VINTAGE

Copyrighted material

Contents

Copyrighted material

About the Author

Dr. Sherwin B. Nuland was a well-known surgeon in the US at the top of his profession. He was the author of *The Doctors*, which explores the ethics of the medical world, *How We Die*, a bestseller in America and Britain and *The Wisdom of the Body*.

Nuland drew on more than 35 years in medicine and a childhood buffeted by illness in writing *How We Die*, an award-winning book that sought to dispel the notion of death with dignity and fuelled a national conversation about end-of-life decisions. He died in March 2014 at his home in Hamden, CT.

Copyrighted material

ALSO BY SHERWIN B. NULAND

The Origins of Anaesthesia
Doctors: The Biography of Medicine
Medicine: The Art of Healing
The Face of Mercy
The Wisdom of the Body

Copyrighted material

To my brothers,
Harvey Nuland and Vittorio Ferrero

Copyrighted material

... death hath ten thousand several doors For men to take their exits.

—John Webster, *The Duchess of Malfi*, 1612

Copyrighted material

Foreword

I attended the Yale School of Medicine when Shep Nuland taught there and, despite our both being surgeons, I know him best in my capacity as a reader. I don't recall when I first read *How We Die*—I was just finishing high school when it came out—but I do know that few books I had read so directly and wholly addressed that fundamental fact of existence: all organisms, whether goldfish or grandchild, die. His description of his grandmother's illness showed me how the personal, medical, and spiritual all intermingled. As a child, Nuland would play a game in which he indented her skin to see how long it took to resume its shape—a part of the aging process that, along with her newfound shortness of breath, showed her 'gradual slide into congestive heart failure ... the significant decline in the amount of oxygen that aged blood is capable of taking up from the aged tissues of the aged lung.'

But 'what was most evident,' he continued, 'was the slow drawing away from life... By the time Bubbeh stopped praying, she had stopped virtually everything else as well.' With her fatal stroke, Shep Nuland remembers Browne's *Religio Medici*: 'With what strife and pains we come into the world we know not, but 'tis commonly no easy matter to get out of it.'

I studied literature at Stanford, and later history of medicine at Cambridge, to better understand the particularities of death, which still seemed unknowable to me—and yet vivid descriptions like Nuland's convinced me that such things can only be known face to face. *How We Die* brought me into medicine to bear witness, as Shep Nuland had done, to the twinned mysteries of death, its experiential and biological manifestations: at once deeply personal and utterly impersonal.

I like to think of Nuland, in the opening chapters of *How We Die*, as a young medical student, alone with a patient whose heart had stopped. In an act of desperation, he cut open the patient's chest

Copyrighted material

and tried to pump the patient's heart manually, to literally squeeze the life back into him. The patient died, and Shep was found by the intern, his supervisor, covered in blood and failure.

Medical school has changed since Shep's time, and such a scene is unthinkable now: medical students are barely allowed to touch patients. What has not changed, though, I hope, is the heroic spirit of responsibility amid blood and failure. This is the true image of a doctor. It is not the idealized happy profession in which we always cure diseases and ease suffering, our patients invariably leaving us better than we found them. It is also doctors facing the enormity of patient problems, seeing the crudeness of our tools, and, inevitably, watching our patients die, usually either in agony or under sedation.

I had dinner with Shep Nuland once, along with several other Yale students. It was right after he had given a talk on his battle with depression, his near complete loss of will. 'All my major surgical cases,' he said, 'I was scheduling them for twelve, one o'clock in the afternoon, because I couldn't get out of bed before about eleven ... I clearly became increasingly depressed until I thought, my God, I can't work anymore.' He was admitted to a psychiatric hospital, where every treatment failed: 'it got so there was a throbbing, there was a ferocious fear in my head. You've seen this painting by Edvard Munch, *The Scream* ... Every moment was a scream.' He nearly had a lobotomy but a resident physician convinced the staff to try electroconvulsive therapy, and after twenty cycles, 'I've never forgotten—I never will forget—standing in the kitchen of the unit ... and thinking, "I've got the strength now to do this."'

Shep's books did not make him immune to tragedy, of course—to address suffering so directly is not to become impervious to it. To ward off depressive thoughts, he used a talismanic phrase: 'Aw, fuck it.' Not the most literary of phrases, and I suspect there is a lesson in that.

We discussed none of this over dinner. I was too shy to speak a word the entire meal. I was still lost in the nakedness of his story—today I remember the talk primarily for its therapeutic effect. My closest friend from this time had attended the talk with me. She had been suffering from depression for years, alone in a crowd, cold in the warmth of friends, as the truly depressed are. Shep's

Copyrighted material

frank description of his experience, and my friend's identification with it, convinced her to seek treatment. She has gone on to live a happy life.

I'm now a neurosurgical resident; I've removed scores of brain tumors, and Nuland's descriptions of cancer often return to me. Cancer took his brother's life, and, finally, his own, and he describes the disease not just as a body eaten away by monstrous forces, but as a civilization crumbling as it is increasingly overrun by ravenous juveniles: 'Cancer cells are fixed at an age where they are still too young to have learned the rules of the society in which they live. As with so many immature individuals of all living kinds, everything they do is excessive and uncoordinated with the needs or constraints of their neighbors ... they are reproductive but not productive. As individuals, they victimize a sedate, conforming society.'

What a perfect metaphor! Every time, a brain tumor looks like a young invader. My goal is eradication. Few things are as satisfying as completely excising a tumor, leaving only the glistening appearance of a healthy brain. Yet, in brain cancer, we know the system carries the disease and the reprieve is temporary: the society will likely be overrun.

Almost exactly twenty years after the publication of *How We Die*, I was diagnosed with lung cancer, and as I looked at images of my own body riddled with cancerous lesions, Shep's compelling descriptions returned to me. His metaphor has helped me see the cancer as part of me, both literally and figuratively. At times I think of it as a native insurgency—something to be quelled. But every so often, my attitude is more charitable, and I'll find myself spastic with pain, pitching a coughing fit, or overwhelmed with nausea, saying quietly, 'Now, now, children ... '

By sharing his life as a doctor openly, Nuland demonstrated the power of honest self-appraisal, something I have always tried to replicate: to admit my failures to myself and others, to recognize that I will fail, to find the dedication to improve. Shep has shown us that writing allows the physician to fulfill another duty. *Doctor* derives, of course, from the Latin *docere*, 'to teach'; he once wrote, 'Only by a frank discussion of the very details of dying can we best deal with those aspects that frighten us the most. It is by knowing the truth ... that we rid ourselves of that fear of the terra incognita

Copyrighted material

of death.' No doctor or writer did more to draw the map. Condolences to Dr. Nuland, his family, and those who knew him. I hope he was one of the lucky few to find death with dignity. But I am glad that, until my own time comes, I have his voice in my head; and if I need an extended conversation, it is just a bookshelf away.

Paul Kalanithi,
2014

Copyrighted material

Acknowledgments

The eighteenth-century novelist Laurence Sterne once remarked that writing "is but a different name for conversation." The content and tone of a book or essay are determined by the author's perception of the reader's anticipated response to each sentence as it is given form on the page—the reader is always present. The book you are about to read was conceived with no other plan in mind than that of conversing with people who want to know what it is like to die. I have tried to hear how a reader might reply to what is being said. By listening well, I hoped to be able to address every response as immediately and clearly as possible.

The dialogue in these chapters, however, is only the culmination of other conversations I have been having most of my life—with my family, my friends, my colleagues, and above all my patients—with those who have been closest to me and whose wisdom I have sought in order to come to an understanding of what our lives, and our deaths, are about. To seek wisdom in another's words is much less difficult than to find it in another's experience. I have looked for it everywhere I thought it could be discovered. Even when I had no idea I was learning from one or another of the vast number of men and women whose lives have entered mine, they were nevertheless teaching me, usually with equal unawareness of the gift they were bestowing.

Although most learning is thus subtle and unrecognized as such by either its recipients or its providers, a great deal of it does grow out of the more usual kind of conversation: direct verbal interchange between two people. In my own case, the most extensive of those dialogues have gone on intermittently for years or even decades, while a few have taken place only during the writing of the book. If "conference [maketh] the ready man" as Francis Bacon claimed, then I have been made ready for *How We Die* by countless hours in the company of extraordinary people.

Copyrighted material

Several of my fellow members of the Bioethics Committee at the Yale–New Haven Hospital have again and again sharpened my comprehension of critical issues faced not only by patients and health professionals but at one time or another by all of us. I am particularly indebted to Constance Donovan, Thomas Duffy, Margaret Farley, Robert Levine, Virginia Roddy, and Howard Zonanna. Together and individually, they have shown me an image of medical ethics that is as humane (and even spiritual) as it is intellectually disciplined.

Thanks go also to another member of the committee, Alan Mermann, a pediatrician who found renewed vigor as a Congregationalist minister and the chaplain of our medical school. He has been generous in helping me understand what it is like for medical students and dying patients to befriend each other and share one another's fears and hopes.

