygen compared with the very thin chart of the first 18 months while receiving oxygen. This impressive demonstration led to the reimbursement of oxygen for this patient by the Colorado Department of Social Services, well before Medicare or Medicaid became established.



Hospital records of early oxygen patient during the 18 months before starting home oxygen, followed by the first 18 months while using oxygen. This led to the reimbursement for oxygen by Denver's Department of Social Services well before Medicare or Medicaid.

We then enrolled many more patients in LTOT when they had chronic stable hypoxemia during our pulmonary rehabilitation program during the recruitment period of 1966-1968. We had a public health service contract to support the demonstration project. Our scientific reports stimulated others to study LTOT at sea level and in Europe.

The most solid science to support LTOT comes from the Nocturnal Oxygen Therapy Trial and the British Medical Research Council, both of which were conducted in the late 1970s. Together these landmark studies showed that oxygen improved survival and quality of life. Continuous oxygen (approximately 18 hours per day) was more effective than part time oxygen (approximately 12 hours per day). These studies represent the state of the art treatment and oxygen technologies of an era that is now four decades old. More research is planned, particularly in milder degrees of hypoxemia. The technology of portable oxygen is advancing steadily. Utilization is increasing about 10% per year. Access to modern technology remains a problem in some areas of this country, and in most parts of the world. Large population areas such as nearly all of South and Central America, Africa, and Asia have no LTOT available for the millions that suffer from respiratory insufficiency on a global basis.

Pulmonary rehabilitation emerged as a form of systematized care in the 1960s, based on observations that exercise is beneficial to most patients with dyspnea on exertion from respiratory causes. The early observations were anecdotal, but added together to suggest real benefit from daily exercise. Various breathing strengthening techniques and breathing training also helped to augment exercise. Oxygen was key to exercise in the hypoxemic patient, and appeared to help some patients who were normoxemic at rest and mild exercise. How oxygen was beneficial in these patients has been extensively studied, but the mechanisms remain unanswered.

In addition to exercise, breathing training and oxygen, pulmonary rehabilitation involves a complete understanding of COPD and how it is treated. Thus patient and family education are integral parts of a pulmonary rehabilitation program.

Finally, patient support groups are highly beneficial to most who suffer from chronic symptoms of disease. There are several support groups, that are national and internet based, such as COPD Alert, and Efforts. There are also many local groups such as the Pulmonary Education and Research Program (PERF) and the Colorado COPD Connection (affiliated with the American Lung Association of Colorado (ALAC)).

Pulmonary rehabilitation (PR) has become the standard of care by which other therapies are judged. This was the case with the National Emphysema Treatment Trial (NETT). NETT studied lung volume reduction therapy. The outcomes were similar between PR and NETT. A subset of patients who derived little relief from exercise, and who had the most extensive destruction of the upper lobes, did show a small survival benefit from lung volume reduction surgery (LVRS).

CHAPTER 4

PASSING THE BATON – SPREADING THE WORD



The 1960s and early 70s brought forth major increases in our ability to manage patients with a variety of acute and chronic respiratory disorders. The mechanical ventilator came into common use for post-operative support and for a variety of acute pulmonary problems, such as status asthmaticus, COPD, overwhelming pneumonia, pulmonary embolism, and less common disorders. The use of mechanical ventilation in advanced stages of respiratory insufficiency, particularly on COPD, though controversial in the 1960s, began to become accepted once it was realized that home oxygen and pulmonary rehabilitation could change the lives of patients who had previously suffered a nursing or home-bound existence.

The knowledge of controlled low-flow oxygen and the emergence of liquid portable oxygen as the way of providing lightweight oxygen for exercisers, including those involved in recreation and work, was a revolution at the time. Our research in pulmonary rehabilitation showed very clearly that a combination of patient education, breathing retraining, physical reconditioning, and in selected cases, oxygen, added to a growing array of pharmacologic agents, could dramatically change the course and prognosis of the majority of patients with COPD.

“Understanding the acute respiratory distress syndrome (ARDS),”

also was considered a revolution at the time and to this very day has stimulated the imagination of basic scientists, intensive care experts, and technological engineers who are continuously able to produce new equipment to provide ventilatory hemodynamic, and monitoring support with sudden acute lung injury from a variety of background factors. The survival rate was only about 50% in the 1950s, which demanded improvements in care, but already what we now know established the widespread application of the principles of management, which originated in Denver. Our first approach in providing comprehensive education in the short-term was to organize a one-week refresher course for pulmonologists from around the country. We had these three times a year, where physicians who were already experts in pulmonary medicine, would be with us for an entire day's activities, which included morning rounds, work in the blood gas and pulmonary function laboratory, in the intensive care unit with active patients receiving mechanical ventilation, and hands-on experience in the animal research laboratory for the purpose of doing invasive procedures. Techniques of tracheostomy, chest intubation, use of mechanical ventilators and resuscitation were taught. Attendance was limited to



Author with three pulmonologists reviewing the details of use of the mechanical ventilator (Bennett PR-1).



*Pulmonologist student in blood gas laboratory actually doing an arterial blood **TEST? EXPERIMENT?** which he had drawn on a patient in the next-door intensive care unit.*

ten physicians in each one-week refresher course. We had no assigned budget or additional personnel for this effort. It is an understatement to say that everyone was exhausted at the end of this experience.

In the 1970s, the Regional Medical Program, a brainchild of the Johnson administration, began to be implemented. It was relatively easy for us to obtain a large grant from the Colorado Rocky Mountain Regional Medical Program, which allowed us to expand our personnel to include a small number of physicians, nurses, and therapists to provide a range of experiences including several days of hands-on experience throughout the region. Most of our efforts were spent in Boulder, Greeley, and Ft. Collins. On the western part of Colorado, we focused on Grand Junction, Durango, Montrose, and Glenwood Springs.

We also traveled extensively in Wyoming, spending significant time in Casper, Cheyenne, and Sheridan. The experience included representatives from these hospitals spending time in Denver for additional brush-up or new hands-on experience. Funding for this very effective method of teaching ended in the late 1970s with a change of administration. This magnificent method had to be abandoned.

Later we expanded our one-week refresher courses to more individuals and they evolved inevitably into the lecture format, where the exchange of knowledge is far less efficient than when one dedicates their entire professional time to learning new techniques. By the 1980s we had to use our refresher course to give it an international flavor. Physicians, whose second language was English, were invited – 20 in all, who spent one week in our growing hospital systems, which then included the University of Colorado, the National Jewish Hospital, the Veterans Hospital, and the Denver General Hospital. With an expanded faculty we could provide more personal instruction. This course was particularly popular with physicians from Japan, Taiwan, Brazil, Argentina, Scandinavian countries, and Eastern Europe.

A highlight of this educational program was a case report “To Stump the Expert” presentation by the visitors to the Denver faculty assembled. This was probably one of the most valuable give and take international efforts ever mounted. Support for this included publicity and some marketing assistance from the American College of Chest Physicians.

How to provide the most effective education in a field of changing technology, continues to stimulate clinical educators today. Some of the models developed in Denver in the 1960s, 1970s, and 1980s might well be repeated on a regular basis, utilizing the major centers from around this country and elsewhere in the world.

In the late 1960s and early 1970s, Louise Nett, RN, RRT, provided a one-week program for a large number of people who were just begin-



Pulmonary division as it existed in the mid 1970s. Includes faculty and fellows.

ning to learn about the new developments in respiratory care. While this training was held in a hotel setting, there was quite a bit of hands-on instructional experience with the new products that were available. The physicians from the Pulmonary Division participated in this program, which was designed primarily for nurses and respiratory therapists. However, a number of physicians came with their nurse or therapist and a few came from long distances, including Europe. This was extremely successful and continued on an annual basis until the mid-1980s. It is hard to evaluate the impact of such a program, or any educational effort for that matter, but the feedback was excellent.

Louise Nett made 13 trips to Japan to train nurses in HOT. Tom taught extensively in foreign countries, most notably in Argentina and Australia, on extensive lecturing circuits.

PART II

MEMORABLE PATIENTS AND EVENTS



INTRODUCTION TO PATIENT VIGNETTES

Following are a few dramatic vignettes about patients whom I encountered, often in a consultative role throughout my career, which taught particularly important lessons about principles in medicine.

Many of these vignettes go beyond the simple use of the stethoscope but the reader will know how important this universal medical instrument can be in many circumstances. These vignettes begin while I was chief resident in medicine in 1962 or in completion of my pulmonary fellowship training. In that era, only one year of fellowship was required for me. It was completed in 1962-1963 under the direction of Roger S. Mitchell, renowned pulmonologist and tuberculologist of the era.

I began my appointment as what is known as Chief Resident in Medicine following my one year formal fellowship in pulmonary disease. The Chief Resident is a junior member who oversees the functions of all of the medical residents in training and coordinates the activities of many of the subspecialties in patient care. I had completed my internship at Philadelphia General Hospital and a one-year residency in general internal medicine at the University of Michigan. This was followed by two years of general internal medicine at the University of Colorado pursuant to board certification in internal medicine, which could not be completed until 1965 because two years post residency training was required by the Boards at that time. Thus, I had my pulmonary fellowship before my chief residency experience, which is not the usual sequence. It is much more common for physicians who do broad-base training in internal medicine, prior to training in pulmonary, to complete their residency and sometimes research before taking a position. Chief residency training may or may not include some research, and is pursuant of a foundation for an advanced degree such as a PhD. Fellowship (specialty) training usually follows these earlier training and research experiences.

C H A P T E R 5

MUFFY



One of the many memorable patients from my chief residency year, was a beautiful young woman aged 25, who went by the nickname of Muffy. She was a beautiful, vivacious, married woman. Both she and her husband worked and planned to start their family the following year. They had bought an old home and began the weekend chore of making various necessary repairs, both inside the house and in a large garage that served as a barn for two horses on the small acreage the couple had purchased.

On a Saturday night, Muffy developed severe sudden shortness of breath, following her shower after working in the dusty barn with her husband on a hot Saturday afternoon. She suddenly became extremely short of breath with cough and wheeze, and was immediately transferred to the Colorado General Hospital, which was the University Hospital of that era where all of our training in internal medicine and the major subspecialties of medicine took place. The resident who admitted Muffy called me at 3:00 am to report on this desperately ill woman with a breathing problem. I immediately went to see her; curiously already in my fishing clothes because I had planned to take a rare day off from my duties to do some trout fishing in the high lakes west of Denver, Colorado. This rare off-duty trip was a matter of “mental health,” which was encouraged by the chairman. Even though not dressed like a doctor, I immediately went to the bedside and spoke with

the husband who described the wife's terrors as she struggled to breathe before the ambulance came and provided the oxygen. The patient had marked shortness of breath and "bubbling" crackles, indicating fluid in the chest, which I could easily hear with my stethoscope. On initial examination in the emergency room, her chest x-ray showed diffuse fluffy infiltrates, shadows (called infiltrates on chest x-rays) indicating pulmonary edema. I carefully reviewed these after examining her. The heart size was normal. By the time I saw her, she had stabilized with the use of some oxygen and diuretics. She was alert and cooperative. After reviewing some more details of the history, which suggested that she had had Rheumatic Fever in childhood, I listened carefully to the heart and lungs. She did not have neck vein distension indicating elevated pressure on the venous system leading immediately to the heart. This suggested that the heart was transporting the blood returned to it with the vessels from head and extremities. Her chest was not too noisy but some diffuse fine "moist" rales were heard on both sides equally. It is hard to describe moist rales. These fine crackles are also known as rales. Trying to explain the sound is like trying to explain how you know your dog is barking outside. Most rales are multiple, soft, and are loudest at the base of the lung. There were no expiratory wheezes. These findings indicated fluid within the fine reaches of the lung; i.e., alveoli, alveolar ducts. She had an elevated pulse of 120. A careful examination over the four valves of the heart revealed absolutely no murmurs. I specifically listened for the murmur of mitral stenosis, since I knew that mitral stenosis; i.e., either on a congenital or rheumatic basis interfered with blood flow from the left atrium chamber to the left ventricle or lower chamber. This is particularly important, since Muffy's mother had remembered rheumatic fever when the child was only four. A description of this rheumatic fever was not very classic, but should carry the diagnosis of rheumatic fever indicating the high likelihood of valvular disease since childhood. Since the patient was improving so rapidly, I went ahead with my planned fishing trip at about sunrise.

By the time I had returned from fishing on Sunday evening, the patient was further improved. Not only was she breathing without oxygen, chest x-rays showed almost complete clearing of the edema within her lungs. I had a momentary recollection of my own pulmonary edema suffered from high altitude, which was a different mechanism I suffered as a medical student or perhaps this was not pulmonary edema, but a severe lung irritation or viral infection. At this time I asked about the possible causes of the lung irritation. Both she and her husband had been spray painting in the old barn on the day before the sudden attack of breathing difficulty began. They had used a mask, but masks are not fully protective. The couple did not have any cough while they were in the barn. The husband did not have any symptoms similar to that of Muffy. I was wondering about the possibility of hypersensitivity pneumonitis, which is an allergic inflammation of the lung, but rarely if ever, produces a finding of acute pulmonary edema. She also had a bit of a cold and we wondered about a viral infection, but it seemed unlikely for a viral infection to clear so rapidly and dramatically. We discharged the patient home the very next day because she appeared entirely well. We had no explanation for her very dramatic presenting symptoms or the rapid complete clearing. We finally concluded that some irritation to some inhaled paint or paint cleaner solvent might have been the cause due to some particular susceptibility or different degree of exposure than the house.

Only ten days later, Muffy returned with exactly the same clinical presentation of sudden shortness of breath, the clinical x-ray picture of acute pulmonary edema. I again examined her and on this occasion heard a distinct rumble of mitral stenosis, that is, blockage of the valve between the upper and lower chambers. My conclusion at this time was that this patient had a rare, benign tumor known as an atrial myxoma or a clot in the left atrium, which would intermittently cause a dysfunctional ball valve mechanism leading to dramatic obstruction and then relief in different positions or because of the flow of the

blood in just a fashion as to dislodge the obstruction. The consulting cardiologists ridiculed my diagnosis because they also heard a small “whiff” of aortic insufficiency thus indicating both mitral and aortic valve involvement, presumably a result of her earlier Rheumatic Fever. I had not heard this aortic sound before they called it to my attention, but could when I first listened to the chest, it was much noisier than when the cardiologists saw her. Nonetheless, I steadfastly believed that the correct diagnosis was atrial myxoma and told this to my chairman, Gordon Meikeljohn. We consulted the cardiac surgeon over the ire of the cardiologists and the great leader in cardiology and my personal mentor and close friend up to the present time, S. Gilbert Blount. Blount believed that the diagnosis was most certainly rheumatic heart disease because of her history and the multiple valve abnormalities. In any case, largely due to my persistence and back and forth discussions among the cardiologists, department heads and surgeons, Muffy was taken to the operating room. Indeed, an atrial myxoma was found and removed. The equipment for heart/lung bypass, which was not generally available in that era, was not used for her surgery.

