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CLINICAL ARTICLE

Risk factors for complications during intracranial electrode recording in presurgical evaluation of drug resistant partial epilepsy

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Abstract
Background Intracranial electrode monitoring is still required in epilepsy surgery; however, it is associated with significant morbidity.
Objective To identify risk factors associated with complications during invasive intracranial EEG monitoring.
Materials and methods Retrospective study of all patients undergoing invasive monitoring at Westmead between 1988–2004. From detailed chart reviews, the following variables were recorded: duration of intracranial monitoring, the site of grid implantation, number of grids and electrodes, seizure frequency, postoperative complications and seizure outcome.
Results Seventy-one patients (median age: 24 years) underwent subdural electrode implantation; 62% had extratemporal lobe epilepsy and 46% were non-lesional. Of the 58 monitored patients who had cortical resections, 45 had good seizure outcomes. Complications related to subdural electrode implantation included transient complications requiring no treatment (12.7%), transient complications requiring treatment (9.9%) and two deaths (2.8%). Specific complications included subdural haemorrhage, transient neurological deficit, infarction and osteomyelitis. The two deaths occurred within 48 h of implantation were related to raised intracranial pressure (one venous infarction, one unexplained). Complications were associated with maximal size of grid ($p<0.001$), greater number of electrodes ($p<0.001$), electrode density per cortical surface implanted ($p<0.001$), right central surface implantation ($p=0.003$) and left central surface implantation ($p=0.013$). Multiple logistic regression identified larger size grids and right central surface implantation as independent predictors of complications.
Conclusion There are significant complications during intracranial EEG evaluations but the majority of these are transient. We found a relationship between the size of the electrode arrays and the incidence of complications. The results of this study have been used to modify our implantation and monitoring protocols.

Keywords Electrodes · Implanted · Electroencephalogaphy · Epilepsy · Risk factors · Postoperative complication

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Introduction

Preservation of eloquent cortex, accurate localization and complete removal of the epileptogenic focus is vital in achieving seizure freedom in patients with medically refractory epilepsy. Despite advances in structural and functional imaging, the epileptogenic focus and the boundaries of a surgical resection cannot always be defined. In patients where no definite anatomical abnormalities are present, the specificity of magnetic resonance imaging (MRI) and positron emission tomography (PET) are less than 25% [36]. When seizures are difficult to localise and neither neuroimaging studies nor neuropsychological data are concordant with the electroencephalogram (EEG) findings, further evaluation with invasive intracranial electrodes is indicated [32]. However, intracranial recordings are associated with significant risks. We conducted a clinical audit of all patients undergoing intracranial recordings to identify complications and determine the risk factors associated with these complications. We hope the understanding of the morbidity and recognition of specific risk factors will minimise future complications.

Materials and methods

We reviewed a database of patients undergoing epilepsy surgery at Westmead Hospital and The Children's Hospital at Westmead between 1988 and 2004. Two hundred and ninety-six cases underwent epilepsy surgery during our study period. Seventy-one patients who underwent 79 intracranial monitoring sessions were identified. Eight patients had two intracranial monitoring sessions each. Four of these patients had separate admissions for the intracranial monitoring sessions because the resection following the first monitoring did not result in seizure freedom, four other patients had two monitoring sessions during the same admission due to re-positioning of subdural electrodes for optimal localization of the epileptogenic zone. None of these eight patients had complications in their second monitoring session. Only the first monitoring session was included in the statistical analysis. Seventeen patients (23.9%) were 16 years old or younger. All patients were referred for intracranial monitoring because either non-invasive investigations did not produce adequate information to localize the focus or the focus was adjacent to eloquent cortical regions. The number and location of the subdural grids to be inserted was determined by consensus at our regular epilepsy surgery meeting based on results obtained from seizure semiology, prolonged inpatient scalp digital video-EEG findings, neuropsychological assessment, ictal and interictal single photon emission computed tomography (SPECT), PET and MRI.

Demographic data and the following monitoring variables were recorded (Table 1): epilepsy syndrome, anticonvulsant medications, duration of implantation surgery, duration of intracranial monitoring, side and site of grid implantation, the number of grids and electrodes, the size of grids and the cortical surfaces covered. Other clinical parameters recorded were the frequency of clinical seizures, maximum temperature and lowest Glasgow Coma Scale (GCS) reached within the first 3 days, presence of previous intracranial monitoring or previous craniotomy, other medical conditions, length of hospital stay and number of readmissions after monitoring. The cortical surfaces were designated as frontal convexity, central convexity, mesial frontal, orbitofrontal surface, parieto-occipital convexity, mesial parieto-occipital, inferior occipital, inferomedial temporal and lateral temporal surfaces. The central region was defined by cortical stimulation producing primary sensorimotor responses. For each patient, all the designated

Table 1 Demographics

Characteristics		
Gender	Male	44 (62%)
	Female	27 (38%)
Age	Median, interquartile range (IQR)	24 (18,34)
Extra-temporal epilepsy		44 (62%)
Non-lesional epilepsy		33 (46.5%)
Hemisphere implanted	Left	22 (31%)
	Right	25 (35.2%)
	Bilateral	24 (33.8%)
Cortical surface covered (left; right; bilateral)	Frontal convexity	11; 16, 9
	Central convexity	15; 14; 4
	Mesial frontal surface	9; 9; 8
	Orbitofrontal surface	9; 6; 5
	Parieto-occipital convexity	10; 11; 3
	Parieto-occipital mesial surface	3; 9; 4
	Inferior occipital surface	8; 6; 0
	Lateral temporal surface	15; 17; 4
	Inferomedial temporal surface	11; 14; 10
Duration of monitoring (days)	Median, IQR	11 (7,14)
No. of electrodes	Median, IQR	62 (32,100)
	Range	8–164
	<40	23 (32.4%)
	41 to 80	25 (35.2%)
	81 to 120	11 (15.5%)
	>121	12 (16.9%)
Maximal size of grids implanted	40–64 electrodes	20 (28.2%)
	20–32 electrodes	28 (39.4%)
	≤16 electrodes	23 (32.4%)
No. of strips/grids	Median, IQR	4 (2,5)
	Range	1–12

cortical surfaces covered by a subdural grid array or strip were identified. This data is presented in Table 1. For the purpose of analysis each patient was then scored as having or not having a subdural grid array or strip over one of these cortical surfaces. The mean density of electrodes per cortical surface was determined by dividing the total number of electrodes implanted by total number of cortical surface covered.

