CHAPTER ELEVEN

Reflex Sympathetic Dystrophy

Reflex sympathetic dystrophy (RSD) is the most dreadful of the painful diseases. In no other illness is the physician so called upon to do something – even if it is wrong.

A painful injury, usually in an extremity, incites an unusual reaction. It recruits the autonomic nervous system, that subcortical apparatus dedicated to the control of blood flow, into misbehavior. Blood vessels constrict. Muscles wither and contract. Bones becomes osteoporotic, and the skin glossy, atrophic, and cold. As with other states of chronic pain, sleep, appetite, and mood are all disordered, but the predominant and most visible effect is reduction in blood flow and with it extravagant pain. Why a painful injury should occasionally incite the autonomic system into dysfunction is problematic. It may have something to do with age and gender because reflex sympathetic dystrophy is usually a disease of young women.

Luz just graduated from high school, found employment in a grocery store. There only a short while, she slipped on a wet place and fell back against her extended arm, breaking her left ankle. The fracture was set in the emergency room. In a few days her foot began to swell. The orthopedist replaced the cast and noted that her pulse was bounding, but that her foot was cool and blanched.

Luz's fracture, even though immobilized by her cast, became progressively painful and kept her awake at night. She began to experience hyperpathic pain. Merely touching the tips of her toes was uncomfortable to her. X-rays showed satisfactory healing of the fracture, but when the cast was removed, the ankle and foot were cadaveric in appearance. Luz's pain was severe and incessant.
Nerve cells are resilient. They have the inherent capacity to reconfigure and change their function, a phenomenon known as neural plasticity. Old cells learn new tricks. Fortunately so, for this is the reason we recover from strokes and other neural injuries. Nature's flexibility, however, is not always adaptive. Sometimes it leads to the brain attacking the body, and this is what occurs in reflex sympathetic dystrophy. The capacity of the brain to actually wither an extremity challenges credulity. But it happens, and by a mechanism we have not yet begun to understand.

That segment of the autonomic nervous known as the sympathetic is housed in the brain stem, and axons from its cells descend and exit the spinal cord throughout its length to synapse (communicate) with other neurons in aggregates known as sympathetic ganglia. These lie adjacent to the vertebral column. Nerve fibers from these ganglia course in company with blood vessels throughout the body and control their reactivity. This, so far as we know, is their sole function. By virtue of their plasticity, however, they can, under provocation, reconfigure and actually generate pain.

Luz was treated with a sympathetic block. A needle was introduced into her neck and directed to the ganglia along the lumbar spine. An anesthetic was injected, denying sympathetic innervation and the power of vasoconstriction to her extremity. As predictably happens, her leg, formerly cold and moist, became warm and dry. Her pain diminished, but it reappeared, as did coldness, when the anesthetic wore off. The blocks were repeated several days running in an attempt to somehow arrest her disease. With each injection her leg would warm and her pain partially abate.

Encouraged by the response, her physicians elected to do a sympathectomy. The ganglia were surgically removed, and Luz's lower extremity was deprived of sympathetic innervation.