Complications of invasive subdural grid monitoring in children with epilepsy

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Object. This study was performed to evaluate the complications of invasive subdural grid monitoring during epilepsy surgery in children.

Methods. The authors retrospectively reviewed the records of 35 consecutive children with intractable localization-related epilepsy who underwent invasive video electroencephalography (EEG) with subdural grid electrodes at The Hospital for Sick Children between 1996 and 2001. After subdural grid monitoring and identification of the epileptic regions, cortical excisions and/or multiple subpial transections (MSTs) were performed. Complications after these procedures were then categorized as either surgical or neurological.

There were 17 male and 18 female patients whose mean age was 11.7 years. The duration of epilepsy before surgery ranged from 2 to 17 years (mean 8.3 years). Fifteen children (43%) had previously undergone surgical procedures for epilepsy. The number of electrodes on the grids ranged from 40 to 117 (mean 95). During invasive video EEG, cerebrospinal fluid leaks occurred in seven patients. Also, cerebral edema (five patients), subdural hematoma (five patients), and intracerebral hematoma (three patients) were observed on postprocedural imaging studies but did not require surgical intervention. Hypertrophic scars on the scalp were observed in nine patients. There were three infections, including one case of osteomyelitis and two superficial wound infections. Blood loss and the amounts of subsequent transfusions correlated directly with the size and number of electrodes on the grids (p < 0.001). Twenty-eight children derived significant benefit from cortical resections and MSTs, with a more than 50% reduction of seizures and a mean follow-up period of 30 months.

Conclusions. The results of this study indicate that carefully selected pediatric patients with intractable epilepsy can benefit from subdural invasive monitoring procedures that entail definite but acceptable risks.

KEY WORDS • subdural electrode grid • invasive monitoring • epilepsy surgery • complication • children

The prevalence of epilepsy is reported to be between 4.3 and 9.3 persons per 1000. Ninety percent of patients with epilepsy suffer their first seizure in the first two decades of life, and in 10 to 20% of this population the disease is known to be refractory to drug therapy or the patients are affected by the serious side effects of the medications. Because of this large number of children with refractory epilepsy, and because there is still a significant mortality rate associated with pediatric epilepsy, there has been growing interest in providing surgical options for patients whose epilepsy can be localized. Although there are reports in which noninvasive surgical protocols have been followed with some good results, increasingly there has been interest in performing invasive monitoring to identify patients with nonlateralized epilepsy who could potentially benefit from surgery. In addition to depth electrodes and multiple strip electrodes, which have been the most frequently used invasive monitoring tools, the introduction of large subdural grids for chronic EEG recordings in the 1980s has had a major impact on identifying patients who are eligible for surgery. Chronic subdural grid recordings provide the epilepsy treatment team with more accurate delineation of regions of eloquent cortex and epileptogenic foci than do noninvasive strategies or awake craniotomy. The information derived from chronic subdural recordings can then be used to plan an aggressive approach to the epileptogenic focus to maximize favorable outcomes.

Although the focus of most publications on chronic invasive monitoring for epilepsy is understandably on seizure outcome, the accumulated data on the adverse events associated with subdural grid surgery are still insufficient. There have been a few reports previously that have emphasized the complications of subdural grid monitoring, however, none of these studies...
and 15 over the right hemisphere. Thirty-four patients had (mean 8.3 years). Twenty grids were placed over the left years). The duration of epilepsy ranged from 2 to 17 years tients, whose ages ranged from 2 to 19 years (mean 11.7 ture outcome data. There were 17 male and 18 female pa-
form the basis of this study. Table 1 describes the patient video EEG-derived data between June 1996 and June 2001 tation of large subdural grid electrodes to record invasive
resection not performed.

