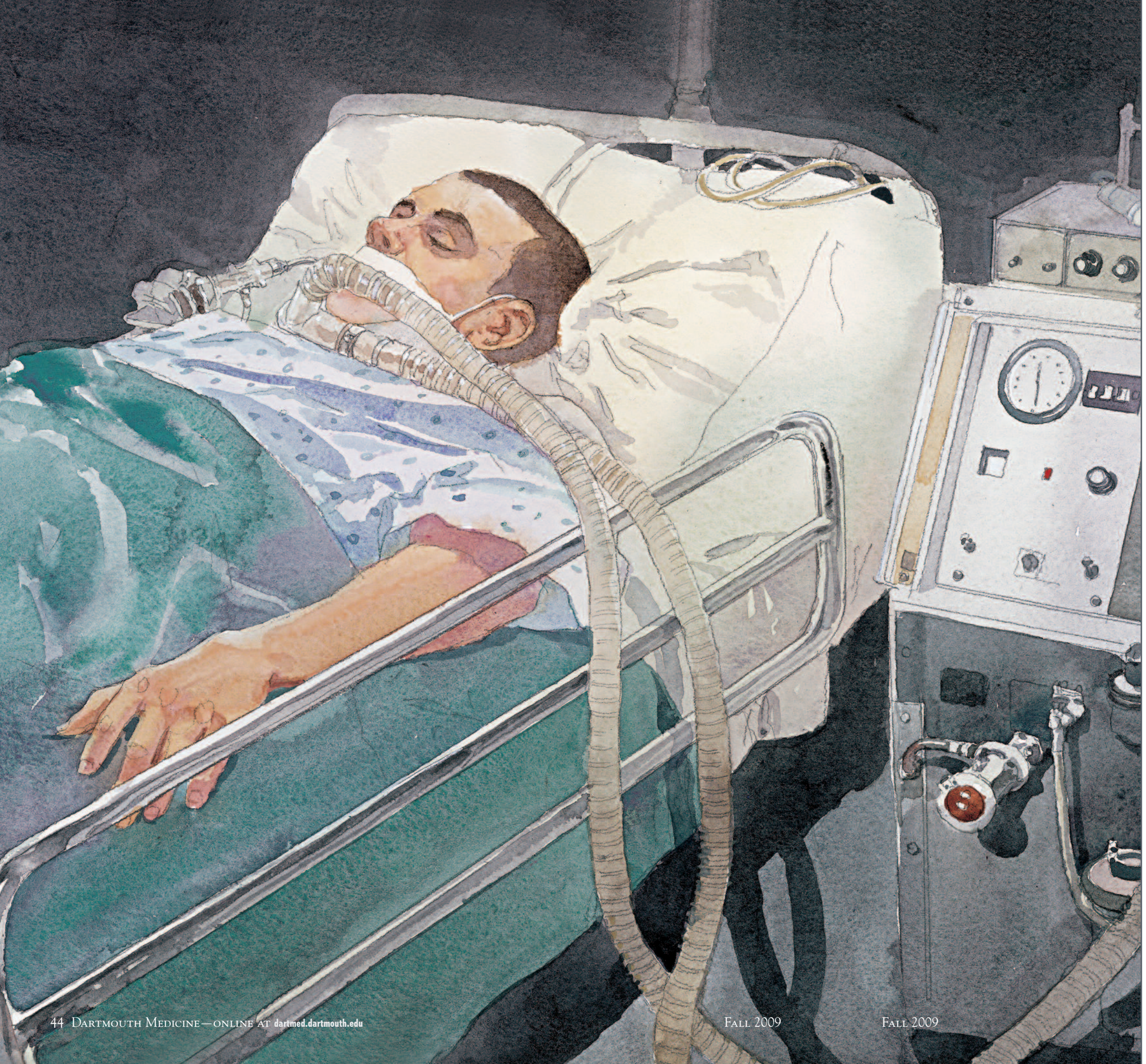

Pondering Personhood

Story by Robert Martensen, M.D. Illustration by Bert Dodson

Two comatose teenage boys, 20 years and 200 miles apart, lead a noted ethicist and medical historian to ponder the nature of personhood—the nuances of determining if and when death is inevitable. This feature is excerpted from a new book by a DMS alumnus.