Ferenc Gyorgyey has made available the vast resources of the historical collections at Yale's Cushing/Whitney Library, but his even greater gift during these many years has been the equally vast resources of his friendship and his wide-ranging intellect. Jay Katz, both in our conversations and in his writings, has taught me a sensitivity to medical decision-making that transcends the mere clinical facts of a patient's illness and even the conscious motivations that would seem to determine choice of treatment options. My wife, Sarah Peterson, teaches me yet another kind of sensitivity, which is sometimes called charity and sometimes called love. In charity or love there is an understanding of another's perceptions and there is also unquenchable faith. In Sarah's tradition: "Though I speak with the tongues of men and of angels and have not love, I become as a sounding brass or a tinkling cymbal." Therein lies a great lesson not only for individuals but for nations and professions—especially my own profession of medicine.

For the past decade, I have benefited from the friendship of Robert Massey. As a practicing internist, a medical school dean, and a historian of medicine as well as a commentator on its present and future, Bob Massey has transmitted to several generations of his physician colleagues a dimension of understanding and a sense of medical obligation that surpass the ephemeral concerns of the moment and the parochial concerns of

Copyrighted material

the guild. I have taken advantage of his friendship by making him my sounding board, my oracle, and even my authority for classical allusions, not to mention Latin grammar. There is almost nothing in this book that he and I have not discussed. His confidence in the value of this undertaking has been a source of quiet energy for me over these many months of work.

Each chapter of *How We Die* has been reviewed by one or more authorities on its content. In every case, important suggestions have resulted from the readings which contributed in significant ways to my ability to clarify the material. The cardiac chapters were critiqued by Mark Applefeld, Deborah Barbour, and Steven Wolfson; the sections on aging and Alzheimer's disease by Leo Cooney; the trauma and suicide section by Daniel Lowe; the AIDS chapters by Gerald Friedland and Peter Selwyn; the clinical and biological aspects of cancer by Alan Sartorelli and Edwin Cadman; the discussion of the doctor-patient relationship by Jay Katz. Specialists in these areas will easily recognize the names of each of my consultants—I honor myself by recording them here. They have been generous beyond my expectation.

A number of people have helped me answer specific queries and track down sources: Wayne Carver, Benjamin Farkas, Janis Glover, James M. L. N. Horgan, Ali Khodadoust, Laurie Patton, Johannes van Straalen, Mary Weigand, Morris Wessel, Ann Williams, Yan Zhangshou, and my great-hearted secretary, Rafaella Grimaldi. G. J. Walker Smith reviewed an autopsy series with me and helped to put its findings into the context of the degenerative processes of aging. A morning spent with Alvin Novick opened my eyes to political and intensely personal aspects of AIDS that I had only guessed at—it could not have been easy for Al to expose to a virtual stranger the pain in his still-grieving heart, but somehow he found the strength to do it, and I will not forget what he taught me. Irma Pollock, whom I have admired since childhood, spoke to me through the anguish of recalling the tragedy of Alzheimer's disease, because she wanted to help others. Her story has strengthened my faith in the power of selfless love.

The entire text of *How We Die* was read by several people of disparate backgrounds, whose comments proved extremely helpful in my own final scrutiny: Joan Behar, Robert Burt, Judith Cuthbertson, Margaret DeVane, and James Ponet. It goes without

Copyrighted material

saying that Bob Massey and Sarah Peterson made numerous critical contributions as they reviewed the evolving work, chapter by chapter. Bob's style is benevolent and diplomatic, but that Peterson woman is unsparing in her pursuit of what I have elsewhere called "the recognition of rambling and the discouragement of drift." I always made the changes when she pointed them out—even *her* charity has its limits.

And finally, to my new friends in the world of publishing. *How We Die* originated in the vision of Glen Hartley—not only the idea but even the title was his. At Dan Frank's suggestion, he and Lynn Chu sought me out and presented me with a mission I could not turn away from. The manuscript that ultimately resulted was passed through the filter of Dan's skillful editorial mind; only his authors can fully appreciate the value of such guidance. Sonny Mehta carried this project in his own gentle hands from inception to conclusion, as its editor, publisher, and chief booster. If there is an all-star team in publishing, this must surely be it.

It is said that in the twentieth century there are no longer any Muses, but I have found one. Her name is Elisabeth Sifton, and I have tried to treat ideas and the English language in ways that will please her. I ask no greater reward than her approval.

There is a second of Laurence Sterne's aphorisms that applies to *How We Die*. It is this: "Every man's wit must come from every man's soul, and no other body's." This is my book. No matter the inspiration and contributions of so many others, I declare every bit of it—every conception and every misconception, every truth and every error, every helpful thought and every useless interpretation—to be my own. They are no other body's. *How We Die* is no other body's because this book comes from my soul.

S.B.N.

Copyrighted material

Introduction

Everyone wants to know the details of dying, though few are willing to say so. Whether to anticipate the events of our own final illness or better to comprehend what is happening to a mortally stricken loved one—or more likely out of that id-borne fascination with death we all share—we are lured by thoughts of life's ending. To most people, death remains a hidden secret, as eroticized as it is feared. We are irresistibly attracted by the very anxieties we find most terrifying; we are drawn to them by a primitive excitement that arises from flirtation with danger. Moths and flames, mankind and death—there is little difference.

None of us seems psychologically able to cope with the thought of our own state of death, with the idea of a permanent unconsciousness in which there is neither void nor vacuum—in which there is simply nothing. It seems so different from the nothing that preceded life. As with every other looming terror and looming temptation, we seek ways to deny the power of death and the icy hold in which it grips human thought. Its constant closeness has always inspired traditional methods by which we consciously and unconsciously disguise its reality, such as folk tales, allegories, dreams, and even jokes. In recent generations, we have added something new: We have created the method of modern dying. Modern dying takes place in the modern hospital, where it can be hidden, cleansed of its organic blight, and finally packaged for modern burial. We can now deny the power not only of death but of nature itself. We hide our faces from its face, but still we spread our fingers just a bit, because there is something in us that cannot resist a peek.

We compose scenarios that we yearn to see enacted by our mortally ill beloved, and the performances are successful just often enough to sustain our expectations. Faith in the possibility of such a scenario has ever been a tradition of Western societies, which in

Copyrighted material

centuries past valued a good death as the salvation of the soul and an uplifting experience for friends and family and celebrated it in the literature and pictorial representations of *ars moriendi*, the art of dying. Originally, *ars moriendi* was a religious and spiritual endeavor, described by the fifteenth-century printer William Caxton as "the craft for to deye for the helthe of mannes sowle." In time, it evolved into the concept of the beautiful death, truly the correct way to die. But *ars moriendi* is nowadays made difficult by the very fact of our attempts at concealing and sanitizing—and especially preventing—which result in the kinds of deathbed scenes that occur in such specialized hiding places as intensive care units, oncology research facilities, and emergency rooms. The good death has increasingly become a myth. Actually, it has always been for the most part a myth, but never nearly as much as today. The chief ingredient of the myth is the longed-for ideal of "death with dignity."

Not long ago, I saw in my clinical office a forty-three-year-old attorney on whom I had operated for an early-stage breast cancer three years before. Although she was free of disease and had every expectation of permanent cure, she seemed oddly upset that day. At the end of the visit, she asked if she might stay a bit longer, to talk. She then began to describe the recent death in another city of her mother, from the same disease of which she herself had almost certainly been cured. "My mother died in agony," she said, "and no matter how hard the doctors tried, they couldn't make things easy for her. It was nothing like the peaceful end I expected. I thought it would be spiritual, that we would talk about her life, about the two of us together. But it never happened—there was too much pain, too much Demerol." And then, in an outburst of tearful rage, she said, "Dr. Nuland, there was no dignity in my mother's death!"

My patient needed a great deal of reassurance that there had been nothing unusual about the way her mother died, that she had not done something wrong to prevent her mother from experiencing that "spiritual" death with dignity that she had anticipated. All of her efforts and expectations had been in vain, and now this very intelligent woman was in despair. I tried to make clear to her that the belief in the probability of death with dignity is our, and society's, attempt to deal with the reality of what is all too frequently a series of destructive events that involve

Copyrighted material

by their very nature the disintegration of the dying person's humanity. I have not often seen much dignity in the process by which we die.

The quest to achieve true dignity fails when our bodies fail. Occasionally—very occasionally—unique circumstances of death will be granted to someone with a unique personality, and that lucky combination will make it happen, but such a confluence of fortune is uncommon, and, in any case, not to be expected by any but a very few people.

I have written this book to demythologize the process of dying. My intention is not to depict it as a horror-filled sequence of painful and disgusting degradations, but to present it in its biological and clinical reality, as seen by those who are witness to it and felt by those who experience it. Only by a frank discussion of the very details of dying can we best deal with those aspects that frighten us the most. It is by knowing the truth and being prepared for it that we rid ourselves of that fear of the terra incognita of death that leads to self-deception and disillusions.

There is a vast literature on death and dying. Virtually all of it is intended to help people cope with the emotional trauma involved in the process and its aftermath; the details of physical deterioration have for the most part not been much stressed. Only within the pages of professional journals are to be found descriptions of the actual processes by which various diseases drain us of vitality and take away our lives.

