

Painful hand and moving finger treated by wearing a glove

Abstract—The authors report a patient with unilateral painful hand and moving finger in whom tactile stimulation interrupted both the movement and the pain. This effect suggests a gating mechanism at a segmental level. The difference between afferent and efferent pathway levels and the delay of several months between trauma and occurrence of symptoms support a central mechanism, most probably involving sensorimotor reorganization at a segmental level.

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Painful hand and moving fingers (PHMF) was first described in 1985 in a patient suffering from radiation-induced brachial plexopathy, with pain and movements of all fingers.¹ Since then, few cases have been reported, generally in patients with peripheral nerve, plexus, or root disease.^{2–4} Deep aching or pulling pain often precedes movements by several months. Involuntary movements are composed of complex sequences of flexion/extension and abduction/adduction, which cannot be imitated. Treatment is unsatisfactory. Similar symptoms occur in the inferior limb in the much more common syndrome of painful legs and moving toes (PLMT).^{5,6} We report a patient with unilateral PHMF after median nerve lesion in whom tactile stimulation in the same territory simultaneously interrupted both the movement and the pain.

Case report. A 55-year-old woman was referred to our center for pain and movement in the left hand. Two years before, she had suffered traumatic fracture of the left distal part of the radius bone. After conservative treatment, she developed continuous pain, hyperalgesia, and sudomotor changes and was diagnosed with complex regional pain syndrome (CRPS), treated by pamidronate and calcitonin for 2 years. She reported no abnormal movement. Nine months before our evaluation, she had median nerve surgical decompression of the carpal tunnel. Two months later, she developed involuntary movement of the third left finger, followed after another 2 months by sharp pain in the hand with third finger predominance. Upon notice that it would help both the pain and the movement, she started wearing a glove.

Physical examination showed continuous involuntary pseudorhythmic and rapid movements of the third left finger, at a frequency of 3 to 5 Hz, with predominance of flexion/extension but

with some abduction/adduction component that sometimes gave it a rotating aspect (see video, segment 1 on the *Neurology* Web site at www.neurology.org). The patient could not willingly stop the movement, nor would contralateral action either reduce or increase it. Tactile stimulation of the palmar aspect of the first to third fingers immediately, simultaneously, and completely stopped both the movement and the pain (video, segment 2), as would pinprick and fork application. In contrast, stimulation outside the median territory on the internal aspect of the hand or on the dorsal aspect of the fingers did not affect either the movement or the pain (video, segment 3). Apart from that, neurologic examination showed slight weakness of dorsal interosseous muscles, and minor sensory abnormalities over the anterior distal parts of fingers 2 and 3.

Nerve conduction studies of the motor and sensory radial, median, and ulnar nerves were within the normal range. Needle EMG showed continuous spontaneous bursts of activity in the extensor digitorum communis (EDC) and in the second dorsal interosseous (2IO) muscles (figure, A and B). Burst duration ranged from 50 to 260 msec, and frequency was 4 to 5 Hz (EDC) and 5 to 6 Hz (2IO). Tactile stimulation in the median territory rapidly interrupted burst activity (figure, C).

Cerebral and cervical spine MRI were unremarkable. Sensory-evoked potentials with stimulation of the median nerve were normal and symmetric. Back-averaging of somatosensory-evoked potentials using surface EMG as pre- and post-trigger (standard 10 to 20 montage EEG, 512-Hz sampling, high-frequency filter (HFF) = 256, low-frequency filter (LFF) = 0.3) did not show any electrical activity with temporal correlation with the movement by using dipole analysis, brain mapping, or phase or source diffusion.

fMRI was performed and first allowed to delineate the primary motor cortex (M1) activated during voluntary movement of the third finger (figure, D). This motor cortex was used as a region of interest in which rest activity (with involuntary movement) was defined as reference for comparison with other conditions. Using a cotton-wool stick, these comprised successive 1-minute periods of tactile stimulation of palmar aspects of fingers 2 (movement interruption) and 5 (no effect on movement), followed by voluntary movement (figure, E, conditions 1 to 3). No significant change compared to rest condition was induced by tactile stimulation of either the second or the fifth fingers (conditions 1 and 2), whereas voluntary movement induced a significant increase in activity (condition 3).

Treatment with gabapentin (900 mg three times daily, higher doses were not tolerated), amitriptyline (10 mg three times daily, stopped because the patient claimed that the pain worsened), and baclofen (50 mg/day) remained unsatisfactory. Geographic factors currently prevent further therapeutic trials, which should include higher doses of sodium channel blocker, anticonvulsants, and IV lidocaine infusion. The best treatment remains wearing a glove or holding a tissue in the hand to provide tactile input that suppresses both movement and pain.