Unfortunately and tragically, the end of this story is not entirely happy, since a piece of the atrial myxoma, which extended from the mitral valve clear out through the aortic outflow tract, became dislodged. The patient’s left leg turned blue below the knee in the immediate post-operative period. There were no surgical procedures to restore arterial flow at that time and this young woman ended with a below-the-knee amputation, but this did not dampen her spirits towards life. She quickly learned to use her prosthesis. Soon she returned to her love of dancing with her husband and other friends, often on Saturday nights after working hard on their recently purchased property.

The lesson here, of course, is careful auscultation, noting changes which, in this case, could be indicative of a very critical differential diagnosis between the rare atrial myxoma and the much more common

rheumatic heart disease. Today the exact diagnosis would have been easily made by ultra sound or MRI, which would have lead to a successful operation and the embolus followed by amputation of her leg would not have occurred. She was lucky in a sense because a complete obstruction could have been fatal had she not been within reach of the hospital on another occasion.

Even though I felt proud about being able to make the diagnosis, I always felt special efforts should have been made to arrange for heart/lung equipment to be used. This would have been difficult in those days (1962), but with complete circulatory support, a greater opportunity for controlled removal of the complex myxoma could have been accomplished and the embolus and subsequent amputation avoided.

C H A P T E R 6

J O Y



On New Year's Eve 1967, I received a call from a nurse anesthetologist at the Aspen community hospital about a young woman, now being supported by her efforts to squeeze an anesthesia bag, as would be done in that era during a surgery procedure. There were few automatic ventilators in those days, and the small community hospital did not have any ventilatory equipment at all. Even four decades ago, Aspen was a playground for the rich and famous, and also the most popular ski resort in the Rockies. But it lacked medical facilities in that era.

Luda, the nurse, described the patient Joy as a young and previously healthy woman who worked as a medical research assistant at New York University Hospital. She and her girlfriend had arrived in Aspen the previous day, and skied only part of the day because they were not in top physical condition and were cautious of the altitude which ranged from 8,000 feet in town to 12,000 feet on the tops of many of the mountains used for skiing.

The two women stayed in a brand new motel, near the ski slopes. The next morning the manager noticed no preparation for skiing by the two and accordingly she entered their room to find Joy unconscious, but breathing, and her roommate dead. It was a tragedy that the motel had not yet been approved by the town's building inspectors. Carbon monoxide was the probable cause. Joy was taken to the emergency

room in the small community hospital and kept alive by the efforts of Luda, who could only maintain ventilation by squeezing the anesthesia bag. The nurse anesthetist was administering 100% oxygen, because of the near certainty of carbon monoxide poisoning. Early in her course the resistance to lung inflation increased and the chest x-ray showed pulmonary congestion; i.e., pulmonary edema. It was possible that this was high altitude pulmonary edema, a common occurrence when non-acclimatized persons exert at high altitude. See Chapter 1.

Luda, asked me for help and I responded by explaining how end expiratory pressure could be done, using her anesthetic bag with the exhalation port connected by tubing to an underwater trap. This was just after we had reported our first series of acute respiratory distress syndrome (ARDS) and the beneficial effects of positive end expiratory pressure (PEEP) in improving oxygenation.

I tried to arrange helicopter transfer via the army base in Denver, but there was such a blizzard in Aspen that landing was impossible. I talked nearly every hour that evening to Luda and into the next day. Joy remained unconscious, but had favorable brain stem reflexes, suggesting that irreversible brain damage had not taken place. Still the blizzard raged, and ground transportation was risky with the mountain passes mostly closed. Maintaining ventilation in an ambulance in that era was also not possible due to the lack of suitable equipment.

On the third day, the weather cleared partly and transfer to my unit in Denver was finally accomplished. Joy was admitted to the ICU accompanied by Luda, whose hands were swollen to twice their normal size. Luda was exhausted but she was amazing. What dedication to a critically ill patient, I thought.

Joy then made a very rapid recovery. She was able to follow commands on the second day at my hospital, and became fully awake and oriented on the next. Neurological tests did not show any abnormality.

She could be discharged one week later, or two weeks after her year end “vacation” began.

We celebrated her recovery during a trip I made to New York later that year, and attended the famed musical *HAIR*. I surprised a few of my friends by getting up on the stage and dancing with the nude performers. What a celebration!

I have been in touch with Joy for the four decades that have followed. She married, was blessed by a son who grew to adulthood and began his own family. She still continues to work as a research assistant at the New York Hospital.



Joy, on recovery in NY with author.

C H A P T E R 7

MR. SMITH



In the late 1960s, it became tradition to end Friday afternoon rounds, with a glass of beer at a local “pub” across the street from the hospital, known as the College Inn, disguised sometimes as the “College Institute.” One afternoon, the fellow, Mike, said there was only one afternoon admission to the general medical wards, an “old man” brought in from a nursing home, whom we could see on Saturday morning, since he had already been seen by the ward staff and had no family with him. Our group was pretty thirsty that hot summer day, but I asked, largely by habit, about his age and condition, and a few questions about his family.

He was described as a demented 57 year-old man who had been in a nursing home for about six months. No family was available. This did not make sense to me. “What is the reason for dementia and nursing home care for such a young person,” I asked. Mike replied that he had no more information, so I insisted on seeing him before we left. Mike said that he could not give a history and was totally deaf.

I approached the man, who was covered with several layers of bedding, lying on his side in a fetal position. Since we knew his name, I yelled directly in his ear, “Mr Smith, what’s the matter?”

There as a long pause, then I heard a weak reply, “I have Heinz disease.”

“What’s Heinz disease?” I again shouted.

There was another long pause and then the reply came, “57 varieties.”

Wow! I pulled off the covers and found a poorly nourished man with several shirts and two sets of pajamas. I checked his heart which was very slow, and his reflexes, which had the classic delay of relaxation, associated with severe hypothyroidism! Hypothyroid patients always feel cold, even in hot weather. He wasn’t demented at all; just very slow, which is typical of hypothyroidism and the so-called “hypothyroid (myxedema) wit.”

It was easy to confirm the diagnosis with the tests of the era, and after slow and deliberate thyroid replacement and physical therapy, plus refeeding, the man made a complete recovery and could soon walk on his own.

For several years we had him return to chest clinic, just to visit with him for fun. No one will ever forget MR Smith, but I imagine the beer tasted particularly good that hot summer afternoon.

R O N



Ron was a heavy smoker, who developed shortness of breath in his mid-thirties. He went to a number of primary care physicians for symptomatic care, but no one ever did spirometry and a diagnosis was never made to explain his symptoms. Ron was an intelligent man with an engineering background and wanted to solve problems for himself whenever he could. He went to the library and found our book on emphysema, *Enjoying Life With Emphysema*. After reading this, he concluded that he must have emphysema by virtue of his heavy cough and progressive shortness of breath on exertion, which was becoming disabling. He then saw one of my former fellows in Denver, who confirmed the diagnosis. Ron immediately quit smoking. At the time of diagnosis, Ron's airflow index, FEV_1 was only 28% of predicted; i.e., extremely low for a man of 50. He immediately enrolled in a pulmonary rehabilitation program. He was dedicated to improving his exercise tolerance and to keeping active. He particularly enjoyed the small mountain cabin, which he built himself, called the "observatory." Now, retired because of disability, he spent enjoyable summers but at 8500 ft. Soon he had to abandon his mountain retreat because of the altitude. Breathing was always better at sea level where he would visit various friends and family. He became very depressed and considered suicide. But his mother reminded him to read the bible about various things that trouble mankind. He concluded from the bible that he

would be denied entry into heaven, should he ever commit suicide. He gave up that idea and decided instead to try and find out whether he could find relief from his suffering or perhaps, in the distant future, obtain a lung transplantation for his advancing emphysema state.

I first met Ron at a meeting of the editorial board of the National Home Oxygen Patients Association (NHOPA). It was apparent that he was quite knowledgeable about his disease. He checked his peak flow, used his metered dose device several times during the two-hour planning meeting. We struck up a friendship and he kept in touch with me about his progress, even though he had now moved to Texas. Over the course of the next four years, his pulmonary function deteriorated rapidly and he consulted Dr. Joel Cooper at the pioneering Barnes Lung Transplantation Unit for possible lung transplantation. He was deemed an excellent candidate but he had no health insurance. Ron asked me about his prognosis on several occasions, and I felt that his only chance for survival was transplantation since he had no other serious comorbidities and was young and intelligent. His entire life savings was \$125,000, which was exactly the amount that lung transplantation was to cost. Ron made this investment and told me, upon recovery, "What else did I need the money for?" He continued, "I have no heirs, after all. All I have is my life and my faith."

Ron was a very spiritual person and we prayed together on many occasions before and after his transplantation. On one amazing occasion, I was in Rochester, Minnesota, at the Mayo Clinic on the evening before my third open heart procedure (*see Chapter 18*) and I received a phone call from the lobby. It was Ron. He had come to pray with me prior to my surgery as a surprise and because of our prayers together on his behalf before his transplantation. As tired as I was from traveling, I went to the lobby and together we held hands and prayed aloud for about 20 minutes about my care and recovery. Ron and I keep each other in our evening prayers and to this day remain very close friends.

Ron has had many bouts of rejection since his transplant nearly seven years ago. He always seems to have some extra stamina or spiritual force that leads to his recovery.

Following his transplant, Ron spent a great portion of his time and most of his available income developing crude but highly effective video tapes called "Teens Speak Out." This is intended for distribution in high schools throughout the United States. Production grade movie equipment and this project never reached its goal. Recently Ron and I began a film on COPD for patients. With the movie now in production, hopefully with appropriate support from the industry, it will become possible for Ron and his teenage message to reach many who, otherwise, could fall victim to the harms of tobacco smoking which affects at least half of those who take up smoking. The message of early identification and intervention also needs widespread publicity. Ron has no money of his own to support his ambitious plans, but with his knowledge and dedication, he may succeed.

A number of fortuitous chances brought Ron and me together, beginning with a book I had written for emphysema patients. Our mutual faith and prayers for each other over the years have been a major factor in both our survivals.

ARNIE



Arnie was an 81 year-old retired farmer from Eastern Colorado, whom I had cared for for nearly ten years. He had advanced emphysema and received oxygen at all times in recent years. He was from rural Colorado, but he had the availability of portable oxygen and with this and some modest training in pulmonary rehabilitation he had adapted well to his limitations. He simply walked as far as he could at a comfortable pace with oxygen twice a day. He led a thoroughly comfortable life in the small community where his children lived. I had cared for him during several exacerbations of disease in consultation with his personal physician who lived in their community some 180 miles away.

His general state of stability with his illness was dramatically disrupted by acute abdominal pain which migrated to the lower part of his abdomen. I had Arnie transferred to the Presbyterian Hospital, where I practiced at that time. I was his primary attending physician. He was seen in consultation by an expert in abdominal surgery, who felt that there was nothing “surgical” to deal with. He did not have anything to suggest acute appendicitis. However, I strongly suspected that he had a ruptured diverticulum; i.e., an outpouching of the colon which is common in the elderly and particularly common for some reason in patients with advanced stages of COPD. I wasn’t happy with the prospects of possible exploratory surgery because of Arnie’s tenuous

pulmonary and cardiac status. I treated him with fluids and antibiotics. At this point in his care I had to leave town to attend an international medical meeting where I was chairing an important symposium. I left Arnie in the care of an excellent colleague in the same office where I did my practice. I knew Arnie would be in good hands. The family felt somewhat insecure over the fact that I needed to be away and for this reason I remained in contact with them on an almost daily basis by phone from Europe. On returning to Denver after one week, I saw that Arnie's condition had deteriorated significantly. He continued with high fever, a very high white count, and tenderness in his abdomen. This had occurred in spite of a broad spectrum of antibiotic therapy for what everyone agreed by now must have been a ruptured, infected diverticulum, which has consequences similar to a ruptured appendix. I was sure that he had a pelvic abscess. I talked with the family once again and they felt that any surgery was out of the question and I reluctantly agreed. It should be stated parenthetically that this was before the advent of the abdominal CT scans which would have made the diagnosis much easier. Here is an example where new technology would have been enlightening, but not necessarily a factor in changing the approaches to management.

On the very next day Arnie summoned me, expressing a great deal of anxiety over how he was going to face what we both knew was his impending death. In fact, we had discussed this before my leaving for Europe, but did not expect that the end would come so quickly. I promised that I would use whatever medication necessary to relieve Arnie's pain. This gave the family, who was in almost constant attendance, a considerable amount of comfort.

I was next called at approximately 2:00 AM the next morning by the nurse, who said that Arnie was "climbing the walls" with pain and anxiety. I quickly dressed and came to see him. It was clear that he was suffering badly. I asked the nurse to get me 8 milligrams of morphine,

which I planned to administer by the intravenous route in order to deal the overwhelming pain. I recognized that high doses of morphine may well depress respirations and hasten to Arnie's ultimate death. I gave the entire 8 milligrams of morphine over approximately ten minutes which brought calm and then slumber. The patient's respiration dropped and it was certain that he would not survive without any further intervention. We had long ago decided that mechanical ventilation would not be used for his terminal illness. It could only delay his death and lead to more suffering.

The patient's extended family had been mobilized and, in all, there were eight children and/or grandchildren including the wife in attendance. I asked everyone to come into the room and said, "Gather around everyone. We are going to watch your Grandpa die and go to heaven." I offered a brief prayer. I sat there in a rocking chair for the two hours that it required for Arnie's respiration to finally cease. I asked the patient's wife and daughter if they wanted the oxygen removed as a final act of defiance against the illness, COPD, which had dominated their lives for so many years. His blood pressure dropped gradually and about 8:00 AM Arnie finally quit breathing.

I attended the autopsy and was not at all surprised to find at least 2,000 ccs of pus and feces filling the abdomen. He had extensive diverticulitis of the lower colon with two areas of rupture. This caused the extensive soiling of the abdomen and abscess formation. This confirmed the fact that no form of intervention would have been life-saving even if it had taken place earlier.

Approximately six months later, the patient's wife called and made another appointment for herself and the oldest daughter. I had some fleeting concerns about what might cause them to drive 180 miles to Denver now that Arnie had died. We had a very good conversation in my office about the diagnosis and management and why surgery was not attempted. I was particularly pleased at the gratitude they

expressed over how Arnie's death was handled. They thanked me profusely for my constant attendance and, in fact, my availability during the time when I was involved in the European conference.

The main factor in this case is trust and the principle is benevolence. The patient and the family had a trust that I would do what was in the best interests of Arnie, without concern about the secondary effects of drugs such as morphine on hastening outcome, etc. It must be emphatically stated that this vignette that I cite is not euthanasia. It is the treatment of a suffering patient until death intervenes.

MARY AND JOHN



In 1984, when ARDS was just being understood as a common medical and surgical emergency, I got a call from John, the husband of a patient from New Jersey. His wife, Mary, had suffered ARDS following a period of prolonged shock associated with a massive myocardial infarction, where a significant amount of the left ventricle was damaged or stunned, so that it could not maintain adequate cardiac output. The patient's circulation was supported for a few hours with a synchronized balloon pump, placed in the aorta just beyond the aortic valve. This helps to supplement the pumping action of the injured left ventricle. Her course stabilized over the next three days, but stiff and difficult to oxygenate lungs was emerging as a manifestation of concomitant acute lung injury, leading to ARDS. This aspect of her complex illness was managed according to the principles that we had established, but the needs for high oxygen concentrations and high inflation pressures made use of the mechanical ventilator crucial for day-to-day survival. The heart function improved, but runs of rapid ventricular tachycardia, remained and were treated with amiodarone, known to help suppress these life threatening rhythm disturbances. A dreaded side effect of amiodarone, seen all too commonly by pulmonologists and cardiologists, is pulmonary fibrosis, which may be progressive and fatal if the amiodarone is not stopped. Pulmonary fibrosis may progress even after the use of amiodarone is stopped.