My career and my lifelong experience of death confirm John Webster's observation that there are indeed "ten thousand several doors for men to take their exits"; my wish is to help fulfill the prayer of the poet Rainer Maria Rilke: "Oh Lord, give each of us his own death." This book is about the doors, and the passageways that lead to them; I have tried to write it in such a way that insofar as circumstances allow, choices may be made that will give each of us his or her own death.

I have chosen six of the most common disease categories of our time, not only because they include the mortal illnesses that will take the great majority of us but for another reason as well: The six have characteristics that are representative of certain universal processes that we will all experience as we are dying. The stoppage of circulation, the inadequate transport of oxygen to tissues, the

Copyrighted material

flickering out of brain function, the failure of organs, the destruction of vital centers—these are the weapons of every horseman of death. A familiarity with them will explain how we die of illnesses not specifically described in this book. Those I have chosen are not only our most common avenues to death, they are also the ones whose paving stones are trod by everyone, no matter the rarity of the final disease.

My mother died of colon cancer one week after my eleventh birthday, and that fact has shaped my life. All that I have become and much that I have not become, I trace directly or indirectly to her death. When I began writing this book, my brother had been dead just a little more than a year, also of colon cancer. In my professional and personal life, I have lived with the awareness of death's imminence for more than half a century, and labored in its constant presence for all but the first decade of that time. This is the book in which I will try to tell what I have learned.

Sherwin B. Nuland

New Haven
June 1993

Copyrighted material

AUTHOR'S NOTE

With the exception of Robert DeMatteis, the names of all patients and their families have been altered to preserve confidentiality. It should also be noted that "Dr. Mary Defoe," who appears in Chapter VIII, actually represents a composite of three young doctors at the Yale–New Haven Hospital.

Copyrighted material

I

The Strangled Heart

EVERY LIFE IS different from any that has gone before it, and so is every death. The uniqueness of each of us extends even to the way we die. Though most people know that various diseases carry us to our final hours by various paths, only very few comprehend the fullness of that endless multitude of ways by which the final forces of the human spirit can separate themselves from the body. Every one of death's diverse appearances is as distinctive as that singular face we each show the world during the days of life. Every man will yield up the ghost in a manner that the heavens have never known before: every woman will go her final way in her own way.

The first time in my professional career that I saw death's remorseless eyes, they were fixed on a fifty-two-year-old man, lying in seeming comfort between the crisp sheets of a freshly made-up bed, in a private room at a large university teaching hospital. I had just begun my third year of medical school, and it was my unsettling lot to encounter death and my very first patient at the same hour.

James McCarty was a powerfully built construction executive whose business success had seduced him into patterns of living that we now know are suicidal. But the events of his illness took place almost forty years ago, when we understood a great deal less about the dangers of the good life—when smoking, red meat, and great slabs of bacon, butter, and belly were thought to be the risk-free rewards of achievement. He had let himself become flabby, and sedentary as well. Whereas he had once directed on-site the crews of his thriving construction company, he was now content to lead imperiously from behind a desk. McCarty delivered his pronouncements most of the day from a comfortable swivel chair

Copyrighted material

that provided him an unobstructed view of the New Haven Green and the Quinnipiack Club, his favorite grillroom for midday executive gluttony.

The events of McCarty's hospitalization are easily recalled, because the startling staccato with which they burst forth instantly and permanently imprinted them in my mind. I have never forgotten what I saw, and did, that night.

McCarty arrived in the hospital's emergency room at about 8:00 p.m. on a hot and humid evening in early September, complaining of a constricting pressure behind his breastbone that seemed to radiate up into his throat and down his left arm. The pressure had begun an hour earlier, after his usual heavy dinner, a few Camels, and an upsetting phone call from the youngest of his three children, an indulged young woman who had just started her freshman year at a fashionable women's college.

The intern who saw McCarty in the emergency room noted that he looked ashen and sweaty and had an irregular pulse. In the ten minutes it took to wheel the electrocardiogram machine down the hall and connect it to the patient, he had begun to look better and his unsteady cardiac rhythm had reverted to normal. The electrocardiographic tracing nonetheless revealed that an infarction had occurred, meaning that a small area of the wall of the heart had been damaged. His condition seemed stable, and preparations were made to transfer him to a bed upstairs—there were no coronary intensive care units in the 1950s. His private physician came in to see McCarty and reassured himself that his patient was now comfortable and seemed to be out of danger.

McCarty reached the medical floor at 11:00 p.m., and I arrived with him. Not being on duty that evening, I had gone to the rush party that my student fraternity held to inveigle entering freshmen into joining. A glass of beer and a lot of conviviality had made me feel especially self-confident, and I decided to visit the care division to which I had been assigned only that morning, the first of my clinical rotations on the Internal Medicine service. Third-year medical students, who are just starting out in their initial experience with patients, tend to be eager to the point of zealousness, and I was no different than most. I came up to the division to trail after the intern, hoping to see an interesting emergency, and to make myself helpful in any way I could. If there

Copyrighted material

was an imminent ward procedure, like a spinal tap or the placement of a chest tube, I wanted to be there to do it.

As I walked onto the division, the intern, Dave Bascom, took my arm as though he was relieved to see me. "Help me out, will you? Joe [the student on duty] and I are tied up down the hall with a bulbar polio that's going bad, and I need you to do the admission workup on this new coronary that's just going into 507—okay?"

Okay? Sure it was okay! It was more than okay; it was wonderful, exactly the reason I had returned to the division. Medical students of forty years ago were given much more autonomy than they are allowed today, and I knew that if I did the admission routines well, I would be granted plenty of work on the details of McCarty's recovery. I waited eagerly for a few minutes until one of the two nurses on duty had transferred my new patient comfortably from the gurney onto his bed. When she went scurrying down to the far end of the hall to help with the polio emergency, I slipped into McCarty's room and closed the door behind me. I didn't want to run the risk that Dave might come back and take over.

McCarty greeted me with a thin, forced smile, but he couldn't have found my presence reassuring. I have often wondered over the years what must have gone through the mind of that high-pressure boss of large, tough men when he saw my boyish (I was then twenty-two) face and heard me say that I had come to take his history and examine him. Whatever it was, he didn't get much chance to mull it over. As I sat down at his bedside, he suddenly threw his head back and bellowed out a wordless roar that seemed to rise up out of his throat from somewhere deep within his stricken heart. He hit his balled fists with startling force up against the front of his chest in a single synchronous thump, just as his face and neck, in the flash of an instant, turned swollen and purple. His eyes seemed to have pushed themselves forward in one bulging thrust, as though they were trying to leap out of his head. He took one immensely long, gurgling breath, and died.

I shouted out his name, and then I shouted for Dave, but I knew no one could hear me in the hectic polio room all the way down the corridor. I could have run down the hallway and tried to get help, but that would have meant the loss of precious seconds. My fingers felt for the carotid artery in McCarty's neck, but it was pulseless and still. For reasons I cannot explain to this day, I was

Copyrighted material

strangely calm. I decided to act on my own. The possibility of getting into trouble for what I was about to attempt seemed a great deal less risky than letting a man die without at least trying to save him. There was no choice.

In those days, every room housing a coronary patient was supplied with a large muslin-wrapped package that contained a thoracotomy kit—a set of instruments with which the chest could be opened in the event of cardiac arrest. Closed-chest cardiopulmonary resuscitation, or CPR, had not yet been invented, and the standard technique in this situation was to attempt to massage the heart directly, by holding it in the hand and applying a long series of rhythmic squeezes.

I tore open the kit's sterile wrapping and grabbed the scalpel placed for ready access in a separate envelope on top. What I did next seemed absolutely automatic, even though I had never done it, or seen it done, before. With one surprisingly smooth sweep of my hand, I made a long incision starting just below the left nipple, from McCarty's breastbone around as far back as I could without moving him from his half-upright position. Only a little dark ooze leaked out of the arteries and veins I cut through, but no real flow of blood. Had I needed confirmation of the fact of death by cardiac arrest, this was it. Another long cut through the bloodless muscle, and I was in the chest cavity. I reached over to grab the double-armed steel instrument called a self-retaining retractor, slipped it in between the ribs, and turned its ratchet just far enough to allow my hand to squeeze inside and grasp what I expected to be McCarty's silent heart.

As I touched the fibrous sack called the pericardium, I realized that the heart contained within was wriggling. Under my fingertips could be felt an uncoordinated, irregular squirming that I recognized from its textbook description as the terminal condition called ventricular fibrillation, the agonal act of a heart that is becoming reconciled to its eternal rest. With unsterile bare hands, I grabbed a pair of scissors and cut the pericardium wide open. I took up Mr. McCarty's poor twitching heart as gently as I could and began the series of firm, steady, syncopated compressions that is called cardiac massage, intended to maintain a flow of blood to the brain until an electrical apparatus can be

Copyrighted material

brought in to shock the fibrillating heart muscle back into good behavior.