All complications were recorded and graded according to a four step scale [18]. Minor complications common and expected in neurosurgery were not included as they are not directly related to the placement of intracranial electrodes. These included mild to moderate headache, eyelid swelling, ecchymosis of one eye, minor swelling of the incision and minor CSF leaks that stopped spontaneously, after reinforcing sutures or applying Histoacryl®. Mild drowsiness during the first postoperative day was not considered significant. However, a GCS less than 12 without obvious attributable cause and requiring investigative action was included as a complication.

The statistical software package SPSS for Windows (Version 12) was used to analyse the data. Two tailed tests with a significance level of 5% were used throughout. Pearson chi-squared or Fisher's exact test as appropriate were used to test for association between categorical variables and the presence of complications. Mann–Whitney test was used to assess associations between the presence of complications and continuous variables. All possible risk factors with $p<0.1$ by univariate analysis were entered into a multiple logistic regression model. Backward stepwise variable selection was used to identify the independent predictors of complications. Odds ratio and their 95% confidence interval were used to quantify the degree of association. Receiver operating characteristic (ROC) curve for complications was constructed using the probability of complication predicted by the logistic regression model.

Surgical technique and monitoring procedures

All patients underwent surgery under general anaesthesia. The scalp and bone flaps were made large enough so that the region of interest could be covered with the subdural electrode arrays. The dura was opened in a cruciate fashion and dural leaflets were tacked in position with a series of stay sutures. After exposure of the hemisphere, subdural electrodes were placed on the cortical surfaces under direct vision. From 1999, we routinely used frameless stereotaxy to plan the craniotomy

Ad-Tech/Wyler® stainless steel subdural strip and grid electrodes were used. The subdural electrodes were 0.7 mm thick. All electrode contacts were 4 mm in diameter with a 2.3 mm centre-contact. The centre of each contact was separated from the adjacent electrodes by 10 mm. The smallest strip inserted was an array of 1×6 electrodes and the largest was a grid of 8×8 electrodes.

The electrode arrays were then anchored in place by attaching a dural stitch through the strips or grids. The positions of the electrode arrays were recorded both by hand drawing and intraoperative photography. The dura was closed with a running 4/0 vicryl suture in a watertight fashion and with purse string sutures around the cables. The bone flap was reapproximated. All strip or grid electrodes have a small diameter wire carrier that was tunnelled through the scalp away from the original incision and secured with sutures to the dura and scalp. A 7 mm Jackson-Pratt drain was placed in the subgaleal space and was externalized through a separate stab incision. The scalp was closed in layers and staples were used for final skin approximation. Between 1988 and 1995, prophylactic antibiotics were administered in 14 (82%) cases. After 1996, prophylactic antibiotic (cefazolin, flucloxacillin or vancomycin) were administered routinely to all patients. Dexamethasone was given in 25 (62.5%) prior to 2002. Starting in 2003, dexamethasone was given routinely to all patients. Following a death during intracranial monitoring in February 2004, an external ventricular drain was placed in the subdural space for intracranial pressure monitoring for 24 h following the procedure. Following the second death in October 2004, further changes were made to the operative technique and monitoring protocols.

Postoperatively, all patients were monitored in a high dependency care setting with hourly observation. At 24 h, patients are transferred to the telemetry unit for VEEG monitoring and had fourth hourly neuro-observations thereafter. A patient's relative or carer was with the patient at all times to alert for a seizure during the period in the telemetry unit. Within the monitoring unit during the period of this study, there was no facility for continuous monitoring of arterial blood pressure, oxygen saturation or intracranial pressure. Additionally, patients were nursed at a ratio of one registered nurse to four patients, two of whom were cared for at some distance from the monitoring unit.

Routine anticonvulsants were discontinued or reduced during the monitoring period in all patients. Intravenous clonazepam was given to abort prolonged (more than 3 min) partial seizures, serial seizures or after secondarily generalized tonic clonic seizures. Head dressings were changed as often as required, but at least once a day, using aseptic techniques. VEEG was programmed to sample 10 min of every hour and 5 min before and 5-10 min after each manual seizure detection.

Cortical stimulation studies were performed at the bedside by the technician (JB) and supervising neurologist (ES, DG or AB) after seizures had been recorded and the patient restarted on antiepileptic medication. Using a GRASS®

S12 Isolated Biphasic Stimulator, reference electrodes were established by bipolar stimulation to 15 mA without causing symptoms or interruption to the patient's motor and language tasks. Regional areas of interest were examined with bipolar stimulation between the reference and individual electrodes using incremental currents, patient reports and observation of patient tasks. Stimulation was conducted over 1–3 days in 1–2 h sessions. At the end of intracranial monitoring, the patients were taken back to the operating room and the electrodes were removed by reopening the craniotomy. A cortical resection was performed in 58 (81.7%) patients.

Results

Complications occurring during intracranial monitoring are shown in Tables 2 and 3. One patient simultaneously had two complications (case 10). Eight patients (11.3%) had complications during the monitoring session that were not directly related to subdural electrodes. These complications included deep vein thrombosis, pulmonary embolism, delirium secondary to acute benzodiapine withdrawal, rash secondary to cefazolin and intravenous cannula (IVC) insertion site infection or sepsis. Complications related to subdural electrodes occurred in 18 patients (25.4%). In these patients, 14 had electrodes placed over the central region; ten were on the right. The complications included osteomyelitis, subdural haemorrhage, cerebral infarction, status epilepticus, transient neurological deficits, significant cerebrospinal fluid (CSF) leak and cerebral oedema.