35 11, F lt frontal lt hemisphere lt frontoparietal cortical dysplasia I 18
34 16, F rt frontotemporal rt hemisphere rt frontoparietal gliosis III 21
33 14, M lt temporal lt hemisphere lt temporal gliosis III 25
32 15, M rt frontotemporal rt hemisphere rt frontoparietal gliosis III 21
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30 2, F rt central rt hemisphere rt central gliosis II 23
29 4, F lt parietooccipital lt hemisphere lt parietooccipital tuberous sclerosis I 57
28 1, F rt parietooccipital rt hemisphere rt parietooccipital Rasmussen syndrome IV 41
27 7, M rt parietooccipital rt hemisphere rt parietooccipital frontal MTS III 39
26 14, F rt central rt hemisphere rt inferior frontal cortical dysplasia I 21
25 14, F lt parietal lt hemisphere lt frontoparietal cortical dysplasia II 40
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23 13, M lt parietooccipital lt hemisphere lt occipital, perirolandic cortical dysplasia IV 25
22 9, M bilat lt hemisphere none performed NA IV 46
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20 19, F rt frontoparietal rt hemisphere rt frontoparietal gliosis III 25
19 8, F lt frontoparietal lt hemisphere lt frontoparietal gliosis IV 36
18 10, M rt occipitotemporal rt hemisphere rt occipitotemporal gliosis III 26
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14 9, F lt frontoparietal lt hemisphere lt frontoparietal cortical dysplasia IV 36
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12 14, F rt frontal rt hemisphere rt frontal gliosis III 26
11 16, F rt perirolandic rt hemisphere rt perirolandic NA IV 60
10 8, M rt temporoparietal rt hemisphere rt temporoparietal gliosis III 24
9 5 16, F lt mesiotemporal lt hemisphere lt temporal cortical dysplasia I 28
8 4 10, M lt frontoparietal lt hemisphere lt frontal cortical dysplasia II 38
7 15, F rt frontotemporal rt hemisphere rt frontotemporal gliosis II 45
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4 10, M lt frontoparietal lt hemisphere lt frontal gliosis II 38
3 13, M lt frontotemporal lt hemisphere lt frontal gliosis II 38
2 6, M lt perisylvian lt hemisphere lt temporal, perisylvian cortical dysplasia I 45
1 15, M rt central rt hemisphere rt posterosuperior frontal astrogliosis I 41

† Outcomes were classified according to a modified Engel scale.

has dealt exclusively with the pediatric population. Our aim in writing this report is mainly to address the complications that can arise directly from subdural grid implantation as well as the anticipated or unanticipated adverse events dependent on the natural course of the disease and surgical intervention.

Clinical Material and Methods

Patient Population

Thirty-five consecutive children who underwent implantation of large subdural grid electrodes to record invasive video EEG-derived data between June 1996 and June 2001 form the basis of this study. Table 1 describes the patient demographics, ictal onset according to invasive video EEG, grid and resection locations, pathological findings, and seizure outcome data. There were 17 male and 18 female patients, whose ages ranged from 2 to 19 years (mean 11.7 years). The duration of epilepsy ranged from 2 to 17 years (mean 8.3 years). Twenty grids were placed over the left and 15 over the right hemisphere. Thirty-four patients had contralateral strips (97%) and 17 patients (48%) had depth electrodes inserted. Fifteen children had undergone at least one previous neurosurgical intervention. Of these, five underwent multiple subdural strip insertions for seizure lateralization; six had resections involving at least one lobe, three underwent biopsy procedures, and one had an anterior or two-thirds callosotomy.

All children had intractable extratemporal epilepsy disrupting their quality of life, with or without temporal lobe localization-related epilepsies. In all patients multiple trials of anticonvulsant therapy had failed. Prior to invasive video EEG monitoring, all children had been evaluated with EEG and video EEG monitoring. Neuroimaging modalities included MR imaging. MEG [$^{19}$F]fluorodeoxyglucose–positron emission tomography scanning and/or single-photon emission CT scanning. Formal neuropsychological tests were performed on cooperative patients. Wada testing, functional MR imaging, or MEG was used to determine hemispheric language dominance.

Subdural Grid Placement

Subdural grids (Ad-Tech Medical Instrument Co., Ra-
Complications of subdural grid monitoring in children

cine, WI) were custom-made for each patient by using data from the seizure symptomatology, interictal and ictal scalp EEG recordings, interictal spike sources, and somatosensory evoked fields documented by MEG. Grid size was ultimately determined using three-dimensional MR imaging as previously reported. The electrodes were 5 mm in diameter and were embedded in a silastic sheet, with center-to-center interelectrode distances ranging from 10 to 13 mm. The grids ranged in size from 40 to 117 platinum electrodes (mean 95). In several cases, unilateral subdural electrode strips and depth electrodes were placed to capture ictal data from larger regions of the hemisphere than could be covered by the grid.