I had read that the sensation imparted by a fibrillating heart is like holding in one's palm a wet, jellylike bagful of hyperactive worms, and that is exactly the way it was. I could tell by its rapidly decreasing resistance to the pressure of my squeezes that the heart was not filling with blood, and so my efforts to force something out of it were useless, especially since the lungs were not being oxygenated. But still I kept at it. And suddenly, something stupefying in its horror took place—the dead McCarty, whose soul was by that time totally departed, threw back his head once more and, staring upward at the ceiling with the glassy, unseeing gaze of open dead eyes, roared out to the distant heavens a dreadful rasping whoop that sounded like the hounds of hell were barking. Only later did I realize that what I had heard was McCarty's version of the death rattle, a sound made by spasm in the muscles of the voice box, caused by the increased acidity in the blood of a newly dead man. It was his way, it seemed, of telling me to desist—my efforts to bring him back to life could only be in vain.

Alone in that room with a corpse, I looked into its glazed eyes and saw something I should have noticed earlier—McCarty's pupils were fixed in the position of wide black dilatation that signifies brain death, and obviously would never respond to light again. I stepped back from the disordered carnage on that bed and only then realized that I was soaking wet. Sweat was pouring down my face, and my hands and my short white medical student's coat were drenched with the dark lifeless blood that had oozed out of McCarty's chest incision. I was crying, in great shaking sobs. I realized, too, that I had been shouting at McCarty, demanding that he live, screaming his name into his left ear as though he could hear me, and weeping all the time with the frustration and sorrow of my failure, and his.

The door swung open and Dave rushed into the room. With one glance he took in the entire scene, and understood it. My shoulders were heaving, and my weeping was by then out of control. He strode around to my side of the bed, and then, as if we were actors in an old World War II movie, he put his arm around my shoulders and said very quietly, "It's okay, buddy—it's okay. You did everything you could." He sat me down in that death-

Copyrighted material

strewn place and began patiently, tenderly, to tell me all the clinical and biological events that made James McCarty's death inevitably beyond my control. But all I can remember of what he said, with that gentle softness in his voice, was: "Shep, now you know what it's like to be a doctor."

Poets, essayists, chroniclers, wags, and wise men write often about death but have rarely seen it. Physicians and nurses, who see it often, rarely write about it. Most people see it once or twice in a lifetime, in situations where they are too entangled in its emotional significance to retain dependable memories. Survivors of mass destruction quickly develop such powerful psychological defenses against the horror of what they have seen that nightmarish images distort the actual events to which they have been witness. There are few reliable accounts of the ways in which we die.

Nowadays, very few of us actually witness the deaths of those we love. Not many people die at home anymore, and those who do are usually the victims of drawn-out diseases or chronic degenerative conditions in which drugging and narcosis effectively hide the biological events that are occurring. Of the approximately 80 percent of Americans who die in a hospital, almost all are in large part concealed, or at least the details of the final approach to mortality are concealed, from those who have been closest to them in life.

An entire mythology has grown up around the process of dying. Like most mythologies, it is based on the inborn psychological need that all humankind shares. The mythologies of death are meant to combat fear on the one hand and its opposite—wishes—on the other. They are meant to serve us by disarming our terror about what the reality may be. While so many of us hope for a swift death or a death during sleep "so I won't suffer," we at the same time cling to an image of our final moments that combines grace with a sense of closure; we need to believe in a clear-minded process in which the summation of a life takes place—either that or a perfect lapse into agony-free unconsciousness.

The best-known artistic representation of the medical profession is Sir Luke Fildes' renowned 1891 painting entitled *The Doctor*. The scene is a simple fisherman's cottage on the coast of

Copyrighted material

England, where a little girl lies quietly, seemingly unconscious, as death approaches. We see her grieving parents and the pensive, empathetic physician keeping his bedside vigil, powerless to weaken the tightening grip of mortality. When the artist was interviewed about the painting, he said, "To me, the subject will be more pathetic than any, terrible perhaps, but yet more beautiful."

Fildes clearly had to know better. Fourteen years earlier, he had seen his own son die of one of the infectious diseases that carried off so many children in those late-nineteenth-century years shortly before the dawn of modern medicine. We don't know what malady killed Phillip Fildes, but it could not have bestowed a peaceful ending on his young life. If it was diphtheria, he virtually choked to death; if scarlet fever, he probably had delirium and wild swings of high fever; if meningitis, he may have had convulsions and uncontrollable headaches. Perhaps the child in *The Doctor* has gone through such agonies and is now in the final peace of terminal coma—but whatever came in the hours prior to her "beautiful" passing must surely have been unendurable to the little girl and her parents. We rarely go gentle into that good night.

Francisco Goya, eight decades earlier, had been more honest—perhaps because he lived at a time when the face of death was everywhere. In his painting, variously called in English *Diphtheria* or *The Croup*, done in the style of the Spanish realist school and during a period of great realism in European life, we see a doctor holding a young patient's head steady with one hand on his neck while preparing to insert the fingers of his other hand down the boy's throat in order to tear out the diphtheritic membrane that will choke off his life if not removed. The original Spanish title of the picture, and of the disease, reveals the full force of Goya's directness, as well as that age's everyday familiarity with death. He called it *El Garrotillo*, for the strangulation by which it kills its victims. The days of such confrontations with the reality of death are long since over, at least in the West.

Having chosen, for whatever psyche-shrouded reason, the word *confrontations*, I need to pause; I need to consider whether I, too, even after almost forty years of James McCartys, do not from time to time still fall into stride with the prevailing temperament of our times, when death is regarded as the final and perhaps the ultimate challenge of any person's life—a pitched battle that must

Copyrighted material

be won. In that view, death is a grim adversary to be overcome, whether with the dramatic armaments of high-tech biomedicine or by a conscious acquiescence to its power, an acquiescence that evokes the serene style for which present usage has invented a term: "Death with dignity" is our society's expression of the universal yearning to achieve a graceful triumph over the stark and often repugnant finality of life's last sputterings.

But the fact is, death is not a confrontation. It is simply an event in the sequence of nature's ongoing rhythms. Not death but disease is the real enemy, disease the malign force that requires confrontation. Death is the surcease that comes when the exhausting battle has been lost. Even the confrontation with disease should be approached with the realization that many of the sicknesses of our species are simply conveyances for the inexorable journey by which each of us is returned to the same state of physical, and perhaps spiritual, nonexistence from which we emerged at conception. Every triumph over some major pathology, no matter how ringing the victory, is only a reprieve from the inevitable end.

Medical science has conferred on humanity the benison of separating those pathological processes that are reversible from those that are not, constantly adding to the means by which the balance shifts ever in favor of sustained life. But modern biomedicine has also contributed to the misguided fancy by which each of us denies the certain advent of our own individual mortality. The claims of too many laboratory-based doctors to the contrary, medicine will always remain, as the ancient Greeks first dubbed it, an Art. One of the most severe demands that its artistry makes of the physician is that he or she become familiar with the poorly delineated boundary zones between categories of treatment whose chances of success may be classified as certain, probable, possible, or unreasonable. Those unchartable spaces between the probable and everything beyond it are where the thoughtful physician must often wander, with only the accumulated judgment of a life's experiences to guide the wisdom that must be shared with those who are sick.

At the time that James McCarty's life came to its abrupt end, the outcome of his heart's misbehavior was inescapable. Although a great deal was already understood about heart disease in the early

Copyrighted material

1950s, the available therapies for it were few and too often inadequate. Today, a patient with McCarty's specific problem may expect to leave the hospital not only alive but with a heart so much improved that years may have been added to his life. So much have the laboratory-based doctors accomplished that one of the approximately 80 percent who survive a first attack has good reason to think of a cardiac seizure as the shiniest silver lining of his life, because it has exposed a condition that might soon have killed him had it not been discovered while still eminently treatable.

Indeed, the balance has shifted so much that the effectiveness of treatment for cardiac disease is far more often on the good side of probable. That should not, however, be taken to mean that the once imperilled heart is now an immortal heart. Although the great majority of cardiac patients today survive their first episode, well over half a million Americans still die every year of some form of McCarty's sickness. Another 4.5 million are newly diagnosed as being afflicted with it. Eighty percent of people whose heart disease eventually kills them are victims of this particular form of it: Ischemic heart disease (or coronary artery disease, or coronary heart disease, as it is variously called) is the leading cause of death in the industrialized nations of the world.

James McCarty's heart died because it was not getting enough oxygen; it was not getting enough oxygen because it was not getting enough hemoglobin, the blood-borne protein whose function is to carry the oxygen; it was not getting enough hemoglobin because it was not getting enough blood; it was not getting enough blood because the heart's nourishing vessels, the coronary arteries, were hardened and narrowed by a process called arteriosclerosis (literally, hardening of the arteries). The arteriosclerosis had occurred because of a combination of McCarty's sybaritic diet, his cigarette smoking, his lack of exercise, an element of high blood pressure, and a certain degree of inherited predisposition. Very likely, the phone call from his pampered daughter had the same spasm-inducing effect on his severely narrowed coronary arteries as it did on his angrily clenched fists. That bit of acute tightening was probably just enough to rupture or crack one of the deposits of arteriosclerosis, called plaques, in the lining of a main coronary artery. Once this

Copyrighted material

occurred, the disrupted plaque served as a focus on which fresh blood-clot formed, making the obstruction complete and choking off the already-compromised flow. This final stoppage caused so-called "ischemia" (pronounced *iskeemeeya*), or blood lack, thereby acutely starving a large-enough piece of McCarty's heart muscle, or myocardium, to disrupt its normal rhythm into the chaotic squirming of ventricular fibrillation.

