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'Duplicate Limbs' Sensation in Acute Traumatic Quadriplegia

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Summary

A 64-year-old man sustained acute quadriplegia due to a traffic accident, while in the midst of a petit mal seizure. After recovery from the initial medical complications he developed a duplicate limb phenomenon. The patient felt that another pair of upper and lower limbs had grown from his body, parallel to the paralysed limbs. To the best of our knowledge this duplicate phenomenon in all limbs has not been described before in a traumatic quadriplegic patient. It is our impression that this phenomenon is a rare example of preoccupation with the paralytic limbs, of sensory deprivation with a (possible) unusual drug reaction.
Key words: *Acute cervical spinal cord injury; Quadriplegia; Duplicative phenomenon; Sensory deprivation.*

Acute traumatic spinal cord injured patients (SCIP) may develop phantom sensations or phantom pain of the paralysed limbs. Sometimes an 'anaesthesia dolorosa' paradox appears among SCIP. Patients may express denial or neglect of the paralysed limbs, or even hatred ('misoplegia' as Macdonald Critchley put it), or preoccupation with them. Philoplegia or plegomania may sound somehow awkward words, but there is an old forgotten term for psychological preoccupation or 'idee fixe' which is 'hyperprosexia'. We describe a patient who developed a very peculiar and rare phenomenon: a duplication of all limbs. To the best of our knowledge, this is the first description of such a phenomenon in a quadriplegic patient. This phenomenon had been described in other patients to a much lesser degree.

Case report

A.G., a 64-year-old man, was involved in a car accident on 30 July 1986, resulting in an acute spinal cord injury below C5. He drove alone and crashed into a rock. For the last 5 years he is known to have had petit mal attacks once a month. He was treated

regularly with Tegretol (Carbamezapine) and Valporal (Sodium Valparate). For many years he suffered from low back pain (spondylosis) and in 1976 he had had a lumbar spine fusion.

On admission he was unconscious. Radiologically no fractures or dislocations were found in the spine, but CAT scan revealed small bone fragments in the cervical canal. The surgeon (in another hospital) decided (wrongly, in our opinion) to 'decompress' the C5 spinal canal by fusion. After the operation he regained full consciousness but could not breathe without the aid of a respirator. Neurologically he developed incomplete spastic quadriplegia below C5. During that phase he was very depressed, wished to die and was uncooperative. Complications included septic shock, pneumonia, leucopenia, pulmonary emboli, cachexia, peripheral edema and pressure sores. At that stage he was transferred to our unit. Slowly, we weaned him from the tracheal cannula, the indwelling catheter, and the intravenous drip. We could not begin with the rehabilitation programme because of his pressure sores, so he was confined to bed for a longer period.

Until the beginning of November the medications administered to him consisted of:

T. Baclofen (Lioresal): 10 mg × 3 daily
T. Tegretol: 100 mg × 3
T. Ditropan: 5 mg × 3, and occasionally Promethazine
T. Phenergan: 25 mg × 1

All the routine laboratory values were within normal limits (including renal and hepatic functions, and full blood count). His body temperature was 37·5°C. At this stage the patient developed a very peculiar and rare picture, although not resigned to the idea of being paralysed. His metabolic, respiratory and general condition were satisfactory. He generally expressed despair, depression and negativism, was very critical and bitter toward the staff, and constantly complained of pain (shoulder and limb spasms etc.). Then he experienced a vivid feeling of limb duplication: a pair of hands was situated parallel to the normal hands and two legs were also situated parallel to the real ones. He recognised the staff and relatives by names, was fully conscious, and experienced nightmares. We did not change his medication. Two months later he dreamed that another pair of upper limbs crossed his chest causing difficulties in breathing. After he woke up all the symptoms disappeared. This last phenomenon was later repeated a few times. EEG tracings showed no pathology. Arterial blood gases were also found within normal limits. A year after the injury the patient died from myocardial infarction at his home.

Discussion

We could not find any similar clinical description in the literature among SCIP. There are a few etiological possibilities for this phenomenon:

1. 'Status petit mal' (PM).
2. Side-effects of drugs; altered drug metabolism.
3. Wishful thinking or 'phantom-like' phenomenon.
4. Sequelae of head injury.
5. Sensory deprivation and cognitive malfunction after long hospitalisation.

