ORIGINAL ARTICLE

Robotic-Assisted and Image-Guided MRI-Compatible Stereoelectroencephalography

Jeffery A. Hall, Hui Ming Khoo

ABSTRACT: ***Background***: Stereoelectroencephalography has been in regular use at the Montreal Neurological Institute since 1972. The technique has been in constant evolution to incorporate advances in materials, imaging, and robotics technology. MRI-compatible electrodes were introduced in 2007 and robotics in 2011. Here we report on the technique, safety, and advantages of our current method of stereoelectroencephalography implantation. ***Methods:*** We retrospectively reviewed all patients who underwent stereoelectroencephalography by the senior author. Technical, clinical, and radiological complications, and postimplantation outcomes were analyzed. Only patients implanted with MRI-compatible electrodes were included to review MRI abnormalities with electrodes in situ. ***Results:*** A total of 53 patients were implanted with 550 electrodes (average = 10.4 per patient), for an average duration of 14.6 days. There was no mortality, infection, or new neurologic deficit. Two patients had a superficial screw plunge without clinical consequence. Four patients demonstrated asymptomatic MRI abnormalities (7.54% per patient, or 0.72% per electrode). MRI with electrodes in situ was used for neuronavigation in all 29 who underwent resection and yielded a histopathological diagnosis of focal cortical dysplasia in 15 MRI-negative patients. ***Conclusions:*** The technique of stereoelectroencephalography described here was associated with no clinical morbidity although not without technical complications or radiologic (MRI) abnormalities. We should therefore remain vigilant in refining the technique and minimizing the number of electrodes required to answer a well-developed hypothesis regarding the epileptogenic zone. The use of MRI-compatible electrodes allowed neuronavigation using the images with the electrodes in situ, which was useful to tailor the eventual definitive resection and in localizing MRI-negative lesions.

RÉSUMÉ: La stéréo-électroencéphalographie assistée par robot et guidée par l'image peut être compatible avec la technique d'IRM. ***Contexte:*** C'est depuis 1972 que l'Institut neurologique de Montréal utilise couramment la stéréo-électroencéphalographie (SEEG). Cette technique n'a cessé d'évoluer en intégrant les avancées effectuées dans la technologie des matériaux, de l'imagerie et de la robotique. À cet égard, précisons que des électrodes compatibles avec l'IRM, de même que l'usage de la robotique, ont été respectivement introduits en 2007 et en 2011. Nous voulons ici faire état des aspects techniques et sécuritaires, mais aussi des avantages, liés à la mise en place de notre méthode actuelle de SEEG. ***Méthodes:*** Nous avons examiné de façon rétrospective tous les patients à qui l'auteur principal avait demandé de passer une SEEG. Tant les complications techniques, cliniques et radiologiques que les impacts consécutifs à la mise en place de cette méthode ont été analysés. Seuls les patients à qui l'on avait implanté des électrodes compatibles avec l'IRM ont été inclus dans notre étude afin que nous puissions nous pencher sur les anomalies techniques propres à cette méthode. ***Résultats:*** On a implanté chez 53 patients un total de 550 électrodes (moyenne = 10,4 par patient), et ce, pour une durée moyenne de 14,6 jours. Aucun cas de mortalité, d'infection ou de déficit neurologique n'est survenu. Fait à noter, deux patients ont vu leur vis s'enfoncer sans que cela n'entraîne des conséquences cliniques. De plus, on a détecté chez quatre patients des anomalies techniques asymptomatiques (7,54 % par patient ou 0,72 % par électrode). Enfin, la technique d'IRM couplée *in situ* à des électrodes a été utilisée dans un contexte de neuro-navigation chez tous les 29 patients qui avaient subi une résection. En outre, grâce à cette technique, on a pu obtenir chez 15 patients dont le cerveau était d'apparence normale un diagnostic histopathologique de dysplasie corticale focale. ***Conclusions:*** La technique de SEEG décrite ici n'a pas été associée à une forme ou une autre de morbidité clinique. Cela dit, des complications techniques et des anomalies liées à l'IRM se sont produites. Améliorer cette technique exige donc de rester vigilants et aussi de diminuer le nombre d'électrodes nécessaires afin d'étayer l'hypothèse, déjà bien admise, concernant les foyers épileptogènes. L'utilisation d'électrodes compatibles avec la technique d'IRM nous a aussi permis d'effectuer un examen de neuro-navigation en utilisant *in situ* des images. Cela s'est avéré utile car on a pu ainsi adapter d'éventuelles résections finales et localiser par IRM des lésions négatives.

