CHAPTER 3

THE SILENT ATTACK

"And just remember—Be Big!"

"Big Jay" Sorensen, morning drive-time radio host, clicked off his microphone, slid off his headphones, and winced. His stomach hurt.

It was a sizable stomach, to be frank. Big Jay had earned both his nickname and the motto that served as his daily sign-off. He was larger than life in personality and in shirt size, and he had the booming voice and gregarious nature to match. He was fifty-two years old and he had been working in radio since he was seventeen, knuckling his way up from news stringer to board operator to program manager to what he liked to call "classic-hits musicologist." He had worked at WNBC-AM, a legendary New York City station that birthed the biggest names in 1970s and '80s radio: Wolfman Jack, Howard Stern, Joey Reynolds, Don Imus. He developed a following of his own, people who recognized the difference between the Dave Clark Five and the Five Man Electrical Band and knew that the first place in the United States where one of the Beatles played in public was an Illinois VFW Hall, not the Ed Sullivan Show. Now he was back in Manahawkin, New Jersey, thirty miles from the oceanfront town where he grew up and got his first job, playing early-rock classics and bantering with Anita Bonita, co-host of "Big Jay and Anita in the Morning" on WJRZ-FM.

To get to the station in time to run through newspapers and websites—fodder for the interviews and calls that were an essential part of the show—Sorensen got up at 2:45 every morning. So when he finished a shift, he expected to be tired and mentally wrung out. But he'd never felt like this before. There was a knot of pain just below his navel, so intense that it was hard to stand up from the big chair he sat in to broadcast. He unfolded himself carefully, straightening as much as he could, and made

his way slowly out of the studio and into his car for the midday drive home. It was late August 2005, and his wife Andrea, who worked part-time at a tax-preparation office, was waiting for him for lunch.

They never got lunch. "We have to go to the hospital," he said. "I don't know what, but something's terribly wrong."

The local emergency room identified the cause pretty quickly: Jay had diverticulitis, small, balloon-like sacs protruding from the surface of his large intestine into his abdominal cavity. One of them was swollen and infected, and on ultrasound it looked like it had ruptured. That was what was causing the piercing pain. The staff admitted Jay to the hospital, and told him and Andrea that they would try him first on antibiotics and pain medication, to see whether the infection would clear and shrink back on its own.

It did not. After a week in the hospital, his own doctor told Jay to prepare for surgery, a bowel resection that would take out two feet of his colon and stitch the snipped ends back together to make his bowel function normally again. Diverticulitis was a common problem, the doctor said reassuringly, and bowel resection was a routine surgery. He would be in the hospital for about a week afterward and then go home. Jay was a healthy man, for all his size, and his doctors predicted his recovery would go smoothly. He would be back to normal in just a few months.

In fact, it would take years.[1]

The surgery took place on a Monday, and by Tuesday Jay was up and hobbling through the hospital hallways, shuffling along in rubber-bottomed booties and hanging on to the IV pole for support. He hoped to go home on the weekend, but on Thursday he started to feel sick, and on Friday, his colorectal surgeon came by, spotted a bubble of fluid behind his stitches, and drained it. By Saturday, Andrea was nervous; Jay kept asking for more pain relief, but once he downed the meds he babbled and made no sense. On Sunday, he seemed better, though, and when Andrea mentioned that their daughter Tracy wanted to bring the grandchildren to visit, Jay levered himself to his feet and hunched over the sink to wash his face and shave.

"He bent over, and all this orangey-brown stuff came pouring out of his stomach, out of the wound," Andrea said. "I made him sit down and I screamed for the doctor. The one who was covering for the weekend came in and looked and said, 'He's got a site infection. Your doctor can look at it tomorrow, it will be fine.'"

The next day, his doctor paged the infectious disease physician on call,

and she arrived quickly and prodded the wound. She ordered some cultures and, knowing they would take twenty-four hours or more, started Jay preemptively on vancomycin, the MRSA drug of last resort.