Discussion. We report a patient with PHMF involving only one finger, which occurred 2 years after traumatic median nerve injury. The overall clinical picture matched previously published cases of PHMF;

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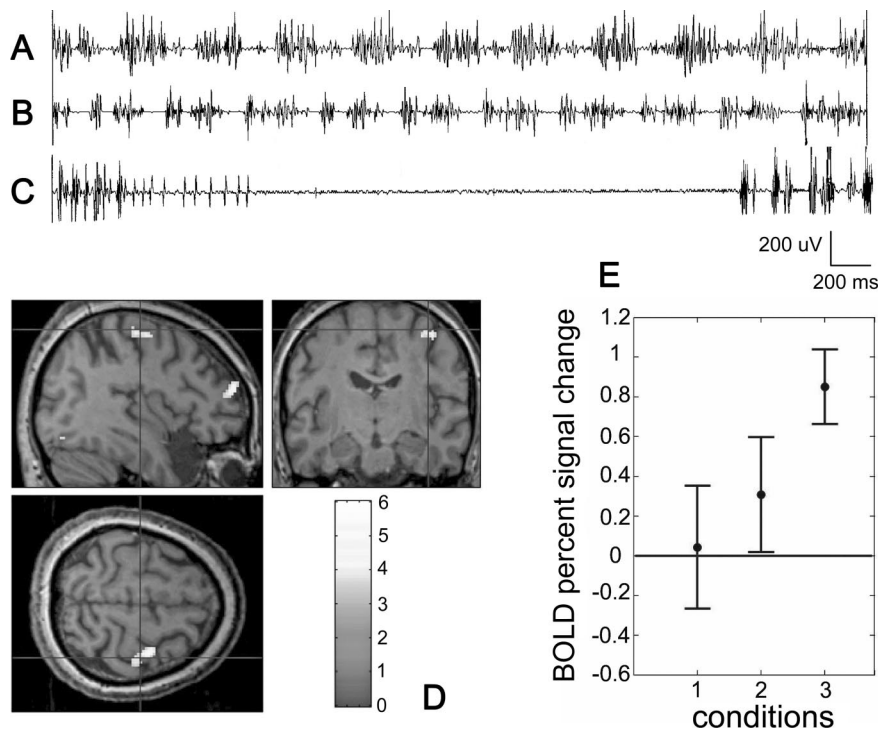


Figure. (A and B) Needle EMG in rest conditions showing continuous spontaneous burst activity in the extensor digitorum communis muscle (A) and in the second dorsal interosseus muscle (B). (C) Interruption of spontaneous activity upon tactile stimulation in the median nerve territory. (D) fMRI during voluntary flexion-extension movements of the left third finger, showing activity in the corresponding primary contralateral frontal cortex. (E) fMRI activity in the region of interest defined in (D), expressed as a percentage of change compared to rest conditions. Condition 1: movement stopped by tactile stimulation of second finger; condition 2: tactile stimulation of fifth finger, not influencing the spontaneous movement; condition 3: voluntary movement (see text for comment).

however, in our patient, both movement and pain could be immediately suppressed by tactile stimulation in the median nerve territory.

Marsden et al.⁷ reported abnormal movement modified by sensory stimulation of muscle spasms and tremor and CRPS. In their patient, an oscillation at 7 Hz could be induced by the application of a fork over the thenar eminence (their case 1). However, this effect was not observed after skin anesthesia, suggesting that some symptom-modifying impulses were conveyed by skin sensation. Also, cases of painless limbs and moving extremities (PlessLME) were described,^{6,8} one of them with a striking suppressive effect of tactile stimulation.⁹ These clinical entities could be viewed as different manifestations of a symptom spectrum, from CRPS to PlessLME, with PHMF and PLMT in between.⁶ Our patient further supports this hypothesis, illustrating the intimate relationship between pain and involuntary movement, through their simultaneous suppression by the same stimulus.