Sixty percent of the 20 monitored sessions with grid size of 40 or more electrodes implanted resulted in complications. The most frequent complication was transient neurological deficit. The deficit occurred within 24 h of implantation and lasted for a median duration of 8.5 days (range 1 to 15 days). Imaging studies performed did not reveal any obvious cause. Each of these patients was on prophylactic dexamethasone.

During the monitoring, one patient had drowsiness that improved after 4 days. Investigations revealed no definite aetiology. Venous stasis and cerebral oedema were thought to be possible causes but neuroimaging did not show any definite cause. One patient had a significant CSF nasal leak 1 day following orbitofrontal grid implantation that was treated with acetazolamide and a CSF lumbar drain. Two patients had osteomyelitis diagnosed on follow up assessment and required removal of the bone flap. *Staphylococcus aureas* and *Staphylococcus enterococcus* were isolated respectively. They were treated with antibiotics and subsequently underwent delayed reconstructive cranioplasty.

Four patients were found to have an asymptomatic subdural haematoma detected either on brain computed tomography (CT) or at surgery during electrode removal. These patients were managed conservatively. One patient had a symptomatic subdural haemorrhage requiring urgent electrode removal and recovered completely without neurological deficit.

Table 2 Summary of complaints following intracranial electrode implantation

	Severity of complication			
	Transient, no treatment required	Transient, treatment required	Permanent	Death
Complications directly related to subdural electrodes				
CSF leak		1 (1.4%)		
Subdural hemorrhage	4 (5.6%)	1 (1.4%)		
Cerebral infarction		1 (1.4%)		1 (1.4%)
Transient neurological deficit	4 (5.6%)			
Motor weakness		3 (4.2%)		
Left homonymous hemianopia		1 (1.4%)		
Drowsiness	1 (1.4%)			
Cerebral edema		1 (1.4%)		1 (1.4%)
Osteomyelitis		2 (2.8%)		
Status epilepticus		1 (1.4%)		
Total	9 (12.7%)	7 (9.9%)	0	2 (2.8%)
Complications not directly related to subdural electrodes				
Acute benzodiazepine withdrawal	1 (1.4%)			
DVT	1 (1.4%)			
Pulmonary embolism		2 (2.8%)		
IVC site infection/sepsis		3 (4.2%)		
Drug rash	1 (1.4%)			
Total	3 (4.2%)	5 (7%)		

Table 3 Individual complications occurring during intracranial monitoring

Patient, gender, age	Grid size	Coverage	Complication	Localization of seizure	Pathology	Engel's seizure outcome	Follow up duration (days)
Case 1, female, 24	1×32, 2×24, 1×16, 4×8	Right: frontal convexity, central convexity, mesial frontal, orbitofrontal, inferomedial temporal, lateral temporal	CSF leak	Right frontal	Neuronal heterotopia	1	373
Case 2, female, 22	1×56, 2×16, 1×8	Right: frontal convexity, central convexity, mesial frontal, parietooccipital convexity, mesial parietooccipital	Asymptomatic SDH	Right parietal	Gliosis	1	342
Case 3, male, 12	1×64, 1×8	Right: frontal convexity, central convexity	Asymptomatic SDH	Right frontal	DNET	1	1,095
Case 4, male, 14	1×32, 2×8	Right: parietooccipital convexity, lateral temporal	Asymptomatic SDH	Right parietal	Hamartoma	1	2,555
Case 5, female, 41	1×32, 1×16, 1×8	Left: inferior occipital, inferomedial temporal, lateral temporal	Asymptomatic SDH	Left neocortical temporal	Non-specific changes	1	365
Case 6, male, 51	1×32, 1×8, 2×4	Right: frontal convexity, central convexity, mesial frontal,	Symptomatic SDH	Right parietal	N/A		
Case 7, male, 28	1×64, 1×16,2×12, 3×8	Left: frontal convexity, central convexity, mesial frontal, orbitofrontal, inferomedial temporal	Venous Infarction causing drowsiness, managed conservatively	Left frontal	Gliosis	1	600
Case 8, male, 43	1×64,6×16,	Left: frontal convexity, central convexity, mesial frontal, mesial parietooccipital Right: frontal convexity, central convexity, mesial frontal, mesial parietooccipital, lateral temporal	Transient right foot weakness	Right frontal	Cortical dysplasia	1	772
Case 9, female, 23	1×40, 5×16,, 1×8	Left: frontal convexity, central convexity, mesial frontal, Right: frontal convexity, central convexity, mesial frontal	Transient right hand weakness	Left frontal	Gliosis	2	885
Case 10, female, 33	1×32,8×16, 1×4	Left: frontal convexity, central convexity, mesial frontal, mesial parietooccipital, parietooccipital convexity Right: frontal convexity, central convexity, mesial frontal, mesial parietooccipital, parietooccipital convexity	Transient bilateral leg weakness; Right popliteal vein thrombosis	Bilateral frontal	Not operated		
Case 11, female, 14	1×64,3×16, 1×8	Right: inferior occipital, mesial parietooccipital, inferomedial temporal, lateral temporal	Transient left homonymous hemianaopia	Right parieto-occipital	Cortical dysplasia	1	324
Case 12, female, 18	1×64, 3×16, 2×4	Left: frontal convexity, central convexity, mesial frontal Right: frontal convexity, central convexity, mesial frontal, inferior occipital	Unexplained drowsiness	Right frontal	Hamartoma	1	1,245

Table 3 (continued)