It is quite possible that none of McCarty's heart muscle was actually killed by its acute blood lack. Ischemia alone may cause ventricular fibrillation, especially in a heart already injured by a previous attack And so may the adrenalinelike compounds produced by the body at times of stress. Whatever the cause, the electrical communication system upon which James McCarty's heart depended for its regularity and coordination broke down, and so did McCarty's life.

Like so many other medical terms, *ischemia* is a word with an interesting history and colorful associations. It will recur again and again in the telling of the stories in this long narrative of death, because it is so ubiquitous—and so insidious—a driving force toward the quenching of life's energies. Though starvation of the heart may offer the most dramatic example of its lurking dangers, the process of choking off oxygen and nutrition is the common denominator in a wide variety of mortal illnesses.

The concept of ischemia and the word itself were introduced in the middle of the nineteenth century by a brashly brilliant little Pomeranian (the word, when applied to dogs, evokes a tiny and intensely spirited bundle of scrappy exuberance, which seems appropriate for the man being described) who began his multifaceted career as a kind of *enfant terrible* of research, and ended it sixty years later universally recognized by the sobriquet "the Pope of German Medicine." No single individual has ever contributed more to the understanding of the ways in which disease wreaks its havoc on human organs and cells than did Rudolf Virchow (1821–1902).

Virchow, a professor of pathology at the University of Berlin for almost fifty years, produced more than two thousand books and articles, not only on medicine but on anthropology and German politics as well. So liberal a member of the Reichstag was he that the autocratic Otto von Bismarck once challenged him to a duel.

Copyrighted material

Being given the choice of weapons, Virchow ridiculed the upcoming encounter out of existence before it took place—by insisting that it be fought with scalpels.

Among Rudolf Virchow's many research interests was his fascination with the ways in which disease affects arteries, veins, and their contained blood constituents. He elucidated the principles of embolism, thrombosis, and leukemia and invented the words to describe them. Seeking a term to designate the mechanism by which cells and tissues are deprived of their blood supply, Virchow seized (this word is chosen advisedly) upon the Greek *ischano*—"I hold in check," or "I quench"—derived from the Indo-European root *segh*, which refers to "seizing" or "holding" or "causing to pause." By combining it with *aima*, or "blood," the Greeks had created the word *ischaimos*, to signify a holding in check of the flow of blood. *Ischemia* was chosen by Virchow to designate the consequences of diminishing or totally stopping blood flow to some structure of the body, whether as small as a cell or as large as a leg or a section of heart muscle.

Diminishing is a relative term, however. When an organ's activity increases, its oxygen requirements go up, and so does its need for blood. If narrowed arteries cannot widen to accommodate this need, or if for some reason they go into tight spasm that further restricts flow, the organ's demands are not met, and it rapidly becomes ischemic. In pain and anger, the heart screams out a warning, and continues to do so until its shrieking exhortations for more blood are met, usually by the natural stratagem of the victim, who—alarmed by the distress within his chest—slows or stops the activity that is tormenting his cardiac muscle.

A ready example of this process is the suddenly overworked calf muscle of a weekend athlete who returns to jogging each year when the weather warms up in April. The discrepancy between the amount of blood required by his out-of-condition muscle and the amount that is able to force its way through his out-of-condition arteries may result in ischemia. The calf does not get enough oxygen and it cries out in an agonizing seizure, to warn the athlete manqué to stop his exertions before a clump of muscle cells are starved to death, the process known as infarction. The shriek of pain in the overtaxed calf is called a cramp or a charley horse. When it originates in the heart muscle, we use the much more

Copyrighted material

elegant term *angina pectoris*. Angina pectoris is nothing else than a charley horse of the heart. If it lasts long enough, its victim sustains a myocardial infarction.

Angina pectoris is a Latin phrase which translates literally as "a choking" or "throttling" (*angina*) "of the chest" (*pectoris*, the genitive case of *pectus*, "chest"). It is to another medical philologist, the remarkable eighteenth-century English physician William Heberden (1710–1801), that we owe not only the term but also one of the finest descriptions of the symptoms associated with it. In a 1768 discussion of the various forms of chest pain, he wrote:

> But there is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned more at length. The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called angina pectoris.
>
> They who are afflicted with it, are seized while they are walking, (more especially if it be up hill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or to continue; but the moment they stand still, all this uneasiness vanishes.

Heberden had seen enough patients—"nearly a hundred under this disorder"—to be able to study its incidence and progress:

> Males are most liable to this disease, especially such as have past their fiftieth year.
>
> After it has continued a year or more, it will not cease so instantaneously upon standing still; and it will come on not only when the persons are walking, but when they are lying down, especially if they lie on the left side, and oblige them to rise up out of their beds. In some inveterate cases it has been brought on by the motion of a horse, or a carriage, and even by swallowing, coughing, going to stool, or speaking, or by any disturbance of mind.

Heberden was struck by the unremitting progression of the disease: "For if no accident intervene, but the disease go on to its

Copyrighted material

height, the patients all suddenly fall down, and perish almost immediately."

James McCarty never had the luxury of a succession of bouts of angina pectoris; he succumbed to his very first experience of cardiac ischemia. His brain died because the fibrillating and finally stilled heart could no longer pump blood to it. The ischemic brain was followed gradually into lifelessness by every other tissue in his body.

A few years ago, I met a man who was miraculously resuscitated from such an apparent sudden cardiac death. Irv Lipsiner is a tall, broad-shouldered stockbroker who has been an avid athlete all his life. Although he requires insulin for long-standing diabetes, the disease has had no physical effects on his vigorous good health, or so it would appear at first glance. But he did have a small heart attack when he was forty-seven years old, which is exactly the age at which his father died from the same cause. That episode left his heart muscle with only minimal damage, and he continued his active life without restriction.

Late on a Saturday afternoon in 1985, when he was fifty-eight years old, Lipsiner was beginning his third hour of tennis at the Yale indoor courts when two of his partners left, necessitating a switch from doubles to singles. The practice rally was just beginning when, without warning or premonitory pain, he slumped to the floor unconscious. Two physicians, by luck playing on an adjacent court, rushed to his aid and found him glassy-eyed, unresponsive, and not breathing. There was no heartbeat. Assuming correctly that he was in ventricular fibrillation, they immediately began cardiopulmonary resuscitation, continuing it for what seemed to them an interminable time, until the ambulance arrived. By then, Lipsiner had begun to respond, even resuming a spontaneous regular heartbeat as his airway was intubated and he was placed in the ambulance. Soon, he was wide awake in the Yale–New Haven Hospital emergency room and wondering, as he put it, "what the fuss was all about."

In two weeks, Lipsiner was out of the hospital, fully recovered from his episode of ventricular fibrillation. I met him some years later, on the horse farm where he lives. Every day, he takes time out from work to go riding or play tennis, usually singles. Here is

Copyrighted material

Irv Lipsiner describing what it felt like to drop dead on a tennis court:

> The only thing I can recall is just—not hurting, but just collapsing. And then the lights went out, as if you're in a little room and you flip the switch. The only thing different from that was that it was in slow motion. In other words, it didn't go out like *that* [here he snapped his fingers]. It went out like this [he made a lazy downward circle with his hand, like an airplane turning gently in descent toward a landing], gradually and almost in a spiral, like—[he hesitated briefly in thought, then pursed his lips and blew his breath out in a slow diminuendo]—this. The change from light to dark was very evident, but the speed with which it happened was—well, gradual.
>
> I was aware that I'd collapsed. I felt like somebody took the life out of me. It felt like—I'm thinking of a scene—I had a dog that was hit by a car, and when I looked at that dog on the ground—he was dead already—he just looked like the same dog, only shrunk. You know, shrunk—uniformly. That's how I felt. I felt like—[he made a sound like air going out of a balloon] "Pffft."

Lipsiner's light went out precisely the way it did because the circulation to his brain had been suddenly shut off. As the oxygen in the organ's now-stagnant blood was steadily used up, the brain began to fail—sight and consciousness were turned down as though by the gradual twist of a dial rather than the suddenness of a switch. That was Irv Lipsiner's slow-motion spiral into oblivion, and almost death. The mouth-to-mouth breathing and chest massage of the cardiopulmonary resuscitation forced air into his lungs and drove blood to his vital organs until his heart decided, for reasons of its own, to resume its responsibilities. Like most sudden cardiac deaths in nonhospitalized people, Irv Lipsiner's episode was caused by ventricular fibrillation.

Lipsiner felt no ischemic pain. The probable cause of his fibrillation was some transient chemical stimulation of a supersensitive area left on his heart muscle by the attack of 1974. As to why the fibrillation occurred when it did, there is no way to

Copyrighted material

be certain; but a quite plausible guess is that it was related to the stress of too much tennis on that Saturday afternoon, which could have caused the release into his circulation of extra adrenaline, and this in turn may have made a coronary artery go into spasm and set off the irregular rhythm. Such are the occasional vagaries of ischemic heart disease that Lipsiner was left with no new damage to his heart, although he never again played more than two consecutive hours of tennis.