It was a smart assumption. When the culture results came back the next day, they verified a MRSA infection. Even worse, they indicated a second, equally serious drug-resistant bug: vancomycin-resistant *Enterococcus faecium*, known as VRE. As its name suggests, E. faecium is a gut bacterium; for about ten years, it had been causing ferocious bouts of hospital infections. VRE no longer responded to vancomycin and could be treated only with older, more toxic drugs or with newer, more expensive ones that not all patients could tolerate.[2]

Jay had just become a victim of both hospital-acquired infection and collateral damage in the battle to contain it. VRE had become vancomycin-resistant in the same years that MRSA had begun to intensify, in hospitals and also in the serious community infections that Robert Daum and others had uncovered. More potent strains of MRSA had led to greater use of vancomycin. That suppressed MRSA to some extent, but encouraged the development of VRE instead.[3] The result was that Jay needed to be loaded immediately with two potent antibiotic regimens, one to combat MRSA, the other to beat back the VRE that the MRSA drugs could not contain.

For two weeks, he stayed in the hospital, watching nurses and technicians shuttle in and out to take his blood for cultures and hang fresh IV bags on the beeping infusion pumps by his head. There was little else to watch, and almost no one to talk to—because to keep the infection from spreading, the hospital had put Jay in isolation. That meant every staff member and visitor had to wash and put on gown, gloves, mask, and shoe covers before entering his room and discard them and wash again after leaving. The routine was so onerous that it discouraged casual visitors and reduced traffic to only the staff who most needed to come in. Andrea was there all the time, day after fifteen-hour day, wrapped in a paper gown over her street clothes, fidgeting with the mask elastic that hooked uncomfortably over her ears. Fearing infection, she forbade friends and family to visit. Grandchildren were out of the question.

On the day before Jay was to be released, she got a call at home. It was early Sunday morning, and she was getting ready for church before heading to the hospital for another long vigil. Jay's surgeon was on the line with bad news: the bugs were not being beaten.

"He said, 'I hate to do it, but we have to go in and clean him out,'"

Andrea said. "So they took him in the next morning and opened up the incision, and basically power-washed his insides."

When the surgical team opened the two-week-old incision, they found that the MRSA infection had gone further than they thought. It was not only immediately beneath the vertical slice through Jay's abdomen; it was also in the stitched-up resection of his colon, on the lining of his abdominal cavity, and on the outer surfaces of his organs. The high-pressure lavage they used dislodged the infection, but it negated most of the healing his body had managed in the past fortnight. When they closed him up again, they realized he was facing several more weeks in the hospital, weeks that he would be spending in isolation until the last-chance antibiotics brought the infection under control.

Jay finally made it home on a Friday, six weeks after he had gone in for surgery. Two nights later, he woke Andrea, worried about a sharp pain in his back. He was having trouble breathing too, but he dreaded going back to the hospital. It took the combined arguments of Andrea and the home-health nurse who visited the next day to get him there. He relented at last, and Andrea drove him, fearing he had pneumonia after so many weeks on his back. But the news was worse than that. He had a pulmonary embolism, a blood clot that starts in a major vein, usually deep in a leg, and travels through the circulatory system toward the heart.

Pulmonary embolisms are a life-threatening emergency and require immediate treatment with clot-dissolving drugs that are given by IV. Jay was admitted, and when his ER bloodwork was returned, was put back into isolation as well, because the cultures showed MRSA was still present in his blood. It took another three weeks before he could leave again.

He had been home several weeks and was dressing carefully to avoid jarring the still-tender incision when Jay noticed a lump in his groin. He went back for a check-up, and his surgeon confirmed it: the bulge was a hernia. The two surgeries through his abdominal wall had weakened the muscles, and his internal organs were protruding through them. Jay was worried by the diagnosis, but more worried about risking surgery again; through the winter of 2005 and the spring of 2006, he nervously waited as the lump got bigger and more painful. By summer, his colorectal surgeon felt it could go no longer. He recruited a plastic and reconstructive surgeon, Mike Nagy, who repaired the hole with a patch of preserved cadaver skin.

It was a solid repair, though it left Jay with a set of scars shaped like

an anchor—the original vertical scar from the previous summer, and the smile-shaped new scar that went from hip to hip. But while the operation was a success, the recovery was not. The MRSA recurred.