The reason for John's call was to plead with me that his wife's care team at the New Jersey hospital could not wean the patient from the ventilator after a period of about six weeks. The chest x-rays continued to show a great deal of shadowing and infiltrations that indicated massive damage with some indication of regeneration from ARDS, I later concluded. The physician in charge decided that any attempts at weaning would be futile and both Mary and John had concluded that continuing to live while supported by a ventilator for the rest of her life was not going to be palatable, and that they would soon decide to sedate Mary and remove the ventilator, or "pull the plug" in lay language.

John wanted me to come to see Mary for one last opinion. I happened to have a scheduled speaking assignment at Rutgers Medical Center, which was about fifty miles from John's hospital. I had scheduled an 8:00 AM lecture and Grand Rounds at noon, followed by rounds with the fellows in the afternoon. As I explained this to John, he said he would work out all the logistics, and get me to the hospital about 60 minutes away in time for a consultation and back to the University for noon rounds. This sounded like an impossibly tight schedule, but John so persisted that I reluctantly agreed to try to make this consultation.

Immediately after the morning conference, I was escorted out a side door of the lecture hall to a waiting limousine, and taken to the hospital in Somerset accompanied by John, who filled me in about Mary's past history and treatment at the hospital. In brief, Mary was 54, somewhat overweight and poor with exercise and diet since it was known that she had elevated cholesterol. She had had no previous cardiac events, and led a happy social life.

At the hospital, I was greeted by the head nurse and one of the junior surgeons who were taking care of Mary. She was awake and very alert and eager to communicate with me, even though she had a tracheostomy tube for use with the ventilator. She could mouth some words

and I was good at lip reading. We used a poster board and were able to communicate quite well. She adamantly wanted off the respirator and felt that she was gaining enough strength to make this a possibility, but each time she was taken off the respirator, she became fatigued and panicked. Just as soon as she began to feel a little stress of breathing, she would immediately be placed back on the ventilator. There was no systematic schedule for her weaning being used by the nurses or respiratory therapists.

Her physicians showed me her chest x-ray that had extensive shadows in both lungs. They had concluded that this was progressive pulmonary fibrosis, due to the scars that may accompany ARDS. My stethoscopic examination of her chest, however, did not suggest fibrosis, where the sounds are like velcro fasteners being pulled apart. I believed the shadows on her lungs represented proliferation, growth of new lung cells, which, according to my research and that of other groups, is indicative of lung repair! I gave a brief lecture at the bedside, about this process, and stated flatly that weaning from the ventilator would be possible if a systemized weaning plan that we had outlined before, was used, and that the patient would be able to be discharged home in a month or so.

Time was running out, and I was then quickly escorted to a waiting helicopter, which took me back to Rutgers accompanied by John, just 10 minutes in advance of my Grand Rounds Lecture. I don't even remember the subject of my talk, but I have vivid memories of the "house call" I had just made to the Somerset hospital.

Over the course of the next month, weaning progressed nicely. I had asked Mary to write me a postcard each week to tell me of her progress, and I always replied to help give her encouragement. She was finally discharged, and gradually returned to all of her previous activities, including very active gardening for a period of over five years after discharge.

For some reason, her cardiologist never did discontinue the amiodarone, and to my horror, the next thing to happen to Mary was the development of rapidly progressive shortness of breath from pulmonary fibrosis. This began about 12 years after the hospitalization. In the interim, Mary had no more cardiac events and did not even have palpitations, but for some reason the amiodarone was continued without a good reason. John contacted me again, and I arranged to look at her chest x-rays, which confirmed my fears. I insisted the amiodarone be stopped and it was, but progressive fibrosis continued in spite of treatment. Soon Mary was housebound and needed nasal oxygen.

This evolution of progressive fibrosis covered about a year. Mary did not want to go to the hospital to die, and John, who had been devoted to Mary all their married life, agreed that he wanted to keep her at home. I arranged with a sympathetic pharmacist to get oral morphine for her terminal care, and talked with John each day as he kept Mary comfortable at home until she died quietly one night.

The tragedy of Mary's case was one of stubbornness, or anchoring to a treatment that not only was probably not necessary, but ultimately shortened Mary's life. John and I correspond to this day, by card or phone, though it has been a decade since Mary died. He always still tells me how great those 13 years with Mary were. It is gratifying to be appreciated by patients and their families. This is the most important compensation a doctor can receive.

C H A P T E R 11

B I L L



Bill was 86 years old when he was referred to me by his primary care physician for progressive dyspnea on exertion due to emphysema. Although Bill had been a heavy smoker in the past, he had quit 12 years before, at the time of a bowel resection for colon cancer. His cancer had not recurred. Bill loved the outdoors. He played golf twice a week, and occasionally went trout fishing in spite of his age and frailty. This was quite an accomplishment considering his physiological impairments. On my initial evaluation, his FEV₁ was only about half of the predicted value, with only slight improvement with an inhaler. His oxygen saturation was 85% while sitting in a chair breathing room air. It fell to 78% after he walked down the hall with me, and his pulse increased from 88 to 136 beats/minute. On the basis of all of our previous oxygen studies, Bill was certainly a candidate for long term oxygen therapy. But he refused, saying that everyone he knew who took oxygen soon died. I pointed out that oxygen could prevent or forestall the progressive deterioration caused by right-heart strain.

It took me many months to persuade Bill to begin to take oxygen at night. His personal physician had also tried to convince him. Later, when Bill complained of progressive tiredness, he finally agreed. When I next saw Bill, he said he felt much better with oxygen therapy. He continued to receive nocturnal oxygen for the next two years. I saw

him quarterly as “co-pilot” with his personal physician.

Bill remained stable until age 89, when he reported feeling worse. He now had clinical signs of right-heart failure. With great difficulty, I persuaded him to take portable liquid oxygen with him when he went fishing and played golf and to use it for all activities of daily living. He finally agreed and found that he could continue to live an active life with this therapy.

Bill eventually had to give up fishing at age 91 because of his unsteadiness while wading in streams and along the banks of small lakes. After he could no longer play golf, his recreational activities were limited to outings at his club to play cards and to visit with his former golf foursome. He always used portable oxygen while driving his car.

By age 94, Bill had deteriorated to the point of being housebound. We arranged for around-the-clock nursing care because his wife, Charlotte, who was also elderly, needed help in getting Bill dressed and to the bathroom. We agreed that his terminal care should be at home and that no resuscitative measures should be instituted. He had a living will and durable power of attorney. Charlotte would be his surrogate decision maker, if necessary.

I continued to see Bill regularly and began to manage his urinary catheter drainage. When I saw him on his 97th birthday, I asked if he wanted to live to be 100. “Absolutely not,” Bill said, “There is not much pleasure in life any more.” After that, I continued to see him as his primary care physician. We often talked on the telephone about his various problems. Over the years of caring for Bill, I found that we had many common interests: fly fishing, pheasant hunting, poker, bridge. After his primary care physician retired, Bill relied more on me for his total care, even though he had another primary care physician in a health maintenance organization who showed no interest in caring for him because “nothing more could be done.”

When Bill was 97, Charlotte called me one morning because Bill had fever, cough, and an increase in sputum. His primary care physician insisted on hospitalization and would not see him otherwise, but Bill refused. On Charlotte's request, I made a house call, and because I identified signs of consolidation with my stethoscope in his right lower lung field, I gave him an oral antibiotic for suspected pneumonia. Bill said, "Just let me die." However, the next day, he had made a remarkable recovery. He was sitting up eating, laughing, and telling jokes. Because the response to antibiotics was so rapid, I concluded that we were probably dealing with pneumococcal pneumonia. He continued to make a rapid recovery from this episode.

About one year later, Charlotte called me at 1:30 AM in a panic. She said Bill was desperately short of breath and was dying. He had fallen upon getting up alone. I went to their house at 2:00 am and I found him awake, alert, and crying for relief. There were no signs of fracture or dislocation. I could not detect the cause of his pain and new severe dyspnea, except that he had a lot of mucus that was difficult for him to expectorate.

Bill was always adamant that he did not want hospitalization. I telephoned a pharmacist to arrange for some liquid morphine to mitigate his pain and dyspnea. I was amused when the pharmacist said, "Now, doctor, let's get this straight. You say you are at the patient's home, right now? You mean doctors still make house calls?" The night nurse went to get the prescription. Within an hour of giving him liquid morphine, he was comfortable, but death was clearly imminent.

Charlotte called me at 7:30 the next morning and said that Bill had died. I went to the house to "pronounce him" and to offer comfort. When I called the mortuary, the funeral director asked, "Did you notify the coroner of death at home?" I chuckled to myself and said, "No, this wasn't necessary." I told him that the patient was nearly 98 and had been under my constant care recently. A priest came, and we had a

celebration of life and death at the bedside. It was a moving experience as the three of us recited the Lord's Prayer. The funeral director came with a hearse and removed Bill. I talked with Charlotte for a while about how she would get along at age 87. She told me that her friends and neighbors could help her.

As a clinical investigator, I took satisfaction from the advances made in the care for emphysema that resulted from our original work, which began more than 30 years before this event. As a physician, I was equally rewarded by what I have learned about caring for dying patients at home in that same time period. Most of my elderly patients prefer to die at home; this is the main reason why I continued to make house calls.

In Bill's case, I had the opportunity to move from consultant to primary care provider, the chance to help my patient deal with the end of his life. The friendship that Bill and I developed over 12 years of "consultative care" was powerful. The astonishment of the pharmacist and funeral director that house calls are still made these days was humorous but reaffirming.

Bill died a comfortable death at home with the assistance of morphine to blunt his excessive respiratory drive. The importance of this aspect of care is often overlooked. Such treatment is not assisted suicide or euthanasia – it is caring for a desperately ill patient at the end of life. Of course, Bill could have been hospitalized to determine the exact cause of the unrelenting dyspnea and whether he had suffered fractures from his fall. I, and Bill, decided against this because I felt there was nothing that I could find that I or he would want to fix. Bill, Charlotte, and I had known for a long time that Bill was near the end of life. Hospitalization and a diagnostic workup in his final days would have been futile and expensive. The cost of dying at home is far less to the health care system; in Bill's case, it was the cost of some house calls, hospice nursing care, and the morphine used in his final

hours. Today, the health care system is burdened by excessive and, at times, inappropriate demands for high technology care. Nursing homes and personal care centers are an alternative, but they remove the patient from their favorite environment. Many patients can best be cared for in their homes.

Adapted from original source: "On Being a Doctor" Ann Intern Med 2005;143:234.

C H A P T E R 1 2

D R . F R E D



Dr. Fred consulted me at about age 68. He was a retired orthopedic surgeon from another state, and had moved to Colorado for the fishing and hunting. He was an avid fly fisherman, bow hunter, muzzle loading shotgun hunter, and North American small and big game hunter, who loved to shoot pheasants, ducks, geese, and other upland bird species. He only hunted big game, deer and elk, with antiquated methods as a matter of sport. He was also a gifted skier and won every race in his age group. In fact he outlived all his competition as he approached his final years.

Fred was troubled with shortness of breath on exertion, which was now interfering with his outdoor activities. A primary care practitioner diagnosed COPD, without any measurement of lung function, and advised Fred to move from his mountain home at 11,000 feet to a lower altitude. Fred hated the thought of leaving his friends and the mountains he climbed, skied, his playground for fishing and hunting. My examination of Fred revealed a very healthy man with good muscles and strength and ideal weight. With my stethoscope I heard somewhat decreased breath sounds and prolonged expiration. I suspected he had asthma, and this is what we easily confirmed by doing spirometry. His flows were 65% of normal, but with an inhaled bronchodilator, they increased to 115% of predicted for his age, height, and sex.

Since all his symptoms were exercise related, I began treatment with cromolyn sulfate, a little-used asthma drug in the current era, and one that is rarely considered in adult asthma, because it most commonly is effective in exercise-related and allergic forms of asthma, and in children. I chose cromolyn also because we had just completed a controlled clinical trial of this drug in late onset adult asthma and found it effective. It has very low toxicities, and is easily inhaled before exercise from a small metered dose inhaler. In a month, Fred reported complete control of his symptoms.

About two years later, Fred called me from his mountain home and said he thought that cromolyn was giving him chest pain. On questioning, the facts were that he had anterior chest heaviness and substernal pain on shoveling snow at his mountain home. I had him come to the office for more questioning and examination, and admitted him for coronary angiography, which showed multiple vessel disease. He had a four-vessel bypass without complications, and returned to altitude to continue his activities.

Over the years that I saw him, I treated Fred for gout, GERD, several bouts of acute bronchitis, and aborted what appeared to be an emerging influenza attack with Tamiflu, which dramatically reduced his myalgia, cough, and fever in a matter of hours.

Fred remained well between these acute illnesses, and would see me annually, for partly social reasons. We liked to exchange stories about fishing and hunting and would occasionally have lunch on a day when I was going to have the afternoon off. We enjoyed a glass of red wine on these days. Sometimes he brought his “girl friend,” a fierce competitor in ski races, but much younger.

Just before his next scheduled visit, Fred called me to say that he was having bad nosebleeds and was very short of breath. He was better when I saw him in Denver, but extremely pale. With my stethoscope,

I could hear the lung congestion associated with heart failure, and a rapid “galloping” heart rhythm, signifying failure of the left ventricle. His hemoglobin was drastically reduced to 6.5 grams (normal is 14 to 16 grams), and I immediately diagnosed pernicious anemia, and so-called high output heart failure. This was all confirmed in the hospital and he responded well to vitamin B12 which he would give himself for the rest of his life.

Fred gradually began to lose some of his physical abilities over the next years as he approached 90. He still wanted to keep living his lifestyle and was regular with his refills for Viagra. I gave Fred oxygen to use at night, which he said he rarely did. Fred also confessed to me that he had carefully sequestered a supply of barbiturates for his personal use in the future, when he no longer could fully enjoy life.

Fred was adamant that he did not want to give up life at his mountain cabin, and I agreed to be his decision maker at the end of his life. He abhorred the thought of moving into an assisted living facility. I made it very clear on his medical record that Fred was fully capable of making his own decisions, including those end-of-life judgments.

Fred took his daughter fishing at an alpine lake last spring, one that he could drive to. He was so tired that he could scarcely fish for 30 minutes. He told his daughter to take him home to rest. The next morning Fred was found dead, sitting in a chair with his oxygen running.

This story nicely exemplifies how a long, friendly, trusting relationship between a doctor and his patient, who also happens to be a doctor, can be successful and fulfilling for both parties. This is medicine at its best.