The fact that Lipsiner experienced no cardiac charley horse before he began fibrillating makes this particular case of heart seizure somewhat unusual—the majority of people who drop dead probably do feel ischemic pain of the characteristic sort. Like its equivalent in the calf, the onset of ischemic cardiac pain is sudden and severe. It has been most commonly described by its sufferers as constricting, or viselike. Sometimes it manifests itself as a crushing pressure, like an intolerable blunt weight forcing itself against the front of the chest and radiating down the left arm or up into the neck and jaw. The sensation is frightening even to those who have experienced it often, because each time it recurs it is accompanied by awareness of the possibility (and quite a realistic awareness it is) of impending death. The sufferer is likely to break out into a cold sweat, feel nauseated, or even vomit. There is often shortness of breath. If the ischemia does not let up within approximately ten minutes, the oxygen deficiency may become irreversible, and some of the deprived cardiac muscle will go on to die, the process called myocardial infarction. If that happens, or if the oxygen lack is sufficient to scramble the heart's conduction system, some 20 percent of the afflicted will perish in the throes of such an episode before reaching an emergency room. That figure drops by at least half if transportation to a hospital is possible within the period cardiologists call "the golden hour."

Eventually, about 50 to 60 percent of people with ischemic heart disease will die within an hour of one of their attacks, whether the first or a later one. Since 1.5 million Americans suffer a myocardial infarction each year (70 percent of which occur in the home), it is not difficult to understand why coronary heart disease is America's biggest killer, as it is in every industrialized country of the world. Almost all of those who survive every infarction will

Copyrighted material

eventually be claimed by the gradual weakening of the heart's ability to pump.

When all natural causes are taken into account, approximately 20 to 25 percent of Americans die suddenly, defined as unexpected death within a few hours of onset of symptoms in persons neither hospitalized nor homebound. And of these deaths, 80 to 90 percent are cardiac in origin, the remaining segment being due to diseases of the lungs, central nervous system, or the vessel into which the left ventricle pumps its blood, the aorta. When the death is not only sudden but instantaneous, there are only a few that are not the result of ischemic heart disease.

The victims of ischemic heart disease are betrayed by their eating and their smoking and their inattention to such simple housekeeping chores as exercise and the maintenance of normal blood pressure. Sometimes pedigree alone gives them away, in the form of family history or diabetes; sometimes it is that driving impetuosity and aggressiveness that today's cardiologists call the Type A personality. In a way, the person whose heart muscle will be anguished by angina is very like the overly ambitious schoolchild who throws a hand aggressively into the air when the teacher looks for volunteers—"Choose me, choose me; I can do it better than anyone else!" He is easy to identify, and death will single him out. There is little randomness in the choices made by cardiac ischemia.

Long before we knew about the lurking perils of cholesterol, cigarettes, diabetes, and hypertension, the medical world was beginning to recognize specific characteristics in those persons who seemed destined for cardiac death. William Osler, the author of America's first great textbook of medicine in 1892, might have been describing James McCarty when he wrote, "It is not the delicate neurotic person who is prone to angina, but the robust, the vigorous in mind and body, the keen and ambitious man, the indicator of whose engines is always at 'full speed ahead.' "By their speedometers shall ye know them."

Despite all medical advances, there are still plenty of people who die with their first heart attack. Like lucky Lipsiner, most of them do not actually suffer death of cardiac muscle but are victimized by a rhythm suddenly made disorderly by the effect of ischemia (or sometimes local chemical changes) on an electrical conduction

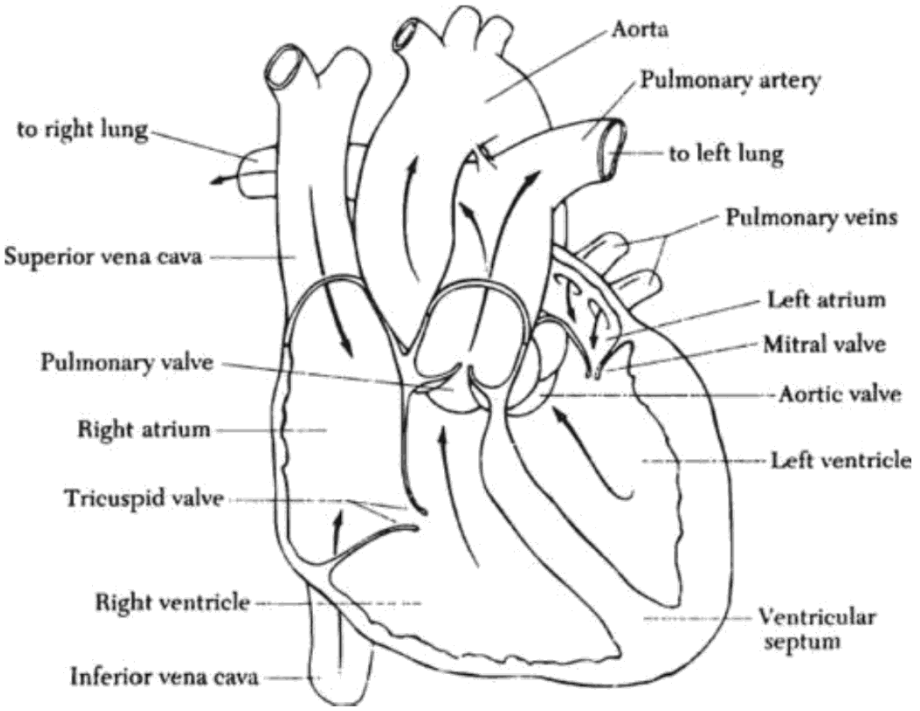
Copyrighted material

system already sensitized by a previous injury, whether it was recognized or not. But the usual way in which people succumb to ischemic heart disease these days is not the way of Lipsiner or McCarty. Decline is most often gradual, with plenty of warnings and much successful treatment before the final summons. The killing off of increments of heart muscle takes place over a period of months or years, until that besieged and enervated pump simply fails. It then gives up, for lack of strength or because the command system that controls its electrical coordination can no longer recover from yet another breach of its authority. Those laboratory doctors who are convinced that medicine is a science have accomplished so much that those bedside doctors who know it is an art can often, by careful timing and skillful choice of what is now available to them, provide victims of heart disease with long periods of improvement and stable health.

The fact remains, however, that each day fifteen hundred Americans will die of cardiac ischemia, whether its course has been sudden or gradual. Although preventive measures and modern methods of treatment have been reducing the figure steadily since the mid-1960s, it is virtually impossible for any slope of decline to change the picture for the vast majority of those who carry the diagnosis today or in whom it will be made in the next decade. This unforgiving sickness, like so many other causes of death, is a progressive continuum whose ultimate role in our planet's ecology is the quenching of human life.

In order to make clear the sequence of events that leads to the gradual loss of a heart's ability to pump effectively, it is first necessary to review some of the wondrous qualities that enable it to perform with such extraordinary precision when it is healthy. This will be the subject of the first pages of the chapter that follows.

Copyrighted material



Diagrammatic section cut through a normal heart with arrows indicating the blood flow

A complete cycle begins with the two large veins that receive dark oxygen-poor blood from the upper and lower portions of the body; the capaciousness, sources, and relative positions of these two broad blue vessels are reflected in the names given them by Greek physicians more than 2,500 years ago: the superior and the inferior vena cava. The two cavae empty their blood into the right atrium, whence it passes down through the valved opening (the right atrioventricular, or tricuspid, valve) into the right ventricle, which pumps it with a pressure equal to the weight of a column of mercury approximately thirty-five millimeters high into a large vessel called the pulmonary (Greek *pulmone*, "lungs") artery, which soon divides into separate conduits leading into each lung. Revitalized in the lungs by oxygen filtered through microscopic air sacs (called *alveoli*, Latin for "small basins or compartments"), the now bright red blood completes the lesser circulation by returning via the pulmonary veins to the left atrium, to be channeled down

Copyrighted material

into the ventricle and thence driven throughout the body, to the most remote living cell in the great toe.

Because it takes a pressure of approximately 120 millimeters of mercury to generate such a forceful squeeze, the left ventricle's muscle is more than half an inch wide, giving it the strongest and thickest wall of all four chambers. Pushing out about 70 milliliters of blood (2⅓ ounces) with each contraction, this vigorous pump drives some 7 million milliliters (more than 14,000 pints) each day, in 100,000 rhythmic and powerful beats. The mechanism of a living heart is a masterpiece of nature.

This complicated series of events requires intricate coordination, which is accomplished by messages sent out along microscopic fibers that originate from a tiny ellipse-shaped clump of tissue near the top of the right atrium, in its back wall very close to the entrance of the superior vena cava. It is at this very spot, where the cava empties into the atrium, that the blood starts out on its circuitous journey through the heart and lungs, and there could be no more appropriate point to position the source of the stimulus that makes it all happen. This little bit of tissue, called the sinoatrial (or SA) node, is a pacemaker that drives the coordinated beating of the heart. A bundle of fibers carries the SA node's messages to a relay station lying between the atria and ventricles (and therefore called the atrioventricular, or AV, node), and from there they are transmitted to the muscle of the ventricles via an arborizing network of fibers called the bundle of His, named for its discoverer, a nineteenth-century Swiss anatomist who spent most of his career at the University of Leipzig.