Keywords: Robotics, Stereoelectroencephalography, Depth electrode, Image-guided

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Introduction

Image-guided placement of intracranial surface and depth electrodes for the characterization of epileptic foci was first promoted 52 years ago[1] and is currently a crucial step in the pre-surgical evaluation of many patients with pharmacoresistant epilepsy. Stereoelectroencephalography (SEEG) was developed in

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Paris using a frame-based method that made use of intraoperative angiography to avoid vascular complications.[2] The aim of SEEG is to determine the epileptogenic zone—that is, the region of ictal onset and immediate spread—in three dimensions. It was first implemented at the Montreal Neurological Institute in 1972[3] and has been associated with a very low rate of morbidity.[4,5] The procedure has been in constant evolution to take advantage of advances in materials and innovation in imaging and robotics technology. We began using magnetic resonance imaging (MRI)-compatible electrodes (DIXI Microtechniques, Besancon, France) in 2007 and a Robotic Surgical Assistant (ROSA, Medtech, Montpellier, France) in 2011. Previous studies have shown a greater degree of accuracy with the robot-assisted technique.[6,7] SEEG electrodes from historical cases were not MRI-compatible, and thus a thorough review of MRI findings was not possible. In our study, for the first time, we showed MRI findings in a relatively large cohort of patients with pharmacoresistant epilepsy who had MRI with electrodes in situ following implantation of SEEG electrodes. We describe the current technique of SEEG implantation at our center and aim to assess its safety and advantages.

Methods

We performed a retrospective chart review of all patients with MRI-compatible intracranial SEEG electrodes implanted by the senior author (JH). The research ethics board of the Montreal Neurological Institute and Hospital approved the review (no. NEU-14-101). All patients had preoperative enhanced MRI for image guidance during implantation of SEEG electrodes as well as postimplantation MRI with the electrodes in situ.

Initially, electrodes were inserted using a frameless technique with a double-chuck articulated arm[8] and the StealthStation neuronavigation system (Medtronic, Minneapolis, MN, United States) with image guidance by global acquisition gadolinium-enhanced MRI. Avascular trajectories were chosen from the scalp entry point to the intracranial target. Once the plane was obtained, a percutaneous craniostomy was performed with a 1.3-mm drill bit. A hollow anchoring screw measuring 2.5 cm in length was then secured in place with an adapted screwdriver (DIXI). The neuronavigation system was used to calculate the distance from the tip of the anchoring screw to the target. This distance was then translated to a measuring tool (DIXI), and a stylet (DIXI) was passed through the hollow screw to make a path for the electrode. Coagulation was sometimes required if the dura had not been breached by the anchoring screw. When the stylet was removed, the electrode was inserted to the proper depth and secured to the anchoring screw. The electrodes measure 0.8 mm in diameter.

Since 2011, we used ROSA for stereotaxy and the StealthStation neuronavigation system for verification and for depth measurement in all cases. Avascular trajectories are planned on the ROSA software with image guidance using global acquisition gadolinium-enhanced MRI coregistered onto global acquisition thin-cut computed tomography (CT) angiography (slice thickness = 1 mm). The stereotaxic craniostomy, anchoring screw, and electrode placement are identical to that described above.

The anchoring screws are made of titanium and the electrodes are made of platinum and iridium (DIXI Microtechniques, Besancon, France). Preimplantation CT angiography for stereotaxy planning was acquired using a Toshiba CT machine (Aquilion ONE, Toshiba, Tokyo, Japan). Both preimplantation MRI for stereotaxy planning and postimplantation MRI (T1- and T2-weighted images) with electrode in situ was acquired using a 1.5-Tesla magnetic field machine (Signa [GE Medical Systems, Chicago, IL, United States] prior to November of 2014 and Ingenia [Philips Medical Systems, Amsterdam, The Netherlands] thereafter). A T1-weighted image was acquired using the following sequences: prior to November of 2014: slice thickness 1.0 mm, repetition time (TR) 23 ms, echo time (TE) 8 ms, flip angle 20 or 65 degrees; from November of 2014: slice thickness 0.78 mm, TR 7.9 ms, TE 3.5 ms, flip angle 6 degrees). A T2-weighted image was acquired using the following sequences: prior to November 2014: slice thickness 1.2 to 1.6 mm, TR 3.6 to 4.1 ms, TE 1.2 ms, flip angle 20 or 65 degrees; from November 2014: slice thickness 2 mm; TR 2800 ms, TE 475 ms; flip angle 90 degrees).

Postimplantation MRI images with electrodes in situ were employed for evaluation of complications and to reconstruct global images for later use in neuronavigation protocols during resection of the epileptic focus. While there is some metallic artifact, the electrodes and electrode contacts are best visualized on T2-weighted images. T1-weighted images were employed as reference in our neuronavigation protocols, and the T2-weighted images were coregistered.

A total of 55 consecutive patients were identified from September of 2007 to June of 2017. Two patients were excluded from analysis: one because not all of the implanted electrodes were MRI-compatible and postimplantation MRI was not acquired, and the other because it was not performed for SEEG (but for thermoablation). This was done to provide a homogenous cohort of patients undergoing SEEG for pharmacoresistant epilepsy, all of whom had MRI with the electrodes in situ. The MRI images with electrodes in situ were used for image guidance at the time of definitive resection in all patients.

The factors studied included technique of implantation (manual vs. robotic-assisted), radiological morbidity, clinical morbidity, and mortality. We also calculated the MRI-positive rate of patients diagnosed with focal cortical dysplasia (FCD) on histopathology. The MRI findings were extracted from the neuroradiology report of our center based on 1.5-Tesla MRI.