RICH AND FAMOUS



From time to time, many physicians are asked to see patients who are well known because of their wealth, their contributions to society, or the political position they occupy. Over the years, I saw a number of these patients, usually on a casual basis over the years that I had an active practice. I saw Willie Nelson on one occasion, when he was recovering from a spontaneous pneumothorax. His lungs had nearly completely expanded. When I elevated him, I was curious about his lung function, since he was an admitted heavy pot smoker. His spirometry was normal with an FEV₁ and FVC of approximately 110% of normal.

I had several detailed phone conversations with Rosemary Clooney, who was dealing with the difficulties of her asthma over a 10-year period. She was under the direct care of my good friend, Dr. Ed Rosenow III, who was responsible for most of the decisions in her case.

Certainly the most notable of the celebrities that I was asked to see was Governor John Y. Brown, then Governor of Kentucky, and Menachem Begin, Prime Minister of Israel in the early 1980s. Each patient presented unique questions and problems, which I will cite for the profound message that they carry.

JOHN Y. BROWN, FORMER GOVERNOR OF KENTUCKY

I had just completed a very pleasant five-day fishing trip with my

family and arrived home on a late Sunday afternoon to hear the telephone ringing. I answered the phone as soon as I could to hear a somewhat irate Phyllis George, a former Miss America and then the wife of John Y. Brown, Governor of Kentucky. She complained bitterly that I was one of the most difficult persons that she had ever tried to reach. I pointed out that I had been in a fishing camp where there were no ordinary telephones and that my medical service had been covered by a colleague who was very capable of handling my calls in my absence. She immediately stated that she had a private jet scheduled for me to leave Denver at 6:00 AM Monday morning for the purpose of making a trip to Lexington, Kentucky, to consult on the case of her husband. I said something like, “Mrs. Brown or Ms. George, there is no way that I can come to Kentucky tomorrow with the backlog of work that I have waiting for me in the morning.” She answered with a very irritable voice that raised several decibels above her usual tone, “Dr. Petty, do we have a bad connection or is there some reason that you cannot understand me? I am going to have you at the airport at 6:00 AM in Denver and you will fly to Kentucky and immediately see my husband, who is a patient at the University Hospital.” After a moment’s hesitation, it was very apparent to me that I was never going to win this argument, so I agreed to come and spent the rest of the evening arranging some additional coverage for the next day when I would be particularly occupied on this “unique house call.”

The Governor had suffered a massive heart attack four weeks before and had a long period of cardiogenic shock resulting in acute lung injury, which ultimately emerged into the acute respiratory distress syndrome (*see Chapter 2*). In fact, it was the report on ARDS, written by me, that led to Phyllis finding me and deciding that she wanted me to be her husband’s consultant. All of the family in Lexington had felt that for family and for “political reasons,” it would be best to transfer the Governor to Houston where the very best “critical care medicine” should be available. On my arrival at the intensive care unit, I went

over all of the details with the Governor's very capable surgeon, Dr. Ed Todd. It became apparent to me that the patient, who had been receiving mechanical ventilation for 13 days, should receive a tracheostomy for comfort and for feeding, and that there was no reason why weaning would not occur, following recovery of acute lung injury, which was an observation that we had made a number of years before.

Together we held a press conference to make the point that there was no need to transfer the Governor to Houston and that his care would predictably continue successfully at the University Hospital, where excellent care was being provided. This is exactly what followed and the Governor was rapidly weaned from mechanical ventilation over the next two weeks. He was discharged home a week later and continued his rapid improvement.

During my many conversations with the Governor that followed this, the question about the need for specialized care facilities for critically ill ventilator-dependent patients came up. The Governor decided to investigate the possibility of launching a special care facility for the catastrophically ill ventilator-dependent patients. He became one of the co-founders of the Vencor Hospital system, now known as



Newspaper clipping of the author at press conference in University of Kentucky Hosital, Lexington

Brown seen making full recovery

From Page D1



Author addressing the press in Lexington, KY about the condition of Gov. Brown.

Kindred Hospitals, that has successfully developed a very reliable and sophisticated method for weaning many patients with far more severe lung impairment than exhibited by the Governor. I have remained in contact with Governor Brown for the many years that followed.

PRIME MINISTER MENACHEM BEGIN

I had just completed my year as President of the American College of Chest Physicians and was sitting in my office on a Sunday morning relaxing and patting myself on the back just a bit because I had saved the entire week to relax and go fishing, following a very arduous and successful convention of the American College of Chest Physicians held in Toronto in October of 1982. My quiet morning was interrupted by a phone call from the Begin family in Israel describing the plight of Mrs. Alicia Begin, the Prime Minister's wife, who was desperately ill and receiving ventilator care for acute respiratory failure at the Telshomere Hospital in Jerusalem. The family requested most



Prime Minister Menachem Begin with author in the Begin's living room in Jerusalem, October 1982.

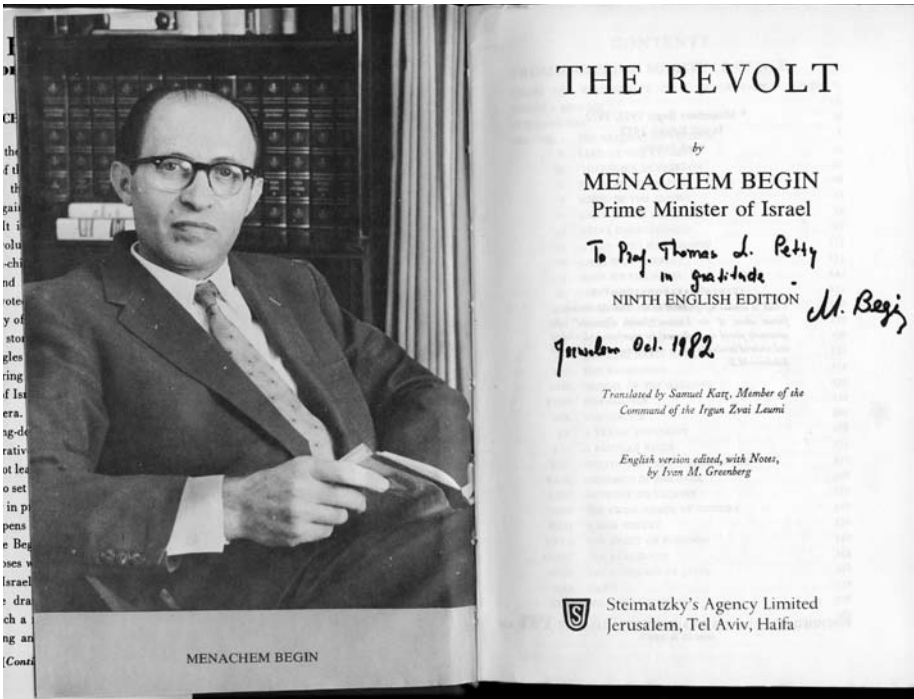
adamantly that I travel immediately to Jerusalem to see if anything could be done to help with the ventilation weaning process. Mrs. Begin had been receiving mechanical ventilation for nearly three weeks, following a bout of severe asthma. On the next day, I made the trip to Jerusalem via Boston and Paris and was quite exhausted when I was met on the tarmac by a group of soldiers, dressed in full uniform and led by Benny Begin, son of the Prime Minister. He helped me off the plane and onto a military truck to avoid any possible interference

by customs or other hassles at the airport. I was also met by my friend, Dr. Jerry Baum of Tel Aviv, a longtime colleague and advisor since the budding of my career in pulmonary medicine.

After going over all of the details of Mrs. Begin's situation, it was very apparent to me that she was suffering from advanced emphysema, incorrectly diagnosed as asthma by the Prime Minister's four physicians. Apparently, they were trying to put forward the most hopeful positive image of her chances for recovery. Mrs. Begin was now suffering from complications from the endotracheal tube, which had been in place for a long period of time. I convinced the doctors and Prime Minister Begin that a tracheostomy should be done to allow for proper nutrition and for the patient to communicate. This was accomplished on the second day of my visit. On the third day it was apparent that things were improving slightly and Mrs. Begin was able to sit up and visit for the first time in over a month. At this time, I spent the entire afternoon discussing Mrs. Begin's prognosis with the Prime Minister, which was clearly dismal by all indicators of disease. His four medical consultants were present during this discussion. To say that the Prime Minister was furious with his physicians would be a gross understatement. He realized that he had been misled about Alicia's diagnosis and prognosis. It is true that she had suffered asthma all her life and had treated this mostly by smoking Stramonium cigarettes, an age-old remedy containing a potent anticholinergic agent. She also smoked cigarettes at other times and had very hyper-inflated, nearly destroyed, lungs by the time that I saw her. I outlined a program, which was not exactly followed in full detail and approximately one month following my return to the United States, I received word that she had died while still on a ventilator, with a respiratory rate of zero. How this was intended to be of assistance to the patient eluded me then, as it does now.

The major messages of these two special patients was a need for an outside expert for further consultation to coordinate care and

to develop a systematic approach, one which would be predictably successful, and the other one not. It is in these settings that the pulmonary physician can provide some of the greatest assistance, not only in dealing with the technological aspects of disease but also in counseling a bereaved family who, by well-meaning family and friends, is often bombarded by conflicting messages about what to do next.



Book, authored by Prime Minister Begin, with a dedication to the author.

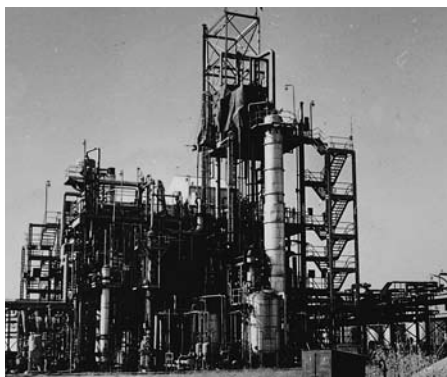
INTERNATIONAL DISASTER



One of the most moving experiences of my medical career happened in 1984, at the time of the toxic environmental disaster which happened in Bhopal, India. A chemical product with several uses in the agriculture industry, methylisocyanate, had leaked from the Union Carbide Plant, forming an irritant gaseous plume. Since the toxic substance was heavier than air, it swept down the valley from the plant, affecting thousands of people who lived near the facility. Often the wall around the plant functioned as part of the shack which served as home for the poverty-stricken population, many of whom worked in the plant. People were seen running away from the barely visible plume and often dropped dead, while others were spared possibly because the concentration of vapor was not uniform or perhaps because they protected themselves by breathing slowly and shallowly as some observers suggested in the elderly, and did not breathe in deeply, thus minimizing over-exposure of this irritant material.

I was originally asked to be part of a triage and medical observer rounp that originally was going to meet at the World Health Organization in Geneva, Switzerland, and then fly to Bhopal on a private jet. When these plans fell through because it was difficult to mobilize doctors to respond, I ended up traveling as a private citizen via Pan American Airlines all the way from New York near Danbury,

Connecticut. I had gone there for a briefing to Bombay, India, via Frankfurt and Dubai. Following a briefing at Union Carbide, I was resting in a Danbury Hotel when I received a phone call from a man with an English accent advising me to vacate my room immediately because it was “soon to be bombed.” Where this terrorist message originated made very little difference to me. It was impossible for me to rest alone in my room and thus I waited for two hours in the lobby before the shuttle came to take passengers to New York’s Kennedy Airport. Once in the airport and checked in, I realized that I was being followed in and out of restrooms, airport bars, etc. I was traveling first class and did not see the man who was following me after I was seated in the small front compartment until I reached Bombay more than 24 hours later and again I noticed him following me into a restroom in that filthy airport. I identified myself to him by passport and he gave me a blank stare. I supposed that he was a CIA agent, because of the mystery surrounding the toxic tragedy and perhaps, since I had worked with Union Carbide before in the development of the first liquid oxygen systems (*see Chapter 1*), I may have been considered some sort of “agent” for Union Carbide. There were no other doctors that I noticed at the airport in Bombay who were trying to reach Bhopal at that time.



Union Carbide plant in Bhopal, India, 1984.



Streets of Bhopal, nearly abandoned on the date the plant was restarted to consume the product that had leaked and caused toxic injuries and death.

I had no hotel reservations in Bombay and, of course, thousands were trying to reach Bhopal because of the magnitude of the tragedy. Many of these were American lawyers who wanted to get in the “litigation queue.” All planes to Bhopal were fully booked and the only way you could check on a booking at that time was to stand in a long line to get to a desk where an official had the names of the passengers entered into a large three-ring notebook. No computers then! I managed to find a cab driver who took me down dark alleys in the heart of Bombay to a dingy and dirty room where I was able to rest for a few hours. I can still see the moths swirling around a dimly lit ceiling fan in the tiny lobby. I couldn’t even take a shower because the water was brown and smelled of sewage. Fortunately, I could order two quarts of beer to my room. After I hydrated myself, I got some sleep for about four hours, and then I went back to the Bombay Airport with some cab driver who was quite helpful. At this juncture, I thought I had lost my “tail,” since the cab driver drove very fast and often down alleys to and from the airport.

(Another person started following me in the Bombay Airport, but I don’t think he ever got on the plane to Bhopal. To my knowledge, I was not followed in Bhopal, but I stayed in the Union Carbide compound, which was a guarded and gated facility.)

I was standing in line at the Bombay Airport and must have had 300 ahead of me, and wondering how in the world I would get on the next airplane – a small, two-motor jet that could not have held more than 120 passengers. I approached a well-dressed Indian Sheik to tell him of my objectives and my plight. He was most kind and helpful. He asked for my business card and 10,000 rupees and soon came back with my permit number which had magically jumped to fourth. Signs cautioning against bribery were everywhere, but soon all of the lawyers behind me began to clamor for positions up the line.

Fortunately the plane took off on time and I was met at the Bhopal Airport by representatives of Union Carbide, who took me imme-

diately to their compound. I took a shower, had a couple of beers, and received a further debriefing about the disaster. Apparently high pressures had built up in the main tank that contained the methylisocyanate and the most likely theory put forward by Union Carbide engineers was that water had been introduced by a saboteur, which created steam and increased pressure, leading to a blow-off of the pressure-release valve. Nothing else seemed to make sense, since this plant had functioned flawlessly for the previous 17 years. This plant was extremely important in the economy of the region and was a major factor in the improved agricultural yield of crops in that part of north central India. By the time I got to Bhopal, it was nearly a week after the tragedy; the Hindus were already cremated and the Muslims buried on the day of the tragedy as custom dictated.

The next day I went to the major teaching hospital where I got a glimpse of two doctors, whom I knew from the Center for Disease Control in Atlanta. They saw me, but made no attempt at a visit. Somehow I was considered an “outlier.”

I was assigned a senior medical student from the Gandhi University and the next morning was taken to the Hamidia Medical College to meet Professor Mizra, then Dean of the faculty. He seemed to gloat over the opportunity this gave him to make himself famous, a fact that really disgusted me both then and on later occasions. I was accompanied by several medical students. There were no other foreign physicians at the hospital while I was there. I was told that Dr. Hans Weill, an occupational medicine expert from Tulane had been there for one day before my arrival. I only saw him on my return trip to Denver about a week later (see below).

I saw many patients in dilapidated wards who were all suffering from moderate to severe respiratory distress. Oxygen was in limited supply and was given to those most severely ill. There were no blood gases, spirometers, or other respiratory equipment. Soon, however,

boxes of blood gas machines, bronchoscopes, ventilators, and other useless equipment began to arrive ostensibly from the World Health Organization, but who knows. These expensive machines certainly weren't useful, but the stethoscope served us well as we examined many patients.