The SA node is the heart's personal internal generator; nerves from outside may affect the rate of beating, but it is the conduction of electricity from the SA node that determines the wondrous regularity of its faultless rhythm. Awestruck anew each time they viewed the proud independence of an exposed animal heart, wise men of ancient civilizations proclaimed that this supernal mechanism of boldly autonomous flesh must be the dwelling place of the soul.

The blood within the heart's chambers is only passing through; it does not stop to nourish the muscular valentine whose syncopated strokes are busily squeezing it along its way through the circulation. For the sustenance required for its forceful labors,

Copyrighted material

the heart muscle, or myocardium, is supplied by a group of separate and distinct vessels, which, because they originate in encircling arteries that wind around the heart like a crown, are called coronary. Branches of the main coronary arteries descend toward the valentine's tip, giving off twiglike branchlets that bring bright red oxygen-rich blood to the rhythmically heaving myocardium. In health, these coronary arteries are the friends of the heart; when they are diseased, they betray it at its most needful moments.

So commonly do the coronary arteries betray the heart whose muscle they are meant to sustain, that their treachery is the cause of at least half of all deaths in the United States. These "now I love you, now I don't" vessels are gentler to the gentle sex than they are to those who have more commonly gone out to hunt and fish—not only is infarction less common in women, it tends also to come later in life. The average age of the first infarction of women is in the mid-sixties, but men are more likely to have that terrifying experience ten years earlier. Although the coronary arteries have by that age reached the critical degree of narrowing necessary to threaten the viability of heart muscle, the process begins when its victims are much younger. An oft-quoted study of soldiers killed in the Korean War revealed that some three-quarters of these young men already had some arteriosclerosis in their coronary vessels. Varying degrees of it can be found in virtually every American adult, having begun with adolescence and increasing with age.

The obstructing material takes the form of yellowish white clumps called plaques, which are densely adherent to the inner lining of the artery and protrude into its central channel. The plaques are made up of cells and connective tissue, with a central core composed of debris and a common variety of fatty material called lipid, from the Greek *lipos*, meaning "fat" or "oil." Because so much of its structure is lipid, a plaque is called an atheroma, from the Greek *athere*, meaning "gruel" or "porridge," and *oma*, signifying a growth or tumor. The process of atheroma formation being by far the most common cause of arteriosclerosis, it is usually referred to as atherosclerosis, hardening by atheroma.

As an atheroma progresses, it becomes larger and tends to coalesce With neighboring plaques at the same time that it is absorbing calcium from the bloodstream. The result is the gradual

Copyrighted material

accumulation of an extensive mass of crusted atheroma that lines a vessel for a considerable distance, making it increasingly gritty, hard, and narrowed. An atherosclerotic artery has been compared to an old length of much-used, poorly maintained pipe whose inner diameter is lined with thick, irregular deposits of rust and embedded sediment.

Even before the cause of angina pectoris and infarction was understood to be a narrowing of the coronary arteries, a few physicians were beginning to make observations about the hearts of those people who died of the process. The same Edward Jenner who introduced smallpox vaccination in 1798 was an inveterate student of disease who made a custom of following to the autopsy table as many of his deceased patients as possible. In those days, doctors performed their own postmortem examinations. As a result of his dissections, Jenner began to suspect that the narrowing he discovered in the death-room coronary arteries was directly related to the anginal symptoms he had elicited from patients during life. In a letter to a colleague, he wrote of a recent experience dissecting a heart during such an autopsy:

> My knife struck something so hard and gritty as to notch it. I well remember looking up at the ceiling, which was old and crumbling, conceiving that some plaster had fallen down. But on further scrutiny the real cause appeared: the coronaries were become bony canals.

In spite of Jenner's observations and a gradual increase in understanding the way in which coronary obstruction injures the heart, it took until 1878 before a physician was able to diagnose a myocardial infarction correctly. Dr. Adam Hammer of St. Louis, a German refugee from the repression following the unsuccessful revolutions of 1848, sent off to a medical journal in Vienna his case report, entitled "*Ein Fall von thrombotischem Verschlusse einer der Kranzarterien des Herzens*," "A Case of Thrombotic Occlusion of One of the Coronary Arteries of the Heart." (Here an interesting twist of language presents itself: The German term for coronary artery is *Kranzarterie*, a *Kranz* being a wreath or a crown of flowers, which thus bestows an entirely new and quite poetic significance on the valentine image.) Hammer had been called in consultation to see a

Copyrighted material

suddenly stricken thirty-four-year-old man who was in such a rapidly worsening state of collapse that death was imminent. Although physicians knew the mechanism of myocardial ischemia, the diagnosis of infarction caused by it had never been made, or even thought of. As he watched helplessly while his patient died, Hammer suggested to his colleague that a completely occluded coronary artery had caused death of heart muscle, and he decided that an autopsy was mandatory to prove his novel theory. It was no easy matter to obtain permission from the grief-stricken family, but the experienced Hammer overcame their objections by the timely application of that perennial solvent of reluctance, a handful of dollars. As he put it so frankly in his journal article: "In the face of this universal remedy, even the most subtle misgivings, including the religious ones, eventually yield." Hammer's persistence was rewarded by finding a pale yellow-brown myocardium (its color signifying infarction) and a completely occluded coronary artery, confirming his insight.

During the following decades, the principles of ischemic heart disease and infarction became gradually established. With the invention of the electrocardiogram in 1903, physicians were able to trace the messages carried by the heart's conduction system of fibers, and they soon learned to interpret the tracings made by electrical changes taking place when the heart muscle is endangered by a decreased blood supply. Other diagnostic techniques were discovered apace, including the fact that injured myocardium releases certain chemicals or enzymes whose identifiable presence in the blood aids in detecting infarction.

An individual infarction involves that part of the muscle wall supplied by the particular coronary artery that is occluded, a part that most commonly measures two or three square inches in surface area. The specific culprit almost half the time is the left anterior descending coronary artery, a vessel that passes down the front surface of the left heart toward its tip, tapering as it gives off subdivisions that enter the myocardium. The frequent involvement of this artery means that approximately half of infarctions involve the front wall of the left ventricle. Its back wall is supplied by the right coronary artery, which accounts for 30 to 40 percent of occlusions; the lateral wall is supplied by the left circumflex coronary artery, which contributes 15 to 20 percent.

Copyrighted material

exertions of the distended, thickened heart require more oxygen than the narrowed coronary arteries can bring it, and the faltering myocardium may be damaged further, or perhaps new abnormalities of rhythm will appear. Some of these abnormalities are lethal—ventricular fibrillation and similar disturbances of rhythm kill almost half of the patients in heart failure. So, no matter how boastful its bombast, the failing heart continues to fail, in a kind of vicious circle of trying to disguise its own inadequacies by straining to compensate for them. As a cardiologist colleague has put it, "Heart failure begets heart failure." The proprietor of that heart is beginning to die.

The afflicted patient becomes increasingly short of breath with even minimal exertion, since neither the heart nor the lungs can respond to the increase in the work demanded of them. Some sufferers have difficulty lying down for more than a short period of time, because they need the upright position and gravity's help to drain excess fluid from their lungs. I have known many patients for whom sleep became impossible unless their head and shoulders were elevated on several pillows, and even then they were subject to paroxysms of frightening breathlessness during the night. Patients in heart failure suffer also from chronic fatigue and listlessness, owing to a combination of the added effort of breathing and the poor tissue nutrition caused by low cardiac output.

The elevated pressure that is transmitted from the venae cavae back into the body's veins causes the feet and ankles to swell, but when patients are bedridden, gravity forces the fluid to collect in the tissues of the lower back and thighs. Although rare today, it was not uncommon in my medical school years to come upon a patient sitting upright in bed, belly and legs swollen with fluid, throwing himself into almost convulsive heavings of shoulders and gaping mouth while struggling fiercely for each individual gasping breath as if it were his last chance to save his own life. In the wide-open mouths of these combatants in losing campaigns against imminent mortality, one could usually detect the blueness of deoxygenated lips and tongues, parchment-dry even though the dying patients were drowning. Doctors feared to do anything that might worsen the already intolerable eye-bulging anxiety of a man being submerged in his own waterlogged tissues, hearing only the

Copyrighted material

horrible wheeze and gurgling of his own death agony. In those days, we had little to offer a terminal sufferer except sedation, with the full and merciful knowledge that every bit of relief brought the end closer.

Although nowadays less common, such scenes are sometimes still enacted. A professor of cardiology recently wrote me: "There are many patients with terminal, intractable congestive heart failure whose final hours—or days—of life are made uncomfortable and even miserable by their drowning, while physicians can only watch helplessly, and use morphine for sedation. It isn't a pleasant exit." Not only the heart itself but the long-range damage inflicted by soggy, anemic tissues has plenty of other ways to kill. Eventually, the abused organs themselves fail. When the kidneys or liver are gone, so, too, is life. Kidney failure, or uremia, is an exit for some cardiac patients and so, on occasion, is inadequacy of liver function, frequently signaled by the appearance of jaundice.