"They'd done some blood work, and he was sleeping," Andrea said. "I saw the nurse come in with an IV bag I recognized, the bag of vancomycin, and I thought, *Oh no.*"

Jay went home after a week, still on IV antibiotics—but the combination of MRSA and the blood thinners he took to prevent more emboli kept the long incision in his abdomen from healing. In September, Nagy had him come back for more surgery, his fourth, to remove dead tissue around the sides of the wound. That left Jay with a hole in his abdomen too wide to close surgically—"You could fit a melon in it," he said, "a cantaloupe"—and so Nagy ordered home care. That meant a "wound vac," a contraption that had to be set up twice a day by a nurse. A thick layer of foam was trimmed to fit the wound and pushed gently inside the hole, topped by a sticky film to seal it. Then a tube with an adhesive collar was pushed into the foam and connected to a portable vacuum the size of a toolbox. When it was turned on, the vacuum hummed and slurped, sucking away wound fluids to encourage new tissue to grow. It made Jay grateful the nerves in his abdomen were dead.

For three months, he carted the box around with him, tolerating the gurgling. "It's my purse," he joked. "My purse that makes noise." He went back to work at the radio station, leaning back as far as possible in the most flexible chair because the wound made it too hard to sit upright, and levering the mike close to his mouth so it would not broadcast the slurping. The machine was a disgusting companion, but it was effective: slowly, the wound was closing. But Jay was still on high doses of antibiotics, and eventually they took their own toll. He developed thrush, a creamy white coat of fungus over his mouth and throat that for weeks made it too painful to swallow anything but ice chips and Gatorade. On a day in January 2007, stopping at his daughter's house on his way home from work, he collapsed. She took him to the ER. Andrea rushed to meet them there and bumped into a friend who is an emergency nurse.

"He's going to be here a while," the friend told her. "Does he have a spare battery for the wound vac? Why don't you go home and get it?"

It was months before her friend confessed what that really meant: she thought Jay was about to have a heart attack, and she didn't want Andrea to see.

* * *

From its earliest emergence, MRSA was a hospital phenomenon, though it took a while for medicine to admit the bug had settled there. The three initial cases found in 1961 by the British national program of microbiological surveillance were assumed to be an anomaly. British scientists put the episode down to random genetic accident, the slot machine of staph's relentless evolution happening to line up in a particular way at a particular time. They were wrong, though. The cases were not a quirk. They were a warning of how quickly methicillin resistance had emerged and was spreading in the country where the drug was used first.[4]

Eighteen months later, in September 1962, doctors at the Central Middlesex Hospital in northwest London gave emergency treatment to a twenty-eight-year-old railway worker. In a terrible accident, he fell on the tracks and was run over by a train that cut off his left leg below the knee, crushed his right shoulder and ribs, and ruptured his spleen. He had been in the hospital for three days when he developed a searing 106-degree fever. The staff rushed to give him antibiotics, and started him on penicillin, streptomycin, and methicillin. Five days later, realizing the infection was subsiding more slowly than it should, the hospital cultured his blood for bacteria and found a staph strain that they judged "weakly resistant" to methicillin. But by only two days later, the same strain had grown significantly more resistant. They dropped the methicillin and switched him to two other drugs, but he had to stay on them for two more weeks before the infection was finally quelled.

There had been no methicillin-resistant staph at Central Middlesex up to that point, and the hospital was bewildered where it might have come from. The staff checked lab results from all the other patients, and found that the same "weakly resistant" strain had been recorded on September 4 in a woman recovering from a surgical hip fusion. That was the same day the railway worker arrived at the hospital. The hospital reluctantly concluded that the staph strain had not arrived with the gravely injured trauma case. The institution had given it to him.

"The appearance within four days of two strains of Staph with identical antibiograms . . . and both resistant to methicillin is unlikely to be coincidental," the hospital pathologist gravely noted in a report. "It is therefore accepted that cross-infection occurred."[5]

Someone must have conveyed the methicillin-resistant staph from one patient to the other, because between the hip fusion and the ampu-

tation, neither the woman nor the railway worker could walk. The hospital said that it was not possible to identify who might be responsible, even though the day of the woman's surgery was an end-of-summer holiday when the institution was running on a skeleton crew. The few staff who worked that long weekend were given days off later in the month, and they were still absent when the two staph infections were diagnosed. When they returned to work, they were checked, but no staph was found.