For grid placement, a generous craniotomy was fashioned, the bone flap was removed, and a large M-shaped dural opening based on the sagittal sinus was made (Fig. 1 upper left). After exposure of the hemisphere, the subdural grid was placed on the cortical surface (Fig. 1 upper right). To prevent displacement, the grid was sutured to the dura. The dura was then closed with large dural grafts, and the bone flap was hinged superiorly. Individual cables were then tunneled separately through the scalp away from the incision, and secured in place with purse-string sutures. Collodion was applied to the exit sites as a sealant; no drains were used. A bulky head dressing was applied.

Invasive Video EEG Recording

A plain skull x-ray film and a head CT scan were obtained immediately after surgery to verify grid placement (Fig. 1 lower). Subdural grids were then used for closed-circuit video EEG monitoring and for extraoperative cortical stimulation of motor, sensory, and language functions while the patient was in the intensive care unit. Any change in the level of consciousness or evolving new neurological deficit was evaluated by CT scanning. Antiepileptic medications were usually reduced and an antibiotic combination of cefotaxime and vancomycin was used during the perioperative period.

Grid Removal, Cortical Resection, and MST

After obtaining sufficient data about functional cortex and regions of epileptogenesis, the subdural grid was removed and epileptic regions were resected or treated with MSTs. After an overnight stay in the intensive care unit, the patient was transferred to the ward.

Postoperative seizure control was categorized using a modified Engel classification system. Patients in Class I were seizure free postoperatively; those in Class II had occasional seizures postsurgery but less than one per month; those in Class III had a greater than 50% decrease in postoperative seizure frequency; and patients in Class IV had a less than 50% improvement or worsening compared with their preoperative status.

Data Analysis

Complications during invasive video EEG and after cortical excision were classified as either surgical or neurological. Neurological deficits were further divided into transient or permanent ones; permanent neurological deficits...
were defined as those that persisted more than 1 year post-surgery. We performed a univariate analysis by using the Fisher t-test to evaluate differences between factors such as demographics, surgical procedure and seizure outcome. Differences that reached statistical significance were defined as those with a probability value less than 0.05.

Results

Surgical Complications

The surgical complications that occurred during and after invasive video EEG monitoring, cortical excision, and/or MST are shown in Table 2. Seven patients (20%) had CSF leaks. In four cases the leaks stopped spontaneously within 2 days; however, the other three patients required reinforcing sutures around the electrode cable exit sites. In one patient a prominent subgaleal CSF collection developed, which required placement of a lumbar drain for 5 days.

During invasive video EEG monitoring, cerebral edema was noted on neuroimages obtained in five patients (14%). One patient suffered profound brain swelling after grid implantation, which became manifest as the dura was being closed. This patient’s bone flap was left out during the monitoring period. The invasive video EEG monitoring was continued for 5 days, and the bone flap was subsequently replaced at the second stage without difficulty.

We detected SDHs in five patients (14%) on CT scans (Fig. 2); none was symptomatic. The SDHs were identified either above the grid (three patients), or under the grid (two patients) (Fig. 3), and were evacuated when the grid was explanted. None of the patients had evidence of blood dyscrasias and none of the SDHs required an additional surgical procedure to that which was planned.

We observed ICHs in four patients (11%), with three lesions diagnosed on CT scans, two of which were managed conservatively. Formation of an ICH after MST in an 8-year-old boy with Landau–Kleffner syndrome was diagnosed intraoperatively by using ultrasonography, and the lesion was removed in the same session. The other ICH diagnosed on CT scanning created an epileptogenic focus that subsided after evacuation with the aid of ultrasonography in the second stage of the operation (Fig. 4). No additional surgical procedures were required in the management of the ICHs.

The most frequent complication in this series was hypertrophic scar formation (nine patients). Even though complaints about a palpable scar were relatively common, only one patient (3%) needed reconstructive surgery involving placement of a tissue expander, scalp expansion, and scalp flap reconstruction. In five patients (14%) alopecia was a delayed postsurgical complaint. Bone loss was detected in five patients (14%), in four at the sites of burr holes.