Patient, gender, age	Grid size	Coverage	Complication	Localization of seizure	Pathology	Engel's seizure outcome	Follow up duration (days)
Case 13, male, 36	1×64, 1×32	Left: frontal convexity, central convexity, orbitofrontal, parietooccipital convexity, lateral temporal	Cerebral edema	Left frontal	Astrocytoma	3	477
Case 14, male, 34	1×64, 1×32, 1×16, 2×8	Left: central convexity, parietooccipital convexity, inferomedial temporal, lateral temporal	Cerebral edema, infarct, death	Left temporal parietal	N/A		
Case 15, female, 24	1×64, 3×16	Right: frontal convexity, central convexity, mesial frontal, mesial parietooccipital	Cerebral edema, infarct, death	Right centrotemporal	N/A		
Case 16, male, 10	1×20, 1×4	Left: central convexity, inferomedial temporal, lateral temporal	Status epilepticus	Right temporal	Non-specific changes	2	730
Case 17, male, 22	2×32,2×16	Right: central convexity, mesial frontal, orbitofrontal	Osteomyelitis	Right frontocentral	Non-specific changes	1	879
Case 18, male, 28	1×32,1×20, 2×16, 1×8	Right: frontal convexity, central convexity, orbitofrontal, parietooccipital convexity, inferomedial temporal, lateral temporal	Osteomyelitis	Right parieto-occipital	Cortical dysplasia	2	577

The most serious complication was death in two patients. The first patient was a 24-year-old female with a 12 year history of daily left sided somatosensory auras progressing to left hemiclonic seizures. Postictally, she might develop a left arm paresis. MRI showed a large area of superficial encephalomalacia extending from the right frontocentral region into the parietal lobe. Scalp video-EEG, ictal SPECT and PET imaging supported a seizure onset in the right central region. She proceeded to intracranial electrode monitoring to define the epileptogenic zone in relation to the sensorimotor cortex. One hundred and twelve subdural electrodes were implanted (1×64, 3×16) over the right frontoparietal cortex, covering motor and premotor area, and over the right mesial frontocentral surface (Fig. 1a and b). In the recovery room, there was mild left upper limb weakness but she remained alert. At 5 h postoperative, she had her typical partial seizure with secondary generalization and developed flaccid paralysis of her left upper limb. CT showed intracranial air, but no mass effect (Fig. 1c). She had four further secondarily generalised seizures within 12 h and became increasingly drowsy and less responsive (GCS=10). Mannitol was administered and an urgent MRI showed extensive right parietal infarction and oedema. Magnetic resonance venogram (MRV) and angiogram (MRA) revealed no vascular occlusion. She returned to surgery for removal of the subdural grids and the bone flap. The patient developed uncontrollable raised intracranial pressure (ICP) despite maximal medical therapy and went on to developed extensive brain oedema and bilateral occipital hemisphere infarction (Fig. 1d). She died 7 days after the original surgery. Autopsy showed diffuse cerebral oedema, transcalvarial herniation and bilateral occipital lobe infarction. The nature of the original pathology could not be determined due to the autolytic changes in the brain.

Following this episode the surgical technique was modified significantly but no other changes were made to the monitoring protocols. All patients underwent placement of a subdural intracranial pressure monitor and this was recorded for 24 h after the surgical procedure. The monitor was removed only if the ICP was normal and after a post-operative MRI was performed 24 h following electrode placement. In addition, titanium fixation plates were applied to the bone flap but were not secured to the skull allowing the bone flap the "ride up" if ICP became elevated.

The second patient was a 34-year-old male who had a Grade 2 astrocytoma removed from the left frontoparietal region at 11 years of age. Complex partial seizures started when he was twenty-six and were never controlled with medications. MRI showed an area of cystic encephalomalacia in the left parietal region and a defect extending into the left temporal lobe with haemosiderin deposition. Preoperative evaluation suggested a left temporal seizure onset. Intracranial monitoring was performed to define the

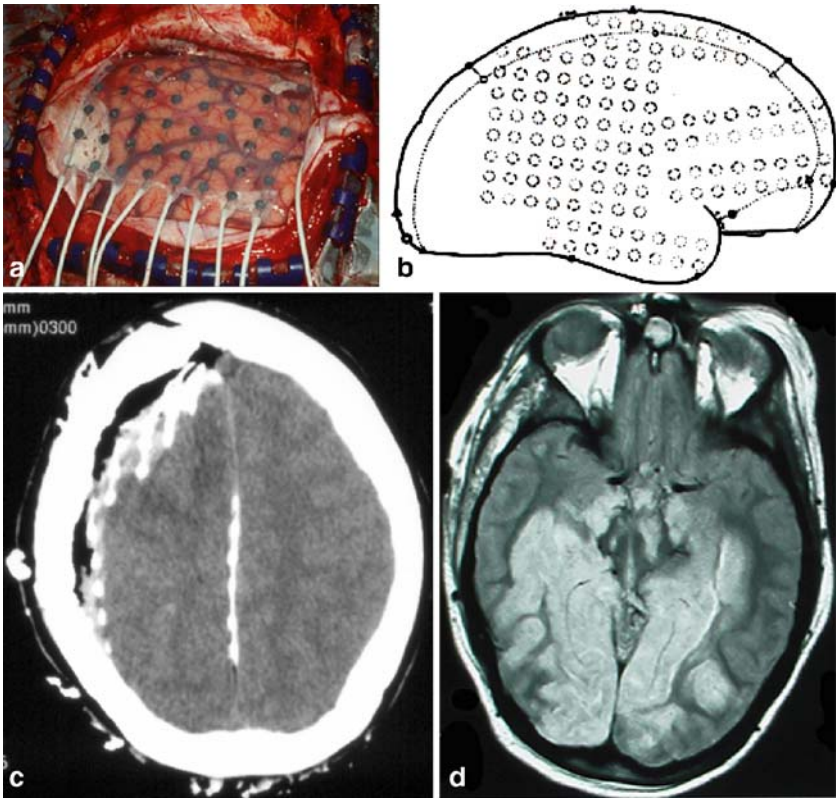


Fig. 1 **a** and **b** subdural electrode placement over cortical surface. **c** CT scan 12 h postoperative showing no obvious infarction or hemorrhage. **d** MRI at 36 h showed diffuse cerebral oedema, transcalvarial herniation and bilateral occipital infarction

extratemporal extent of the epileptogenic zone in relation to language cortex. One hundred and twenty-eight electrodes (1×64, 1×32, 1×16, 2×8) were implanted over the area of encephalomalacia and the surrounding regions (Fig. 2a,b).