Ryan: Boston, 1993

I was the staff emergency physician on duty one Saturday morning at a Boston-area hospital when an ambulance brought in an injured boy. Ryan, who was 17 and went to high school in Boston's western suburbs, had woken up that morning eager to play soccer, which he did. On the way back from the game, a truck rammed the passenger side of the car in which he occupied the front seat. Now he lay motionless in the hospital's trauma room. What I saw was this: a fair-haired, pale teenage boy of a slender and wiry build, about five feet eight inches tall, who weighed around 125 pounds. He had bruises on his right forehead and temple, and his right upper arm and leg showed obvious deformities, though the skin was not broken. Ryan's airway, neck, chest, abdomen, and pelvis seemed normal on my emergency survey. Based on initial x-rays, Ryan's cervical spine looked good to me, and the orthopedic resident said the fractures—of an arm and a leg—did not require surgery and should heal well. X-rays of his chest, abdomen, and pelvis suggested that they were not injured, as did the absence of blood in his urine.

It was Ryan's brain that was the problem. Though his pupils responded normally to light, he

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moved his good arm in an uncoordinated way when his chest was pinched. Despite breathing on his own and maintaining a stable pulse and blood pressure, he remained unconscious and unresponsive. Could a mass lesion—a bleed—have been causing Ryan's unresponsiveness? CAT scans of his skull showed no pool of blood or clots for the neurosurgeons to evacuate, and Ryan's ventricles and cisterns—the cavities in the brain that contain cerebral spinal fluid—were not obliterated, though two might have been compressed. (Compression of the ventricles is an ominous sign, especially when it is not caused by a discrete mass, for it suggests that a diffuse injury is causing the brain to swell. Too much swelling in a closed compartment like the skull frequently leads to irreversible brain damage or worse, the upper brain forcing the lower brain downward against bony protuberances inside the base of the skull, a situation that often leads to death if it is not halted promptly.)

Although Ryan was breathing on his own and maintaining a decent level of blood oxygen, we intubated his trachea and put him on a ventilator in case his brain swelled. If the ventilator was set to stimulate his breathing slightly, the level of carbon dioxide in his blood would fall, and studies on patients with severe head injuries as well as animal studies suggest this would likely lower his intracranial pressure. We also elevated the head of Ryan's bed 30 degrees, and I put an intravenous catheter in his left jugular vein to monitor his blood levels of carbon dioxide and oxygen. Our care was "supportive," and it did not feel like much.

We speculated that Ryan's brain had sustained a diffuse axonal injury. The back-and-forth collision of his head into solid parts of the car had profoundly, if subtly, damaged his brain's cortex, even as his brain stem was capable of directing his vital functions. An MRI would be able to detect focal abnormalities in his frontal and temporal lobes as well as in the corpus callosum, the structure that connects the right and left cerebral hemispheres. Ryan's family had not yet arrived, and I turned to other patients as he went for his MRI.

"Will he recover?" I asked the neurosurgical resident.

"Hard to say," he responded. "He's a teenager, so he'll probably do better than either an infant or adult, but we'll just have to see. It depends. His ventricles look a little compressed, not a good sign. About 40 percent with his injury die in the hospital."

What I knew about long-term outcomes for severely brain-injured people was that many apparent recoveries turned into tragedies, as about 40 percent end up in a persistent vegetative state (PVS), often after an initial lengthy coma. The resident suggested I call the organ transplant coordinator; "better to start with the family early," he said. To look at Ryan, relatively unmarked except for the forehead and temple bruises, one would not have perceived him to be at the end of his life. His skin was pink and warm and his breathing regular, and he looked asleep. Indeed, he looked very much like my older son, who was 15, slight and wiry, and devoted to soccer. I did not call the transplant coordinator.

About two hours had passed since Ryan arrived. He was back from his MRI, though I had not yet seen it, and his family was waiting for news in the so-called Quiet Room. On the way to see them with a nurse, the transplant coordinator found us. Her position was new, part of the hospital's push to become dominant in the Boston organ transplant "market." She wanted to come in with us to talk with the family, "after you do, of course, Dr. Martensen." I demurred; the timing was not right. I told the coordinator that Ryan was stable now; she could talk with the family in another setting later. But the coordinator, not pleased, switched into earnest mode: "You *do* know, don't you, Dr. Martensen, that our new program involves early intervention in the ER. It's been shown to increase the harvest rate if we talk to the families early. I saw you at the presentation we did for your ER group."