Wound infection was observed in three children (9%). One of the two superficial wound infections required excision along the incision line when antibiotic agents failed; the other infection was managed conservatively with antibiotics and healed well. The most serious infection in the series was observed in an 11-year-old boy who underwent a right hemispheric craniotomy that was complicated by scalp infection, osteomyelitis, and epidural abscess. Two months postsurgery, a third intervention consisting of reopening of the craniotomy, evacuation and debridement of the epidural abscess, and curettage and internal fixation of the bone flap was performed. Results of wound cultures revealed a Staphylococcus aureus infection that responded to long-term antibiotic therapy. No signs of recurrent infection were detected after 48 months of follow up. One patient (3%) had suspected postoperative meningitis—this was a child with Landau–Kleffner syndrome who was readmitted with headache 2 days after discharge. High-dose intravenous ceftriaxone therapy for was administered for 10

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**Table 2**

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<th>Adverse Event</th>
<th>No. of Patients (%)</th>
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<tr>
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</tr>
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<tr>
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**Fig. 2.** Axial CT scan obtained after right cortical resection, demonstrating a small right frontal acute SDH for which no treatment was required.
days even though the culture results after lumbar puncture showed no infection.

**Blood Loss and Blood Transfusion**

Table 3 summarizes the data relating to grid size, blood loss, and requirements for transfusion. We monitored patients implanted with subdural grids for 4 to 10 days (mean 5.1.7 days). We inserted subdural strips over the contralateral hemisphere in 34 patients to evaluate contralateral ictal discharges. In 17 patients, we placed additional depth electrodes and subdural strips in the mesial temporal, interhemispheric, or basal regions. Total blood loss during the two-stage operations ranged from 150 to 1250 ml (mean 756 ± 308 ml), and the amount of blood transfused ranged from 175 to 1020 ml (mean 521 ± 368 ml). In 14 patients, the total blood loss was 1000 ml or more. The blood loss in 19 patients with fewer than 100 subdural grid electrodes was 608 ± 282 ml. In 16 patients with 100 or more electrodes, the blood loss was 943 ± 218 ml. Twelve (63%) of 19 patients who had grids with fewer than 100 electrodes required blood transfusion. All 16 patients (46%) with grids having more than 100 electrodes required blood transfusions. Of the 28 patients who received blood transfusions, 12 received them during the first craniotomy, eight during the monitoring period, and eight during the second stage, when resection or MSTs were to be performed. There were no apparent complications related to blood transfusions in this series.

**Neurological Adverse Events**

The neurological deficits related to invasive monitoring in this series are shown in Table 4. The adverse event with the highest documented frequency was hemiparesis. Twenty-four (69%) of 35 patients had mild to moderate postoperative contralateral hemiparesis, which typically resolved within the first week after surgery. No permanent hemiparesis was encountered in this series. The second most common neurological deficits were dysphasia and facial weakness, in 18 (51%) and 17 (48%) patients, respectively. Thirteen (37%) of the 35 patients had postoperative visual field defects ranging from minimal field alterations to significant contralateral hemianopsia. All visual field defects were permanent and expected, considering the targeted surgical approach to the epileptic foci. Five patients (14%) experienced an acute postoperative increase in seizures.

**Seizure Outcome**

Follow up ranged from 18 to 60 months (mean 30 ± 13 months). Nineteen patients (54%) achieved good seizure control and were either seizure free (14) or had occasional seizures (five) (Engel Classes I and II; Table 1). Nine patients (26%) had a greater than 50% reduction in seizure frequency (Engel Class III); the remaining seven patients (20%) had residual seizures without improvement (Engel Class IV). In two of these patients, resection or MSTs could not be offered because we failed to localize the ictal onset zone by using invasive video EEG.

**Results of Statistical Comparisons**

On univariate analysis, age, sex, total amounts of blood loss, and blood transfusion were not significantly different between the good seizure outcome (Engel Classes I and II) and poor seizure outcome groups (Engel Classes III and IV). Only the relationship between number of grid electrodes (using grids with 100 electrodes as a reference) and blood loss reached statistical significance (p < 0.001).

**Discussion**

In recent years, early epilepsy surgery for children has been advocated by many groups. The main rationale for this trend is the favorable response

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**Fig. 3.** Intraoperative photograph showing a linear SDH under the grid, which caused partial interference with electrophysiological recordings.