Postoperatively, his ICP was monitored for 24 h via a subdural catheter and showed normal ICP. Twenty four hours following the surgery, he underwent MRI which showed no evidence of intracranial haematoma, significant mass effect, cerebral edema or infarction. The electrodes were in good position and anticipated postoperative changes were seen. He returned to the telemetry unit at 24 h and was ambulant and well. He had two complex partial seizures at 25 and 32 h postoperatively. At 42 h, he was found unresponsive with fixed dilated pupils. No clinical seizures had been witnessed by the carer that was with him at all times. When examined by nursing staff he was breathing spontaneously with pulse oxygen saturation at 89% that improved to 98% with mask 6 L/min and hemodynamically stable (heart rate 60 beats per minute, blood pressure 110/60 mmHg). The EEG monitoring showed a burst suppression pattern had developed over all of the grids 2 h before he was found unconscious. CT scan of the brain showed left hemisphere swelling and midline shift and subfalcine herniation (Fig. 2c). He was treated with mannitol and returned to surgery for subdural electrodes removal. Intraoperatively, there was no evidence of any significant haematoma or cortical vein thrombosis. Brain MRI, MRA, MRV performed a day later showed extensive infarction of the entire posterior cerebral artery territory and scattered areas of infarction in the midbrain, hypothalamus and right thalamus but no evidence of venous or arterial occlusion (Fig. 2d). He died 3 days later. A post mortem examination was not performed.

Results of statistical analysis

On univariable analysis, the factors significant at $p \leq 0.1$ were as follows: size of grid ($p<0.001$), total number of electrodes ($p<0.001$), density of electrodes per cortical surface ($p<0.001$), electrode implantation over the left central convexity surface ($p=0.013$) and electrode implantation over the right central convexity surface ($p=0.003$). The number of grids arrays implanted was not significant ($p=0.076$). All these factors were entered into a multiple logistic regression model. Only the size of the grids implanted and right central surface implantation were found to be independent predictors of complications (Table 4). Inclusion of the eight patients with medical complications did not change the relationship.

Using the linear predictor based on this model, the area under the Receiver Operating Characteristic (ROC) curve for complications was 0.81 (SE 0.05, $p<0.001$), indicating that this is potentially a good classifier. The predicted

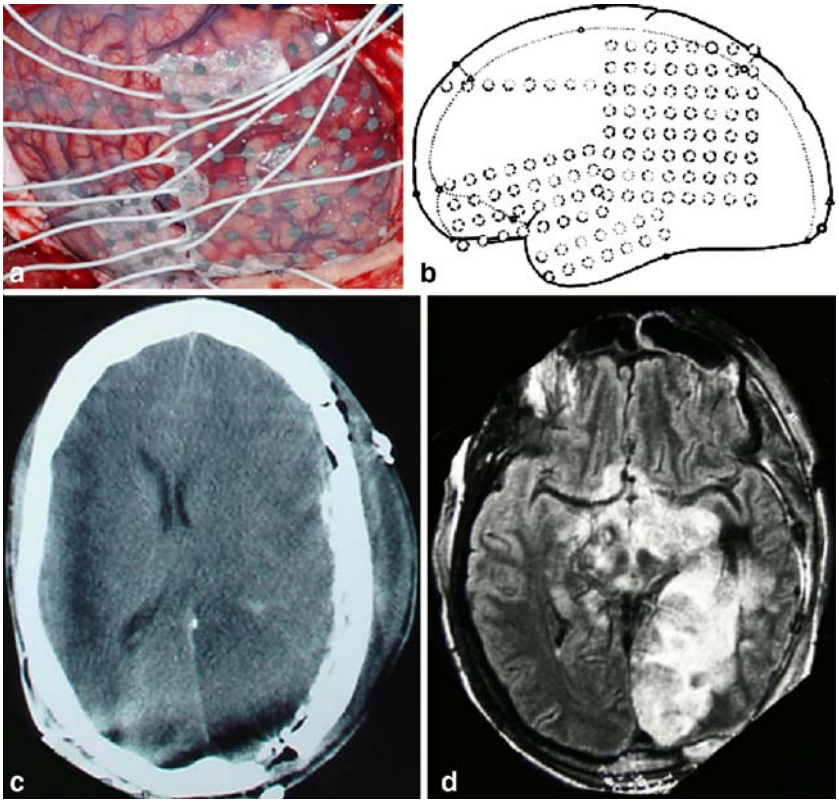


Fig. 2 **a** and **b** subdural electrode placement over cortical surface. **c** CT scan at 43 h postoperative showed left hemispheric swelling and midline shift to the right. **d** MRI at 48 h showed extensive infarction of the thalamus, midbrain and occipital lobes

probability of an adverse event when grid arrays of 40 electrodes or more were implanted on the right central region was 79.5% compared to 47% if implanted elsewhere and decreased for each site with grid size (Graph 1).

Seizure outcome

A resection was performed in 58 patients (81.7%). Follow up duration ranged from 300 to 5,400 days (median 906, IQR 491; 1440). Good seizure control was achieved in 77.6% (Engel Classes 1 and 2) and 63.8% became completely seizure free (Table 5) [13]. The size of the grid, total number of electrodes and grids implanted and the site of implantation were not associated with a poor seizure outcome.

