Editorial commentary

The central effects of peripheral injury

Oliver Sacks

I am indebted to Drs Stone, Perthen and Carson for their thoughtful consideration of my book A Leg to Stand On, although I do not agree that the experience was a functional or conversion phenomenon. This was, however, my own first thought when I discovered that the leg had gone ‘dead’ on me. I asked to be seen by the psychiatrist at the hospital, and he was struck by the co-occurrence of many factors, but he said that it did not resemble any hysteria he had come across. My surgeon spoke, vaguely, of ‘inhibition’ in the spinal cord, although he agreed that the distribution of sensory and motor loss did not follow any clean neuro-anatomical pattern. I too had observed this—in particular the flaccidity of some of my hip muscles and, in great contrast, the preserved and free movement of my foot, which felt very much part of me, albeit separated from my hip by a senseless, immobile and alienated leg, encased in its long, white cylindrical cast.

One of the orderlies at hospital told me that he had fractured both legs and part of his pelvis in a parachute accident, and had long casts on his legs, both of which seemed to him ‘dead’ or alienated. He gained much reassurance, during these weeks of immobility, from being able to touch his normal-feeling feet together. My friend Jonathan Miller told me that when he had been in medical school (we qualified at much the same time), one of his fellow students allowed himself to be put in a full body cast, an experiment suggested by a neurologist at the hospital. The young man, normally of a somewhat phlegmatic character, panicked when the cast was completed, and said he could no longer feel his body below the neck; he insisted that the cast be taken off. During my hospitalisation, the physiotherapist at the hospital remarked that she wished transparent casts were available to reduce the feeling of alienation.

I too was suspicious of the cast which prevented any movement or proprioceptive feedback from the knee and cut the leg off from sight and touch. And since body image is dependent on the collated information from proprioception, touch and (in the sighted) vision, the cast itself, I think, contributed to my difficulty in affirming the leg as mine.

Whatever the cast may have contributed to this sense of an alien leg was heightened by the damage to the quadriceps and its innervation, both sensory and motor. Recently I had a total knee replacement on the same side, and the regional anaesthetic used for this surgery paralysed the quadriceps and rendered it atonic for about 24 h. In contrast, with the injury I described in A Leg to Stand On, atonia of the quadriceps lasted about 14 days, and the muscle had mild persisting abnormalities for at least a year. During this period I found, for example, that if I swam for too long or too strenuously, the quadriceps would go atonic, so I had difficulty climbing out of the swimming pool—the leg would recover normal tone in an hour or so. I would also get sudden, flash-like proprioceptive illusions, feeling that the leg had moved to another position; I suspect there was some cross-wiring as the innervation healed.

There was, as Stone et al emphasise, some apparent spread of motor and sensory problems to the hip. But part of the quadriceps—the rectus femoris—is a hip flexor muscle, indeed the main one, so it is not surprising that it too was atonic, undercutting movement and proprioception at the hip. But even when there is no anatomical connection, it is common to see distant effects of a local injury. Even with a simple bony injury like a Colles fracture, there is apt to be ‘sparing’ of the arm and the shoulder. AR Luria, writing to me in 1974 about my leg experience, spoke of ‘the central effects of a peripheral injury.’

The sorts of complex perceptual and relational difficulties described in A Leg to Stand On are increasingly recognised as normal brain responses to peripheral injuries, as many pain neurologists and neurophysiologists have observed, and there is a large amount of literature on the subject.

Another factor, which I discussed in the 1990 afterword to my book, was not being allowed to stand for 14 days after my 1974 operation. It was only realised in the late 1970s that it is crucial to get limbs back into action as soon as the requisite surgery and splinting is done. Otherwise, their action-patterns may be (temporarily) lost. When I ruptured the quadriceps of my other leg, in 1984, I was stood up in the recovery room as soon as the spinal anaesthesia wore off. In this instance, there was no alienation or paralysis.

Taking so robust a neurological basis into consideration, there is no need to postulate a dissociative or functional disorder, although, of course, it is possible. I would be the last to deny this, and I think that there may have been elements of functional overlay superimposed on a very real neurological condition, the spinal and cerebral response to an injury involving the integrity and innervation of a major postural muscle like the quadriceps.

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