What the hospital was looking for, in checking the heath care workers, was staph colonization, the asymptomatic carriage that was the key to Eichenwald and Shinefield's treatment for penicillin-resistant staph 80/81. It had been known since the 1930s that people could carry staph without being made sick by it, usually on the mucous membranes just inside the nostrils.[6] Staph can also survive on the moist skin of the groin and armpits, and on dry exposed skin as well if the skin's immune protection has been disrupted by eczema or similar diseases.[7] Up to 30 percent of adults carry staph persistently, and many more can be transiently colonized for days or weeks if they are exposed to the bug.[8]

Despite the revelation of the cloud babies, it was not yet understood how often colonization led to disease. People who developed staph boils and sties tended to be infected with the same strain they were carrying in their noses—suggesting they had infected themselves—and also were more vulnerable to wound infections after surgery.[9] And carriers could infect others even if they were experiencing no symptoms themselves. In 1958, the brand-new U.S. Centers for Disease Control determined that the source of thirteen staph infections at St. Joseph's Hospital in Atlanta—several of them life-threatening, one of them so serious that the patient lingered in the hospital an additional five months—was a surgeon who was an unwitting nasal carrier.[10]

Staph colonization is remarkable. There is no other disease-causing bacterium that is carried, without causing infection, by such a substantial slice of the population. The arrival of antibiotics altered that stable balance. They put the bacteria under what scientists call "selective pressure," creating a microbiological battlefield on which the ones that developed resistance would be more likely to survive. Researchers soon noticed that the hospital patients who were colonized with drug-sensitive staph became carriers of drug-resistant staph instead once they were given penicillin.[11] Then they found that hospital workers, working

in an environment where both pathogens and antibiotics were common, were even more likely to carry drug-resistant staph than their patients were.[12] It was too soon to know whether new methicillin-resistant staph strains might colonize patients more quickly, or persist in their nostrils more durably, or move from one person to another with more facility than penicillin-resistant strains had. But it was evident that broad use of antibiotics not only caused drug-resistant infections; it also made people who had no symptoms of infection into silent carriers of drug-resistant strains. The first hospital outbreak of MRSA demonstrated how significant that would be.[13]

Queen Mary's Hospital for Children, the first dedicated pediatrics hospital in England, boasted an odd but beautiful layout, the relic of an Edwardian-era hospital-building boom. It comprised three ranks of twenty-four single-story buildings, laid out on 136 acres of lavish park-like grounds, and housed 850 children undergoing medical care, tuberculosis quarantine, and long-term rehabilitation. It also had a busy and curious microbiology lab that began scouting for methicillin resistance as soon as the hospital began to use the drug.

In March 1961, the hospital found its first methicillin-resistant isolate in a baby who had been admitted in January with a drug-sensitive staph infection. She got penicillin, and the infection resolved, but she remained in the hospital for unrelated reasons—and when she was checked two months later in a routine survey, she had become a carrier of methicillin-resistant staph. The next month, the same strain had spread to four other children. Two of them were only colonized, but two were infected, one within a patch of eczema and the other with an ear infection. The hospital tried to cordon off the strain, isolating the children with infections, closing the building where they lived to all visitors, bathing the patients and staff with antiseptics and inserting antibiotic ointments into all of their noses. It was too late, though: by May 1962, the bug spread through the hospital, to eight of its forty-eight wards, infecting thirty-seven children and one nurse. One child died.

The hospital had mounted what it called, in a research report, a "fairly vigorous prophylactic campaign" to contain the strain. Yet the outbreak persisted for fourteen months and spread with ease between widely separated buildings on a campus where few patients were well enough to walk that far. That raised the possibility once again that an unwitting carrier—a patient, a parent, or a health care worker—could be the vehicle for transporting a resistant strain.

* * *

Throughout the 1960s, British and European hospitals reported increasing amounts of MRSA, and so did Australia. Sydney's Royal Prince Alfred Hospital built a new surgical ward expressly to reduce outbreaks.[14] But by January 1967, there had been no MRSA outbreaks in any U.S. hospital. Doctors at Boston City Hospital, the overcrowded charity institution in the city's South End, even crowed in a medical journal that hospital infections caused by staph had "decreased markedly."[15]