Results

The details pertaining to semiology, MR findings, FDG–PET, and neuropsychology findings of the patients are summarized in Table 1. Both of the techniques described here allowed successful implantation in all cases. Between September of 2007 and March of 2011, 20 patients (11 males; mean age = 34.6 ± 9.8 years, range = 18–53) were implanted using the manual technique with a total of 184 electrodes (Table 1, patients 1–20). Between July of 2011 and June of 2017, 33 patients (23 males; mean age = 30.1 ± 9.4 years, range = 14–53) were implanted using the robot-assisted technique with a total of 366 electrodes (Table 1, patients 21–53). Therefore, among the 53 patients included in our analysis, 550 electrodes were implanted, for an average of 10.4 per patient. The duration of implantation ranged from 7 to 31 days, with an average of 14.6 days.

Placement of the anchoring screw requires gentle forward pressure with the screwdriver to secure it to the bone. On two occasions, both associated with the electrodes placed through the temporal bone for hippocampal targets, the screw penetrated the cortex. In these two cases, a 1-cm incision and enlargement of the craniostomy was required to retrieve the screw found at the level of the inner table. While this technical complication resulted in no clinical

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Table 1: Summary of semiology, imaging, neuropsychology data, and preimplantation hypothesis ($N = 53$)

Pt	Age/ gender	Seizure semiology	MRI findings	FDG-PET	Neuropsychological data	Pre-implantation hypothesis
1	44/M	No aura. Arrest of activity, unresponsive, oral and manual automatisms. Post-ictal confusion, fatigue and L side headache	L T intraventricle cyst with discret wm gliosis. Bil shape Hc abnormalities	Not performed	Bil T dysfunction L > R	Bil TLE
2	47/F	Aura: epigastric and numb sensation in L forearm. Stares, laughs, oral and gestural automatisms. SGTC with apnea and cyanosis. Prolonged postictal confusion	Normal	Not performed	Bil T dysfunction	R TLE
3	32/F	Aura: rising epigastric sensation, déjà vu or premonition 1. Arrest of activity, chewing, moaning, some restlessness 2. Stares, terror, high pitch moan, bi-manual automatisms, R arm dystonia. Post-ictal amnesia.	Normal	Not performed	Bil T dysfunction	FTLE
4	27/F	Aura: headache L OF (root of the nose), sometimes epigastric with palpitations. Stares, immobile and mute, amnesia. Occ SGTC	L sphenoid fibrous dysplasia, and OF encephalocele (post-op)	Not performed	Mild dysfunction L mesial T	L FLE
5	31/M	No aura. Nocturnal. Arousal, motor agitation, horizontal body mvts, with or without head deviation, occ. posturing of L > R arm and chewing	L inf. P FCD	Not performed	L FT dysfunction	L PQE
6	53/F	1. Aura: Rising chest sensation, fear 2. No aura. Loss of awareness, vocalization, L face and arms jerking, agitation. Post-ictal fatigue and speech impaired	R Am and Hc atrophy and L OF FCD	Not performed	L mesial T dysfunction	L FLE
7	16/M	No aura. Asymmetric tonic posture with head turning to L	Normal	Diffuse hypometabolism	Diffuse dysfunction	L FLE
8	28/M	Aura: déjà vu, dizziness and hot flashes. Hesitant, oral automatisms, sometimes speech disturbance but alert	Mild diffuse brain atrophy	bil T hypometabolism	Bil mesial T dysfunction	Bi TLE
9	42/M	No aura. Nocturnal. Hypermotor seizures with agitation, complex gestural automatisms, tonic phase (head and eyes to L). Rapid recuperation but amnesia, fatigue and stuttering	Normal	R FT abnormalities	Widespread dysfunction, possibly more on R	R FLE
10	25/F	No aura. Nocturnal, vocalization, clonic contractions of face, dystonic posturing	Normal	R post F hypometabolism	Normal profile	R FLE
11	43/F	No aura. Nocturnal. Frequent SGTC. Stares, oral automatisms, R arm flexion and stiffening, blinking, facial flushing. Post-ictal amnesia, confused and fatigue	L Hc atrophy and signal change	bil T hypometabolism	Bil mesial T dysfunction	Bil TLE
12	33/M	No aura (occ. olfactory aura and déjà vu). Stares, automatisms and frequent SGTC. Post-ictal amnesia	Hydrocephalus	not performed	Non-dominant mesial T dysfunction	L mTLE vs bi TLE
13	40/M	No aura. Confused, stares, unresponsive, oral and manual automatisms. Ictal and post-ictal coughing. Post-ictal amnesia and confusion	L MTS	not performed	Bil T dysfunction	Bil TLE
14	21/M	Aura: bad taste, nausea and epigastric sensation. Chewing, swallowing, moaning, L hand dystonia, confusion. Occ blurry vision. Occ SGTC	R MTS, L O uligyria/ encephalomalacia, L P gliosis/ dysplasia	not performed	Diffuse interference with bil T dysfunction	R TLE
15	48/M	1. Brief absence-like episodes with grimacing. Drooling and postictal amnesia. 2. Confusion, head version to R, R hemibody tonic convulsion	Normal	not performed	L T dysfunction	L FLE
16	22/F	Aura: numb feeling, foggy vision. Gestural automatisms, rubs frontal area and scalp with R hand or both, eyes and head to L, L arm elevation and SGTC	Normal	not performed	Diffuse interference (FT max), not lateralizing	R FLE
17	33/M	No aura. Asymmetrical tonic posture, L arm stiffening and elevation with or without L facial pulling, head and body turn to R. Occ SGTC	R hemimegalencephaly	not performed	Diffuse interference	R FLE
18	38/M	No aura. Nocturnal, arousal, grimace, facial flushing, contraction of facial muscles, smiling and giggling, shakes head	Normal	R FT hypometabolism	Non-dominant disturbance FC	R FLE vs TLE
19	26/F	Olfactory aura, déjà vu, anxiety, staring, anxiousness, oral automatism	R Hc atrophy	R FT mild hypometabolism	Bil mesial T dysfunction	R TLE