I visited several families at home to hear firsthand about the tragedy, saw quite a few patients at the bedside and spoke with some of the young doctors in training. Based on my experience with acute lung injury (ARDS), it was apparent that most of the injured would recover. I gave the patients and families as much comfort as I could. It was also obvious upon reviewing stacks and stacks of chest x-rays that pulmonary disease was very common in the region before this disaster and I was struck by the number of apical cavities and pleural effusions that I saw prior to the tragedy. This part of India, of course, has a high prevalence of tuberculosis and other infectious diseases.

On Sunday I attended Hindu services along with local Hindus of Bhopal. It gave me comfort to stand in a long, slowly moving single file line of people, waiting to take communion from the "Priest." The Hindu religion believes in reincarnation, and this concept presented the quiet reassurance which I overheard from the throng assembled in the temple.



Signage in the Hamadia Hospital, Bhopal, 1984

I was quite relieved when my work in Bhopal was complete after about five days, and I returned via New Delhi. At the airport, I met my old friend, Hans Weill, who had been touring the north of India and the Taj MaHal, during the time I was in Bhopal. After take-off, we shared several glasses of

champagne together as we began the long trip retracing our steps across the country. Champagne never tasted so good.

After a very long journey and the usual customs delays, I was met by a limousine in New York, taken to my hotel, and given my room keys by the limo driver. I was quite tired, but not as exhausted as when I arrived in Bombay. A representative of Union Carbide was scheduled to meet with me shortly, but upon answering a knock at my hotel room door, I encountered a person who identified himself as an FBI agent, who said that I was under arrest because I had registered under an assumed name in New York. Since I was told that I was pre-registered, I was thoroughly bewildered by these events. The matter was quickly resolved by the Union Carbide doctor who had originally contacted me to go to Bhopal. He did not explain this needless emotional trauma and, in fact, I was not interested in any more discussion of the matter. I was mentally and physically beat.

We spent an evening of debriefing over a few beers and some hamburgers, and I finally got a good night's sleep before returning to Denver.

Three years later I participated in a major scientific conference in Bombay sponsored by the Lung Division of the National Heart, Lung and Blood Institute, but by this time Professor Mizra was no longer in good health and could not attend the meeting. It became apparent that there were a number of issues relating to numbers of people suffering from cough, wheeze, and dyspnea, and some with measured obstructive disease and others with restrictive disease. Whether or not these illnesses were caused by the toxic exposure will never be answered on the basis of any rigorous epidemiological study but many patterns emerged that strongly suggested that this was the case. No one from Bhopal had received any compensation for death or injury, even though a large "settlement" was paid.

I used my stethoscope a lot on this occasion, which was the one instrument that was understood by the medical students and doctors involved. In fact, it was the only instrument they had to evaluate their patients and they seemed well trained in its use. They were behind in technology, but not in their interest in caring for their patients.

This disaster, which sounds like a script for a James Bond movie, was a powerful experience as it brought me face to face with suffering and multiple dying people of another culture. These victims had suffered pain, grief, mental anguish, and threat to life. Their human strivings for hope, relief of fear and pain, and faith proved to be similar to my own and in parallel with all other nationalities I had previously encountered while on medical missions.

C H A P T E R 15

ANNIE



More than 20 years ago I received a handwritten letter from a woman who had suffered from a lung disease and gotten my name from the library of her community, Selma Alabama. She described herself as a poor black single mother, with shortness of breath and cough. She had smoked very little in her lifetime. I answered her questions, and very shortly received a second letter with more questions. She had many symptoms, affecting most organ systems. I thought she may have sarcoidosis, and she had this checked at the local community hospital with negative results. On a frequent regular basis, I received very legible and intelligent questions and became more and more curious. She offered her phone number, and I called her one morning, just to learn more about her. I found her charming and engaging.

From then on, we became “pen pals.” She related to me her years of struggle, her grief over bearing a stillborn son, and the refusal of medical personal to let her hold the dead infant, before relinquishing the body. This thought plagued her entire life. She told me about her teenage daughter and the problems with community life in her humble surroundings. She also tried to give care to her nephew, who was already into the drug scene, in his teenage years. She told me about participating in the march from Selma to Montgomery with Martin Luther King.

It became a custom for her to call me on my birthday and I called her on hers. She once “borrowed” \$200 from me for her phone bill, which I reluctantly sent as a gift, since I said I did not lend money to patients, friends, or family. I would usually send her a little cash for Christmas.

On one occasion, I found myself in Birmingham, with a schedule to also speak in Montgomery. Fortunately my host, a pharmaceutical representative, was willing to drive me to Selma to meet Annie one summer, a few years ago. I had little trouble finding her small, and poorly repaired house, in the poorest part of Selma, because everyone I asked directions of, knew where “Ms Annie” lived.

She awaited me on the porch, accompanied by her daughter, who was now in her 20s. She called the neighbor kids for her nephew, who reluctantly came, for a minute. He was surly and rude, and I knew he had trouble ahead. After her daughter left us alone, we discussed how to foster her education and growth as an individual, with the goal of living outside of the ghetto. We discussed this frankly.

After about a two-hour visit, we said goodbye but remained pen pals. Once, when I moved files, Annie’s was about 10 inches thick representing two decades of correspondence.



Newspaper clipping of death of Annie’s nephew. Annie died shortly thereafter following a wasting disease and depression.

Annie continued to write, and now about weight loss and poor appetite. Her doctor diagnosed a “stomach ulcer,” but I feared cancer. She continued to lose weight. Then her nephew was murdered in a drug deal. A newspaper clipping carried the story. Afterwards, she gradually became deeply depressed and deteriorated rapidly. Shortly thereafter I received a phone call that “Annie had passed.”

I learned so much about her and the poor black culture, the frustrations of trying to live in dignity, and to raise children in our troubled society. She was a friend. I miss her.

PART III

EXPERIENCES AS A PATIENT



C H A P T E R 1 6

VIEW FROM THE BED

*(Adapted from an editorial originally published
in CONTEMPORARY INTERNAL MEDICINE September 1992)*



An attack of severe angina at the Frankfurt Airport in 1992 accompanied my effort to make a tight connecting flight from Barcelona to Chicago/Denver. As angina is supposed to do, the substernal chest pain stopped when I did. It returned when I got going again, and stopped again as I reached the ticket counter.

At that moment I had to decide whether to learn about the German healthcare system firsthand or get back to more familiar surroundings. Despite my respect for the German system, I opted for home territory. Through providence, good luck, or both, I returned to Denver uneventfully, and then quickly got the message through a series of near catastrophic events that I needed to be in an ICU – immediately.

INITIAL EVENTS

February 14, 1992, was not a particularly frivolous day for me, although I was supposed to be cheered by the St. Valentine's messages displayed on the walls of ICU. The decision to bypass some short, but very narrow regions of atherosclerosis and possibly repair a regurgitant mitral valve had already been made jointly by my cardiologist, my surgeon, and me while observing my coronary anatomy on the catheterization table.

The hospital routine was beginning to feel more natural and I was ready to receive instructions and explanations about “how it will be from now on” or some similar pronouncements from the evening nurse. When told that I would be restrained with hands and feet tied down following surgery so that I would not pull out any tubes or otherwise injure myself, I stated emphatically that I did not want to be restricted under any circumstances once I became awake and alert.

I emphasized my right to privacy and bodily self-determination, and my right to refuse or accept any or all medical care. I documented on the chart that I categorically refused to be restrained unless totally irrational. (In more than 30 years of providing postoperative care I had never had to use restraints except in such extreme situations as delirium tremens or the development of convulsions.)

On being wheeled into surgery the next morning I was very optimistic about the success of the surgery and my survival. Following surgery I awoke unrestrained in the ICU, but struggling to breathe on the respirator because the inspiratory flow rates were set too low. As an expert on the patient-machine interface, I tried to communicate what was wrong, but I had too much tremor to write on a clipboard. After about 2 hours, my surgeon and a consulting pulmonologist removed the endotracheal tube and respirator. Although I didn't need the respirator in the first place, there was an established routine of ventilating all patients overnight following open heart surgery. My objection to being restrained and to the routine use of the ventilator were the first in a series of events that labeled me a “bad patient.”

RECOVERY BEGINS

My earliest recollections of recovery are fragmented due to the use of hypothermia during the operation. I'll cite just a few. Knowing you are awake . . . a familiar smiling face . . . ice chips. Being left alone –if only for a moment . . . Beginning to feel stronger . . . Morning coffee

sneaked in by a friend . . . First food . . . Bladder catheter and chest tubes removed . . . Transfer to the wards . . . Standing for a moment . . . First shower . . . Walking in the hall . . . A friend brings a beer.

I also became more aware of ward procedure. I was startled when a night nurse on my first day in the monitored unit came to tell me that evening vital signs were taken between 10 and 10:30 PM, and that pain and sleeping pills were given only at that time. When I told her that I usually fall asleep at 8:30 PM and always sleep well for 3 or 4 hours, she said, “No way am I going to disrupt my ward routine.” I fired her and spoke to the evening supervisor who explained that individuals had to adjust to protocol.

My response was to use a magic marker and make a sign that said, “When this sign is displayed, kindly respect the patient’s right to privacy – do not enter room.” This caused all sorts of problems. The issue seemed to be one of control. I said to a head nurse I had known for some time, “Why can you do nothing at all for your patients’ needs for rest, privacy, and some level of self-determination?” I began to see that the system is set up to serve the healthcare hierarchy rather than the patient.

ON BEING SENT HOME

Everyone who has had a life-threatening illness looks for the day of discharge. Being home, sitting in a favorite chair, and sleeping in your own bed can only be described as wonderful. Walking outdoors and sensing that recovery is right around the corner build hope for a return to zestful living.

Fortunately, I improved rapidly. I persuaded my physicians, who were reluctant to let me even look at the daily office mail, that I would be better off doing some office work and feeling productive rather than being bored or stewing about myself. I was able to catch up on mail and take the time I needed for correspondence, both of which added to my

confidence about full recovery. I rested, took my afternoon naps and walks, and made plans to resume some speaking engagements, including those set more than a year in advance that involved overseas travel.

My physicians were somewhat hesitant. I remained in atrial fibrillation and was anticoagulated, but felt reasonably well with a slow controlled rate. Conversion to normal sinus rhythm was delayed by some remaining fluid in my chest.

SETBACKS AND RENEWED HOPE

One night I found I couldn't sleep on my left side: I was short of breath. Being a pulmonologist, I immediately realized that something must be wrong with my left lung, since the down lung does most of the breathing and gets most of the blood flow. I had no trouble sleeping in other positions.

I had been monitoring my breathing capacity with a simple incentive spirometer for vital capacity and a pocket-sized peak-flow meter, and noted a fall in pulmonary function that was then confirmed by formal study. By self-percussion I had detected a flat percussion note on my left chest. I requested a chest X-ray, which confirmed the presence of a large pleural effusion on the left side. This was tapped and revealed all the characteristics of an exudate. The effusion was only slightly bloody and contained no malignant cells. No mycobacteria were found on culture. These were good signs.

Nonetheless, my doctors and I were concerned: A late exudate some 3 months after surgery, particularly one that interferes with comfortable breathing, is not normal. A small pneumothorax following the thoracentesis forced me to cancel a trip to the annual meeting of the American Thoracic Society (ATS) where I was to moderate two panels. I had not missed an ATS meeting in 30 years. I did breathe better, though, and was even able to go fishing.

I decided to have a radiologist check my diaphragmatic function by fluoroscopy. He said both diaphragms moved nicely, but I pointed out that the “sniff test” is the correct way to look for diaphragmatic paralysis. By sniffing as if your nose is running, each diaphragm should pop down. In my case, the right diaphragm responded properly, but the left showed paralysis by popping upward. I told the radiologist that loss of one diaphragm compromises about 20% of ventilatory function – and felt almost smug in participating in my own diagnosis.

A former fellow told me that exudative effusions on the left side are common when the internal mammary artery is used for coronary artery bypass. Of course, the phrenic nerve is sometimes injured where it runs together with the internal mammary artery in the same sheath. My surgeon said regeneration of the nerve is common in 6 to 12 months. Hope returned.

Then my pulmonary function began to fall quite dramatically. In addition to not being able to complete my afternoon walks, my forced expiratory volume in one second and forced vital capacity were in the range of disabling dyspnea. Was I another physician who had developed a disease associated with his own specialty? Could I have rapidly progressive bronchiolitis obliterans and obstructive pneumonitis?

I didn't respond to bronchodilators, but on the repeat X-ray, which I requested, I noted some Kerley's B lines on the right that indicated fluid retention. The left pleural effusion had increased. Although my cardiologist insisted that this was not heart failure, much of the fluid cleared nicely with diuretics, and my pulmonary function returned toward levels I had determined prior to surgery. I could be hopeful again. I could also walk unlimited distance in Denver. Strength and stamina had increased. At a recent Aspen Lung Conference, I felt comfortable at 9,000 feet and could fish, both of which meant a lot to me.

BASIC POINTS

In our routines and very scheduled lives, we physicians can forget some basic truths of care:

- Everyone who enters a hospital has fears and personal needs. The right to privacy and self-determination must not be ignored.
- Even though a physician is transformed into a patient by disease, he continues to utilize his medical knowledge. That must be recognized.
- A person who has had a brush with death can withstand almost anything once aware that he or she has not died.
- Encouragement from friends, family, and the people providing healthcare really matters.
- Hope is the major driving force to recovery.

PERSONAL MISERY



All physicians, no matter how broad their experience, should have their own personal physician whom they see on a regular basis for their symptoms and for preventive care. Physicians make poor doctors for themselves. They cannot be objective and often jump to the wrong conclusions. They are poor self counselors. It takes an outside friendly, interested, and knowledgeable person to guide a patient, including a physician through the fears, anxieties and procrastinations associated with the onset of a new symptom or laboratory finding that has been obtained through screening or sometimes by accident.

More than ten years ago I learned from my personal physician that my prostate antigen known as PSA was “a little high.” He had added the PSA test to my blood exam at the time of a regular checkup, but had not told me. His rectal exam did not reveal any nodular or suspicious areas in the gland. Normal PSA is less than four for men under 60 and with a family history of cancer; probably less than two should be considered the normal range. I had a family history of a father with prostate cancer and a grandfather said to have died of prostate cancer at a very young age, but he had other co-morbidities that were more likely the cause of death. Thus, with this history I was concerned and had the PSA repeated. The surprising report was 16, which was in a clearly elevated range. A repeat test was 20. I consulted another

colleague, an urologist, who did an ultrasound of my prostate, which had no palpable nodules. This was negative. Nonetheless, six biopsies were taken of the prostate gland and seminal vesicle. All six proved positive for cancer grade 6, which is the most common grade, range three through ten. The PSA was high enough to suggest metastatic disease and the seminal vesicle biopsy was normal, which was reassuring for the possibility of still having a local lesion. A total body scan, including bone and liver scan, was negative.

Accordingly, the next consideration was treatment. At the age of 64, my life expectancy was probably more than ten years, even though I had had prior heart surgery five years before. Little was I to know that I would have three more open heart surgeries subsequent to the prostate cancer, as well as other life-threatening diseases documented in this book.