**Fig. 4.** Axial CT scan obtained in a patient in whom a large right subdural grid and contralateral strip electrodes were implanted. Note the ICH in the right temporal lobe. The presumed mechanism of this hematoma was occlusion of venous outflow by the grid, which led to hemorrhagic infarction.
of the child’s brain to cortical resection. After successful epilepsy surgery, the pediatric patient may demonstrate enhanced neurocognitive development because of the elimination or decreased frequency of epileptic discharges. In addition, a benefit is derived in this group from the tapering or cessation of anticonvulsant drugs, many of which lead to significant complications with long-term use.28,37

Despite advances in preoperative imaging modalities such as positron emission tomography, functional MR imaging, and MEG, these imaging methods do not as yet provide an unequivocal localization of the seizure focus.40,41,43,52,55 Nevertheless, promising results have been reported in carefully selected patients with epilepsy in whom invasive monitoring was used.2,3,10,19,48,49,58,61,64,68 Bruce and Bizzi10 recently reported a success rate of between 80 and 90% in identifying the epileptogenic foci in children in whom invasive monitoring strategies were used.

Reports on the complications related to invasive monitoring are far from uniform.31,16,20,49,53,54,61,64,65 With the exception of a few reports specifically on this topic, in which results in the adult population are primarily discussed,34,39,78,107 the adverse effects of invasive monitoring in a series of pediatric patients have not been critically analyzed and reported systematically. In our series, we categorized adverse events caused by subdural grid implantation as either surgical or neurological; the latter were either transient or permanent. For the purposes of this report, emphasis has been placed on the surgical complications related to subdural grid implantation.

Many of the adverse events reported in our series were directly related to the surgical site and to the size of the craniotomy. One of the most common complications of invasive subdural grid monitoring is CSF leakage. Adelson, et al.,2 reported on four (13%) of 31 patients with transient CSF leakage through electrode exit sites. These patients were all successfully treated with collodion application and monitoring was continued. In the study by Swartz, et al.,56 19% CSF leakage was reported as an expected adverse event in their series of invasive monitoring procedures. Subgaleal or epidural fluid collection with subsequent surgery for drainage has been reported in some series.28,37

### Table 3

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<th>Case No.</th>
<th>MP (days)</th>
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<th>Type of Epilepsy Op</th>
<th>Blood Loss (ml)</th>
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* AH = amygdalohippocampectomy; CR = cortical resection; front = frontal; FTO = frontotemporoccipital; lob = lobectomy; MP = monitoring period; none = no cortical resection or MSTs performed; temp = temporal; — = no transfusion.*
Complications of subdural grid monitoring in children

ly 20%; fortunately, the CSF leaks stopped spontaneously in four of seven patients. To prevent CSF leaks from occurring, we have found that purse-string sutures tied tightly around the electrode cable exit sites and reinforced with collodion work well. None of our patients developed meningitis.

Complications such as wound infection, meningitis, osteomyelitis, and epidural abscess formation are also reported in several invasive monitoring series.6,10,19,28,29 In their series of 85 children who underwent invasive monitoring, Bruce and Bizzi10 reported on three patients (4%) who needed bone flap removal because of delayed infection. In their series there were two additional superficial wound infections (2%). In his experience with epidural electrode arrays, Goldring19 reported one bone flap removal without infection. In one of his first patients to undergo invasive monitoring, the bone flap was removed and steam-autoclaved before its insertion during the second procedure; however, aseptic necrosis developed and it had to be removed. Kim, et al.,28 reported on three children with postoperative wound infection, two requiring bone flap removal and the third with meningitis. Two cases of major infection (4%) in 49 patients with 50 subdural grids and/or strips or depth electrodes were reported by Lee, et al.29 One was osteomyelitis necessitating bone flap removal and the other one was a brain abscess requiring stereotactic aspiration. Behrens, et al.,6 reported a 3.5% rate of wound infection in their series. They reported four cases of meningitis during electrode implantation despite negative results on microbial cultures. Uematsu, et al.,58 described two cases of postoperative wound infection (7%) in a series of 28 patients who underwent grid implantation. Morrison, et al.,37 described two superficial wound infections, one sterile abscess, and three bone flap removals requiring late cranioplasties in their pediatric epilepsy experience. Recently, Hammer, et al.,26 reported a 12.1% infection rate in their large series of 187 patients with complications caused by invasive video EEG monitoring. In their series there was a 7.1% meningitis rate and a 3% osteomyelitis rate.

