

# Metabolic Imaging Correlate of Truncal Onset Seizures

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**Objective:** To denote the metabolic imaging localization of seizure onset with ictal and interictal Neurolite single-photon emission computed tomographic scans and difference images made by subtracting activity at individual voxels of these images, which ultimately identify intense focal uptake in the trunk motor region and other, probably secondarily, propagated pathways.

**Design:** Retrospective review of a unique case report.

**Setting:** A suburban epilepsy referral center.

**Patient:** A 59-year-old man with truncal-onset seizure.

**Interventions:** Ictal and interictal metabolic imaging.

**Main Outcome Measures:** Onset location of ictal events.

**Results:** The patient had a gliotic lesion in a focal region on magnetic resonance imaging undercutting the trunk motor area in the cortex of the precentral gyrus with concordant single-photon emission computed tomographic imaging.

**Conclusions:** While truncal-onset seizures have been described previously in a few case reports, they are clinically rare. As far as we know, this is the only case report in the literature in which metabolic imaging was carried out with this entity and is consistent with the anatomical localization of seizure onset in the trunk motor area.

*Arch Neurol.* 2011;68(2):251-253

## REPORT OF A CASE

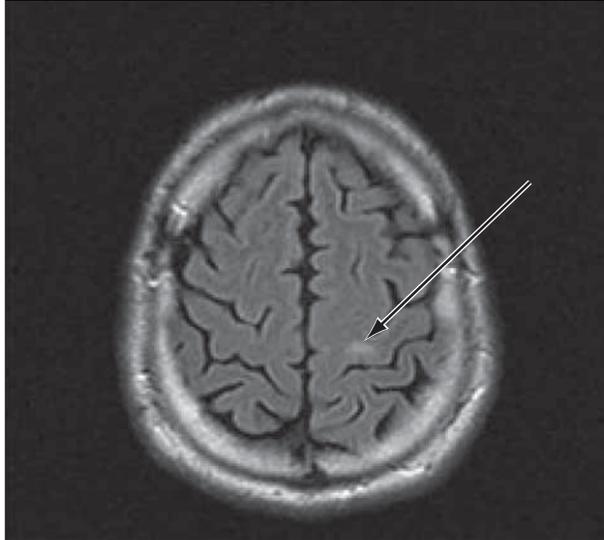
A 59-year-old man with history of atrial fibrillation, radiculopathy, mild anxiety, depression, and no family history of seizures or any preceding neurological illness developed abnormal involuntary nocturnal movements that started as a spasm in the right upper and lower quadrants with fleeting, painful cramps and muscular contractions. These occurred at short intervals of approximately 30 to 60 seconds. The patient initially had clonic contractions of the abdomen and torso and could become tilted in such a way that he might lean paroxysmally toward the right; subsequently, his arm and leg would have clonic contractions. The patient was generally alert, although he could not control the noted movements; taking benzodiazepines at night helped control the movements. During the past 6 months, the episodes had become more intense and frequent. The patient had only rare episodes of secondary generalization resulting in generalized tonic clonic seizures. There

were no reports of status epilepticus or other varied clinical manifestations.

Magnetic resonance imaging illustrated a gliotic area in the precentral gyrus of unclear etiology. We suspect the lesion could be a cortical dysplasia or a remote small embolic stroke from atrial fibrillation. The patient was admitted for video electroencephalogram (EEG) telemetry to verify the semiology of the episodes and to determine whether there were any EEG correlates. During admission, the patient had approximately 15 events, and the EEG record was obscured by muscle and movement artifacts during nearly all of them; there were no other notable focal findings on EEG. During the seizures, the video was not sensitive enough to reveal the initial clonic contractions that the patient described but the remaining semiology described by the patient was verified. The incidents were, on average, just less than 1 minute in duration. An ictal and interictal Neurolite single-photon emission computed tomographic scan series was carried out. Subsequently, a differ-

