

Thalamic tremor as onset of vascular parkinsonism

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Introduction

Thalamus is the main output of the basal ganglia and it's implicated in the genesis of various abnormal involuntary movements, including cerebellar signs, tremor, dystonia, asterixis, myoclonic jerks, and myoclonic dystonia. In particular tremor, myoclonus and asterixis have been related to dysfunction of posterolateral nucleus.

Case Report:

The patient is a 75 years-old diabetic and hypertensive man who presented to our center with a history of 2 years of tremor on his left hand. He reported that his tremor starts 1 year after an ischemic stroke, which involved right middle cerebral artery and which begun with left hemiparesis. The neurological examination showed a left resting myoclonic jerky which became a rhythmic tremor associated with myoclonic jerky with posture and goal-directed movements on his left hand. Extraparallel signs are mild (walking with short shuffling steps, decreased left arm swing, mild rigidity on his left elbow, slowness of left hand movement). We started diagnostic iter with electromyographic recording of tremor that showed a synchronous activation pattern of flexor and extensor muscles of his left forearm with myoclonus jerky; then, somatosensory evoked potentials and recovery cycle of blink reflex which were normal; DAT-SPECT demonstrated normal striatal binding and brain MRI showed vascular leukoencephalopathy with right **lacunar posterolateral thalamic infarction**. Diagnosis of vascular parkinsonism was made.