With such diverse results in these heterogeneous series, it is hard to reach reliable conclusions regarding the infection rate after invasive monitoring. Nonetheless, our results, in which three patients (9%) had wound infections, one of them accompanying osteomyelitis, are in accordance with those reported previously. The patient with osteomyelitis described here had a large hemicalvarial bone flap that was affixed to the skull with absorbable sutures. This complication occurred early in our series, and we have since changed our method of bone flap replacement to rigid fixation with titanium microplates and screws. We hope that such rigid fixation systems accompanied by the use of prophylactic antibiotics, in our case a third-generation cephalosporin and vancomycin, will help further decrease the rate of surgical infections.

A potentially serious problem in patients with implanted grids is cerebral edema. Morrison, et al.,37 reported on one child in their series who died after placement of subdural electrodes. This child went into status epilepticus and died of malignant cerebral edema despite a large frontal lobectomy. In the same article, Morrison, et al., reported on four (8%) of 48 patients with cerebral edema who underwent invasive monitoring and in whom the second-stage operation was performed sooner than planned. Lee, et al.,28 reported on a patient with a brain tumor who developed signs of increased intracranial pressure caused by cerebral edema after grid implantation, with resultant emergency grid removal and reinsertion 1 week later.

In our series, postoperative neuroimaging-verified cerebral edema was encountered in five cases; no patient required an earlier second-stage procedure because of cerebral edema. Only one patient developed moderate acute brain swelling during dural closure after grid implantation. This patient’s bone flap was temporarily removed during the monitoring phase without serious sequelae. To prevent clinically significant cerebral edema from occurring in patients who undergo grid implantation, we have found that the use of large duraplasties and hinging the bone flap at its superior margin with sutures is quite effective. Care must also be taken to ensure that the edges of large grids do not compress and impede the outflow of major draining vessels such as the vein of Labbé or Trolard as they enter the dural venous sinuses.

Several patients in our series suffered hemorrhagic adverse events, none of which required an additional surgical session. Five patients had SDHs and four had ICHs. Two patients had epidural fluid collections. Three of the SDHs and two of the ICHs were evacuated at the time of the second craniotomy and grid removal. In six (55%) of the 11 patients in this group the complications were managed conservatively, with no related morbidity observed on follow-up review, and 45% were treated surgically. In one of the patients with Landau–Kleffner syndrome who underwent extensive MST, an ICH was detected using ultrasonography and evacuated intraoperatively. A second case of ICH diagnosed on CT scans after grid implantation was epileptogenic on EEG, and was thought to be caused by compression of the vein of Labbé by the edges of the grid. This hematoma was evacuated at the second operation with the aid of ultrasonography, and this case illustrates the practical use of intraoperative ultrasonography, especially after extensive MSTs. Once again, avoidance of venous compression by the edges of the grids is emphasized to avoid complications such as ICH.10,28,36