Not only does the heart fool itself into overactivity, it may also fool the organs that might be able to help it out of its troubles. The kidney should be able to filter enough extra salt and water out of the blood to decrease the load on the heart, but congestive failure causes it to do just the opposite. Because the kidney correctly senses that it is getting less blood than normal, it compensates by producing hormones that actually cause reabsorption of the salt and water it has already filtered, so that they are returned to the circulation. The result is to increase the body's total fluid volume instead of decreasing it, thereby adding to the problems of the already-overworked heart. The failing heart thus outsmarts the kidney and itself at the same time; the self-same organ that is trying to be its friend becomes its inadvertent enemy.

Heavy, wet lungs with a sluggish circulation are an ideal breeding ground for bacteria and advancing inflammation, which is why so many cardiac patients die of pneumonia. But the heavy, wet lungs do not need the help of bacteria to do their killing. A sudden worsening of their waterlogged state, called acute pulmonary edema, is the frequent final event for patients with long-standing heart disease. Whether due to new cardiac damage or a temporary overload resulting from unexpected exercise or emotion, or perhaps just a little too much salt in a sandwich (I know of a man who died of what some might call acute pastrami-

Copyrighted material

generated heart failure), the excessive fluid volume dams up and floods the lungs. Severe air hunger rapidly supervenes, the gurgling, wheezing respirations begin, and finally the poor oxygenation of the blood causes either brain death or ventricular fibrillation and other rhythm disturbances, from which there is no return. All over the world, at this very instant, there are people dying in this way.

The final passage of some of them is epitomized in the case history of another man whose death I witnessed. In the reference frame of chronic heart disease, Horace Giddens might be called Everyman. The details of his illness graphically depict one of the common patterns in the inexorable downhill course of cardiac ischemia.

Giddens was a successful forty-five-year-old banker in a small southern town when his path crossed mine in the late 1980s. He had just returned home from an extended stay at The Johns Hopkins Hospital in Baltimore, where his physician had sent him in desperation, hoping that the progression of his increasingly severe angina and heart failure might be slowed, or at least ameliorated; virtually every known treatment had already failed. Trapped in a strife-ridden marriage, Giddens had made the difficult journey to Baltimore as much to separate himself from the enervating enmity of his wife, Regina, as he had to seek some relief for his heart. But it was too late—his disease was found to be so far advanced that he was beyond help from any available therapy. After all the tests and consultations, the Hopkins doctors told him, as sensitively as they could, that even they could not help him—he was no candidate for any treatment other than palliative medication. For Horace Giddens, there would be no angioplasty, no bypass, no heart transplant. I was making a purely social visit to his home on the evening he returned from Baltimore courageously facing the certainty that he would soon be dead.

Although it was understood that Giddens was on his way home, his unfeeling wife seemed not to know or even care about the exact time of his arrival. When he actually entered the house, I was sitting quietly in a chair, listening to the family's conversation but not partaking in it. That entrance was a difficult moment to watch. The tall, gaunt Giddens came shuffling into the living room, grimacing with breathlessness, his narrow shoulders held firmly in

Copyrighted material

the supporting grip of the adoring family maid. From a large photograph on the piano, I could tell that he had once been a robustly good-looking man, but now his grayish face was tired and drawn. He walked stiffly, as if with enormous effort, and carefully, seemingly unsure of his balance; he had to be helped into an armchair.

I knew of Giddens's history of angina, and I also knew that he had already sustained several full-blown myocardial infarctions. Watching the small shoulder-heaving struggle of each paroxysmal breath, I tried to imagine the condition of his heart and also attempted to put together in my mind's eye the various elements of the way it had failed him. After nearly forty years as a doctor, this kind of conjecture is a common preoccupation of mine when I find myself socially in the presence of the sick. It is an automatic drill, a self-testing, and in its own peculiar way, a kind of empathy as well. I do it always, almost without thinking. I'm sure many of my colleagues do the same.

What I visualized behind the breastbone of Horace Giddens was an enlarged, flabby heart that was no longer able to beat with anything resembling vigorous energy. More than three inches of its muscular wall had been replaced by a large whitish scar, and there were several other smaller areas of scarring as well. Every few beats, there was an irregular spasmodic contraction that originated from one or another rebellious focus on the left ventricle, intruding on the muscle's ineffectual attempt to maintain its steady rhythm. It was as though various parts of the ventricles were trying to break free of the intrinsic automaticity of the process, while the SA node struggled to maintain its declining authority. I knew the process well: The severity of the ischemia had cut off the regular messages that Giddens's SA node was trying to transmit to his ventricles. Unable to get their accustomed call, the ventricles feverishly begin to initiate beats on their own, starting each pulsation from whichever spontaneous spot on the myocardium chooses to meet the challenge. Any small increase in stress or decrease in oxygenation leads to a state of what the French so aptly call "ventricular anarchy," as disordered, ineffective contractions spread every which way through the heart muscle, giving way to the totally uncoordinated rapidity known as ventricular tachycardia and then fibrillation. As I watched

Copyrighted material

Giddens's uncertain movements, I could easily tell how close he was to this series of terminal events.

The vena cavae and the pulmonary veins were distended and tense with the pressure of the blood backed up into them because of the heart's weakness. The leathery lungs resembled gray-blue water-soaked sponges, overloaded with puffy edema and barely able to rise and fall like the gentle pink bellows they once were. The whole blood-choked image reminded me of an autopsy I once saw of a man who had hanged himself—his livid purplish face was engorged and bulging, its plethoric features almost unrecognizable as human.

Giddens had lived his life well, and borne with philosophical resolve the slings and arrows fired at him by his malicious wife. He had devoted his life to the seventeen-year-old daughter who idolized him, and to the fulfillment of the trust put in him by the people of his town, whose admiration and respect he had earned by dint of simple probity and the wisdom of sound financial management of their savings. But now he had come home to die.

As I watched his nostrils flare with each difficult breath, I could not help but notice that the very tip of Giddens's nose was just a bit blue, and so were his lips—the wetness in his lungs was preventing proper oxygenation. The laboriously shuffling gait was the product of ankles and feet so swollen they seemed to bulge out over the tops of shoes made too small by the tightly constrained wet flesh within them. Every organ in the man's waterlogged body had some element of edema in it.

Pump failure was only part of the reason that walking was such an enormous effort for Giddens. He must have been agonizingly aware of the effort expended in each step he took, knowing that even the smallest increase in activity might bring on the dreaded pain of angina, since the hair-thin channels of his rigid coronary arteries were incapable of delivering any added requirement of blood.

Giddens sat down in the armchair and spoke briefly with his family, seemingly unaware of my presence. Tiring in both body and spirit, he then climbed laboriously up the staircase to his bedroom, stopping several times to look down and say a few words to his wife. As I watched him do this, I was reminded of a practice commonly resorted to by so-called cardiac cripples in order to

Copyrighted material

ACKROYD ALLENDE ALLINGHAM AMIS ATWOOD
AUSTEN BARNES BARRY BINET BOLAÑO BORGES
BULGAKOV BURNSIDE BYATT CALVINO CARROLL CARTER
CARVER CHANG CHATWIN CHEEVER CLEGG COETZEE
CONRAD DARWIN DE BERNIÈRES DE WAAL DIAMOND
DI LAMPEDUSA DICKENS DOSTOEVSKY DOYLE ECO
ENRIGHT FAULKNER FAULKS FIELDING FITZGERALD
FLANAGAN FORSTER FOULDS GIBBONS GRASS GREENE
GROSSMAN GROSZ HADDON HARDY HARARI HELLER
HEMINGWAY HIGHSMITH HILL HOUELLEBECQ HUXLEY
ISHERWOOD JACOBSON JONES JOYCE KAFKA KENNEDY
KNAUSGAARD KUSHNER NESBO LEE LENNON LOMAX
MACDONALD MAK MANKELL MARÍAS MATTHIESSEN
MAXWELL McCARTHY McEWAN MISHIMA MOGGACH
MORGENSTERN MOORE MORRISON MUNRO MUKHERJEE
MURAKAMI MURDOCH MYERSON NADAS NÉMIROVSKY
NESBO NIFFENEGGER O'CONNOR OGAWA OKRI
ONDAATJE OZ PASTERNAK PENROSE PEREC PETTERSON
POLITKOVSKAYA PROUST PYNCHON QUINN REMARQUE
RIVAS ROTH RUSHDIE SARAMAGO SCHAMA SEBALD
SHUTE SNYDER SOLZHENITSYN STEVENSON STYRON
TAN TANIZAKI THIONG'O THIRLWELL THORPE THUBRON
TOLSTOY TREMAIN TYLER VARGAS **VINTAGE** VONNEGUT
WARHOL WELSH WESLEY WHEELER WILLIAMS WEIR
WINTERSON WOLFE WOOLF WYLD XIAOLU YATES ZOLA

Copyrighted material