At the time, the most popular treatment strategy for cure was radical prostatectomy. The alternative was external beam radiation therapy, which avoided the surgery but had many complications; i.e., incontinence, sexual impotence and inflammation of the bladder and bowel. A third and not very popular alternative was brachytherapy; i.e., the placement of radioactive seeds directly into the prostate substance. This was not a popular procedure in Denver at the time. Many reports in the literature suggested watchful waiting, which did not appeal to me. My radiologist, through his contacts, arranged a visit with one of the pioneers of this procedure, Dr. Gordon L. Grado of Scottsdale, Arizona. He did a very careful evaluation and review of my situation and felt that I was an ideal candidate for brachytherapy which I had under his direction some three months after the diagnosis was originally made. I was in the hospital only overnight, largely because I had been on aspirin and Coumadin before, although the Coumadin had been adjusted to normal prior to the surgery to avoid excessive bleeding. I didn't have a great deal of pain and was very pleased that urination was only a little bit painful following the procedure. This

got a lot worse, however, as bladder irritation began to take over and I developed what patients refer to as a “flappy bladder.” With a “flappy bladder,” you feel like you need to pee every 15 minutes or so but produce very little urine. This can be embarrassing when driving on the freeway or in crowds. I developed some interesting highway techniques of stopping, pulling my car over, and elevating the hood to relieve myself with the warning light flashing. I was caught only once by a highway patrolman since I had not pulled completely off the shoulder of the highway. He didn’t quite understand what I was doing but let me go without a citation.

Fortunately I have had no recurrence of the prostate cancer. In the more than ten years following the procedure, my PSA is normal and barely detectable. Thus, I am “cured.” I have no bowel or bladder complications now, but the infected hemorrhoids that followed the brachytherapy most likely led to life-threatening endocarditis (*see Chapter 19*). I am extremely lucky because prostate cancer still kills approximately 35,000 men a year. Widespread use of screening could change this. The procedures are also improving and the outlook is favorable for the future.

We also need to promote screening for patients at high risk of lung cancer, which has been another campaign of mine for many years. These efforts to change practice patterns are very slow to be successful but some progress is made through persistence.

Personal Comment: There is some luck involved in my early diagnosis since the check-up with my personal physician was done without knowledge of the prostate screening component. This sort of communication or lack of communication can greatly strain the trust between physician and patient for obvious reasons. I was fortunate to be referred to an expert radiation oncologist, who could recommend the best therapy for me. My personal urologist, Dr. Bill Manniatis

of Denver, whose standard treatment for his patients with prostate cancer was radical prostatectomies, recommended the brachytherapy for me. Thus, he chose what was best for me and not where he would benefit financially from the surgery. It was also fortunate that my immune mechanisms very likely dealt with micrometastasis that was strongly suggested by the very high PSA levels prior to therapy.

C H A P T E R 1 8

BACK FROM THE BRINK

(Originally Published in *IM INTERNAL MEDICINE* May 1999/Vol. 20, No. 5)



In the summer of 1998, I was in immediate need of a second open-heart operation because of severe mitral insufficiency. This condition had been worsening over the previous three years, ever since I'd undergone emergent coronary bypass surgery in 1992 for angina and cardiac arrest.

Now I was developing significant pulmonary hypertension. My most recent catheterization revealed pulmonary pressures of 75/35 mm Hg (normal is 25/10). A transesophageal echo showed reversal of blood flow.

I contemplated the risks involved in my next operation, which would be a mitral valve replacement or, preferably, a repair. There was the risk of death, first of all, which my surgeon confidently put at only 5% to 6%. I was concerned about being on a mechanical ventilator again, dependent on the very technology that I'd helped to develop. I was worried about postoperative complications, especially acute respiratory distress syndrome (ARDS). Ironically, I had just returned from attending the 41st Aspen Lung Conference, where I'd presented an historical perspective on ARDS and new information on the cascade of inflammatory cytokines released in states of shock, aspiration, and other massive insults to the body.

I had expressed my concerns to Bill, my pulmonologist and a former

fellow. I asked him to monitor me closely in the immediate postoperative period. He reminded me that if I did get into trouble, I wouldn't be aware of it – not with modern pharmacology. I found this a scary thought, being totally out of contact with reality, not being able to participate in my own recovery. I made Bill promise me that paralytic or sedating agents would not be used to adjust me to the mechanical ventilator postoperatively.

“If I get into trouble,” I told him, “I want to know what’s going on. If I’m dying, I want to experience it.”

A TERRIBLE DREAM

About two nights before my surgery, I had a terrible dream. In this dream I was walking outside the hospital where I would have the operation. It was dawn and very foggy. I stopped at a newsstand and bought a paper. I opened it to what I thought was the sports section. Instead, I came across an obituary: “Denver Physician Dies.” I read in horror that this physician, whose initials were TLP, had died of complications from surgery.

The dream exacerbated the ominous feeling I had about what might happen to me postoperatively. The day of the surgery, I expressed my concerns once again, this time to the anesthesiologist just before the procedure began. He assured me that with pressors, fluids, and mechanical ventilation, I'd do fine.

When I regained consciousness that afternoon, my friend Louise, a nurse, told me that the surgeon had been able to repair my mitral valve. I was delighted. She said that everything looked good. I was still on the ventilator, however, and restrained. I made it clear that I didn't like this, but Louise explained that I'd been thrashing around quite a bit and needed to be restrained.

I still felt uneasy. I moved my arms and legs; everything seemed fine.

I hadn't suffered any neurologic injury such as spinal cord ischemia, I concluded.

After about an hour, I was extubated. This was an important step in my recovery. I could breathe on my own but needed high-flow oxygen by nasal cannula. The oxygen burned my nostrils a little, but otherwise I felt fine for about three hours after being extubated.

FEELING OF IMPENDING DOOM

Then, out of nowhere, it seemed, a feeling of impending doom swept over me. I told Marsha, my other nurse, that a catastrophe was about to happen. I was sure I was about to die.

Marsha was very kind and patient. She pointed out that my vital signs were stable, that everything looked great. In fact, they were going to order a liquid diet for me to have in about an hour.

The thought of eating something nauseated me.

I vomited some fluid, which I was careful not to aspirate. I feared aspiration because it's a major cause of ARDS. I also felt a little guilty because I had violated my NPO-after-midnight orders by taking two ubiquinone tablets around six o'clock that morning. I chewed up the tablets with some peanut butter to aid absorption.

Ubiquinone is a powerful antioxidant. I wanted to have my antioxidant defense mechanisms turned up as high as possible before surgery to avoid acute lung injury if I went into shock or aspirated.

About five minutes after I vomited, my blood pressure began to drop – to 90/55, then 50/palp. Marsha had my IV fluids running wide open. My bloody chest drainage was now being infused through a major central vein. I heard another nurse order the blood that I had stored before the operation. Albumin was given.

“Are you still with me?” Marsha was saying as she put me in deep

Trendelenburg position.

I could see on the cardiac monitor that my left atrial pressure was now 5. Then I saw runs of ventricular tachycardia for fifteen to twenty seconds. Someone brought in a defibrillator.

“I’M DROPPING OUT . . .”

Bill Pluss, the pulmonologist, came in the room. I told him I was “dropping out.” He looked terribly concerned. I kept asking him questions. Could I be tamponading? Was I septic? The nurse said that my temperature was 103.

A chest x-ray was taken. Bill showed me that my cardiac silhouette was normal, with mediastinal tubes in place. No pneumothorax was present.

Now Louise was holding my hand, telling me to hang on. I never lost consciousness, but my vision became gray and pulsatile. It was like an old flickering black-and-white newsreel. Then I felt as if I had slipped into a dark whirlpool, as if I were literally going down the drain, feet first. Whatever I was descending into felt hot.

“I’m dropping out, I’m dropping out,” I kept saying over and over again.

Then they started Levophed. When I was an intern, we used to call this potent peripheral vasoconstrictor “Left for dead” because so many patients who got it went on to die. In my case, however, it turned out to be the turning point, the pharmacologic hand that pulled me out of the jaws of death.

Hours later, after massive blood replacement and infusions of fluids, albumin, lidocaine, magnesium sulfate, and other medications, my condition slowly started to improve. By midnight, my pressure had risen to 100/50. Kern Buckner, my cardiologist, came in to check on

my hemodynamic status. Because my left atrial pressure was still low, he ordered more fluids and albumin.

Eventually my pressure reached 125/70. Words cannot describe how much better I felt. It was truly a miracle, I thought.

TALKING THROUGH THE NIGHT

Julie took over my nursing care after midnight. By now, I was afraid to go to sleep, for fear of never waking up. Julie stayed with me and we talked through the night – about politics, religion, raising children, medical experiences, anything.

In the early morning hours, the Levophed was tapered and the fluid infusion slowed. By morning, I was out of the Trendelenburg position and sitting up slightly in bed.

The oxygen burned my nose. My sternal incision hurt like hell, and the bladder catheter irritated me immensely. But I was hungry and could sip some tea. Then they gave me Jello. I have always hated Jello. I think of it as sick people's food. But this time I ate it with relish because it symbolized recovery.

Amazingly, I was transferred to the ward the next morning. I had gained 27 pounds from my preoperative weight. I was jaundiced. I had a brief period of oliguria. My creatinine had risen to 2.0. I needed six liters of oxygen to keep my saturation level above 90%. Although I'd suffered multiorgan system damage, I never developed ARDS.

Within 24 hours of my near-death experience, I was taking a few steps with a walker and a nurse on each side. The next day, I could walk to the nursing station by myself with the walker. By the third day, I could walk the length of the hall and back alone. On the fourth day, I was able to walk slowly without oxygen and a saturation level between 85% and 90%.

I was discharged on day 5. The next day, I walked around the block. And the day after that, I went to my office for a short time. Within a month, I was back to working full-time. After eight weeks, I traveled with other pulmonologists and friends to Great Slave Lake in the Canadian Northwest Territories for our annual fishing retreat.

Why was my life spared? I really can't say. Maybe God was trying to give me a wake-up call. Confronting one's mortality is not a pleasant experience, but we must all do it sooner or later. We never know when the bell will ring and we'll have to come in off the playground. I thought my time had come, but I was lucky enough to be sent back from the brink – and I planned to do something very special in the new time granted me.

TRIPLE JEOPARDY, ABANDONMENT,
AND MIRACLES



My series of heart problems began with angina, requiring multiple bypasses in 1992 (see Chapter 16), and continued with a mitral valve repair in 1998 (see Chapter 18). Two open heart operations are enough for most! But averages are for other people. I noted the occurrence of ankle edema while hunting in the fall of 2002, but not much, if any, reduction in exercise tolerance. My cardiologist diagnosed a recurrence of severe mitral valve regurgitation late that year. More surgery would be needed.

I postponed making a decision about this next major event in my life because I had a conference to attend and chair in Amsterdam in January 2003. I also had a series of visiting professorships that would take me through the end of May. My cardiologist considered the risks of waiting with me and we decided it was okay for me to keep my schedule intact. I always hated breaking commitments, even for health reasons.

Because of the complexities involved in a third surgery, and with a lot of advice from physician friends, we decided to consult Dr. Hartsell Schaff, famed cardiovascular surgeon at the Mayo Clinic in Rochester. He concurred that I needed a mitral valve replacement on this third encounter with heart disease. He carefully reviewed the CD of the cardiac cath done by my cardiologist in making his decision. He was

okay about my delaying the surgery to keep my schedule. On returning home, I continued to work at my usual schedule, but began feeling anxious about what was ahead. Accordingly, I moved up the date for surgery to early May, and made apologies for having to postpone my scheduled speaking and visiting professorships set for that month.

At surgery, the annulus placed in the mitral repair process was about to rupture! This came as a complete surprise, but it was fortunate, because a leak or tear would have been fatal. Dr. Schaff did not do a replacement, but felt he could do another repair using an annulus prosthesis of a different design. I had no specific complications from this surgery, and was discharged on the 5th hospital day, after an echo showed good functioning of the mitral valve, but wide open tricuspid regurgitation and significant pulmonary hypertension. Unfortunately, these problems were not addressed at the time of surgery, and the opportunity to do a simple procedure to repair the tricuspid valve was missed! No treatment for the pulmonary hypertension was offered.

My recovery from this third operation was slow and I could not increase my walking as I had following the earlier procedure. I developed ankle edema. But I could still walk comfortably for several blocks. One day, after meeting a friend for lunch about six weeks after I returned from Mayo's, I got very short of breath on walking back to my office. I had just walked the same distance briskly, but now was struggling for each breath. I was not coughing not wheezing, and my breathing became comfortable again on stopping to rest back at my office. To shorten this a bit, I had tipped into right heart failure, due to my pulmonary hypertension, which had always been present at each cardiac catheterization and was always attributed to the mitral valve regurgitation, so-called passive pulmonary hypertension. I was to learn in the period to follow that my pulmonary pressures remained high, and I had very little tricuspid valve function from the wide open valve.

My treatment for this was ambulatory oxygen, which improved my exercise tolerance considerably.

Another nagging problem that had gone on throughout the past three years described above, was gross rectal bleeding from hemorrhoids. I had been left with considerable irradiation proctitis from my prostate cancer procedure, and had admonitions not to have large hemorrhoids banded, for fear of developing fistulae. My radiation oncologist was quite adamant about this. I saw an excellent proctologist, who tried injections without much effect, and I continued to lose blood and developed iron deficiency anemia.

As I struggled with all of this, I became very fatigued and began to have a fever and sweats at night. This turned out to be endocarditis, an infection of the repaired mitral valve. An esophageal echo easily demonstrated the telltale vegetations of endocarditis, and the organism was of bowel origin, enterococcus faecalis. I began on a course of double antibiotics, ampicillin and gentamicin, but developed a rash and renal failure with a rapidly rising creatinine. As I was being indoctrinated for hemodialysis, my kidneys finally opened up and the indices of renal function improved. The culprit was believed to be ampicillin, but when gentamicin was again given as a trial, renal failure again occurred. "No more gentamicin" was stamped on my medical chart.

A mitral valve replacement was now indicated, and following personal phone calls to the surgeon, I returned to the Mayo Clinic for what I believed would quickly be a valve replacement. Much to my horror, the experts at Mayo's doubted the diagnosis of bacterial endocarditis and began on a fishing expedition to find out the source of infection. I had had a hemorrhoidectomy in Denver before going to Mayo's. This had showed infection and, of course, the echo was diagnostic of endocarditis with large vegetation, but all of this was ignored at Mayo's.

This hospitalization was the most miserable of all of my previous

experiences. All of the doctors in attendance refused to talk to me about my fears of gentamicin toxicity. Even though I knew better, I agreed to let them try low doses of gentamicin which they gave with vancomycin. Of course, I developed renal failure again! The infectious disease experts refused to consider any other antibiotic including daptomycin, which is the drug that saved my life ultimately. All of the specialists refused to talk to me about the surgery and only insisted that I could be on dialysis for renal failure. I was essentially abandoned and thus decided to return to Denver to die.