Though I loathed her agricultural euphemism—*harvest* was the new buzzword in the transplant world—I thanked her for coming in on a Saturday. Then the nurse and I left her outside the door of the Quiet Room and went in to see Ryan's family. When we were all introduced to each other—they numbered 14 or 15—we sat down. I talked about what had happened to Ryan.

"What should we do?" his parents asked the nurse and me.

"Talk to him," I said. "Talk to him as though he can hear you. We do not know his future, what his function will be. It may well take weeks to sort out." The ER was busy, and I left them as a priest came in.

As I continued seeing patients that day, what moved in and out of my mind were memories of another young man who went into a prolonged coma 20 years earlier, while I was studying to become a doctor and "persistent vegetative state" was a new phrase in the medical vocabulary.

Caleb: Dartmouth, 1973

Caleb was a teenager and breathing on a ventilator and I was a 26-year-old Dartmouth medical student on my pediatrics rotation when we first encountered each other in August 1973. He looked as though he were sleeping, and he had been that way for almost three months. His eyes used to open and close spontaneously two months earlier. But no movement since, just a flaccid paralysis. All Caleb's deep tendon reflexes—the doctor's tap below the kneecap—were gone. Talking to him or pinching him or giving him a whiff of ammonia elicited nothing. Indeed, only his pupils responded reflexively: when one shined a light in one pupil, it contracted, and so did the other. That, and the fact that his pupils maintained normal diameters, were probably the only good neurological signs Caleb had, for they showed his midbrain was working. In every way possible, though, he depended on critical care nurses, doctors, and machines. In the meantime, he was excreting and even growing. Caleb's puberty had just kicked in.

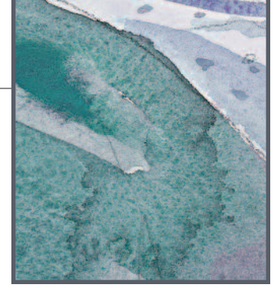
He was a big-boned youth, about six feet tall and 170 pounds, preternaturally pale and with scattered pimples on his puffy face. Whenever I saw Caleb, I also saw his parents. He was their only child. Day in and day out and on most nights, one or the other stayed by his side in the small ICU room where Caleb now lived. Their names where Stephan and Sofia, and, like their son, they were big-boned and had fair skin and dark hair and eyes. They might have been brother and sister, so alike did they look. Originally from Hungary, they had come to this country as newlyweds in 1956 to get away from the Russians. Caleb was conceived shortly after they arrived in Boston, which was why they had given him a New England-style name, not a Hungarian one. He would be a "true American." Since Caleb had been in the ICU, they had switched around their jobs—his with a nearby fire department and hers with the hospital food service department—so that one or the other, and ideally both, could be with their son. What else could they do?

Baffled, the doctors did not know what to tell Caleb's parents. They did not know when Caleb would come out of the coma—if in fact he was in a coma—nor did they know why he had fallen into the coma in the first place. They suspected he was experiencing an extreme case of Guillain-Barré syndrome. But no test existed then (or now) to rule Guillain-Barré in or out. Compared to a disease such as childhood diabetes, which has biochemical markers, the boundaries of a syndrome, whether severe or mild, remain fuzzy by definition. Diagnosis of Guillain-Barré is suggestive and wholly clinical, which means it fits a picture composed of symptoms and signs that doctors can observe. Although almost everything about the course of Caleb's problems fit Guillain-Barré, Dartmouth's neurologists, an experienced group, had never seen or read about a patient with Guillain-Barré going into a coma before. Before Caleb had descended into a coma, he had leg weakness, followed by weakness in his arms and facial muscles, followed by increasing paralysis, all symptoms typical of Guillain-Barré. Before Caleb had developed these symptoms, he had experienced a cold, which is also characteristic of the syndrome.