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Table 1. *Continued*

Pt	Age/ gender	Seizure semiology	MRI findings	FDG-PET	Neuropsychological data	Pre-implantation hypothesis
20	39/F	Olfactory aura with déjà vu. Arms and hands automatisms, oral automatisms. Post-ictal speech deficit and often L frontal headache	L OF encephalocele	L T anterior and mesial hypometabolism	L FT dysfunction	L FLE vs TLE
21	23/F	Feeling of a presence, sensation over dorsolumbar spine. Tonic symmetrical posture with both arms flexed, fingers automatisms, grimaces, apnea, rubefaction. Cluster of spasms	Normal	Not clearly lateralizing or localizing	???	FLE
22	36/M	Aura: tachychardia, ascending or descending body sensation, echoing, dizziness. Agitation, rotatory and horizontal mvts of body, grimaces, R UE dystonia, speech arrest. Rare SGTC	Normal	L FT hypometabolism	Bil F L > R (parasagittal), and R T dysfunction	FLE
23	25/M	Aura: feeling sick, fear, complex visual hallucinations. Head deviation to R, loss of awareness, oral and R arm automatisms. Post-ictal fatigue and R side headache	Normal	R T hypometabolism	Normal T function, atypical speech	R TLE
24	25/M	No aura. Asymmetrical tonic posture, head deviation to L, agitation. Rapid recuperation, discrete R side weakness	Normal	Not localizing	L FC dysfunction	L FLE
25	41/M	Auras: rising epigastric sensation, bladder fullness. Arousal, stares, asymmetric tonic posture, apnea. Rapid recuperation, post-ictal exhaustion, hyperventilation, no deficit	Normal	Not clearly lateralizing or localizing	Not clearly lateralizing or localizing	FLE
26	36/M	Nocturnal. No clear aura. Arousal, stares, tonic posture, pout, blinks. Rapid recuperation. Goes back to sleep. Fatigue next morning	Normal	R T mild questionable hypometabolism	Bil F dysfunction	FLE
27	36/M	Mostly nocturnal attacks. Aura: cephalic sensation, palpitation, embarrassed feeling. Hypermotor seizure, screams and intense agitation, fear. Post-ictal amnesia	Normal	L F hypometabolism	FC, not lateralized	FLE
28	45/M	Aura: epigastric, pressure or pain or discomfort. Loss of awareness, restless, unresponsive and mute. Occ SGTC. Post-ictal confusion and amnesia	RF encephalomacia and bil Hc atrophy	not performed	Diffuse interference	Bil TLE
29	31/F	1. Unresponsive, eyes wide opened, pupils dilated, immobile, no automatic behavior, occ fall 2. L arm sensory aura, sometimes loss of awareness 3. R arm sensory aura, speech impairment, loss of awareness	Bil Hc atrophy with increase signal	R hemisphere diffuse hypometabolism	Diffuse interference	R PQE
30	21/F	Vestibular aura: impression of movement or unsteadiness. L UE increased tone, loss of awareness, L arm clonus, occ SGTC	Normal	not performed	bil post Q. dysfunction	R PQE
31	29/F	Aura: unwell, hot sensation in the chest, rising epigastric sensation; changes of luminosity and "flashes"; sometimes hyperacusis. Staring, oral automatisms, and occ. head version to L and SGTC	Normal	R anterior T hypometabolism	R FT dysfunction	R FLE
32	53/M	Aura: tingling sensation, cephalic, sometimes smiling and giggling. Arrest of activity, hesitant, speech arrest. Occ head version to R, R hemibody clonic mvts, and SGTC	Hypothalamic hamartoma	not performed	L FT dysfunction	hypothalamic hamartoma
33	47/M	No aura. Arrest of activity, staring, oral automatisms, elation, mute or laughing. Post-ictal dysphasia, confusion, amnesia, frequent L F and peri-orbital headache. Occ SGTC	Normal	not performed	F > T dysfunction	FLE vs TLE
34	37/M	Aura: auditory, deja-vu, speech and activity arrest, staring, loss of awareness	L Hc atrophy	Bil T hypometabolism	bil T dysfunction	Bil TLE
35	36/M	Verbal vocalization and hand rubbing	Bil multiple NH	Not clearly lateralizing or localizing.	Bil FT dysfunction, L > R	L TLE
36	33/F	Aura: déjà vu. Loss of awareness, arrest of activity, staring and mute, chewing, blinking, bi-manual automatisms, smiles or grimaces. Occ SGTC. Post-ictal amnesia, confusion	Normal	L anterior and mesial T hypometabolism	R T dysfunction	L PQE