Although the patient had suffered from rare PM attacks in the past, and had repeated normal EEG tracings, we could not attribute the duplicative phenomenon to a possible epileptic fit, neither blame any drug effect. Neuropsychologic examinations excluded any kinds of organic brain damage. Bizarre and mysterious sensory phenomena which occur in the traumatic SCIP are still a subject of intensive research. In patients with complete lesions we may assume that sometimes there is unawareness of the paralysed part as if it had been amputated (Bors, 1963) but, on the other hand, the centrally stored body image may remain intact.

In our experience, the anosognosic phenomenon in SCIP is quite rare. The

patients concentrate on the paralysed limbs. They are even preoccupied with them. There are paraplegic patients who are not only aware of the paralysed part but also 'feel' and know that 'these are fixed in a definite position of moving in a certain direction' (Ettlin *et al.*, 1980; Pichler, 1955). These authors had the impression that amputation illusions predominated in the early stage of spinal shock, and phantom illusions were common in the later 'hyperesthetic' phase. Ettlin *et al.* (1980), interviewed 37 SCIP and concluded that (1) the disturbances of body image do not depend upon past experience or upon personality, but are rather influenced by pain ('distraction, occupation or drugs'), (2) amputation illusion and phantom position illusion never succeed each other; half of the patients were unaware of the paralysed part and the others felt phantom limb sensations, (3) the position of the phantom limb is related to the position which the body had been in at the moment of spinal injury.

In their dreams and sometimes in their thoughts, the patients continue to walk and run. The change in the perception of their body image takes time and sometimes phantom pains demonstrate their inability to cope or to accept their disability. Bors (1963) claimed that phantom sensation in SCIP occurs in practically every patient. They are divided into two groups: surface and postural phantoms. It is quite clear that there is 'organically-based pain which is exacerbated by psychosocial stress which in turn increases the pain' (Richards *et al.*, 1980). Melzack and Loeser (1978) state: 'there is convincing physiological evidence to show that differentiation produces abnormal physiological activity in spinal cord cells deprived of input. The cells fire spontaneously in high frequency burst from adjacent structures, the abnormal firing may persist for 3 hours after a single brief triggering stimulus, and abnormal activity has been observed to persist for months'. In our patient, a persistent sensation of duplication of the four limbs occurred. In clinical neuropsychology we well know this phenomenon in 'status PM' migraine or in brain injured patients (Lishman, 1978). The reduplicative phenomenon or Capgras Syndrome is a very rare one: the patient recovering from a severe head injury believes that familiar persons have been replaced by doubles. The delusions of reduplication are often accompanied by impaired memory, right frontal damage, confusion and later on, euphoria or apathy (Alexander *et al.*, 1979). It is considered a form of denial of illness (Weinstein *et al.*, 1955), which also appears in schizophrenia, Alzheimer's disease, etc. (Mack, 1985).

It is quite clear that this last syndrome does not fit our case. In our opinion, our patient after a long hospitalisation period developed this illusion of limb duplication due to 'wishful thinking' or adaptation to quadriplegia, or a combination of sensory deprivation and the toxic side-effects of drugs. Both elements may change the cognitive functioning during the long hospitalisation.

Bors (1951) in his extensive review on the phantom limbs of patients with SCIP, expressed the fact that there was no telescoping effect or shrinkage of the phantom limb; dissociation of position occurred in a large minority and disappeared with time; some volitional movements of the phantom were reported by the patients.

An old man with tetraplegia, hospitalised a long time with altered nutrition and other physiological changes, may react more strongly to a certain dosage of medication, but it is hard to prove these assumptions.

Bors does not mention any symmetric duplicative phenomenon of all limbs. Davis (1975) mentioned 1 patient with complete C5 quadriplegia who felt he had an extra pair of legs which at times were tied up in knots. The symptoms were abolished following physiotherapy.

In our patient, the phenomenon reappeared even after 1 year post-injury, and did not respond to any physical, pharmacological or psychological treatments. Shelley (1792–1822) wrote: 'thy deep eyes, a double planet Gaze the wisest into madness . . .' but it was Melville (1851) who, unaware of the future psychologic implication of his words, successfully described this phenomenon: 'O nature and O soul of man! how far beyond all utterance are you linked analogies! not the smallest atom stirs or lives on matter but has its cunning duplicate in mind'.

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