One month later, a courageous Denver surgeon, Myles S. Guber, M.D did a successful mitral replacement, using a pig valve, under the antibiotic cover of daptomycin, to which my organisms had been shown to be susceptible. Slowly I recovered from the surgery and began to participate in life again. This was in June 2004.

This is truly a horror story. How the Mayo Clinic could ignore all the evidence of endocarditis sent by my cardiologist in Denver that showed the presence of infection on the valve, and how the surgeons and the Mayo Clinic could abandon me and deny life-saving surgery, is astonishing. Why drugs that had proven toxic to my kidneys were imposed on me and new drugs not offered is amazing and simply malpractice! This was clearly malpractice and on advice of counsel, I set down a lawsuit, but later decided to abandon it, to avoid all the mental anguish it would entail and the fact that “nobody beats the Mayo Clinic in court in Minnesota.” It’s like the IRS.

This is one of the seamiest stories of medicine that anyone can imagine, but it happened, and I suffered along with my friends and loved ones. Fortunately I was salvaged by the doctors in Denver, who were willing to use a newly available antibiotic and do risky, but life-saving surgery!

AN ARRESTING MOMENT
(SUDDEN DEATH)



Sudden cardiac death occurs in about 100,000 patients a year, usually in a non-hospital setting. This sudden cessation of heart activity is usually due to ventricular fibrillation, which happens when the electrical signs from the heart become deranged and rapidly discharge at the rate of about 180 to 220 per minute, followed by a complete cessation of the electrical activity that controls the muscle force of the heart. This is known medically as ventricular fibrillation. The use of new and dramatic defibrillator techniques, in and out of hospitals, (the external cardiac defibrillator) has now begun to improve the survival rate of such patients. Yet the recovery rate of out-of-hospital cardiac arrests, when no medical team is available, remains about five percent.

Since I have had four open heart surgeries, the question of whether or not I should have an implanted defibrillator to prevent ventricular fibrillation or to restart a heart that suddenly stopped would be in order. However, I did not fulfill the criteria imposed by Medicare and I continued my activities as much as possible, limited by my poor cardiac output, which was due to the continued strain from high pressure in the pulmonary circulation, so-called “pulmonary hypertension.”

For background of the day, I was involved in a very contentious class action legal suit and was prepared to testify in the large conference room of the basement of my building, a non-medical building,

for the five hours that began on December 27th, 2005. My secretary was not in the office on this particular day and my assistant, Louise, was planning to go shopping for some office supplies, shortly after the deposition began. I was positioned at the head of the table with several lawyers and three cameras and started into the deposition in the identification phase, when I suddenly felt a browning out and dimming of vision. Although I did not know exactly what was happening, I told the lawyers to try and find my assistant, which they quickly did. Louise rushed down the stairs one flight, to see me slumped over the table and not breathing. When prompted, the lawyers finally helped Louise get me on the floor and she began closed-chest massage for over ten minutes until the emergency medical technicians finally reached the office. I had no heart beat at the time. They shocked me three times and got cardiac action started. I was quickly placed in an ambulance with a tube inserted to support my breathing and I was taken immediately to St. Joseph's Hospital, a private hospital that deals with a great number of cardiac problems. I have a few flashes of memory of being in the ambulance on the route to the emergency room where I awoke. I had been able to deduce what had happened and was pleased when the attending pulmonologist, a former fellow of mine, extubated me so that I could talk. Apparently I simply slumped in the middle of a question, which I can still remember. In any case, I was transferred to another hospital where my cardiologist does his work, and an implanted defibrillator was placed the next day. This never discharged until about a year later when, following a walk, I felt rather strange; and, on the next occasion when I had the device interrogated, learned it had indeed discharged after a period of 20 seconds of ventricular fibrillation. It was set to discharge at a rate of 180. This becomes important because, as the next chapter on Hip Replacement (*Chapter 21*) indicates, wrong settings at a lower threshold may be caused by benign rhythms, such as atrial fibrillation, and may trigger a discharge when one is not dealing with ventricular fibrillation.

There is a great amount of press about the lives saved by ventricular defibrillators today. There are some technological problems and some difficulties with the connection wires from time to time. But the risk benefit ratio remains very much in favor of having a defibrillator in place, and mine has served me well on at least one occasion and was involved in an episode from hell on another (*see Chapter 21*).

Patients and their families can put a great amount of confidence in the modern technology that has led to the defibrillating device and probably all patients with lowered cardiac function or anyone suffering a prior cardiac arrest should have one in place.

GETTING HIP



Hip replacement therapy has become common place for many who develop degenerative hip disease, which begins in the latter decades of life. The techniques of replacement and prosthesis complete joint (ball and socket) replacement evolved and represent major advances in orthopedic surgery in the past few years. New anterior approaches cause less muscle damage and are associated with a more rapid recovery, although the possibilities of hip dislocation are somewhat greater. Nonetheless most physicians and patients choose the new operation because of the reduced morbidity involved.

Because of my polymyalgia rheumatica, I had had a multiplicity of joint pains over the years. However, remissions under therapy were incomplete in the left hip and as time drew on my left hip caused significant pain on all walking, requiring use of a cane. I consulted a chiropractor, an osteopath, and finally had a definitive diagnosis made by my rheumatologist with simple films showing complete bone on bone degeneration of the left hip in late 2006. I decided to have my hip replacement after several discussions with a popular and experienced orthopedic surgeon in Denver, Dr. Brian Haas, who led a hip and knee center of excellence at Porter's Hospital. This was done in February 2007 under spinal anesthesia. The procedure itself took only about 55 minutes. The surgeon who visited me immediately after the operation

was standing over me in the recovery room. He bragged over the ease of placement and alignment of the total hip prosthesis, but he looked alarmed as he looked at my left ankle. I thought nothing about it at the time, but the next morning, I had severe pain just above my ankle caused by a huge hematoma that took place during surgery. Apparently excessive pressure or traction had occurred without realization that a great amount of tissue damage was being inflicted on my ankle. The surgeons scoffed it off, saying this was minor and of no real concern, although it was of considerable concern and pain to me. I got good relief from all my pains with morphine, but the ankle hematoma continued to grow. Many consultants looked at it and there was no single opinion about what, if anything, to do about it. A nurse friend came to visit and said that this should certainly be drained or infection would take place (as it quickly did with multiple drug resistant staph infection).

I was discharged on the eighth hospital day and again the surgeon said the hematoma was of no great concern and would soon resolve. This was a bold-faced lie! The following weekend, a visiting nurse who came to look at the ankle said it looked “terrible.” What she lacked in tact, she made up for with emphatic advice that I should do something immediately. Accordingly, I returned to the orthopedic surgeon’s office, but he did not see me. His physician assistant, whom I liked very much and who was trying to be helpful to me, was obviously minimizing the complexity and suggested that I contact a plastic surgeon. Since I knew one in another hospital, I went there that very day. This surgeon, with extensive experience in vascular surgery and trauma, Dr. Charles Brantigan, was aghast at the appearance of the ankle. He just stared at it and shook his head. A huge area of tissue surrounding the left ankle was dead and needed to be drained immediately. This was done later that evening under general anaesthetic. By the next morning, all the pain was gone and a new automatic wound pump was placed to help evacuate the wound. However, this became infected over the weekend, along with my right ankle, which had been scratched by

the fingernails of a nurse who was replacing Ted hose as a prevention against pulmonary embolism. Although the scratch didn't look very serious, the right ankle soon became infected.

To summarize a series of rapid and terrifying events, I developed a multiple drug-resistant staphylococcus in both the left hematoma and right ankle region as a result of negligence.

These complications required a multiplicity of hospitalizations the year following surgery. I had a period of a cellulitis on the right, which responded to daptomycin, a drug which had saved my life during the time I had enterococcal endocarditis (*see Chapter 19*). The graft of the left ankle was done along with a complex muscle rotation graft on the right. Both appeared to be healing nicely until one night, immediately following a check-up in the surgeon's office. Severe pain developed, which was excruciating. I was admitted again the next morning, again with cellulitis and blisters over the region of surgery. This was rapidly and unceremoniously drained by sharp incision and no anesthesia that evening. Pain and fever continued. There were fears of the dreaded "flesh-eating bacillus," a streptococcus, which proved not to be the case. Otherwise, amputations would have been necessary. Even though the most horrible of infections was not present, discussions of amputation of my right foot took place, much to my horror.

One particularly dreadful episode happened when I was in the bathroom, straining from the constipation of morphine. Passing golf ball size stools was itself excruciating. Then, suddenly my defibrillator discharged. This felt like getting hit in the chest and head by a baseball bat. Then another – and bang, bang, bang with two second intervals. I was sure that I was dying from these heart rhythm disturbances and asked the gathering crowd of doctors, nurses, and therapists to hold my hand and pray with me as I died. But they were only interested in the IVs and other tubes that were in place, and almost no one helped me back in bed. I had a leg cast on and could do little to help myself. Bang,

bang, bang – a total of eleven (11) consecutive discharges occurred before a cardiologist realized that the trigger rate for the discharge had been set too low and was picking up the signal of “atrial fibrillation,” with a slower rate and not ventricular fibrillation from heart disease. The cardiologist got the painful discharges stopped and a calm spread throughout the room. I continued my prayers of asking for forgiveness and praise to God that I was still alive, joined only by Louise, my life-long friend and spiritual partner.

Finally the infections settled down. I was left with a four inch in diameter wound over a totally exposed Achilles tendon on the right. The treatment that followed included a series of 62 sessions in the hyperbaric chamber of two hours each, which aimed to stimulate granulation tissue to cover the gaping lesion. This gradually occurred over a course of the months that followed. And finally the total Achilles region was covered with granulation tissue. This was followed by a split thickness graft which somehow failed to take for a variety of reasons, some which I think were in the realm of negligence on several peoples’ part, who dressed the wound and prematurely removed the staples. There was also the question of infection although the organism recovered was not an invasive staphylococcus on this occasion. In any case, this attempt at grafting failed but subsequent semi-synthetic grafts, known as apple grafts, were placed and appeared to accelerate the epithelialization of the wound. This finally took place over the course of the 13 months that followed.

Now, at this writing, a year and a half after surgery, I am able to walk with a cane again and gradually am recovering my conditioning, which became very poor during the periods of immobilization and hospitalization. During this near-year of ups and downs, I suffered significant periods of depression and a multiplicity of fears and anxieties over what might happen next.

I have not gone into all of the details of numerous things that

happened to me during the 52 days in the hospital, such as calling for a bedpan when in need of defecation with no answer and getting myself out of bed at great peril in order to avoid staining the bed. I did stain the bed linens on several occasions because of inadequate assistance from the nursing staff. I had a variety of drug toxicities, which included bone marrow suppression from the one of the many antibiotics I was given, among a long list of drug reactions that are a constant peril should I ever have an infection in the future. The most concerning thing, of course, is the neglect and near abandonment by my original orthopedic surgeon, which is certainly negligence, which will be dealt with appropriately in the future. I really hate to sue members of my own profession, but when harm and negligence arise through doctors' inaction and lack of judgement and care, justice must be done. I have related this horrible experience, most of this without bitterness, but with a great amount of concern about what is happening to many people who are candidates for routine elective surgery with assurances that their recovery will be uncomplicated, which is usually the case, but not always. A "center of excellence" does not insure even average care, on some occasions!

BEETS OR BLOOD



I happen to like cooked beets, laced with vinegar. You have to be careful with your spoon, because the red pigment stains easily. You also need to remember that you ate beets the next time you use the toilet. The pigment may remain in the stools or be cleared in the urine. It can actually look like your urine is stained with blood, if you have consumed a lot of beets with your meal.

Good that I remembered this one morning, because my urine was distinctly stained red but kind of a dark red. “Beets!,” I said to myself with some considerable relief. But while traveling a couple of months later, I had the same appearing urine, and had not had any beets to explain the color. Since this recurred, I became panicked, as would be reasonable, since blood in the urine is always due to some cause, often a cancer. I finally saw my original urologist, Bill Manniatis, who put a scope into my bladder via my penis, with a lot of Xylocaine, to make it tolerable. A tiny spot was seen on the bladder. My urologist wondered if this might be remotely related to the radioactive seed therapy I had received for prostate cancer, now 11 years ago. He scheduled a biopsy, just in case we were dealing with an early cancer.

I had this done as an outpatient procedure. I needed a catheter for a day, because of blood clots. These things are never comfortable for either men or women. The surgeon was reassuring when he said

it looked like an early stage low grade cancer. He had also lasered to the base of the lesion. But the pathology report indicated a high grade malignancy, which sounds scary. I had another bladder exam a month later to search for any recurrence. None was found. Six months later, a repeat procedure also showed no cancer.

And why did I get five cancers in my life? Prostate, bladder and three different skin cancers? I never did smoke nor have other contact with other carcinogens, except in construction as a teen.

Anyway, I am back to eating beets again now that I am not worrying about cancer. I will still need more checks in the future, however.

PART IV



WHAT WENT WRONG WITH MEDICINE



The United States spends by far more money today than any other country on medical care: some 16% of the gross domestic product (GDP). Other advanced countries spend far less than this, often less than half, no matter what system they offer their population. The universal care offered in most Western countries, Australia, and Japan, (8.9% in Japan of GDP) is still expensive and supported by taxation, but usually considered “free” by the public.

It is difficult to refer to our health care services as a system since most patients, including me, see very little that is systematic about much of what is going on in medicine today. We basically have a patchwork set of services that includes multiple sources of payment: insurance, private pay, and government. Most insurance pays as little as possible and patients’ needs are rapidly becoming “irrelevant” as technology continues to advance rapidly. A recent headline says it all, “Health plans say they’ll risk losing members to protect profit margins” (*AIGA News* May 19, 2008)!

Although it is certain that our healthcare system will change in the future, it is surprising that approximately 75% of our population is actually happy with what they have right now! The exception is concern and stress over rising premiums that are imposed by the present health care insurance industry. Forty-seven million Americans are uninsured

or underinsured, but this is by choice in all but about ten million people. The majority of the uninsured make a good income. Seventy percent (70%) of the uninsured have at least one full-time worker and 10% a part-time worker. Nine point three (9.3) million households have income in excess of \$75,000 (reference: Kiplinger Letter June 20, 2008). Illegals get better insurance coverage than citizens!

What kind of a health care system we will have in the future will be determined in large part by the platforms of the presidential candidates in 2008 and, in the final analysis, by the voters themselves. Less than 50% of our eligible voters will cast a ballot which will determine our healthcare system. It is unlikely that the majority of our citizens will be pleased with any dramatically new “system,” no matter what form of medical care is offered. Health care delivery will be the major domestic issue in the 2008 campaign.

I have one general comment about a single payor system such as exists in Canada. I have watched the Canadian system evolve over the past 30 years, during the time that I taught in most of the universities and large, private hospitals in Canada. It used to chill me to come to a 7:00 AM conference and walk past hundreds of patients sitting on hard wooden benches, carrying their sack lunches. Here I am preparing to give a lecture at 8:00 AM and a large number of patients have brought their lunch for their afternoon clinic. I don't know how many Americans would put up with such waiting and the relatively mediocre care that is provided to virtually all Canadians since no private medicine is allowed by law in Canada. I have several physician friends in Canada who receive their care in the USA. Some with birthright ties, such as in Eastern Europe, actually returned to their home country for urgent coronary artery bypass surgery, in places such as the Czech Republic and Slovenia.