Guillain-Barré syndrome, which was named after two French physicians in 1916, is an example of the body's unpredictable reaction to an event, in Caleb's case most likely the recent cold. As the syndrome runs its course, it is as though the body becomes allergic to part of itself, which is why it is considered an autoimmune disorder. People with severe Guillain-Barré become paralyzed when their own immune systems, rather than toxic proteins from some bacterial invader like tetanus or rabies, inflame their peripheral motor nerves at their intersections with muscle cells. Most of the time the inflammatory reaction is weak, sensory nerve roots are spared, and symptoms are mild and disappear over weeks. Sometimes, however, sensory nerves are affected, which is probably why Caleb reported numbness and tingling early on. In some percentage of those affected, Guillain-Barré paralyzes the muscles of the chest wall, and the patient may die. Nothing cures the syndrome, just as nothing cures tetanus; unlike tetanus, however, there is no vaccine for Guillain-Barré. But if Caleb did have Guillain-Barré, his brain was likely functioning, for the syndrome is not thought to involve neurons in the central nervous system. He would be sensing and thinking, but his total paralysis would prevent an observer from knowing for certain that he was. He would exist in a frightening (and rare) carapace, in which he could feel and sense and think but be incapable of expressing anything.

Was Caleb, in his paralyzed state and seemingly asleep, still capable of perceiving the external world? Not knowing what he might or might not be perceiving, his doctors told his parents and nurses that they should just talk to him in ordinary ways. This meant having technical discussions out of Caleb's earshot on the off chance that he might hear and be disturbed by them.

Sofia and Stephan took this to mean they should keep Caleb up on his regular interests—TV's *Twilight Zone* and the Red Sox being the principal ones. They turned on the TV and radio when either was being broadcast, and they read him the sports pages from the *Boston Globe* and most of *Sports Illustrated*, spiced with *Popular Mechanics* and the occasional car magazine. Caleb's best friend from school came by often and told him jokes and what everyone was up to. And so the weeks passed. As the summer flowed on and Caleb remained in a coma, his parents would start talking about Caleb finishing high school and going to college before their voices trailed off. All of us were concerned that he was not waking up. Since an extended coma was not typical of Guillain-Barré, we had no idea if, or when, Caleb might reenter the regular world. *When will Caleb wake up?* became, without anyone ever mentioning it,



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Will Caleb wake up? No one wanted to put the question in words, but one could see the shift in everyone's face. By September, the question of his future had become acute.

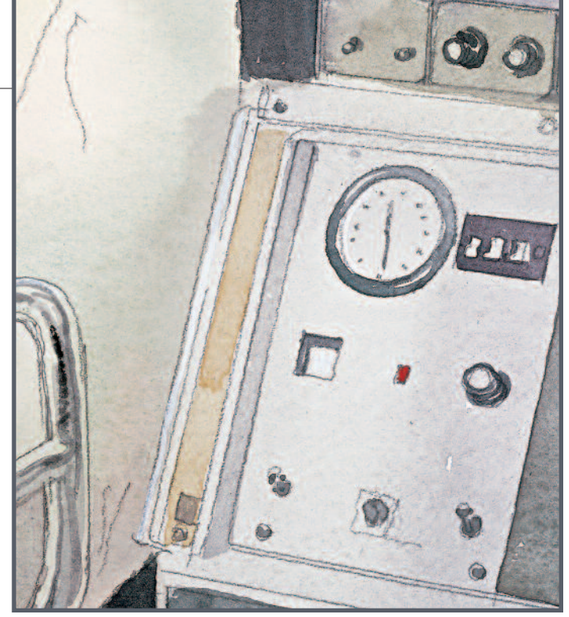
Neurologists were and are brilliant examiners, but before CAT scans (and then MRIs and PET scans), they gained most of their knowledge not by studying images of functioning brains, but through close observation of people's exteriors, awareness, and capacities. Simple x-rays have never been very good for delineating subtle aspects of soft tissues, especially when a process is diffuse. Unlike today, when a PET scan can light up exactly which part of the brain is active in a Buddhist doing a specific type of meditation, neurologists then had no way of directly visualizing what brains are doing. For generations they relied instead on questions and taps and tests of individuals' motor and sensory functions and mental states to correlate seemingly tiny signs of neurological dysfunction with diseases whose outer signs might otherwise have remained obscure. Autopsies, not images of living brains, provided the diagnostic confirmation.