Just weeks after publishing that account, Boston City discovered the first MRSA cases in the United States' first outbreak, which would spread through the hospital for almost a year. It started with patients in a single, small medical ward. One arrived coughing from a lung infection, and two days later the hospital found the same strain in another patient in a nearby bed. Ten days after that, a third patient in the same ward was found to be infected, and a fourth patient in a separate ward. MRSA percolated through the building: one case in March, two in May, five in June, one each in August, October, and November, two in December, and three in January 1968. The outbreak comprised two waves of infections—bronchitis, pneumonia, urinary tract infections—that each seemed to be triggered by elderly patients who had arrived sick from nursing homes. But the bacterium also seemed to be moving back and forth between patients and staff. The eighteenth case in the outbreak was a nurse who was only colonized, with no symptoms, who had treated the previous four cases. The patients were all men, housed in one small room with four beds. Two had pneumonia and were coughing, and one had a tracheostomy that needed regular suction. It was impossible to determine who was infecting whom. Tracing the bug's path through the building, the hospital's investigative team issued a report that sounded like a prophecy: "It seems reasonable to predict that methicillin-resistant staphylococci will become more widespread in the United States, and may present some clinical and epidemiological problems in the future."[16]

They were right. MRSA vanished from sight for several years, but in the mid-1970s it began to hopscotch the country. In 1976, twenty-three patients at Hartford Hospital in Connecticut, almost all of them recovering from surgery, became infected in less than a year and developed very serious illnesses: pneumonia, osteomyelitis, lung abscesses, bacteremia. Three died.[17] In 1978, sixty-one patients at Memorial Hermann Hospital in Houston contracted MRSA, most of them surgery patients or burn victims.[18] That same year, the University of Virginia's hospital

admitted a patient with a chronic skin condition and a heart-valve infection who brought MRSA with him. The bug leapfrogged through the hospital, from that patient to a second patient via their shared physician; from the second patient to a third, fourth, and fifth by the close proximity of their beds; and from the fifth patient to five more when he was transferred into intensive care. Within six months, sixteen patients were infected, fourteen more became carriers, and three died.[19]

By 1980, 109 hospitals that responded to a national survey had treated at least one case of MRSA. Dismayingly, many of those cases had blown up into outbreaks. Thirteen institutions said they had had fewer than ten cases; but thirty-four said their outbreaks had involved fifty cases or more. Only thirteen could say they were confident that they had chased the bug completely from their buildings, and twelve of those were hospitals where the outbreaks had been very small.[20]

A picture was starting to emerge of who was most vulnerable to MRSA. They were patients who were very young or elderly, or severely or chronically ill, all conditions in which the immune system is not robust. In one outbreak at St. Louis University School of Medicine, 70 percent of the fragile newborns in the neonatal intensive care unit were colonized.[21] Patients were in greater danger if the protective barrier provided by their skin had been breached by surgery or burns; at the City of Memphis Hospital in Tennessee, burn victims were infected by each other and by the hydrotherapy machinery used to debride their wounds.[22] And they were more likely to be colonized or become infected if they had received any antibiotics, because any sensitive bacteria they carried were killed by the drugs and replaced by the hardier resistant organisms.[23,24]

It was also becoming clear how MRSA reached vulnerable patients in order to infect them. The organism colonized patients, but it also survived on hospital surfaces—not just hard surfaces such as counters and bed rails, but bed curtains and doctors' coats as well.[25] And as Queen Mary's Hospital and Boston City Hospital had suspected, it colonized health care workers, sometimes transiently, sometimes long-term. An outbreak among sixty-six patients at two Oregon hospitals was traced to a single colonized physician who worked at both of them.[26]

There was suddenly so much MRSA, in so many different hospitals, that patients' own colonization could no longer be blamed for it. Something was transporting the bug from its many hiding places—patients, staff, the hospital enviornment—to the many vulnerable patients it attacked. With outbreaks increasing across the country, hospitals reluc-

tantly faced a difficult realization: sometimes the vehicle was their own health care workers. Even when there were MRSA outbreaks in their own institutions, health care workers were not washing their hands.[27]

All of those factors—rising incidence, vulnerable patients, unrecognized colonization, inattentive care—came together in one terrible outbreak that began in 1980. It forever established MRSA as a deadly hospital threat.

In June 1980, a sixty-three-year-old man from Washington State who had been badly burned in Texas and was treated at a Houston hospital that had been combating MRSA in its burn unit, was transferred to Harborview Medical Center, the public hospital in Seattle. Harborview knew he had MRSA; it grew from a wound culture taken when he was admitted, and from his sputum and blood in the days that followed. But MRSA was not new in Seattle; doctors there had experience treating it, and the hospital must have believed it knew what to do.