A mechanism involving sensorimotor reorganization secondary to disturbed sensory input may cause PHMF, but also CRPS, Déjerine-Roussy syndrome, and spinal myoclonus and focal dystonia and was long ago evoked as an explanation for involuntary movement after peripheral lesions.¹⁰ It gained support from experimental data that showed changes in the pattern of neuronal activation after peripheral nerve injury to happen anywhere along the sensory pathways, from the dorsal horn to the somatosensory cortex. However, other authors have advocated supraspinal mechanisms⁴ or the combination of both central and peripheral lesions.²

In our patient, a central mechanism is strongly supported by the different levels of afferent (median nerve, roots C6 and C7) and efferent (radial and ulnar nerves, roots C7 to T1) pathways, along with the delay of several months between trauma and occurrence of symptoms. Sensory-evoked potentials, back-averaging, cerebral MRI, and, above all, fMRI speak against a movement generator situated above the spinal cord. In particular, a cortical origin seems improbable, as one would have expected a reduction of activity in the third finger motor cortex region of interest upon tactile stimulation of the second finger (which interrupted the movement) compared with rest condition, when the involuntary movement was present.

This therefore suggests that the critical events leading to pain and involuntary movements probably took place in the spinal cord and were induced by sensory input alterations due to peripheral nerve damage of traumatic or surgical origin. The clinical picture may have been favored by previous CRPS, in which abnormal movements are not uncommon. The inhibitory effect of the sensory stimulus on both pain and movement could be viewed as a gating mechanism, tactile information acting on neuronal networks at a spinal cord level, inhibiting the transmission of pain to upper centers, and acting on interneurons implicated in the genesis of the involuntary movement.

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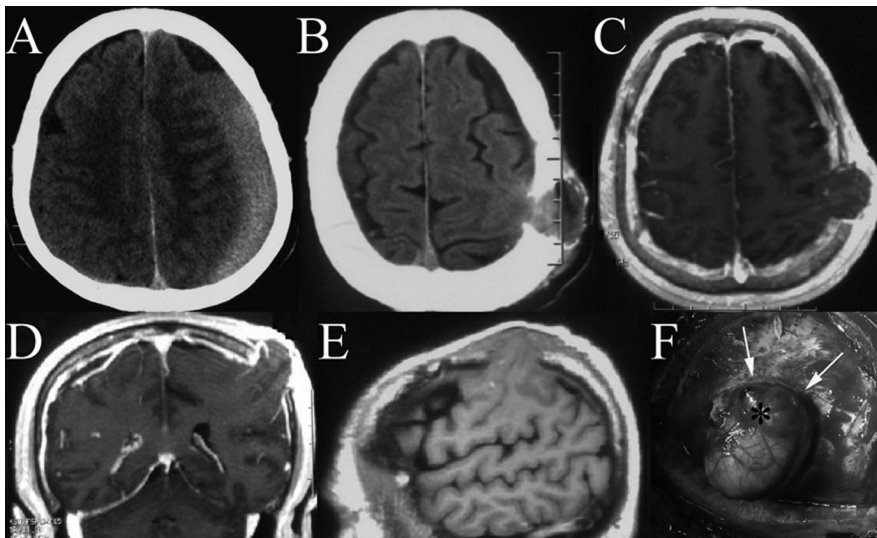


Figure. (A) CT scan demonstrating the left subdural hematoma, which was treated at another institution, with a small craniectomy and no dural closure. The patient had presented with right hemiparesis which had almost completely resolved postoperatively. (B–E) CT scan (B) and MRI examination (C, D: axial and coronal T1-weighted post-contrast; E: sagittal T1-weighted) documenting the cerebral herniation through the craniectomy: the herniation was in the region of the left motor cortex. (F) Intraoperative finding after enlargement of the previous craniectomy: the dura had not been sutured in the previous craniectomy (arrows pointing to dural edges) and brain had herniated through the opening (asterisk).

isk). A possible explanation of the cerebral herniation is local brain edema due to the intervention or drainage removal, causing a minor herniation, which worsened due to the subsequent venous congestion.

Transcranial cerebral herniation after chronic subdural hematoma treatment with no dura closure

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Ten days after surgical evacuation of a left hemisphere chronic subdural hematoma (figure, A), a 57-year-old man presented with sudden right arm monoparesis and dysarthria, associated with

local scalp swelling. Neuroradiologic examination documented a cerebral herniation through the previous craniectomy (figure, B through E). Urgent enlargement of the craniectomy and duroplasty were performed (figure, F). The patient's condition improved dramatically, though fine movements of the hand were still impaired at 6-month follow-up.

Craniectomy and no dural closure have been suggested as treatment for chronic subdural hematoma.^{1,2} Local cerebral edema is possible after hematoma surgical evacuation and cerebral herniation should be considered a possible complication if the dura is not closed when a craniectomy is performed.

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