



CASE REPORT

# Applications of Intraoperative Ultrasound in Epilepsy Surgery for Focal Cortical Dysplasia



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The definite margins of focal cortical dysplasias (FCDs) are usually not clear, and all epileptic surgeons have tried to identify them by advanced navigation, fusing magnetic resonance imaging and positron emission tomography, intraoperative magnetic resonance imaging, and intraoperative mapping in past decades. In this report, we present a convenient tool to define the boundary of FCD and improve the surgical outcome. The safe and effective method of image guidance by intraoperative ultrasound was demonstrated, and is time-saving and of a high resolution. In the future, intraoperative ultrasound will be useful in defining subtle epileptogenic lesions, especially when the surgeons encounter FCDs.

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## Introduction

Focal cortical dysplasia (FCD) is a congenital malformation, in which the neurons in certain parts of the cortex of the brain failed to migrate in proper formation. FCD is a common cause of intractable seizure in children and a frequent

cause of epilepsy in adults. Neurosurgical resection is the only choice of treatment for an effective cure. Early surgical intervention is also suggested nowadays because the longer seizure attacks, the more medications the patients take, and the lower intelligence performance. To achieve a good seizure outcome, the extent of resection should be as complete as possible. In recent years, intraoperative tools, including image navigation systems with multiple fusion strategies, intraoperative magnetic resonance image (MRI), and electrocorticography (ECoG), have facilitated complete resection of FCD. However, these tools have their own respective disadvantages.

With its higher resolution and real-time images, intraoperative ultrasound (IUS) has introduced a new vision of

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FCDs to neurosurgeons. Patients with FCDs can be treated effectively and safely, even if these FCDs are located near eloquent areas. Herein, we report this novel technique of IUS to define the margin of a FCD; this is the first FCD type IIa case using IUS in the literature.

## Case report

An 18-year-old male presented with medically intractable seizures since he was age 8 months. The seizure pattern was stereotypic: a bilateral limbs tonic posture with cyanotic lips for 20–30 seconds and a lapse of consciousness during sleep. Because of the frequent attacks (2–3 times/day) and use of multiple anticonvulsants, mental retardation, and language impairment caused dysfunction in the patient's daily life. Although a blurred lesion was seen on the right frontal opercular and premotor gyrus (Fig. 1), callosotomy was performed when he was age 10 years due to diffuse spikes on bilateral hemispheres. After callosotomy, the frequency of the attacks decreased, but focal spikes became more concentrated on the right opercular lesion. In the most recent year, progressive left-side weakness and dysarthria forced the family and physicians to ask for surgical intervention.

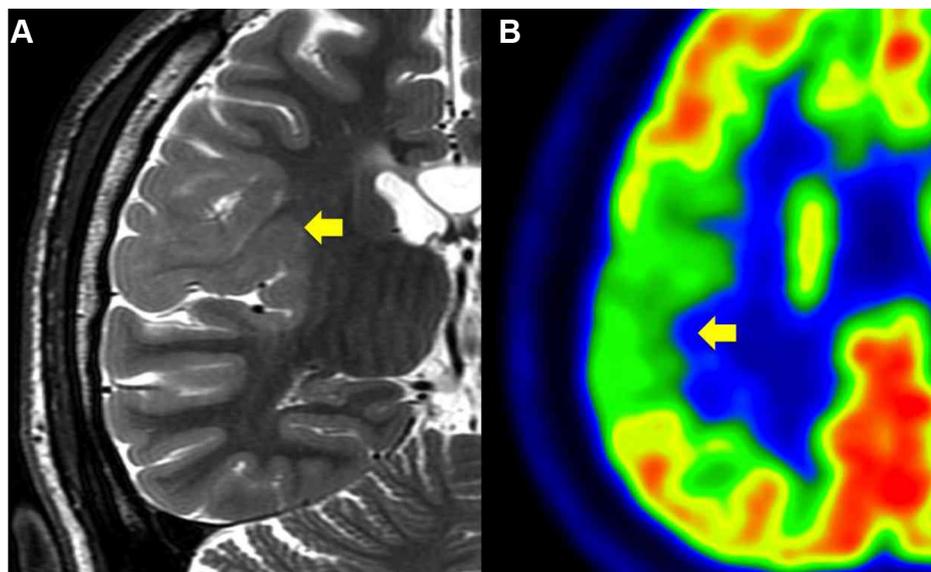
The patient underwent ECoG using two subdural grids: one  $4 \times 8$  contacts, and one  $4 \times 5$  contacts, which were placed on the right frontal lobe and temporal lobe, covering the lesions. Recordings demonstrated significant and frequent focal spikes from the electrodes directly covering the lesion (Fig. 2). Before lesionectomy, functional mapping was also performed by triggered electromyography, which consists of applying an electrical stimulus directly on the motor cortex to elicit complex muscular action potentials that are recorded in the corresponding muscle channels. In addition, the intraoperative electrophysiological survey included sensory evoked potential for central sulcus localization (by phase reversal), and motor

evoked potential and corticospinal D-wave for motor cortex mapping and monitoring. Transcortical ultrasound (SonoWand Invite; SonoWand, Trondheim, Norway) was used for lesion definition. Probes with a 6–12 MHz frequency were placed on the malformed cortex and displayed the lesion with high resolution (Fig. 3). Preresected FCD can be defined more easily than postresected FCD. So the exact depth and width of FCD were measured and recorded prior to resection, and several pictures taken to confirm the anatomic relationship with the adjacent vessels, ventricle, and sulcus. In this way, ultrasound-guided tailored resection was sequentially done according to these measurements. After resection and hemostasis, the IUS probe can be put on the bed of FCD to reconfirm the lesion location and related anatomical landmarks. Because overestimation of the residual of FCD might occur due to a small air bubble in the water, oozing blood, hemostatic agents, and uncertain artifacts, we removed the lesion according to the interpretation of preresection IUS images. During the resection, the eloquent cortex was preserved anatomically, and intraoperative motor evoked potential and D-wave collection confirmed the functional aspect of the eloquent area. The histopathologic features were compatible with that of FCD, type IIa (Fig. 4).

After surgical resection, immediate left hemiplegia was noted and supplementary motor area syndrome suspected. The flaccid status lasted for 12 hours, after which his muscle power returned to the preoperative status (Grade 3/5). The patient stopped drooling, and his appearance was brighter, even though we still maintained the same dosage of anticonvulsants. He has had no seizures up to this writing (6 months after operation).

## Discussion

FCD is the most common cause (around 50%) of focal intractable epilepsy in the pediatric group [1], and at least



**Fig. 1** Radiological features of the focal cortical dysplasia. (A) T2-weighted magnetic resonance coronal image of the focal cortical dysplasia demonstrates the blurring of the gray/white matter junction (arrow). (B) Positron emission tomographic scan with fludeoxyglucose  $^{18}\text{F}$  shows hypometabolism on the axial image (arrow).

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