Although Dartmouth was then about to install a CAT scanner, one of the nation's first, we could not have slid Caleb into its narrow chamber because we could not have maintained his ventilation while he was inside it. Electrical recordings—EEGs—of the brain's activity often provided useful information, but their analysis then was not as refined as it is today, especially in comalike states that had been going on for a while. We performed EEGs on Caleb regularly, but they were not specific for anything, displaying only diffuse slowing and dampened amplitudes consistent with someone on sedatives. Caleb's neurologists talked about doing a brain biopsy on him but ruled it out because of the risks. Was his brain functioning or not? And if so, was his cortex—the part we have come to think of as containing our identity as a person—working? We simply could not tell.

We students began to wonder if Caleb could be in a state that we were just starting to hear about—a "persistent vegetative state" (PVS). That term had been coined by Dr. Fred Plum, a 1945 Dartmouth Medical School graduate and the coauthor of a widely used text on stupor and coma, and Dr. Bryan Jennett, a Scottish neurologist. Spurred by our teachers, we mused on Caleb's state in light of their new definition. As in his previous writing on stupor and coma, Dr. Plum was pithy in the 1972 article on PVS that he and Dr. Jennett wrote for *The Lancet*: "New methods of treatment may, by prolonging the lives of patients with conditions which were formerly fatal, result in situations never previously encountered." He continued: "Few would dispute that in this condition the cerebral cortex is out of action."

In other words, the lower brain could be working, as Caleb's papillary reactions demonstrated, but the brain's upper regions, its cortex or cerebral hemispheres, might be persistently nonfunctional. As Drs. Plum and Jennett emphasized, determination of PVS was clinical. No single test or set of tests could confirm it, and in those days no consensus existed about its timeline—the point when one could say someone was not in a coma but was persistently vegetative. PVS was simply too new for anyone to know anything definitive about it.

Could this be our Caleb? We—all of us medical students, nurses, doctors, technicians, and his family and friends who spent time by his heaving ventilator—moved through our days on the edge of despair about him. Perhaps Caleb despaired, too. From my pediatric clerkship, I moved on to surgery and anesthesia, but in free moments I con-



tinued to circle back to the ICU to see Caleb and his parents. A number of other students who'd spent time with them also did so. Our little "Caleb group" sometimes gathered at his bedside at the end of the day. We would watch him and look for signs. Stephan and Sofia still read to him each day, still turned on the TV, and still talked to him about the Red Sox, but their voices became increasingly listless.

During those gray winter days, Caleb's mother and father began to notice something. When they entered the room and started talking to their son, he seemed different, more alert somehow, though they could not put their finger on how. Then, one morning in late January, approximately nine months after Caleb was first admitted to the emergency room, a member of the Caleb group, an intern, hurried over at lunch to say that Caleb's fingers and toes were twitching. Caleb had moved! His parents, usually so stolid, were overwhelmed. Leaning forward earnestly, they whispered to us that they had hoped against hope for this moment and now they were afraid to hope for too much. As word got around, the nurses began enforcing the visitor limit. Two days after he twitched his fingers, Caleb began moving his arms with purpose; a couple of days later he moved his leg. Along the way, he experienced that immensely poignant moment when he opened his eyes and looked around. It was late at night, and only his parents were there. He saw them! He knew them! It was enough for them: their stolidity gave way to broad smiles when I saw them the next day. No longer content to sit still on either side of his bed, they caressed his face with its new peach fuzz and asked everyone, "What should we do now to help?"

Within a week Caleb no longer needed a ventilator, though it stayed in the room in case he relapsed, as some with Guillain-Barré were known to do. And then he began talking. "What happened to me?" he kept asking his mom and dad. Like many other patients who go into a coma and recover, he had little idea of time. He still thought it was early spring, that he had been "out" for maybe a couple of weeks. He remembered that the Red Sox had swept a four-game series against the Yankees over the Fourth of July and that it happened on the Yankees' turf. But Boston's glorious run had happened long after Caleb's collapse. Although he had appeared to be unconscious, he had actually been hearing and understanding some things around him. He also remembered his mother crying one day, but he had no idea when. Fortunately, like many who experience severe trauma or go into a coma and then come out, he had little memory of any significant pain. What mattered to him most, he said, was getting back on his feet and eating. A month after he twitched, he was home, nicking himself with his new razor and bemoaning his loss of a school year. On his follow-up visits to the pediatric clinic—at least those that I managed to attend—Caleb exhibited no cognitive deficits. Aside from occasional numbness and tingling in his fingers, he said he felt fine. According to our new CAT scanner, whose tube Caleb was now able to enter, his brain showed no atrophy or loss of cortical mass—a loss that is typical of PVS. His muscle conduction studies and an EEG were normal.

