



Case Report

Finger snapping during seizures

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ABSTRACT

We describe two patients who showed snapping of the right hand fingers during invasive intracranial EEG evaluation for epilepsy surgery. We correlated the EEG changes with the finger-snapping movements in both patients to determine the underlying pathophysiology of this phenomenon. At the time of finger snapping, EEG spread from the supplementary motor area towards the temporal region was seen, suggesting involvement of these sites.

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1. Introduction

Surgical intervention in epilepsy is a widely accepted, effective therapy in patients with pharmacoresistant partial epilepsy. Seizure semiology correlates with the seizure origin, spread, and release phenomenon and can help in the estimation of the anatomical brain localization [1]. Recognition of seizure semiology is important for correct diagnosis and surgical treatment of epilepsy.

Finger snapping has been noted as a common act for centuries and is employed in securing someone else's attention. The sound of the snap is created by forcing air out between the fingers with an audible crack. Automatisms involving the distal segments of the upper or lower limbs are frequent during seizures [1] and, when unilateral, often indicate ipsilateral seizure onset, mostly in the temporal lobe or in the orbitofrontal region [1,2]. Finger snapping could be interpreted as a temporal lobe automatism, but the fierceness of the finger-snapping movement associated with tonic abduction of the upper limb could indicate a focus in the supplementary motor area (SMA) [3,4]. We describe two patients in whom invasive intracranial EEG evaluation was performed to localize the seizure onset for surgical intervention. Both patients showed ictal snapping of the right hand fingers, which has not previously been described in literature.

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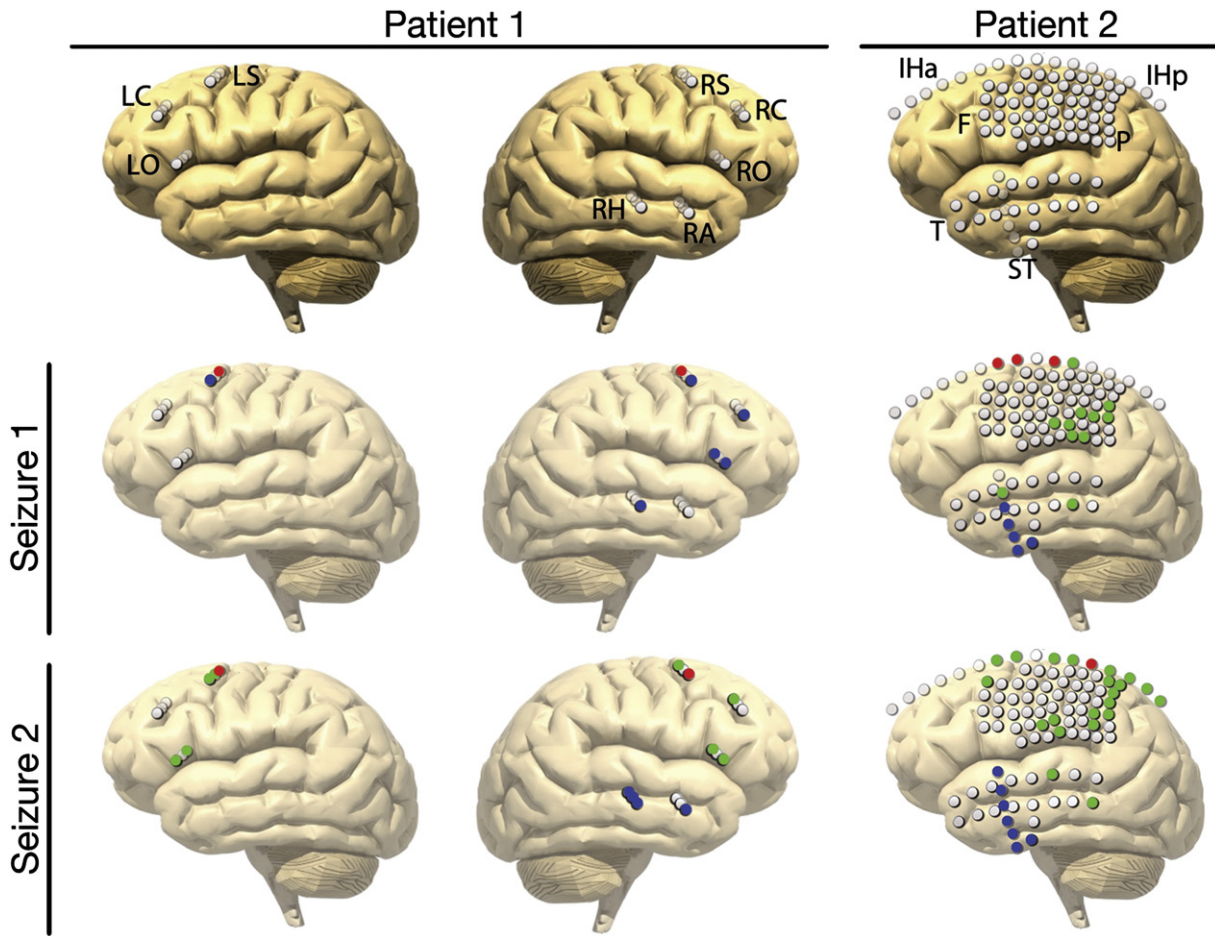
2. Case reports

2.1. Patient 1

A 34-year-old right-handed man was seen at the Montreal Neurological Hospital for investigation of refractory seizures. His seizures began at the age of seven, two years after a right-sided head trauma. A maternal great uncle had unspecified epilepsy. Early EEGs revealed an active epileptiform anomaly over the right midfrontal region. Over the years, he showed fluctuation in frequency and severity of seizures, which eventually remained refractory. 1.5-Tesla MRI showed no structural abnormalities.

During admission for video-EEG, typical clinical seizures occurred predominantly during sleep, without aura, and were defined as tonic posturing with fencing, extension of the right arm, snapping of the right hand fingers, and some pedaling movement. Electroencephalography telemetry showed several seizures not only with bilateral background activity changes, such as generalized epileptiform anomalies, but also with focal attenuation over the right frontal or temporal regions.

For nine days, intracranial stereo-EEG exploration with 11-contact depth electrodes was performed, exploring both frontal lobes and the right temporal lobe (Fig. 1). Active interictal epileptiform discharges were seen in the right hippocampal formation, and an independent, less active interictal epileptiform anomaly was found in the mesial aspect of the right orbitofrontal lobe. Twenty-five seizures were recorded, mostly nocturnal, with a semiology typical for this patient's seizure



Patient I (SEEG - depth electrodes)	Patient II (subdural grids)
LS = Left SMA LC = Left cingulate gyrus LO = Left orbito-frontal RS = Right SMA RC = Right cingulate gyrus RO = Right orbito-frontal RA = Right amygdala RH = Right hippocampus	IHa = Interhemispheric anterior IHp = Interhemispheric posterior F = Frontal P = Parietal T = Temporal ST = Subtemporal
● = Gamma onset ● = First spread ● = Activity during typical Finger Snapping	

Fig. 1. Schematic figures representing the seizure onset and spread.

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