Writing Tremor After Discrete Cortical Infarction

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Background: Hand tremor is a rare manifestation of stroke, and writing tremor has not been reported to be produced by stroke. We describe a patient who developed unilateral hand tremor mimicking primary writing tremor after discrete cerebral cortical infarction.

Case Description: A 67-year-old man developed mild right hemiparesis. Brain magnetic resonance imaging showed a discrete cortical infarct in the left frontal area. After recovery of motor power, the patient showed significant right hand tremor exclusively during writing or similar motor activities including tooth brushing or shaving. The tremor was temporarily alleviated by clonazepam but persisted until 7 months of follow-up. Medical history suggested that the patient had mild essential tremor, but he did not experience tremor on writing before the onset of stroke.

Conclusions: This observation suggests that unilateral hand tremor mimicking primary writing tremor may be produced by cortical infarction. It remains unclear whether the patient's previous essential tremor played an additional role in the development of this symptom. (Stroke. 1994;25:2280-2282.)

Key Words: cerebral infarction • tremor
mg three times a day relieved the tremor markedly, although mild WT was still observed (Fig 2). However, he complained of somnolence and took medication irregularly. The WT persisted, although slightly diminished in severity, at 7 months of follow-up.

**Discussion**

Primary WT is a task-specific tremor mainly induced by the act of writing or similar motor activities. Our patient developed unilateral hand tremor most markedly during writing but also during active movements involving pronation of the right forearm. During writing, jerky, myoclonic hand movements, symptoms commonly seen in patients with primary WT, were occasionally added. However, dystonic hand posture was never observed in this patient. Therefore, the characteristics of the patient's tremor are similar to those of primary WT.

**Fig 1.** T2-weighted magnetic resonance image shows discrete cortical infarct in left frontal area (arrowhead).

Despite occasional unilateral clinical presentations, most of the reported patients with WT did not have any cerebral lesions that were responsible for the tremor, although a few patients had a seemingly unrelated history of meningitis or perinatal hypoxia. Our patient is the first who developed unilateral WT after cerebral infarction. Although there have been a few reports of stroke-related tremor, these were delayed-onset resting or postural hand tremors caused by basal ganglionic or thalamic infarcts. Our patient was unique in that he presented with task-specific tremor after an infarct in the cortical area. The cause of the infarction in this patient remains unclear, although embolism was suggested as a possible pathogenetic mechanism.

The pathogenetic mechanism of primary WT remains obscure, and that of WT shown in this patient also remains speculative. Rothwell et al found that WT was temporarily abolished by partial motor point anesthesia of pronator teres in their patient and suggested that abnormal responses to muscle spindle input from pronator teres may be responsible for primary WT. In our patient unilateral hand tremor was noted during tooth brushing soon after the patient's motor weakness improved. Unfortunately, writing was not tested at that time, and we do not know the exact time interval between the onset of stroke and development of WT. Brain magnetic resonance imaging showed that the lesion was situated on the frontal cortex between the central and precentral sulci, a portion of Brodmann's area 4 that probably represents hand area. According to previous experiments with monkeys, there are substantial interconnections and feedback systems between sensory and motor areas in the sensorimotor cortex. Group 1 muscle afferents reach to area 3a in advance of similar sensory evoked responses in area 4, and perturbations of muscle spindle discharge during the performance of learned movements in conscious monkeys affect the neurons of the motor cortex. Jones et al even suggested that area 3a may be a part of area 4. Therefore, it may be speculated that the discrete lesion in our patient may have interrupted or reset the spindle impulses to area 3a or 4, and consequent alterations of motor discharges might have manifested as WT in our patient. Alternatively, the cortical lesion in this patient might have affected cerebellothalamocortical or frontopontocerebellar circuits. However, this possibility appears unlikely since our patient did not show ataxia in the right extremity.

Considering the patient's history and examination, it appears that this patient had essential tremor before the onset of stroke. However, the patient's bilateral postural hand tremor was very mild, and the unilateral, coarse hand tremor developed after the stroke was clearly task specific. Therefore, the patient's WT should not be a simple exaggeration of the previous essential tremor. However, it remains possible that the cortical infarct may somehow have altered the characteristics of essential tremor, thus producing WT in the right hand.

**Fig 2.** Handwriting of the patient before (A) and after (B) clonazepam medication. Upper rows, Normal writing; lower rows, fast writing.
References
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