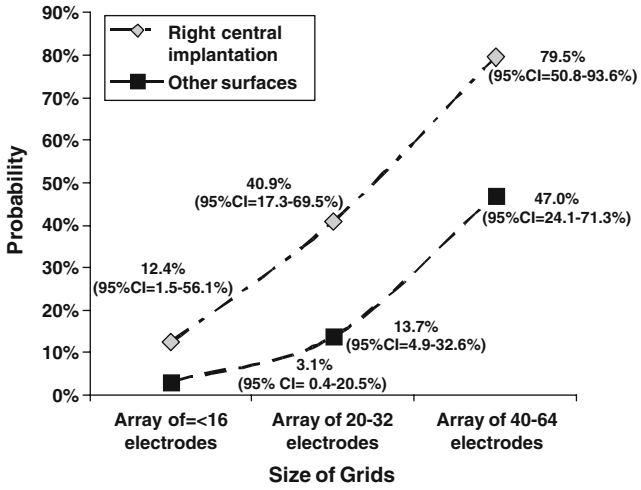
Discussion

As epilepsy surgery is increasingly offered to patients with extratemporal non-lesional epilepsy, invasive subdural monitoring will continue to play an important role in determining the epileptogenic zone and adjacent functional eloquent cortex [2, 23]. A thorough understanding of adverse outcomes and potential risk factors associated with the implantation of subdural grid electrodes is vital for early detection and successful management [15].

In our series, 68% had extratemporal epilepsy and 45% of cases were non-lesional. Our complication rate was similar to that reported by Hamer et al. but was higher than those reported in other studies [2, 10, 15, 18, 33, 40]. Table 5 presents those studies in the literature reporting complication rates of between 0–32%. Common complica-

Table 4 Best fitting multiple logistic regression model

	Coefficient	SE	Sig.	Odds ratio	95.0% CI	
					Lower	Upper
Grids with 40–64 electrodes	3.309	1.142	0.004	27.4	2.9	256.7
Grids with 20–32 electrodes	1.585	1.144	0.166	4.9	0.5	45.9
Right central convexity	1.414	0.677	0.029	4.4	1.2	16.5
Constant	−3.429	1.061				



Graph 1 Predicted probabilites of complications

tions include epidural haematoma, subdural and intracerebral haemorrhages, cerebral infarction, infections, transient neurological deficit, status epilepticus, cerebral oedema and CSF leak. Other less commonly reported complications consist of cortical contusion, brain prolapse, tension pneumocephali, non-habitual atypical seizures and hypersensitivity-type meningovasculitis [9, 14, 15, 29, 35, 37, 41]. Direct comparisons between these studies is hampered by the varying criteria used in the definition of complications. In addition, most of the published series report patient populations monitored only with subdural strip electrodes or combinations of subdural strips and depth electrodes.

The extent of surface contact and the total volume of implanted material had been suggested to be positively associated with complications [2, 7, 18, 40]. A prospective study of 38 patients identified significant correlation with risk of infection when greater than 100 electrodes were implanted or greater than ten cables were used [40]. In a paediatric series, Onal et al. noted that all patient with more than 100 electrodes implanted required blood transfusions [29]. Univariate analysis by Hamer et al. showed a higher number of implanted electrodes was a risk factor for complications, particularly neurological deficits [18]. Similarly, our univariate analysis showed that the larger grids and increased number of electrodes correlated with complications. However, only the size of the grid was shown in our study to be an independent predictor of complication. This is not surprising as larger grids were used in patients requiring larger number of electrodes. In a Swedish multicentre study on complications after epilepsy surgery, 14.3% of those implanted with subdural grids had complications as compared to 3.8% implanted with strips [33]. Of the four haematomas reported after grid implantation, all required surgical evacuation. None of the three haematomas reported after strip implantation required intervention. A study by Brehen et al. whereby 25 of their 189 patients monitored had subdural grid implantation, six (24%) of those with subdural grid had either subdural haemorrhages or transient neurological deficit. In contrast to the patients implanted with strip electrodes, only four (2.2%) cases of meningitis were reported. Similar findings were also reflected by Burneo et al. [10] where 3% of patients implanted with subdural strips had complications as compared to 13% of those implanted with grid arrays. Although the results did not reach statistical significance because 86% were monitored exclusively with subdural strips, subdural grids appeared to be associated with higher complication rate. [10]. Grid arrays covering a wide surface may be more rigid, exerting greater surface tension over more of the cortical surface and providing less accommodation to any cerebral oedema arising from subclinical seizures or the trauma of the surgery [17, 34]. These factors could lead to increased intracranial pressure and interference with both arterial and venous circulation.

The site of implantation as a risk factor for complications had not been previously addressed. Some studies had suggested that left-sided and bilateral hemispheric implantation caused a higher rate of neurological deficit or subdural haemorrhages [18, 24]. We found a statistically significant increased complication rate in patients implanted over the right central cortical surface. The central region is responsible for important motor and sensory function and may therefore be more sensitive to trauma. The reasons why right central implantation should increase risk are not clear. Increased mortality has been associated in patients following right sided cortical resections in epilepsy surgery [19, 28]. Interestingly, right hemispheric strokes have also been found to have a poorer outcome [5, 11, 39]. Further investigations are warranted to understand the differential physiological and cellular vulnerability between the hemispheres.

The use of dexamathasone was not found to be associated with increased risk of complications in our study. Other authors had suggested that the use of prophylactic steroids decreases complication rate [18]. A recent study by Araki et al. found that 9% of those receiving prophylactic steroid experienced cerebral oedema compared to 21.6% of patients not on steroids in patients undergoing invasive monitoring [4].