The man died twelve days after arriving. In those twelve days, he set off a chain of transmission that would spread MRSA to thirty-three other patients across fifteen months, grievously sickening twenty-seven of them, and killing or contributing to the deaths of seventeen.[28]

Reconstructed, the outbreak looked like this: The man transferred from Texas—call him patient 1—was admitted to a thirty-bed burn unit. Patient 2 was already in the unit, and then became infected. Patient 1 died in early July; a few days after his death, Patient 3 was admitted to the unit, and also became infected in turn. Then came patients 4, 5, 6, 7, and 8, successive admissions to the burn unit who all became infected with MRSA in their debrided wounds. Patient 9 was also admitted to the unit, but then developed MRSA pneumonia so severe that it required a transfer to the ten-bed surgical intensive care unit on a different floor. After patient 9 left the burn unit in October, patients 2, 7, and 8 died; patient 4 had died earlier.

Susceptibility tests early in the outbreak had shown that this MRSA strain was resistant to a number of drugs: methicillin, tetracycline, erythromycin, and clindamycin. In the surgical intensive care unit where patient 9 had been sent, the hospital was conducting a drug trial of an older antibiotic called chloramphenicol. When the MRSA strain passed from patient 9 to patient 10 in an adjacent intensive-care bed, it picked up resistance to chloramphenicol as well. Patient 10 had been in the intensive care unit since September, before patient 9 came up from the

burn ward. Because he now had multidrug-resistant MRSA, he would remain in the unit until the following January. While he was there, his MRSA strain would infect fifteen additional patients, and nine of them would die. In January 1981, patient 10 was sent to a rehabilitation ward, where the strain passed to three more patients; two died.

Toward the end of 1980, Harborview tried to shut down the outbreak by putting any infected or colonized patient in isolation. In March 1981, the hospital also closed down the surgical intensive care unit, and additionally opened a new burn unit. Of the outbreak victims, only patient 10 remained in the hospital, in the rehab ward. All the rest were dead or had been discharged. And yet, in May and June, six new infections surfaced in patients in the new burn unit. They had no connection with the previous patients—no family relationships, no crossing paths on wards—and yet their strain was essentially the same.

The only thing the patients could have in common was the health care staff who treated them. Harborview checked everyone who might have had contact with patients in both waves of the outbreak. Out of 182 nurses and physicians and respiratory, occupational, and physical therapists, they found one: a nurse who worked in the new burn unit. She was colonized in her ears, nose, armpits, and groin with the same MRSA strain that had infected the six new burn patients. Further, she had worked in the old surgical intensive care unit where patient 9 had passed the strain to patient 10. And as a student nurse, she had cared for patient 4.

The six newest patients all recovered, but only after lengthy treatment with vancomycin and rifampin. By the time an account of Harborview's epidemic appeared in a medical journal in September 1982, there had been no new cases for a year. But the outbreak could not be declared completely over: patients 10 and 12 were still taking antibiotics, not yet free of the serious infections that they already had been enduring for two years.

Harborview's experience finally persuaded American medicine that MRSA was a burgeoning threat. But action still did not come quickly, and the bacterium did not wait. In 1975, when hospital laboratories around the country tested the staph in their institutions, they found that only 2.4 percent of staph strains were methicillin-resistant. In 1991, in the largest hospitals, the proportion rose to 38 percent.[29] By 2003, in intensive care units, it was 64.4 percent.[30]

By every measure, the situation got dramatically worse as time went

on. In 1993, there were fewer than 2,000 hospital stays for MRSA infection in the United States; in 2005, there were 368,600, and each hospital stay cost twice as much and lasted twice as long as one where MRSA was not part of the diagnosis.[31] In 2005, the CDC estimated, 94,360 invasive MRSA infections occurred in the United States, most of them caused by or linked to health care. "Invasive" infections meant only the most serious, including pneumonia, osteomyelitis, and bacteremia; they caused 18,650 deaths that year, more than were caused by AIDS.[32]

Behind every statistic, there lay shattered trust, enormous bills, and irrevocably altered lives.