<table>
<thead>
<tr>
<th>Complication</th>
<th>Total (%)</th>
<th>Transient (%)</th>
<th>Permanent (%)</th>
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<tr>
<td>hemiparesis</td>
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<td>24 (69)</td>
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<td>dysphasia</td>
<td>18 (51)</td>
<td>17 (48)</td>
<td>1 (3)</td>
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<td>facial weakness</td>
<td>17 (48)</td>
<td>14 (39)</td>
<td>3 (9)</td>
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<td>visual field defect</td>
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<td>13 (37)</td>
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<tr>
<td>dysnomia</td>
<td>12 (34)</td>
<td>10 (28)</td>
<td>2 (6)</td>
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<td>neglect syndrome</td>
<td>10 (28)</td>
<td>8 (22)</td>
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<td>hemisensorial deficit</td>
<td>7 (20)</td>
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<td>2 (6)</td>
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<td>postop increase in seizures</td>
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<td>5 (14)</td>
<td>0 (0)</td>
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<tr>
<td>gait disturbance</td>
<td>5 (14)</td>
<td>5 (14)</td>
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<td>skew deviation</td>
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<td>1 (3)</td>
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<td>diplopia</td>
<td>2 (6)</td>
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<td>1 (3)</td>
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TABLE 4 Neurological complications in patients in whom subdural grids were implanted
Whereas Lee, et al.,29 reported hematomas between the dura mater and subdural grid in some cases, two of our three SDHs were found beneath the grid. Adelson, et al.,2 reported on one case of subacute SDH (3%) in 31 children after subdural grid placement. This patient had undergone previous intracranial surgery and the hematoma was thought to have been caused by subdural scar formation. Behrens, et al.,7 described one patient (7%) who had a clinically silent SDH after grid implantation among 15 such cases; the clot was removed during the second procedure. Kim, et al.,28 reported one hematoma in 27 patients who underwent invasive monitoring. Lee, et al., described four cases of SDH (8%) overlying the grids; these lesions required evacuation, and the electrodes were left in situ for further monitoring. Two cases of transient epileptic foci with intracranial hemorrhage in patients with subdural and epidural electrodes were reported by Wennberg, et al.,62 one of them an SDH overlying the grid and the other a focal intraparenchymal hemorrhage underlying the affected epidural electrodes. Bruce and Bizzi10 emphasized that, to prevent cortical laceration, there should be no sharp edges on the grids if they are cut to a smaller size to fit the targeted region.

Because of the large grids that were fashioned in most of our patients, and the large craniotomies that had to be performed as a result, the majority of patients required a blood transfusion at some point during their hospitalization (only 20% of all patients did not require blood transfusion). Blood loss and subsequent transfusions correlated directly and significantly with the size and number of electrodes on the grids. These interesting preliminary observations should be confirmed in larger series. We have found that most of the blood loss in these cases occurs when the large bone flaps are turned at the initial surgery, especially along the most medial parasagittal cuts. Care should be taken in children to strip the dura at the coronal and lambdoid suture lines, because the dura can be quite adherent to the overlying bone in these locations. The large bone flaps should be fashioned quickly, and the medial edges of the craniotomy secured with bone wax and dural tenting sutures to control blood loss. As an alternative to these suggested methods, one could fashion smaller grids routinely, although such smaller grids might not capture critical data regarding the spread of the seizure focus and secondary zones of epileptogenesis.

The neurological complications reported here are most likely the result of the extent and location of the cortical resections and/or the MSTs rather than the implantation of the subdural grids or strips. These complications are similar to those reported in the literature for surgery to relieve seizures, such as we have performed.6,20,24,25,37 Seizure outcome in the present series revealed that 80% of patients benefited from surgery, with at least a more than 50% decrease in seizure frequency. Eighty-seven percent of the patients reported on by Adelson, et al.,4 and 80% of the patients by Keene, et al.,25 had outcomes categorized as modified Engel Classes I to III. Using the same scale, Bruce and Bizzi10 reported 73% success in their pediatric extratemporal epilepsy surgery series. Kim, et al.,24 reported 68% good results, with outcomes of Engel Classes I to II in 38 pediatric patients. Favorable results have been reported in pediatric epilepsy surgery series, with the percentage of seizure-free patients ranging from 52 to 78%.3,24,25,28 Fourteen (40%) of 35 patients in our series became seizure free. The relatively low percentage of such patients in our group may be attributed to the relative absence of patients with tumors, and to the complex nonlesional epilepsy for which invasive monitoring was required.

Conclusions

Although serious adverse events related to surgical intervention and chronic invasive monitoring have been noted in this series and in the literature, invasive subdural grid monitoring is a valuable method for localizing the epileptogenic zone and for performing functional mapping in children with epilepsy. Given the acceptable complication rate overall in our series, which spanned 5 years, we believe that carefully selected pediatric patients with intractable epilepsy can be well served by chronic invasive monitoring with subdural grids and/or strips or depth electrodes, and by the planned cortical resections that result from information gathered during this monitoring. It must be acknowledged that the procedures discussed here are significant ones, especially in young children. When possible, complication avoidance strategies for these procedures have been described. Attempts to find accurate, noninvasive strategies to manage seizures in these patients, such as with MEG and new technologies as they evolve, should continue.

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