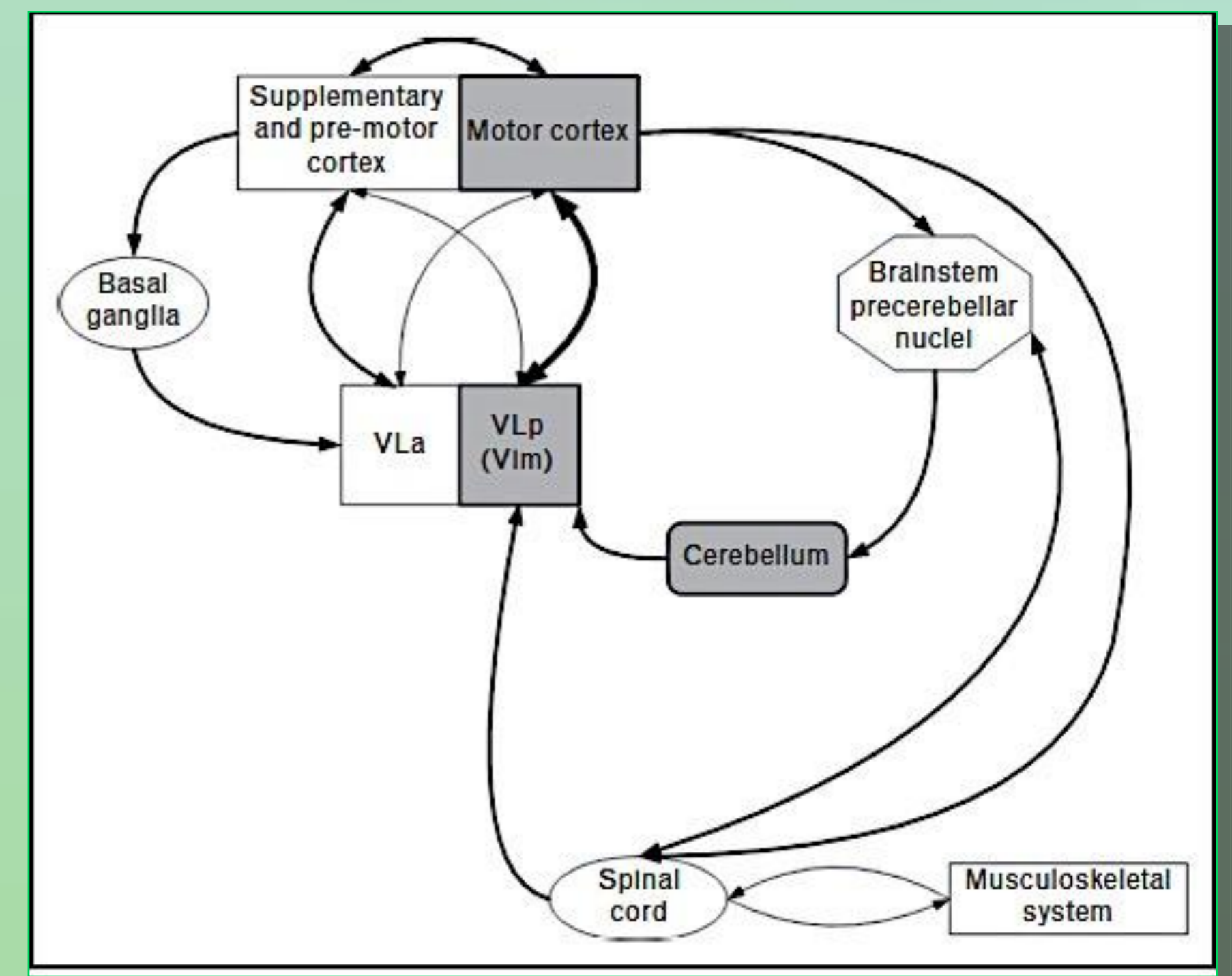


Fig. A: Principal anatomical pathways involved in tremorogenesis

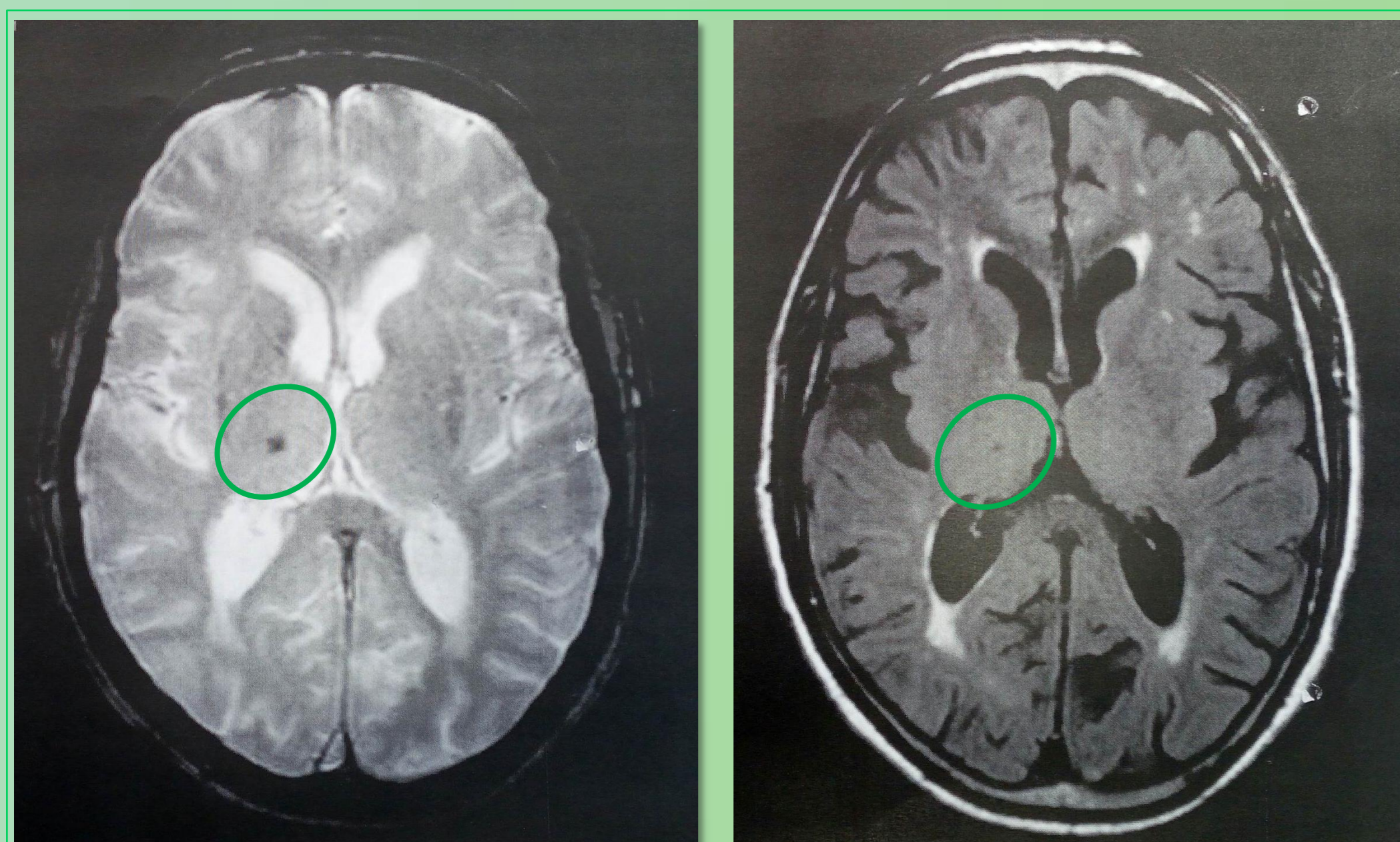


Fig. B: RMI T2W-emosiderina

Fig. C: RMI FLAIR

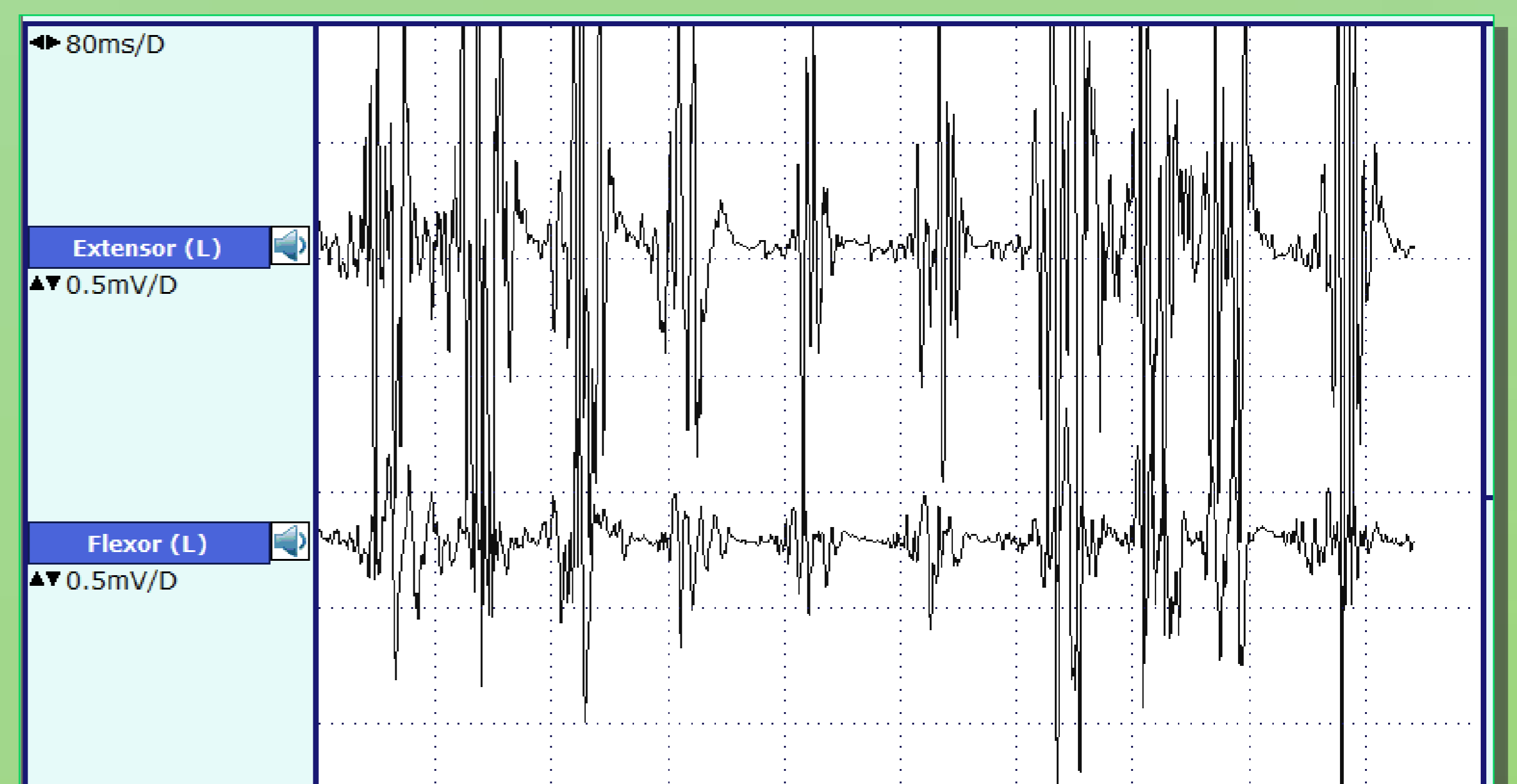


Fig. C: electromyographic recording of tremor that showed a synchronous activation pattern of flexor and extensor muscles of his left forearm with myoclonus jerky

Discussion:

According to literature, these irregular burst, with fluctuations between an alternating and a synchronous pattern in agonist/antagonist muscles, present at rest, but especially increased by posture and goal-directed movements, are described as irregular jerky tremor. Furthermore, evidences about synchronous activation pattern of antagonistic and agonist muscles of tremor associated with DAT-SPECT normal striatal binding can help clinicians to exclude Parkinson's disease. Our patient shows all these features and also a particular type of tremor myoclonic, observed in patients with thalamic posterolateral or posterior lesion. These lesions are responsible of damage of cerebello-thalamus-cortical pathway. For this reason, focal thalamic lesion should be considered in differential diagnosis when a patient shows asymmetrical tremor myoclonic.

References:

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