Jay Sorensen did not have a heart attack; he survived his collapse. His job did not. He had missed so much work that the radio station let him go, on Andrea's birthday, in January 2007. The hole in his abdomen healed in early February. But in April, he began to feel a bulge again. He went back to Nagy, who sent him for a CT scan. As Jay feared, it was another hernia—not in the recent surgical repair, which had held, but in a new weak spot in the muscle, above his original surgical scar. His fifth surgery was scheduled for June. Then, in May, Andrea discovered a lump in her breast. She had a needle biopsy at a local walk-in surgery center and it went smoothly—but two weeks afterward, she woke up on Memorial Day weekend, teeth chattering with chill, and realized she had a fever.

She called her doctor, but he was headed out of town. He sent her to the ER, where they looked at her breast—bright red, swollen, sparking with pain whenever her arm or clothing brushed against it—gave her an IV of pain medication, and drained the infection with a syringe as long as her hand. It took two syringes to get all the pus out. Some was sent to be cultured. The infection was MRSA—but whether she picked it up at the surgical center or from Jay was impossible to know. It took several more doctor's visits to clean and close the abscess, along with an expensive course of linezolid. It left her with a breast so badly scarred that Nagy recommended reconstructive surgery, but her infection and Jay's many recurrences left her too distrustful of health care to attempt it.

Jay's fifth surgery went ahead in June 2007, closing the latest hernia as well as another new weak spot in the muscles that Nagy found. Yet again, the MRSA recurred. That meant another hospital stay and more vancomycin, and this time heavy-duty pain medications that left him in a junkie-like state of withdrawal. Through August and September, cellulitis—a rough red inflammation of the skin that signals infection but

produces no pus to culture—bubbled up near the surgical scar. He took oral antibiotics at home to combat it. The new surgical wound was slow to close as well, and a wound-care nurse came to clean the incision and pack and bandage it where the edges had not healed. In December, Jay stepped into the shower to clean up for the nurse's visit, and to soften up the packing so he could remove it and wash the wound before she arrived. He pulled, and felt something give way internally. A piece of packing had been left in the incision; fluid had developed behind it and then worked its way to the outside of his body. It left a channel that could be vulnerable to infection. He went back to Nagy for his sixth surgery, to close the newest wound, in February 2008.

"He has probably the worst wound healing that I have ever encountered," Nagy said. "It's hard to say why; it could be a lingering, subacute infection in the soft tissue, but it's difficult to test for that. Once you cut through those muscles, even when they heal, they will not be as strong."

Throughout 2008, Jay examined his healing incisions anxiously, panicking at the sight of any redness, popping another course of antibiotics whenever his physicians thought something might be blowing up again. In September, his abdomen began to ache and he felt a new lump emerging, close to his groin and below the surgical scars. Whenever he moved or shifted, it bulged. His doctors referred him on to another team of surgeons, in Philadelphia, who had more expertise operating low in the body close to the genitals. On December 30, 2008, Jay had his seventh surgery, a multihour procedure with several surgeons that involved unwinding a loop of intestine that had slipped downward out of his abdomen and was wrapped around one testicle. It took more than one hundred staples to close the incision. It was more than three years since his first infection had turned an uncomplicated surgical recovery into an indefinite ordeal.

Jay and Andrea estimated that, from 2005 to 2008, his insurance spent more than $1 million on his care. But to them the true cost went far beyond that number. It was the irritable bowel syndrome Andrea developed from worrying about Jay and the one hundred pounds she lost as a result. It was the visits with their children and grandchildren that they sacrificed for fear of infecting them. More than anything, it was the irrevocable change in Jay: still big—though nowhere near as big as he had been—but no longer optimistic or confrontational or bold.

"I don't have words for what this has done to my psyche," he said in New Jersey, one day in 2008. "I think of my mortality, a lot. I think of

what this has done to my wife. She has posttraumatic stress disorder from this, I'm sure of it. I dread looking down at myself in the shower, wondering if it's going to start again. I think I have a fear that, if I get it another time, none of the antibiotics are going to work."

That fear was not unreasonable. In Chicago, Robert Daum's team had been patiently unwinding the genetic mechanisms that had allowed hospital-acquired MRSA to flourish and community-associated MRSA to emerge. The team's contention that community MRSA was a different entity requiring different control strategies was about to be bolstered—not by scientific agreement, but by a wave of community infections that would sweep the United States.