Intruder in the Heart

Miguel was wheezing like a worn-out radiator. He could no longer pretend that nothing was wrong.

BY CLAIRE PANOSIAN

MIGUEL NEVER RECALLED when they actually began—those feeble, quivering heartbeats that snaked through his chest like a high-speed train. The first attacks were so brief he forgot them as soon as they passed. Then, over time, they came more often and lasted longer, until one day a friend at work saw the whole crazy business, the sweat on the face and the fear in his eyes. He thought Miguel was having some kind of dizzy spell and made him lie down. Soon after, Miguel's wife drove up in their truck and took him to the emergency room at L.A. County Hospital. That's when they did an electrocardiogram, his first ever. "There are extra heartbeats," said the nurse, "but no sign of heart attack." The intern on duty added, "You're 38 years old, your blood pressure's great, you don't even smoke. You've got a lot of miles left on that heart." So Miguel decided it was nothing. From then on he pretended he was fine.

The following winter Miguel caught a cold from his daughter. The sore throat and aching muscles kept him in bed. Soon his feet and ankles began to swell, and he couldn't get air. Finally he spent an entire night bolt upright (since he could no longer catch his breath lying down), wheezing, coughing, and gurgling like a worn-out radiator. That night he also worried. He could no longer fool himself. He could tell that something was terribly wrong.

I'm an infectious-diseases specialist in Los Angeles, and I first met Miguel while doing rounds with students and residents in our hospital's Coronary Observation Unit. That's where "medium sick" heart patients are sent, as opposed to the sleek Coronary Care Unit, which is filled with sufferers of crushing chest pain and out-and-out cardiac disasters. I guess Miguel was only a slow-motion disaster. Over a period of months, he had gone from clinic to clinic. Finally he received a heart catheterization. In this imaging procedure, dye is squirted through a tube placed in the heart to reveal its structure and function. The results showed no coronary blockage. After that, Miguel carried the diagnosis of congestive cardiomyopathy and ventricular tachyarrhythmia, idiopathic. Basically, that meant he had a flabby balloon of a heart beating dangerously out of control, and no one knew why. His heart and circulatory system were functioning so poorly that fluids were building up. He had come to us for a possible heart transplant.

Before the students and I visited him,

PHOTO ILLUSTRATION BY SCOTT FERGUSON

I scanned Miguel's chart. Since his first attack of waterlogged lungs, he had taken digitalis and diuretics. The digitalis helped his heart pump more efficiently, and the diuretics helped flush out the fluid that was building up from the failing pump. He also stopped working—"Too tired and short of breath," read the social worker's note. On his current chest X-ray, his heart was a large baggy mass in the middle of his chest. But at least his lungs were clear; thanks to more potent diuretics, in the last few days they had been wrung free of fluid like a wet mop.

We left the nursing station and walked to Miguel's room. Seated in bed, oxygen tubing dangling from his neck, he was speaking Spanish on the phone and looked comfortable enough. When he caught sight of us, he hung up. Then I saw his face. "My God," I thought, "he's my age, but what old eyes." They were dark, deep pools with just a glimmer of hope. It was the hope that scared me.

With the help of a Spanish-speaking student, I launched into my polite prologue: who I was, what I did, who sent me. The usual formalities.

Miguel replied, "*Señora*, thank you for your time, but are you sure you're in the right room? You're the fifth doctor today, and not even a heart specialist. What's this about infection? I'm clean. Faithful, too. Not that it matters much, the way I feel now."

Thus began the intricate dance between doctor and patient. Language was the least of our barriers. My arrival made no sense to Miguel, and why should it? Even to many M.D.'s, microbiology is a far stretch from heart disease. True, we're all taught that viral infections can produce heart inflammation. Often, however, cardiomyopathy patients' damaged muscle fibers are eventually blamed on clogged arteries or too much booze.

The puzzle in Miguel's case was that none of these theories fit. Not that he cared. As he lay slowly dying, he wanted a mechanical fix, not a medical lecture.

Mechanic I'm not. What I am is a parasite sleuth. And today my quarry was *Trypanosoma cruzi*, a single-celled protozoan that can burrow into heart muscle. An ancient, sickle-shaped organism indigenous to Central and South America, it remains in the 1990s one of the leading causes of cardiac death in young and middle-aged people south of the border. Right now, the invader inhabits 16 to 18 million humans in Latin America and more than 300,000 U.S. immigrants. But most victims are unaware of their infection, as are their doctors. Hence, even in southern California, *T. cruzi* infections are missed more often than they are diagnosed. I didn't want to miss the diagnosis in Miguel. And so I pressed for answers.

"*Doctora*, these questions about my home—what does it matter?" Miguel asked. "All right, Jalisco was the place. My family has a small farm with goats and pigs and chickens. Our house? Oh, adobe—mud and straw—very poor. You'll find no fancy homes there. And yes, there are bugs in the house. Naturally. They have to live, too."