Exactly what happened to Caleb remained unclear. After an extensive review of his case and the world literature, his neurologists stayed with their midsummer diagnosis, which was that Caleb had endured an extreme form of Guillain-Barré syndrome, complicated by a low-level autoimmune inflammation of his brain whose origins remained a mystery.

Although Caleb was never in PVS, he might easily have been. If he had gone into respiratory arrest at home or on the pediatric ward, where the hospital had originally thought to place him instead of in the ICU, his brain might have spent several minutes without oxygen before he was resuscitated. The very intervention that had kept him alive—emergency intubation of his airway and mechanical ventilation—would have served to get him back only to the partial life of someone who was persistently vegetative. It struck us students as curious that if that had occurred, we might not have been able to tell the difference for months.

Is this a person?

Three weeks after that initial Saturday with Ryan, I ran into Ryan's father in the hospital lobby. The previous week Ryan had seemed to squeeze his father's finger when he had stroked his hand. There had been no purposeful movement since. The organ donation people had been by again, "just to have another chat" with the parents. Ryan continued to maintain his vital functions, and his fractures showed signs of healing on subsequent x-rays. The hospital's discharge planner was preparing for his transfer to a chronic care facility.

"What are Ryan's chances?" his father asked me.

"I don't know what his chances are right now," I said. "The neurologists and neurosurgeons will have a much better idea."

"What do you think we should do?"

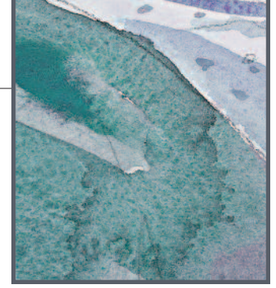
"Stay by your son," I said. "Keep talking to him. It's only been a few weeks since the injury. A recent study of people in comas who subsequently recovered showed that some of them remember bedside conversations. Talk to Ryan as though he is aware, even though he may not be. The study didn't explore people with injuries like your son's, but it's possible that at some deep level he may be aware." I added that the longer Ryan remained unresponsive, the less likely it was that he would recover significant function, though we did not really know.

"And if he doesn't wake up?" Ryan's father asked.

At one of our weekly ER physician staff meetings not long thereafter, Dr. Meldrum, our new ER director, brought in the transplant coordinator to give us an update on the program in relation to the ER. She reminded us that the hospital had been a pioneer in transplant surgery. She really needed us "to talk with those families." The hospital had been losing transplant "market share" to other area hospitals. As she wound up her talk, she noted that some of us had not been requesting her presence as early as we might. She and Dr. Meldrum would like us to call her at *any* time—a pause as she provided us with her cell phone number and Meldrum nodded—when patients with major trauma looked as though they might not survive.

During the Q&A that followed her presentation, I raised my hand. Transplants are great, I said. But isn't our mission in the ER to spend all our efforts on supporting the life of our patients? Aren't families experiencing enough shock at the news that their loved one has a serious, maybe fatal injury? Can't she wait until a little later than inside the ER to talk with families? For one thing, it puts us ER physicians in an awkward role with the families. How can they be sure we are doing our utmost for their loved one as we deliver the news, if we quickly switch to talk with them about organ donation? I knew a number of my colleagues shared these views, but none of them spoke. She and Dr. Meldrum did not respond directly, but their body language suggested my queries were not the response they'd hoped to receive.

What she and Dr. Meldrum did not say was that major transplant operations, as a colleague at another medical school put it, have become the "financial lifeblood" of academic hospitals. Even though this hospital was a nonprofit, dedicated to teaching and research, the "operating surpluses" generated by expensive, well-reimbursed proce-



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dures—and nothing surpasses major organ transplants in this regard—can mean the difference between red and black on a hospital's annual report. A successful program can fill up surgical ICUs and even support the development of new ones.