37	23/F	UE flushing sensation. Dystonic posturing of hands, blinking, R facial contraction, salivation, strange feeling on the chest, dissociative experience, loss of consciousness and fall	Normal	Not performed	Generalized slowing. R FP dysfunction; R mesial T dysfunction	R PQE
38	20/F	Vision lost (L > R), light flashing, blinking. Occ SGTC	Normal	Not performed	Dominant hem dysfunction (F-P circuits)	R PQE
39	22/M	No aura. Loss of awareness, oral automatisms, extension and elevation of both arms, agitation and ambulatory automatisms. Occ fall, rare SGTC. Post-ictal dysarthria and dysphasia	Bil Hc atrophy	Not performed	bil T dysfunction	Bil TLE
40	21/F	"Zap" sensation in the head. Head and eyes deviation to L, L arm elevation, upper body rotation to L. SGTC with vocalization	Normal	R F hypometabolism	Dominant F dysfunction (bil language representation)	R FLE
41	14/M	Aura: ill-defined cephalic sensation. Hypermotor seizures with loud vocalizations and screams, agitation, short, repetitive and mostly sleep-related. No post-ictal deficit	Normal	R F and P hypometabolism	Preserved F function	FLE
42	29/M	Aura: dizzy, epigastric or abdominal sensation. Loss of awareness, gestural automatisms, chewing and L hand dystonia. Post-ictal speech deficits	L inf F convexity PMG; L MTS	not performed	L FT dysfunction	L FLE vs TLE
43	31/M	Usually nocturnal, short, no aura. Arousal, pause and immobile, vocalizations, gestural automatisms, pedaling, anxiety or restlessness, then mute and chewing. Post-ictal amnesia and dysphasia	Normal	L FT hypometabolism	L F dysfunction	L FLE
44	23/M	Aura: vague dizzy and spinning sensations. Unresponsiveness, slow mentation, immobile, gestural automatisms, sometimes head deviation to L. Post-ictal amnesia and fatigue	Normal	R TPO hypometabolism	R F lateral T dysfunction	R PQE
45	24/M	Aura: cephalic and whole body sensation, déjà vu. 2 types of seizures, with or without speech problems, chewing	Smaller R Hc and malformed L Hc	R neocortical and mesial T hypometabolism	R mesial T dysfunction	Bil TLE
46	14/M	Aura: palpitations. Pale, grimace, arrest of activity, stares, raises L arm, pointing, shivering, sometimes cyanotic. Occ. nocturnal SGTC. Occ TLE-like seizures (staring, chewing)	Multiple tubers over both hem	R T hypometabolism	Data not available	PQE
47	43/F	Luminosity change, anxiety/palpitation. Staring, both arm dystonic posture (L > R) with fist clenching, sometimes legs pedaling, head and body turn to R. Occ SGTC	L lateral ventricular ependymal cyst	R mesial T hypometabolism	L F dysfunction	R TLE
48	45/M	Aura: feel dizzy. Speech arrest, non verbal vocalization, unresponsiveness	L temporo-parieto-occipital lobar hemimegalencephaly	L TO hypometabolism	L T posterior dysfunction, verbal memory deficits, speech dysfunction	L PQE
49	30/F	Staring, slight eye deviation to the left, lip smacking	Bil trigonal NH	Not performed	Dominant FT dysfunction	Bil PQE
50	14/M	Sleep-related. Arousal, eyes wide-opened, R-side neglect, mildly agitated or restless, some pedaling or non-purposeful arm mvts, increase heart rate and hyperventilation	Normal	L FP hypometabolism	L FTP dysfunction	L PQE
51	28/M	No aura, staring, facial pallor, speech arrest, oral and manual automatisms. Rare SGTC	Normal	Not clearly lateralizing or localizing.	R T dysfunction, poor non-verbal memory	R PQE
52	32/M	Aura: auditory, staring, occ head and eye turning to the right, automatisms, hands stiffness	Normal	L T hypometabolism	F T dysfunction	L TLE
53	29/M	Nocturnal, arousal, looking around, slow/non-forceful eye and head deviation to L, automatisms of legs, no or very subtle oral and manual automatisms	Normal	L T widespread hypometabolism	R T dysfunction	R TLE

Ant: anterior; Bil: bilateral; C: central; F: female (in the column of gender), or frontal; FC: frontal-central; FCD: focal cortical dysplasia; FLE: frontal lobe epilepsy; Hem: hemisphere; Hc: hippocampus; inf: inferior; L: left; M: male; max: maximum; MTS: mesial temporal sclerosis; mvts: movements; NH: nodular heterotopia; NL: non-lesional; O: occipital; occ: occasional; OF: orbitofrontal; P: parietal; PMG: polymicrogyria; post: posterior; PQ: posterior quadrant; PQE: posterior quadrant epilepsy; Pt: patient; R: right; SGTC: secondary generalized tonic clonic seizure; T: temporal; TLE: temporal lobe epilepsy; UE: upper extremity.

consequences, its description is mentioned here due to highlight the need for very gentle pressure upon placement. We had no complications associated with removal of the electrodes. In all cases, they were removed with the short-handled screwdriver or the spanner made by DIXI to avoid plunging.