The following is just a brief synopsis about my views concerning what is wrong with medicine from several points of view.

A. PATIENTS

Patients have come to expect too much from medical care in the United States. They are heavily influenced by television dramas or events in their own lives or that of family members or friends that are truly miraculous. They have developed a belief that a miracle is the norm. Many patients are too demanding of the services available to them, particularly on nights, weekends, and holidays. They flood emergency rooms for expensive care when very often this care is non-emergent and could easily be postponed or scheduled for regular office hours. Patients are often very demanding and expect unlimited refills of their prescriptions without appropriate review by doctors in order to save upon “medical fees.” Many are intolerant over the fact that busy doctors are scheduled well in advance and generally good physicians will not let “patients buy their way” to the front of the line during busy office hours or operating room schedules. Patients want high technology care but are not willing to pay for it. Many patients expect what they regard as “free medicine,” failing to recognize that all medicine has costs. All countries that provide a single payor private system are offering “free care” to their patients as an entitlement. Yet countries (such as the Scandinavian countries) levy huge taxes, sometimes more than 50% of earnings, to cover the costs of medical care. Medical care is often rationed in these countries.

Patients want and need a doctor who treats them with respect (77%), a doctor who listens to their concerns with patience and understanding (67%), and cares about the patient’s emotional well being (64%). They want doctors who welcome the asking of patient questions (57%), and physicians who make an effort to get to know the patient (42%) (source: *Consumer Reports* Feb. 2007).

B. DOCTORS

I believe it is fair to say that a growing number of doctors today are

too income-oriented and less giving, compared with the dedication to medicine which prevailed when I began my training and during most of my professional life, which covers nearly half a century. The growing proportion of women in medicine has reduced the average professional hours considerably because of most women's family responsibilities.

Giving away free care to the indigent has become a matter of history. Some form of social or entitlement system will often pay for the care that doctors used to give away freely with compassion and fulfillment. A thing as simple as professional courtesy to colleagues and colleagues' families, particularly in time of financial stress, has become almost non-existent. While doctors are well paid for their services, of course they have extremely high investments in their medical education.

A survey from the February 2007 Consumer Reports shows the basic levels of dissatisfaction between patients and doctors. *See Table.*

C. LAWYERS

Trial lawyers and their clients have created a litigious society, often filing nuisance suits or suing for unreasonable amounts. This has caused doctors and hospitals to practice "defensive medicine," which often results in unnecessary tests to counter risks of inappropriate lawsuits. Many lawyers have become millionaires in such practices. Gradually tort reform is occurring to help quell such inappropriate and excessive awards by the courts.

One of the most spectacular examples of lawyer abuse in medicine is that end-of-life care decisions are often brought before the court. This robs patients of their dignity, right to privacy, and self determination. The most colossal abuse of the law in end-of-life care was that of Terri Schaivo. Although a dispute over her wishes about life support became a matter of national focus, the court finally embraced the principle of reason, which, in this case involved a calm discussion with her husband about her wishes. Thus Terri's husband was forced to continue her

death process with a maximum of press coverage and other astonishing meddling such as from the federal government (Terri's Law, which was found unconstitutional). The simple process of having a written living will could have essentially avoided this tragedy.

D. HOSPITALS

Hospitals are more interested in new bricks and mortar and high cost technology than they are in providing appropriate salaries and working conditions for their employees, particularly the nursing and technical staff who must work odd hours. Literally billions of dollars are spent to rebuild or repair hospitals that are in perfectly adequate condition so they remain "competitive." Hospital administrators are frequently paid far more than they are worth and often more than most busy doctors on their staff and they are bottom-line, not humanistic oriented.

E. INSURANCE INDUSTRY

Our private insurance industry is out of control, charging exorbitant fees for the inadequate coverage that is offered. The development of health maintenance organizations is probably the worst thing ever to happen to American medicine. To me HMO means "health mischief organizations," where a large part of the revenue which comes from the provision of services goes to stockholders and chief executive officers. Several plans, such as Kaiser Permanente, give reasonable care, at least in some parts of our country.

F. PHARMACEUTICAL INDUSTRY

Our pharmaceutical industry has provided truly miraculous drugs for many diseases that are so threatening to our society, but these companies will only invest in medications or devices that will have widespread use. They are very reluctant to make investments in rare or so-called orphan diseases simply because of bottom line consider-

ations. Patent protection is sought to keep products paying a high profit, rather than becoming generic, which are less expensive. They tweak molecules to make them slightly different, thus affording patent protection for many established products which still bring excessive profits. This greedy maneuver retains their profit margin.

G. GOVERNMENT

Today more than 50% of medicine is already a government function, when one considers the Veterans Administration Hospitals, Public Health Service, and investment in medical schools. Today there are 125 allopathic medical schools and 24 osteopathic schools. The medical schools are doctor factories of the future. These medical schools deserve more financial support for teaching and probably somewhat less, relatively speaking, for research. Often medical schools are judged by their research productivity and government grant status, and not for the quality of physicians that they produce for the community.

Whether any of the many things that are wrong with American medicine will change rapidly is highly unlikely. Since there will always be suffering patients and doctors, nurses, and technicians to provide care for them, medicine will continue to function, but a shift back to patient-oriented care and education is a critical need and should be supported by everyone.

— TABLE —

DISSATISFACTION

A *Consumer Reports* survey found that doctors and patients don't always see eye-to-eye. Here are some of the most common gripes:

What bugs patients about doctors:	
Keep me waiting 30 minutes or longer	24%
Could not schedule an appointment within a week	19%
Spent too little time with me	9%
Didn't get test results promptly	7%
Didn't respond to my calls promptly	6%
What bugs doctors about patients:	
Don't follow prescribed treatment	59%
Wait too long before making appointment	41%
Are reluctant to discuss symptoms	32%
Request unnecessary tests	31%
Request unnecessary prescriptions	28%

HOW TO THRIVE AND SURVIVE
WITH MEDICINE TODAY



Last week at the 50th reunion of the class of 1958, we recalled the thrill of entering medical school 54 years earlier. We reviewed the awe of the first physicians we saw, now walking the hospital corridors in their white coats. Our first experience with the cadaver we dissected was a most memorable experience. Our feeling and respect for the human body and human tissue began on this day! In our first year, we observed autopsies, surgery, delivery of babies, and conducted our first group interviews with patients.

In our second year, we purchased and learned to use a stethoscope, which remains one of the most useful medical instruments, even though many doctors have never mastered the art of auscultation. We learned to use them in a course using real patients, called physical diagnosis. Sadly this course is not even taught in many medical schools today!

In the clinical years we were assigned our first patients to completely “work-up,” which started with a complete history and physical examination. My first patient had a tremendous impact on my career (*see Chapter 1*). In those days, the patient always came first. This was what medicine was all about, the healing of suffering patients, and the prevention of disease.

After medical school, we went our separate ways into internships,

residencies and, in some cases, fellowship training on the way toward becoming specialists. I chose pulmonology, but from the base of general internal medicine. It is not possible to assign some diseases to one organ system, as is the case with tuberculosis and many other diseases commonly seen by the pulmonologist.

No one of that era was concerned about who would pay for the medical care that was needed. It was our oath to care for the sick. We were respectful of the privacy of patients and were quick to aid colleagues in need. We were paid a meager salary, but that was more than enough compensation. We were not concerned about the length of the service assignment, and often worked all night without sleep. We became seasoned to the stresses that we faced. We did not suffer the limitations of regulations of training hours, and accordingly, we gained a vast experience taking care of our patients. We always gave them hope if we could not cure them.

Sadly, medicine has changed dramatically in the past half century. We have gained massively in technology, but have lost our humanity, indeed our calling, in the morass of the multiple forces that have and continue to destroy our relationship with the patients we have a duty to serve, and have sworn to protect.

Medical care, as we have known it, is gradually but inexplicably changing for the worse. The patient is no longer the focus of the health care delivery system. In fact, the patient is almost irrelevant. Some doctors find patients a “nuisance.” Doctors are not in control of medicine and are vulnerable to the power and control of insurance companies, the pharmaceutical industry, and government. In the USA we spend over twice as much on so-called health care as any other advanced country and with no better outcomes in length or quality of life. What are we to do now to protect our health and welfare? We simply cannot rely on the government to remedy our health care system. In fact, we cannot afford “free” government care!

1. PRESERVATION OF HEALTH.

It is each person's responsibility to maintain the health that is God-given. Congenital defects and diseases are beyond our control and must be dealt with medically. We need to maintain a normal weight, get regular exercise such as in walking or its equivalent, and not smoke or take any medication without good reason. We must drive responsibly and wear seat belts. These simple measures will prevent many diseases, such as heart and vascular diseases including stroke, many malignancies, and type two diabetes. We need to have regular checks of blood pressure and blood sugar in order to be able to treat hypertension and diabetes early and more effectively. We should be alert to the presence of blood in body secretions such as in the mucus we cough, or in urine and stool. We should see a doctor if such an alarming sign is noted. Do not procrastinate! All diseases are more effectively cured or managed if detected early!

2. VACCINATIONS.

We need to inoculate our children for the common childhood diseases, and adults for many preventable diseases such as influenza, women for papilloma virus, herpes zoster. We need special inoculations against diseases that are risks during foreign travel, such as meningitis, malaria, and others, as recommended by travel experts.

3. MEDICATIONS.

We must take only medications that are prescribed or recommended for over-the-counter use by doctors and societies. Naturally it is stupid to take any so-called recreational drug including pot, and any narcotics, meth, LSD, and the nonsense junk that is contrived by the many morons in our society.

4. DOCTORS.

We need to insist that doctors take enough time to get to know

us and the way we respond to disease and fears of diseases, as well as how we handle anxiety and depression. We need to choose a personal physician that we like and trust, and who will take our problems seriously. There will always be such doctors.

5. INSURANCE.

We must all have personal and family health insurance. It should be mandatory, just as car insurance is required for the privilege of driving. It may be employment-related or government-subsidized. We need regulation of the insurance industry if it continues to take huge profits for the CEOs and stockholders.

6. HOSPITALS.

We need to curtail the building of more and more hospitals that resemble the Taj Ma Hal. Bricks and mortar do not make good medicine. Hospitals must be taxed heavily if their bottom line allows for excessive building and purchase of unnecessary medical equipment. Hospitals need to be regulated, if they cannot control themselves.

7. PATIENTS.

Patients must be respectful of the demands and time limitations of their doctors. They must be reasonable about when they call their doctors. They must keep or reschedule appointments. Patients, however, must take the upper hand and begin to redirect medicine!

8. RETURN OF SPIRITUALITY TO MEDICINE.

The return of spirituality to medicine: This aspect of medicine has sadly been slipping away during the 50 years I have been in medicine, as we embrace the growing technology that now dominates our profession. Spirituality offers a strong antidote for hopelessness. I have experienced many life-saving miracles in my life and have seen the hand of God touch others. Whenever I have been in touch with the

sick, I never end the day without a prayer of thankfulness for the bountiful gifts bestowed on my patients and to me and my loved ones.

I must end this book on an optimistic note. As long as there are suffering patients, doctors will find some way to care for them. This is the way it was in the beginnings of medicine, and this truth will remain.

STETHOSCOPE GLOSSARY

Auscultation: the act of listening to the sounds of the chest, by using a stethoscope

Bronchiolitis obliterans: an inflammatory damage and obstruction of the small airways of the lungs

Dyspnea: the feeling of labored breathing, ie, shortness of breath

Exudate: inflammatory fluid that accumulates in an organ or drains externally

Hypoxemia: low oxygen in the arterial blood

Mediastinal tubes: tubes placed in the middle of the chest for the purpose of draining pus, blood or air.

Myalgia: pain in the muscles

Mycobacterium: a family of bacteria which includes tuberculosis and environmental mycobacteria

Pleural effusion: an accumulation of fluid in the chest cavity

Polycythemia: excess red blood cells in the blood

Pulmonary hypertension: high resistance and pressures within the circulation of the lung, between the right and left ventricles

Thoracentesis: the removal of fluid or air from the chest cavity

Trendelenburg position: placing the legs higher than the head on a tilt table or bed, this help encourage blood flow to the lungs

Ventilatory dynamic: the phenomenon of lung over-inflation due to too rapid breathing and not allowing enough time for exhalation

Glossary courtesy of Edna Fiore

P O E M S



Levophed is a powerful “vasopressor,” used to raise blood pressure in shock states. This venerable drug has been available throughout my career in medicine. I first used it in desperate situations. All of my patients and those of my colleagues died. We used to jokingly call this drug, “left for dead.” But now we know that with proper use it is lifesaving.

LEVOPHED

When they started Levophed
I thought that, I would soon be dead
My legs elevated above my head
But a miracle arrived instead!

Nurses are the closest person on the health care team. Their role is to be “patient advocate.” Nurses are either angels or bitches. Fortunately, 90% are angels. I have personal knowledge about how it feels to be abused and abandoned by bitches, or touched by angels.

MIDNIGHT NURSE

Night is Deep and,
My patients
Beep, Beep, Beep,
Sans Sleep.

And Weep With Sorrow.
But, I Will Help them
See Tomorrow.

The gurney is the foldable cart used to transport patients in and between hospitals and ambulances. They are padded just enough to be tolerable and are wide enough for a single patient of increased size and sometimes they must be used to transport corpses. Hopefully this will be the method of transportation for bed patients to facilitate diagnosis and care.

JOURNEYS ON THE GURNEY

How long this ride will last?
Around corners, bumps and noise
Lights above me there,
People who stare,
Toe out of blankets,
The elevator door and,

Now in the OR
Psychedelic lights,
And alarming sights
Then chill,
Moments of sleep and,
Bleep Beep Beep

Is it over?
Did I make it through
and who are you?

Wheeling on again,
to my room
Will the gurney take me home?

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I entered medical school the fall of 1954, full of enthusiasm for my professional training which was about to begin. I walked down the corridors to my first class, along with my eager classmates, wearing a long starched white coat, symbolic of the physician. I can remember my first use of the stethoscope in 1955 in a series of tutorial and physical diagnosis sessions given to the sophomore students. I well recall touching my first patient as I tried to hear and understand the sounds that were transmitted to my naïve ears. From the beginning of my career, I found use of the stethoscope to be a critical part of physical examination. It also provided a spiritual bonding with my patients as my hand placed the instrument over the heart, lungs, and abdomen, as I was touching or holding the patient with my other arm.

From Both Ends of the **STETHOSCOPE**

Now, some half century later, I have many memories of the fascinating and challenging patients I have examined and treated. Also, during this 50-year period, I have had four open heart operations, five cancers (prostate, three skin, and bladder cancer). Some of these experiences as both a physician and a patient, both good and bad, are captured in this book that I have written for both doctors and the patients they serve. To do this, I have had to use a moderate amount of medical terminology, but my messages have been and will be understood by physician and patients alike.



Dr. Thomas Petty is an international authority on respiratory disease. He has published over 800 articles in journals and is author or editor of 46 books or editions. Dr. Petty is a Professor of Medicine at the University of Colorado Health Sciences Center in Denver and the Rush-Presbyterian-St. Luke's Medical Center in Chicago.

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