Slowly the clues were adding up: farm, animals, insects. The natural reservoir of *T. cruzi* is warm-blooded animals—lots of animals, currently numbering more than 100 species. House-dwelling insects, specifically known as conenose, or "kissing," bugs (entomologists call them reduviids) transfer *T. cruzi* from animals to man. The final clue was poverty. That's because reduviids often nest in the nooks and crannies of the poorest rural dwellings, wattle and daub houses. From this retreat, the bugs emerge at night to binge on humans and purge: they first suck blood, then dump parasite-laden feces next to the skin punctures they've just created. Most victims sleep through the insult.

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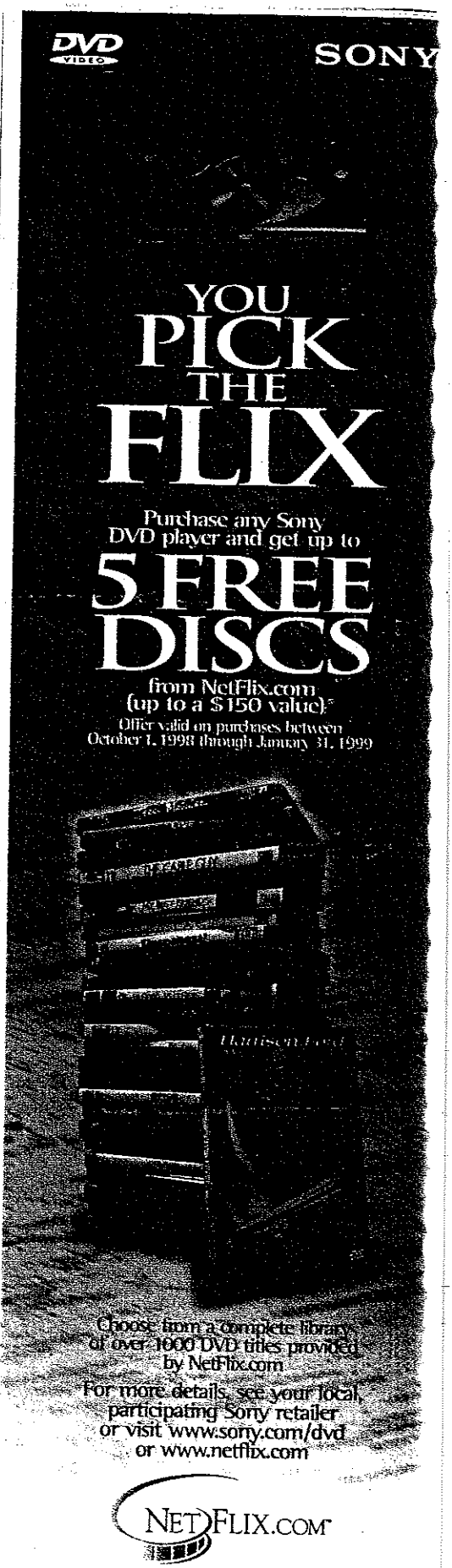

Carlos Chagas was the first scientist to expose the secret life of *T. cruzi*. Sent to central Brazil in the early 1900s to battle malaria, he spent his spare time viewing the intestinal contents of reduviids through his microscope. What he found were crescents that looked like *Trypanosoma brucei*, the then recently discovered cause of African sleeping sickness. Chagas later saw similar forms in the blood of other mammals, local residents, and nearby railway workers. He suspected the parasite was linked to the heart malfunction among the infected. Though the pathogen's taxonomic name, *Trypanosoma cruzi*, pays homage to Chagas's mentor, Dr. Oswaldo Cruz, most doctors today know the infection as Chagas' disease.

By now we were ready to examine Miguel. The team watched as I bent over his chest with my stethoscope. I was ready to hear the soft syncopation of a failing heart, but when I closed my eyes to focus, another image came to mind. I envisioned the moment years ago when the microscopic creatures entered Miguel's body and were swept through his blood, settling into tissues and building cystlike homes. Over the years, they reproduced in his brain, nerves, skeletal muscles, and heart. I pictured the protracted war they waged with his body's immune system, leaving heart and other tissue strewn with dead and dying cells. I tried to explain to him that this simple one-celled organism gave 25 to 30 percent of its victims chronic heart problems just like his.

Miguel did not understand. "*Doctora*, please excuse me, but you must be mistaken. I have been to hospitals many times in the past year. If I had *animales* in my heart, don't you think they would have known by now?"

Trusting Miguel. I would not burden him with studies that told otherwise. But later that day I resolved to show my team an article published in 1991 in the *New England Journal of Medicine*. In it, cardiologists at the biggest public hospital in Los Angeles hypothesized that a few patients with Chagas' disease could easily be lost among the far larger tribe of atherosclerosis sufferers patronizing county clinics. They proved their theory by finding 25 patients with Chagas' on their rolls who had previously been labeled "coronary artery disease" or "dilated cardiomyopathy" with unknown causes. Some of these *T. cruzi* victims had carried the misdiagnosis up to nine years.

