PHANTOM LIMB IN PARAPLEGIC PATIENTS

REPORT OF TWO CASES AND AN ANALYSIS OF ITS MECHANISM*

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The persistence of established knowledge concerning the body schema after removal of a part or all of an extremity has been termed the phantom limb syndrome. Adherence to this definition arbitrarily excludes from the present discussion those individuals with phantoms associated with lesions at various levels of the nervous system, but in whom there has not been an amputation. Also omitted from consideration are the so-called phantom limbs of some psychotic patients.

Mitchell17,18 Leriche,13 and Livingston16 all recorded the presence of phantom limbs in over 90 per cent of all patients who had had an amputation. Pain is not a constant feature of this syndrome, as indicated in the report of Browder and Gallagher,5 who found it to be present in approximately 30 per cent of their patients. Furthermore, only 20 per cent of this group of patients, or 6 per cent of their total series, experienced pain of an enduring character. In spite of the common occurrence of the phantom syndrome little attention had been directed to its pathophysiology until the introduction of various surgical procedures designed to relieve the associated pain. Among the first surgical endeavors aimed at the relief of pain were resection of neuromata of the neural stumps and division of peripheral nerves.19 Subsequently, anterolateral4,7,23 and posterior cordotomies,2,3,20 sympathetic ganglionectomies,23 and ablations of a part of the parietal cortex5,9 were performed. Although some success was obtained with these procedures in certain instances, the results in general left much to be desired. The great difficulties encountered in dealing with this problem have been vividly illustrated by the report of Lhermitte and Puech.14 In a period of 18 years a patient with pain in a phantom limb was not relieved by a resection of a neuroma, paravertebral novocain block of sympathetic nerves to the part, posterior myelotomy, and finally resection of a part of a parietal lobe. In the past few years Riddoch,21 Stone,22 and Henderson and Smyth11 have attempted to establish the basic physiology of sensations of phantom limbs. The latter authors believed that the characteristics of a phantom part may be assigned to three levels of neural activity, namely "psychogenic, sensorimotor cortex,

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and peripheral nerve.” They introduced the term “natural phantom” to describe the persistence of a painless normal body pattern after amputation of a part, therefore setting apart this aspect of the problem from the various epiphenomena such as severe unrelenting spontaneous pain, distorted posture and involuntary spasmodic movements. The latter were considered to be related to “psychological” factors whereas the natural phantom was dependent on an intact sensorimotor cortex. More recently, Li and Elvidge have chronicled observations on a phantom limb in a paraplegic patient. Multiple procedures directed at the lower levels of both the somatic and vegetative nervous systems were of no avail, finally, in assaying the pathways of afferent impulses from the ghost part or in changing its character. Bors, also, has documented the existence of phantoms in paraplegic patients below the site of injury and has postulated a mechanism that requires further consideration.

It is because of the many and apparently contrary views related to certain aspects of phantom limbs as well as our interest in the pathophysiology of this phenomenon that the following examples of the phantom limb syndrome in paraplegic patients are presented.

CASE REPORTS

Case 1. E.G., a 24-year-old pharmacist mate in the United States Navy, was injured on Feb. 19, 1945 when a mortar shell exploded within 2 feet of him. Shrapnel entered his spinal canal at the levels of the 7th and 11th thoracic vertebrae, rendering him paraplegic.

Clinical examination at this time showed evidence of a complete physiologic interruption of the spinal cord at the 11th spinal cord segment. This level changed slightly during hospitalization in that the motor and sensory loss became absolute only in the lower extremity with recovery ensuing in the lower abdominal segments.

After recovery from the initial injuries he suffered from the usual sequelae associated with the paraplegic state, including paroxysmal spasm of the lower extremities. On May 7, 1948, both obturator nerves were crushed in an attempt to relieve these spasms. Since this did not entirely relieve the recurring contractions, part of the motor components of the cauda equina were divided on June 23, 1948. The anterior roots of lumbar nerves 2, 3, and 4 were sectioned under direct visualization at their point of emergence into the dural sleeves. The remainder of roots comprising the cauda equina were stimulated with a current of 5 volts and in those instances in which gross muscle contraction resulted, the stimulated root was sectioned. Thereafter, the lower extremities were flaccid and the spasms were abolished. On Nov. 8, 1949, a right mid-thigh amputation was performed because of persistence of a large infected decubitus ulcer of the knee.

On the first postoperative morning the patient first complained of a phantom limb. As with many patients with this syndrome he was, at first, unable to reconcile the objective absence of his limb with the fact that it felt as if it were still present. This conflict must have been particularly disturbing since he said, “This one (left lower extremity) is here and dead, but this one (amputated extremity) is gone, yet it feels alive.” From his description there was apparently a distinct sensation of viability in the phantom, and, subsequently, he explained that it felt as if he could