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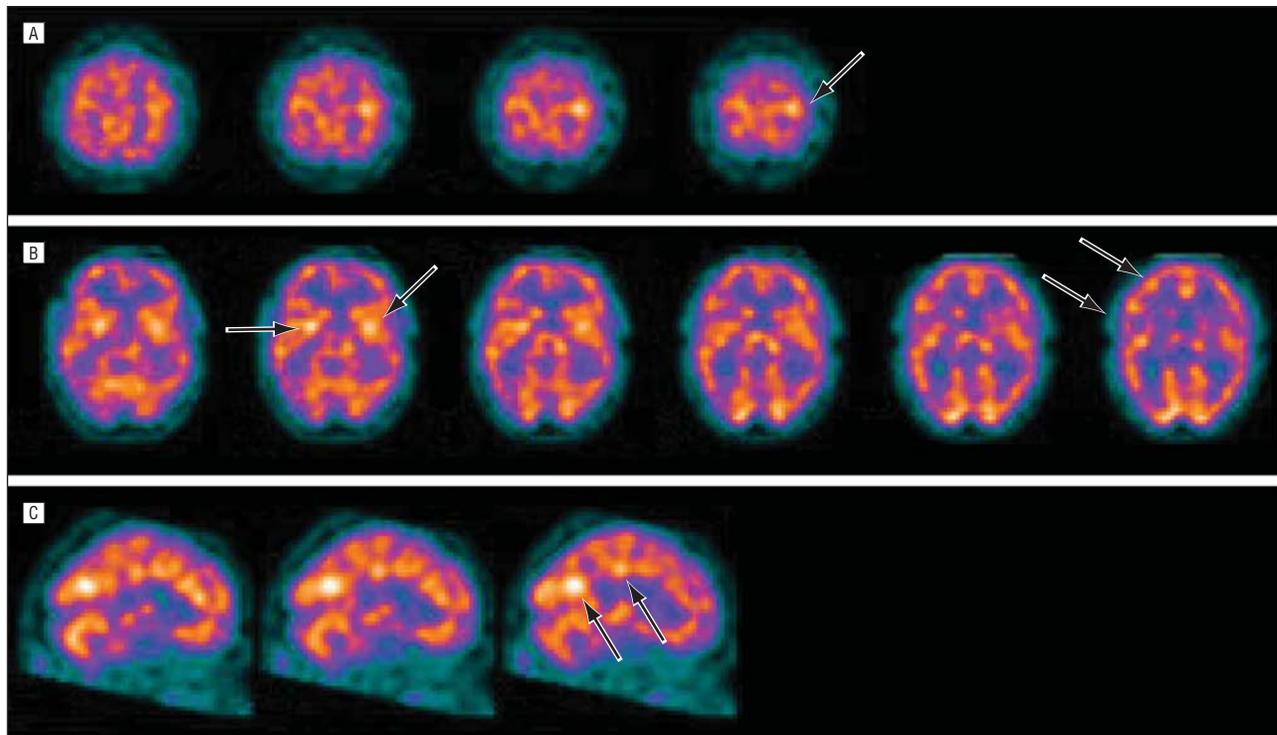
ence image made by subtracting quantitative activity from voxel to voxel was carried out after acquisition of the ictal and interictal images. Ictal injection occurred within 15 seconds into the seizure. The results of these studies are shown in **Figure 1**, **Figure 2**, **Figure 3**, and **Figure 4**. The figures illustrate a gliotic region in the trunk motor area and corresponding hypermetabolism that localizes to that region at seizure onset.



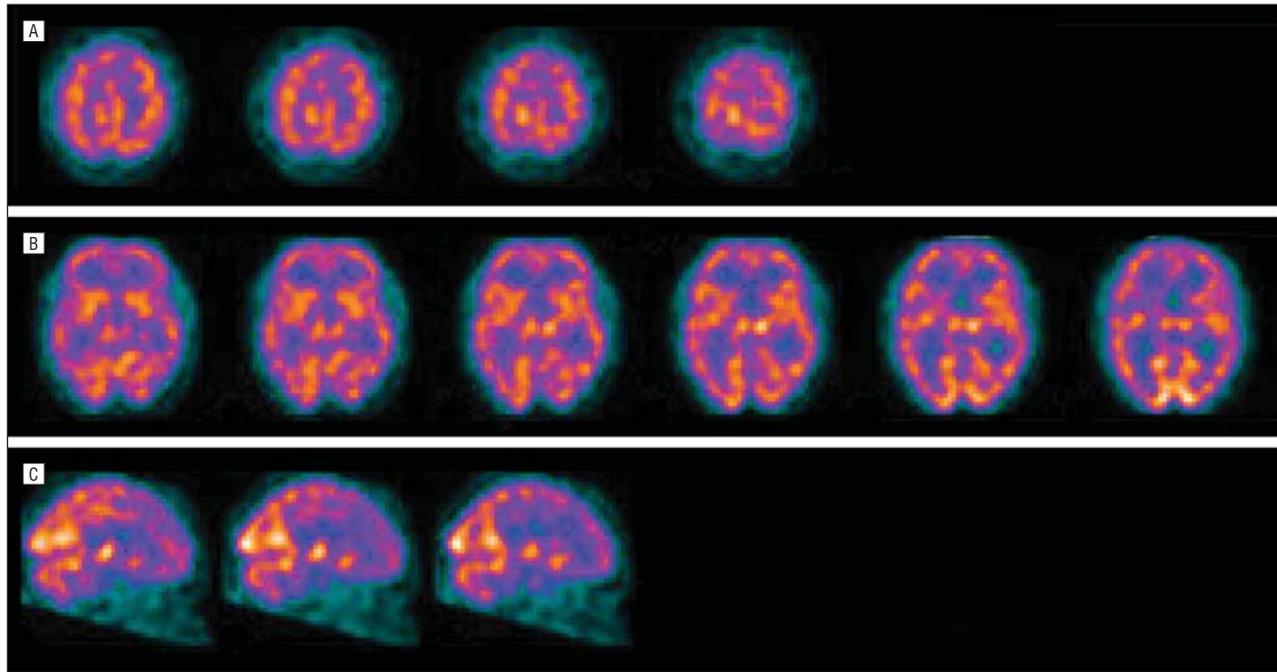
**Figure 1.** Axial fluid-attenuated inversion recovery magnetic resonance imaging illustrating increased signal in the frontal white matter undercuttering the precentral gyrus (arrow). The lesion did not enhance with contrast, and no other lesions were identified.