Mortality in all neurosurgical procedures is reported to be approximately 1% [3, 38]. The exact mortality rate from intracranial monitoring is not known as many centres have not published their experience. From the limited published reports, the mortality rate is between 0.4% and 1.4% [12, 15, 18, 26, 30, 34]. The causes of death reported have included cerebral haemorrhages, status epilepticus and cerebral oedema. Two cases of sudden unexpected death (SUDEP) have been reported during intracranial monitoring [8, 22]. Our two patients have probably suffered raised intracranial pressure due to venous stasis/infarction and postictal central

Table 5 Summary of published papers

	Type of electrodes	Reported overall complication rate related to subdural implantation	Transient neurological deficit	Cerebral haemorrhage	Infection/ aseptic meningitis	Infarction	Status epilepticus	Cerebral oedema	Other complications related to subdural implantation	Comment
Fountas 2007 [15] 185 patients studied	Grid ± strip ± depth	17 (9.2%)	2 (1.1%)	3 Symptomatic epidural (1.6%) 2 SDH (1.1%) 4 CT negative SDH	2 Osteomyelitis (1.1%) 3 positive culture only (1.6%)			2 (1.1%)	5 non-habitual atypical seizures (2.7%)[a]	2 death (uncontrolled brain oedema; stiff lung syndrome from aspiration pneumonia[b])
Alarcon 2006 [2] 105 patients studied	Grid and/or strips and/or depth	7 (6.6%)	1 (1%)	3 SDH (2.9%) 1 ICH from depth implantation (1%)	2 osteomyelitis (1.9%)	None	None	None	None	Study concluded that patients with normal neuroimaging had favourable surgical outcome equal to those with abnormal MRI
Rydenhag 2001 [33] 205 monitoring sessions	Grids, strips, depths, foramen ovale and epidural	13 (6.3%)	None	1 Epidural (0.48%) 6 SDH (2.9%)	4 infections (2%)	None	None	None	2 cases of significant electrode dislocation	Haematoma (n=7) occurred only in the 173 cases implanted with subdural electrodes. Evaluation with grids had a highest complication rate.
Wiggins 1999 [40] 38 patients studied	Grids, strips and depth	4 (10.5%)	None	1 Epidural (2.6%)	3 (7.9%) of the 5 positive culture were considered significant and treated with antibiotics	None	None	None	None	A prospective study examining infection rate during invasive monitoring
Fullagar 1993 [16] 567 patients studied	Strips	9 (1.59%)	None	None	2 meningitis 1 brain abscess 1 wound infection 1 cerebritis 1 subdural empyema 2 cerebral abscess 1 nonspecific inflammation (Antibiotic arm 0.85% vs no Antibiotic arm 2.76%)	None	None	None	None	Extension of study performed by Wyler 1991 (42) found that prophylactic antibiotics reduce infection rate by threefold.
Wyler 1991 [42] 350 patients studied	Strips	3 (0.9)%	None	None	2 meningitis 1 brain abscess	None	None	None	None	3 superficial wound infections were considered as minor complication and not included
Rosenbaum 1986 [31] 50 patients studied	Strips	None	None	None	None	None	None	None	None	
Wyler 1984 [41] 30 monitored	Strips	2 (6.7%)	None	None	1 brain abscess (3.3%)	None	None	None	1 cortical contusion (3.3%)	Subdural strips were inserted through blur holes

sessions in 28 patients										
Burneo 2006 [10][c]	Grids, strips and/or depth	6 (5.2%)	1 (0.9%)	1 Epidural (0.9%)	1 meningitis (0.9%; associated with transient neurologic deficits)	none	1 (0.9%)	none	none	Complications were seen in 3% with subdural strips and 13% in subdural grids.
116 monitored sessions in 115 patients				2 ICH (1.7%)	1 aseptic meningitis (0.9%)					
Hamer 2002 [18][c]	Grids and strips	52 (26.3%)	22 (11.1%)	5 SDH (2.5%)	6 osteomyelitis (3%)	3 (1.5%)	None	16 (8.1%)	1 intraoperative death (0.5%) from sagittal sinus injury causing uncontrolled bleeding	All cases of SDH, cerebral oedema and superior cavernous sinus syndrome caused transient neurologic deficit
198 monitored sessions in 187 patients					14 meningitis (7.1%) 3 wound infection (1.5%) 1 sepsis (0.5%) 30 positive cultures only (15.2%)				1 superior cavernous sinus syndrome 5 had altered consciousness with unknown cause	
Lee 2000 [24][c]	Grids, strip and depth	8 (16%)	N/A	1 Epidural (2%)	1 osteomyelitis (2%)	0	None	1 (2%)	None	Surgical evacuation was required in all cases of SDH. The study lead to procedural operative modification with placement of subdural drainage catheter.
50 monitored sessions in 49 patients				4 SDH (8%)	1 brain abscess (2%)					
Behren 1997 [7][c] 279 monitored sessions in 189 patients	Grids, strips and/or depth	10 (3.6%)	2 (0.7%)	4 SDH (1.4%) (2 asymptomatic; 2 transient hemiparesis)	4 meningitis (1.4%)	None	None	None	2 permanent deficits occurred when grids were placed scarred cortex	Of the 10 cases that had complications, 6 had grid electrodes (out of the total of 25 subdural grid implanted). Of 186 strip implantation, 4 had meningitis.
Johnston 2006[d] [21]	Grids, strips and depth	26 (21.3%)	4 (3.3%)	1 SDH (0.8%)	1 osteomyelitis (0.8%)				1 fractured strip electrode,	7 patients with electrode re-implantations were also included by the authors as a complication.
122 monitored sessions in 112 patients					6 wound infections (4.9%) (3 required debridement) 3 aseptic meningitis (2.5%)				2 CSF leak requiring surgery, 1 symptomatic pneumocephalus	
Musleh 2006[d] [27]	Grids or depths	4 (12.1%)	None	None	1 infected subgalael fluid	None	None	None	1 unexplained fever	Infection risk was found to be significantly higher in reimplantion
33 monitoring sessions in 29 patients					1 wound infection				1 case of prolonged prothrombin time of unknown etiology	
Leijten 2006[d] [25] 22 patients studied	Grids	4 (18.1%)	4 (18.1%)	1 SDH (4.5%)	4				4 patients had increased seizures requiring intravenous anticonvulsant	