Overall, there was no mortality, no neurologic deficit, and no infection observed. One patient developed headache 3 months after explantation of SEEG electrodes due to a venous thrombosis that resulted in edema in the left parietal lobe. However, an association between implantation and the venous thrombosis is unclear. The patient was treated with anticoagulants for 6 months and is now symptom-free.

A typical T2-weighted axial image is shown in Figure 1A. When seen along its plane, the electrode and each electrode contact are well-visualized. Figure 1B shows a sagittal T1-weighted image demonstrating the degree of metallic artifact. The actual diameter of the electrode is 0.8 mm.

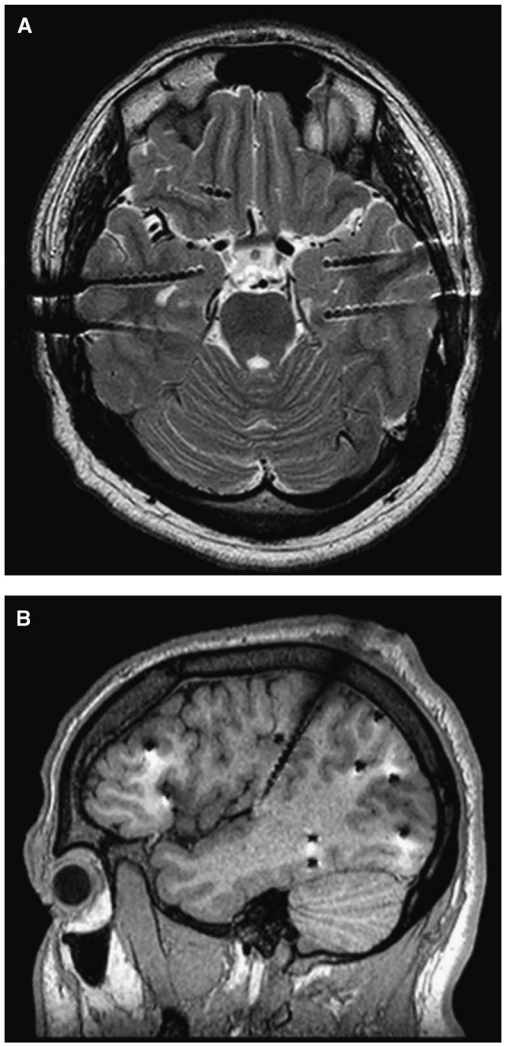

Figure 1: *(A) An axial T2 section from an SEEG study for bitemporal epilepsy. When seen along its plane, the electrode and each electrode contact are well-visualized. (B) A sagittal T1 section from an SEEG study for frontal and posterior quadrant epilepsy. Electrodes were aimed at the orbitofrontal, temporal, and occipital areas from a lateral approach and the insula from a superior approach. A metallic artifact (appeared larger than the electrode diameter, 0.8 mm) is appreciated.*