In 1902, Jackson and Singer<sup>1</sup> postulated that the origin of truncal seizures is localized within the brainstem. The patients they described had status epilepticus; after admission, they developed bilateral seizures of the face and abdomen with loss of consciousness. Treiman<sup>2</sup> explained these phenomena as a subtle seizure subtype occurring within focal status epilepticus. Four patients described by Nathanson et al<sup>3</sup> also had bilateral axial seizures that included the face, palate, tongue, pharynx, and abdomen. Those patients were deeply comatose, with evidence of diffuse brain injury. Findings of EEG showed burst suppression. Nathanson and colleagues believed that the seizure originated in the brainstem, although proof was lacking. The clinical phenomena were consistent with late-stage status epilepticus, as were the EEG findings in that series. Matsuo<sup>4</sup> described unilateral truncal seizures in 3 patients. In 2, computed tomographic scans showed contralateral parietal lesions, but no ictal EEG recordings were available. The third had 2 seizures without electrical accompaniment during EEG, but mild post-ictal contralateral slowing was evident. Their findings suggest that the published EEG segments demonstrate phase reversal between superior frontal and central electrodes or are nonspecific.

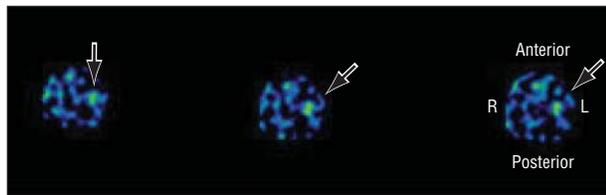
In 1990 Rosenbaum and Rowan<sup>5</sup> described a 65-year-old patient who was admitted for left leg weakness and a draining cystic lesion on the right side of the scalp. One day after admission, the patient developed left-sided seizure that involved the face, arm, and abdominal muscles.



**Figure 2.** Neurolite ictal single-photon emission computed tomographic study. A, Intense uptake is seen in the left superior lateral frontal lobe in coronal sections. B, Other areas of moderate uptake are shown in the right anterior frontal lobe and temporal lobes in axial sections. C, Uptake is shown in the cingulate region with sagittal sections to the left of the midline plane. We speculate that the primary focus of seizure onset occurs in the left motor cortex, which correlates with the intense focal uptake in the left motor area. The areas with the moderate uptake might represent secondarily propagated pathways. Arrows indicate regions of hypermetabolism.



**Figure 3.** Neurolite interictal single-photon emission computed tomographic study showing baseline metabolic uptake in phases with no seizures.



**Figure 4.** Neurolite axial single-photon emission computed tomographic difference images of Figures 2 and 3. Orientation is denoted on the figure. The arrows indicate the location of the greatest difference of the quantitative metabolic activity at individual voxels on these 3 successive axial sections correlating with the lesion seen in Figure 1.

A computed tomographic scan showed a 5-cm parasagittal mass on the right side destroying the parietal bone and compressing the underlying brain. The precentral gyrus was displaced posteriorly, and the brain exhibited extensive frontoparietal edema. The EEG showed continuous quasiperiodic bursts of high-voltage delta- and sharp-wave complexes of 1.5 to 2.5 seconds originating from the right centroparietal area.

These are the only articles and cases mentioned in the current literature that delineate truncal-onset seizures. We speculate this literature is sparse because partial-onset seizures that occur in the trunk muscles are rare.<sup>3,4</sup> The threshold of the truncal area is postulated to be high, and therefore may not be seen with seizure activity; this area is also very small.<sup>4,6</sup> Most cases were not identified until postmortem examination, and there are no other cases in the literature with concomitant metabolic imaging such as ictal and interictal single-photon emission computed tomography as in our case.

While our case identifies a focal intense uptake in the cortex of the left motor region, other less intense regions are also involved. Previously, it was observed that seizures in this region either propagate rapidly or could

be due to the bilaterality of corticospinal connections rather than activation of the contralateral or distant sites.<sup>4,6</sup> We speculate that the other regions noted on single-photon emission computed tomographic imaging could be reflective of secondary rapid propagation into the right frontal, cingulate, and bitemporal areas. We suggest that further observation and research is needed.

**Accepted for Publication:** May 21, 2010.

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**Author Contributions:** *Study concept and design:* Oster and Cosgrove. *Acquisition of data:* Oster and Aljumairi. *Analysis and interpretation of data:* Oster, Aljumairi, and Cosgrove. *Drafting of the manuscript:* Oster and Aljumairi. *Critical revision of the manuscript for important intellectual content:* Oster and Cosgrove. *Administrative, technical, and material support:* Oster and Aljumairi. *Study supervision:* Oster and Cosgrove.

**Financial Disclosure:** None reported.

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