Table 5 (continued)

	Type of electrodes	Reported overall complication rate related to subdural implantation	Transient neurological deficit	Cerebral haemorrhage	Infection/ aseptic meningitis	Infarction	Status epilepticus	Cerebral oedema	Other complications related to subdural implantation	Comment
Bauman 2005[d] [6] 30 monitoring sessions in 15 patients	Grids and strips	2 (6.7%)	None	None	1 empyema (3.3%) 1 aseptic meningitis (3.3%)	None	None	None		Multi-stage approach (monitoring – resection-reimplanation—final resection
Onal 2003[d] [29] 35 monitored session in 35 pt	Grids, strips and depth	N/A	N/A	5 SDH (14%) 3 ICH (9%)	1 aseptic meningitis (3%) 3 wound infection (9%) (1 of the patient with wound infection also had osteomyelitis and epidural abscess)	None	None	5 (14%)	1CSF leak requiring surgery (3%)	28 patients required blood transfusion. The authors found that size and number of electrodes correlated to blood loss.
Zaccariotti 1999[d] [43] 45 monitored session in 44 patients	Grids and strips	2 (4.4%)	1 (2.2%) (secondarily to SDH)	1 SDH (2.2%)	1 positive culture only (2.2%)	None	1 (2.2%)	None	None	Age was not a risk factor in paediatric group
Silberbusch 1998[c] [35] 54 monitored sessions in 51 patients	Grids	17 (31.5%)	1 (1.9%)	4 SDH (7.4%) 2 ICH (3.7%)	1 probable brain abscess (1.9%) I cerebritis (1.9%)				4 brain herniation (7.4%) 2 tension pnuemocephali (3.7%) 2 extraaxial CSF collection (3.7%)	In all but 4 cases, the detected complications were not clinical apparent and did not require specific treatment
Adelson 1995[d] [1] 31 patients studied	Grids and strips	1 (3.2%)	None	1 SDH (3.2%)	None	None	None	None	None	
Morrison 1992[d] [26] 79 patients had surgery of which 48 had subdural electrode placement	Grids and strips	14 (29.2%)	N/A	None	6 wound infections	None	1 (2.1%)	4 (8.3%)	3 subgaleal fluid collection	1 patient (2.1%) went into status epilepticus and died with malignant cerebral oedema

SDH subdural haemorrhage, *ICH* intracranial haemorrhage

[a] Four of the five patients had subdural haematoma identified at surgery but had no detected collections on MRI and CT

[b] Not considered to be related to subdural grid implantation

[c] Paediatric and adult population

[d] Paediatric centres

apnoea. Both of our patients had devastating cerebral infarction likely related to posterior cerebral artery occlusion structures in the setting of this raised ICP. The first patient appeared to have suffered cerebral infarction under the parietal grids; this may have been due to a venous occlusion although a thrombosis was not demonstrated. We incorrectly interpreted the upper limb weakness as a postictal hemiparesis and managed her conservatively. The outcome may have been different if we had removed the grids on the evening of the procedure. As a result of that case we monitored intracranial pressure in the first 24 h postoperatively in subsequent cases. The second patient was found deeply unconscious without a witnessed seizure. The lack of continuous EEG monitoring precludes interrogation of the related EEG sequence and the subsequent burst suppression pattern is consistent with his coma. We propose he developed postictal central apnoea following a subclinical seizure; this had lead to raised ICP and venous stasis in relation to the implanted grids and in turn led to a further rise in ICP, herniation and posterior cerebral artery occlusion. The urgent CT scan showed midline shift although no fixed venous or arterial thrombosis could be demonstrated in subsequent MRI, MRA and MRV.

Both of our patients with catastrophic cerebral infarction had large areas of encephalomalacia covered by the intracranial electrodes arrays. Brehen et al. reported two patients who had undergone previous surgery suffered permanent deficits following subdural grid placement over the scarred area [7]. It is of note that two patients with previous cranial radiotherapy had been reported to have developed severe neurological deficits with subdural electrode monitoring [20]. The authors suggested that in a patient with pre-existing cerebral tissue damage, the placement of subdural grid electrodes might cause additional vascular or glial compromise, resulting in neurological decompensation [20].

We have endeavoured to investigate risk factors associated with complications and performed a detailed literature review. As a result of our experience, we ceased monitoring with intracranial electrodes and invited internal and external review of the protocols in 2004–2005. We put in place a new protocol for the management of video EEG monitoring with invasive EEG that includes; one to one nursing ratio, hourly observations for the initial 48 h postoperatively and continuous heart rate and blood pressure, oxygen saturation and intracranial pressure monitoring throughout the course of the evaluation. The video-EEG evaluation is now continuously recorded and stored. An external ventricular drain is inserted routinely so that all patients have continuous ICP monitoring throughout their monitoring session. The post-operative MRI is preformed 2–4 h following implantation of the electrodes before the patient is transferred to the monitoring ward. The patient's relative or carer has been replaced by a registered nurse trained in the detection of seizures. We are now using smaller subdural electrode arrays. Most often 2×8 and not larger than 4×8 grid arrays. A prospective study following these changes is in progress.

The effects of uncontrolled seizures are debilitating. Although invasive monitoring provides the opportunity of seizure freedom by precisely identify the seizure focus, it can be associated with significant complications. It is therefore of paramount importance to thoroughly understand all of the potential risk factors associated with complications.

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Comments

The authors report on the complications with subdural electrode recording in 89 presurgical epilepsy patients. The study is well written. The surgical technique and its complications are presented well and conclusive. The pictures and tables are well selected. I congratulate the authors to the honest presentation of a rather high complication rate. Because of this honest presentation, it will be of particularly interesting to the neurosurgical readership which conclusion were drawn and what changes of protocol were performed after careful analysis of the data.

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