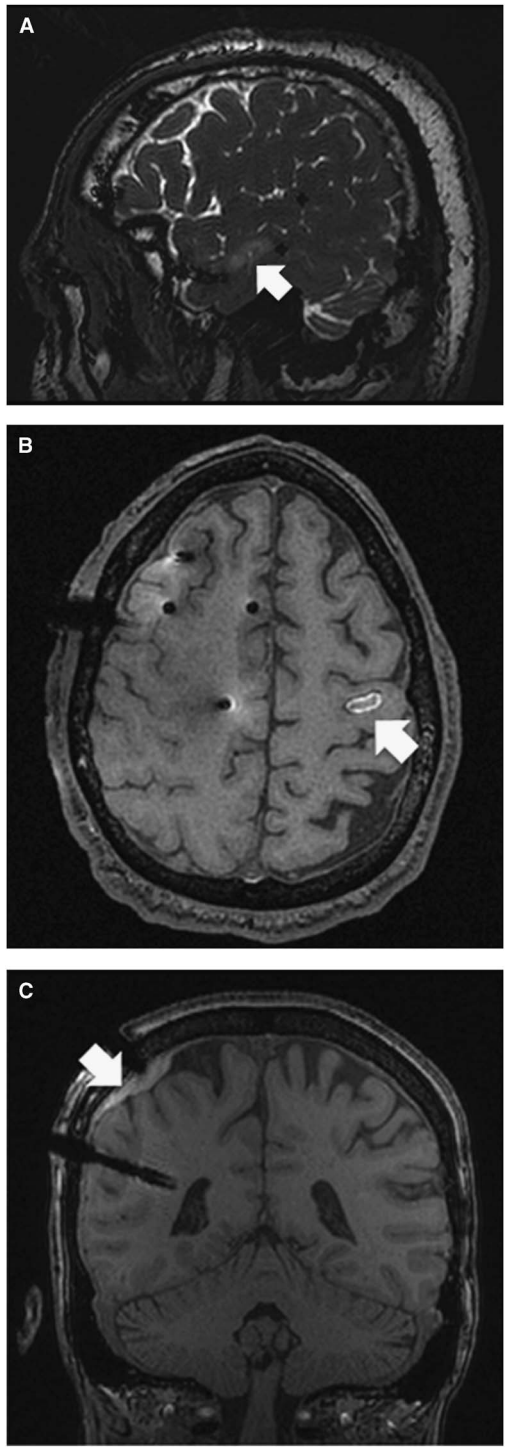

Figure 2: *(A) Patient 12. T2 sagittal section showing a non-hemorrhagic contusion (white arrow) on the second temporal gyrus. The bone had been weakened by prior pin fixation where the anchoring screw penetrated the cortex. A small skin incision was required to retrieve the anchoring screw. (B) Patient 17. T1 axial section showing a hemorrhagic contusion most likely due to penetration of the cortex with the drill bit. This was not caused by insertion of the stylet or electrode since it was the intended site of an epidural contact. (C) Patient 6. T1 coronal section showing a subdural hematoma most likely due to unintended penetration of the dura and/or laceration of the cortical vessel.*

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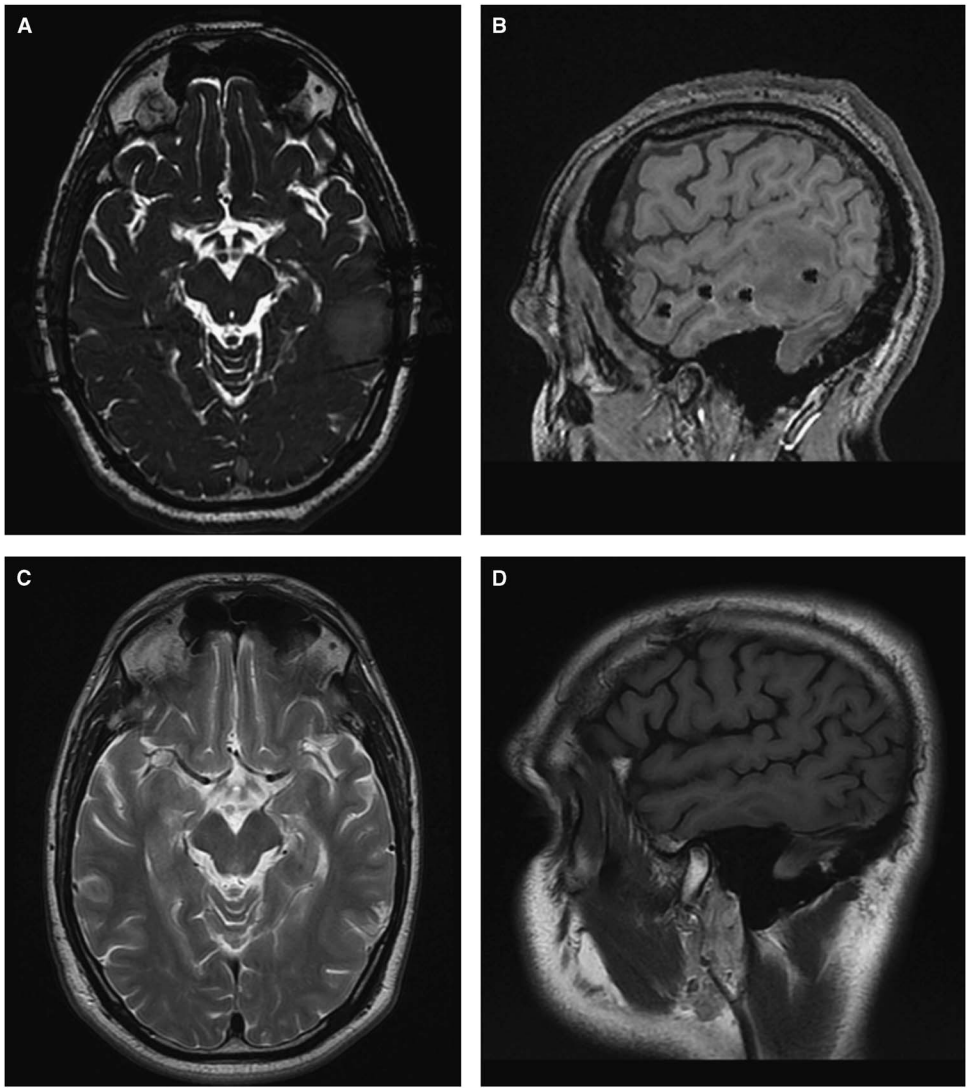

Figure 3: *Patient 34. (A and C) T2 axial section. (B and D) T1 sagittal section. A and B show venous congestion in the left posterior temporal area most likely caused by compression or coagulation of a vein. C and D show these changes to be largely resolved at 3 months post-explantation.*