The hallway scuttlebutt was that the hospital was experiencing operating losses and dipping into capital for the first time to make ends meet. Which was why the hospital was undertaking "marketing initiatives" in many areas, with increased performance of major organ transplants at the top of the list.

During our annual reviews that year from Dr. Meldrum, a number of us ER doctors heard that we were not being sufficiently supportive of the hospital, especially in marketing. In our individual meeting, Meldrum told me to "get with the program." "Really?" I responded. My feedback from patients had been pretty good, I reminded him, not to mention feedback from fellow physicians. I had also managed to publish quite a bit. "That's not what I'm talking about," he said.

We physicians work with patients one at a time, and in the ER we do so in a unique moral space—24/7, with no questions asked about immigration status, nor any requirement for ability to pay, and with every effort made for the well-being of that individual. Serving emergency patients is our end; for us they are not a means to something else. To consider an emergency patient in the context of a financial or utilitarian calculus regarding the transplant potential of his or her organs is not for the treating physicians to do. Moreover, at the time of severe brain injury of metabolic insult—the two general conditions that may lead to PVS—medical science has no reliably predictive tools to determine who may or may not end up in PVS or a minimally conscious state. Whatever we know about these conditions, we do know this: they only become apparent after months have elapsed, and even then physicians cannot definitively say who is vegetative and who is minimally conscious.

As PVS has become more common and the demand for organs from living donors grows, a number of bioethicists, notably those connected with active transplant programs, have argued that the definition of brain death should be expanded to include PVS. The critical biological feature of personhood, they maintain, is a functioning cerebral cortex. Absent that—as is the case by definition in PVS—they assume a person is alive in name only and would be better off dead.

So how was I to respond to Ryan's father as he contemplated his son's fate? We sat down together in the lobby, and I told him what I knew of the science at that time. If Ryan was in PVS, which could not be determined so soon, then his outlook was grim. Since he was on the cusp of adulthood, I mentioned the outcome studies of adults who entered PVS due to traumatic brain injury. Ryan's chances of making it for a year and coming out with some function and awareness were a little better than one in four. The longer he remained unresponsive, the worse his long-term prospects, with 95 percent of those dying within five years of its diagnosis. After a year in PVS, though, anyone who "recovers" without fail demonstrates severe disabilities of function and awareness. Ryan's father said his son's current physicians had told him more or less the same. He wept—and I did, too.

About Ryan's status as an organ donor, which was the question behind his father's question, I kept quiet. Not only did I not know the answer, I doubted that medicine and its modern ethics could generate a universal one. Who are we in biomedicine to claim that we know the meaning of human identity? Despite the elucidation of biological mechanisms that have proven to be universal in humans, biomedical formulations of personhood are just one of many that have crossed and will continue to cross the world's stage. So how can contemporary physicians legitimately claim absolute privilege in determining the boundaries of personhood, including when it ends? When biomedical leaders maintain that their instrumental rationality is the only legitimate means of experiencing the world, including illness and disability, I would argue they step outside medicine's boundaries to the verge of scientistic imperialism.

Even within medicine's legitimate boundaries, physicians and ethicists who maintain that those living in PVS and related states are mere bodies and not persons have their work cut out for them. Less is known about PVS and related diagnostic categories, such as minimal consciousness, than they let on. Even with fresh donors whose brains are deemed wholly dead, transplant physicians commence their "harvests" of the "dead" person's parts by administering general anesthesia. Why do they need to do so if the brain-dead person is, in fact, dead?

A British anesthesiologist faced the issue head-on in a 1999 letter to the *Journal of the Royal Society of Medicine*: "The greatest misconception is that the donor will be dead in any ordinary sense of the word." As the anthropologist Lesley Sharp recently noted (and as I have witnessed): "A brain-dead body [that has not been anesthetized] will move in a lifelike way when nerves are pinched or cut. [It] may seem to shrug or kick or even signal."

As a society we need to engage in reasonable discussions about how we respond ethically, legally, and financially to the challenges posed by PVS and minimally conscious states. At a minimum, the interested parties, especially the transplant lobby, should candidly disclose their philosophical assumptions, financial interests, and lacunae in scientific understanding of brain death, cortical and whole. Organ transplantation is an invaluable way to extend life. But constructing fictions about its underlying realities renders sordid what ought to be resolved in clear light. ■