I turned to Miguel's abdomen. First I felt for his liver. It was easy to find, a fleshy peninsula three inches below the ribs. It was slightly enlarged, confirming a blood buildup from an ailing heart. As my fingertips continued to search, they also sensed fullness of the colon. Did he have constipation, I inquired through the student. No, the answer came back, not usually.

I was relieved. In my mental cinema, I had imagined single-celled squatters, this time ensconced in the nerve clusters that lace around the digestive tract. Parasite nests and inflammation located here produce "mega syndrome," Chagas' disease's strangest hallmark, seen in 6 to 10 percent of its victims. The gut enlarges because the nerves that control the rhythmic squeezing of peristalsis die. Such nerve damage can afflict other hollow organs too. Autopsies on mega patients play like bad horror flicks. With the knife's first stroke, the overstretched organ spills out like a membranous balloon. On top of heart disease, this was almost too cruel to contemplate. Thank goodness Miguel had been spared the indignity.

Autopsies can play like a bad horror flick. With the first cut, the overstretched organ spills out like a membranous balloon.

Now Miguel spoke up, and for a moment, we glimpsed his private storehouse of thoughts and fears.

"*Señora*, if you are right, could I give this infection to my family?"

I was relieved that on this point, at least, I could reassure him. Except for occasional cases of maternal-fetal transmission, intrafamilial spread of *T. cruzi* does not occur, except by the feces of reduviids. But blood donation is a different story. In Santiago, Chile, the prevalence of *T. cruzi*–positive blood is 2.6 percent, and in Buenos Aires, Argentina, 4.9 percent. These and other Latin American countries have made screening blood for Chagas' disease compulsory by law. Not so the United States. Admittedly, the prevalence of antibody in blood donors is lower here. But it was recently pegged at 1 in 8,800 blood units in both Los Angeles and Miami. Considering that anywhere from 13 to 23 percent of those antibody-positive units harbor enough organisms to transmit Chagas' disease, a lot of folks in my field find our lack of testing wholly unacceptable.

But pondering the transfusion issue was not our task. The time had come to leave Miguel's bedside and answer the fundamental question: Did he have Chagas' disease?

Our best bet for starters was the antibody test for *T. cruzi* performed at the Centers for Disease Control and Prevention in Atlanta—it is simple, clean, and fast. I couldn't imagine describing the alternative to Miguel. A few years earlier I had been one of several doctors huddled around a Peruvian woman about to undergo xenodiagnosis, once considered the gold standard for diagnosing chronic Chagas' disease. Understandably, the woman was queasy. Xenodiagnosis requires roughly three dozen hungry, laboratory-reared reduviid bugs to serve as biological incubators. I recalled the shades being drawn, the hospital lights dimmed. Then the insects were allowed to probe and siphon from our patient's forearms. After drinking their fill and defecating, the hostages were returned to their cages. Weeks later, their diminutive guts were dissected and searched for *T. cruzi* parasites.

I never had to discuss xenodiagnosis with Miguel. One week after drawing his blood, I heard from a technician in Atlanta. Miguel's serum was unequivocally positive. Then the technician posed a question. Since Miguel was from Mexico, a country thought by some to have less virulent strains of *T. cruzi*, how sick was he, really?

Everyone in medicine has strengths

and weaknesses. As a clinical specialist in tropical medicine, I do not pretend to know about regional differences in parasites. But I do know patients. No, Miguel would not die tomorrow. But, I added, a 40-year-old day laborer with end-stage Chagas' cardiomyopathy facing possible heart transplantation was, by any criterion, sick. After hanging up the phone, I imagined the conversation I would like to have had with Miguel.

My friend, it's as we thought: unknown to you, a migrant and his family have been living in your heart. Years ago, these creatures came looking for a home for their descendants, and in you they found the perfect place. The good news is this: we have a strong medicine. With a few doses, the trespassers will be evicted, and you'll be as good as new. Back to work, back to your wife, back to Sunday *menudo* with all the salt you like.

That was my dream. Sadly, it was far from reality. In truth, medical treatment had never been the issue. The cardiologists who asked me to see Miguel suspected Chagas' disease long before I appeared. They also knew there was no magic bullet for this stage of *T. cruzi* infection. There are drugs to treat Chagas' disease, but they are toxic and not very effective—and even less so when the infection has progressed. At this stage, the real question facing Miguel was heart transplantation. If Miguel had a transplant, he would need to take drugs to rein in his immune response and prevent rejection of his new heart. What would that do to the delicate balance of power between parasite and host? Would a transplant cause more harm than good?

I could tell him that patients with end-stage Chagas' disease had gotten new hearts—as of a few years ago, in Brazil there were 31 recipients, to be exact. But early results of transplantation had been dismal. Most patients died within a year from new colonies of *T. cruzi* sprouting not only in their new heart but in their skin, brain, and other organs. The drugs used to prevent organ rejection created an open playing field for parasite growth.

In the end, Miguel made his own decision. When he heard all the facts, he declined to be placed on the transplant list, and that was that. I didn't see him again. Months later, I heard he had died. He was one of more than 40,000 who lost their lives that year to a hidden intruder in the heart. D

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