A careful review of each patient's MRI with the electrodes in situ revealed four unexpected findings (three in the manual group and one in the robot-assisted group, $p = 0.287$). Figure 2A demonstrates an area of contusion without hemorrhage that resulted from a penetration of the anchoring screw through bone that had been weakened by previous pin fixation with the manual technique. Figure 2B shows a hemorrhagic contusion that likely occurred from an unintended perforation of the dura and cortex at the time of craniostomy with the manual technique. Figure 2C shows a subdural hematoma that likely occurred from an unintended perforation of the dura and/or laceration of a cortical vessel at the time of craniostomy with the manual technique. Figures 3A and 3B show venous congestion in the left posterior temporal area, most likely secondary to venous compression or coagulation with the robot-assisted technique. Figures 3C and 3D demonstrate its resolution at 3 months post-explantation.

Among the 49 patients in whom the SEEG findings were finalized, 31 (63%) underwent therapeutic intervention (29 resections and 2 thermocoagulations). The MRI images with electrodes in situ were used for neuronavigation in all 29 who underwent resection. Seventeen patients were diagnosed with FCD on histopathology, while only 2 were positive for FCD on multiple preoperative MRI.

Discussion

The goal of the present study is to assess the safety and advantages of the current SEEG electrode implantation methodology in our center. Accuracy of frameless SEEG techniques has been extensively reported,[6,9,10] and this is not our aim here.

Among the 53 patients who were implanted for an average duration of 2 weeks, with an average number of 10 electrodes, there was no mortality, no infection, and no new neurologic deficit. This rate of clinical complication is lower than that associated with other types of intracranial recording such as subdural strips or grids.[11,12] Although the number of patients in this study is

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relatively small, the low rate of clinical complications with our technique is encouraging, as it is comparable to large series reported recently.[7,13,14]

Careful review of postimplantation MRI with electrodes in situ revealed four patients with asymptomatic radiological findings on MRI: three in the manual group and one in the robot-assisted group. The overall rate of asymptomatic radiologic complication was 7.54% per patient, or 0.72% per electrode. The overall rate of radiologic complication is slightly higher than that reported in a recent study.[15] However, the modalities used for postimplantation evaluation of complication were different: CT in previous studies and MRI in this study. The difference in radiologic complication rate may be explained by the difference of sensitivity in detecting abnormalities between MRI and CT: MRI (in this study) is more sensitive than CT (in previous studies). Nevertheless, only one hemorrhagic complication was found in this series (1.9% per patient, or 0.18% per electrode) and this rate is the same or even lower than the hemorrhagic complication based on CT findings in previous studies.[4,7,9,15] While the use of robotics in neurosurgery remains in its infancy,[16] the application to stereotactic procedures is advancing rapidly.[17-19] The present study revealed that robotic assistance is safe to apply to stereotactic procedures.

A novel advantage provided by this technique is the ability to use the MRI with electrodes in situ for image-guided neuronavigation at the time of definitive resection. Under the guidance of MRI with electrodes in situ, resection yielded successful histopathological diagnosis of FCD in 15 patients who were MRI-negative for FCD (88.2% of all patients with FCD in this series). This suggests that neuronavigation using MRI images with electrodes in situ was useful to tailor definitive resection of the so-called "non-lesional" cases, which are often shown to be FCD on histopathology.

The safety of MR imaging of implanted depth electrodes has been a matter of debate.[20] Despite evidence of studies demonstrating its safety under certain conditions,[20-22] postimplantation MRI is still less popular to date because of safety concerns among some neuroradiologists and neurosurgeons. In agreement with previous studies, we did not find any complication after MR imaging in this series of patients. This further supports the safe use of MR imaging in localizing the implanted SEEG electrodes.

Limitations of the Study

While many centers are now moving towards 3-T MR imaging capabilities, the use of 1.5-T MR imaging in our presurgical workup is a limitation in terms of identification of focal cortical dysplasia, or any potential epileptogenic lesion. This may in part explain the patient cohort in whom our preimplantation MRI failed to identify focal cortical dysplasia.

Other limitations include the small number of patients and the retrospective nature of our study. Hence, the safety and complication rate figures reported here need to be taken with caution. The safety of our SEEG implantation methodology and the usefulness of MRI with SEEG in situ in clinical practice will be better demonstrated in a larger controlled prospective study.

Conclusions

We reported the technique, safety, and advantages of the current SEEG implantation methodology in our center. This is also the first reported series of MRI findings of patients with SEEG electrodes in situ. While the radiological changes were few and none were clinically significant, it nonetheless suggests that we should remain vigilant in refining the technique and restrict the number of electrodes to the minimum required to study a well-defined preoperative hypothesis regarding the seizure onset zone and propagation pathway. The use of MRI-compatible electrodes allows neuronavigation using the images with the electrodes in situ, which is useful to tailor the eventual definitive surgical resection.

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Disclosures

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Dr. Hall has nothing to disclose.

Statement of authorship

JAH was responsible for conception and design of the study. JAH and HMK were responsible for acquisition and analysis of data, drafting a significant portion of the manuscript, and the figures.

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