RESEARCH PAPER

Unilobar surgery for symptomatic epileptic spasms

Carmen Barba1, Roberto Mai2, Laura Grisotto3, Francesca Gozzo2, Simona Pellacani1,4, Laura Tassi2, Stefano Francione2, Flavio Giordano3, Francesco Cardinale2 & Renzo Guerrini1,4

1Neuroscience Department, Children’s Hospital Anna Meyer-University of Florence, Florence, Italy
2Claudio Munari* Epilepsy Surgery Center, Niguarda Hospital, Milan, Italy
3Department of Statistics, Computer Science, Applications “G. Parenti”, University of Florence, Florence, Italy
4RCCS Stella Maris, Pisa, Italy
5Neurosurgery Department, Children’s Hospital Anna Meyer-University of Florence, Florence, Italy

Correspondence
Renzo Guerrini, Neuroscience Department, Children’s Hospital Meyer-University of Florence, Viale Pieraccini 24, 50139, Florence, Italy. Tel: +39055 566 2573; Fax: +39055566 62329; E-mail: renzo.guerrini@meyer.it

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Abstract

Objective: To assess factors associated with favorable seizure outcome after surgery for symptomatic epileptic spasms and improve knowledge on pathophysiology of this seizure type. Methods: Inclusion criteria were: (1) age between 6 months and 15 years at surgery; (2) active epileptic spasms; (3) follow-up after surgery >1 year. Results: We retrospectively studied 80 children (aged 1.3 ± 2 years at seizure onset; 5.8 ± 4 years at surgery, 11.7 ± 5.7 years at last follow up). Magnetic resonance imaging (MRI) revealed structural abnormalities in 77/80 patients (96.3%; unilateral in 69: 89.6%). We performed invasive recordings in 24 patients (30%). In 21 patients in whom MRI or histopathology detected a lesion, electrodes exploring it constantly captured initial ictal activity at spasm onset. Fifty-eight patients (72.5%) underwent unilobar and 22 (27.5%) multilobar or hemicerebral procedures. At last follow-up, 49 patients (61.3%) were in Engel class I. Multivariate logistic models showed completeness of resection of the seizure onset zone (OR = 0.016, 95% CI: 0.002, 0.122) and of the MRI visible lesion (OR = 0.179, 95% CI: 0.032, 0.999) to be significantly associated with Engel class I A outcome. Unfavorable outcome was associated with an older age at surgery, when it reflected a longer duration of epilepsy (OR = 1.383, 95% CI: 0.994,1.926). Interpretation: Data emerging from invasive recordings and the good seizure outcome following removal of discrete epileptogenic lesions support a focal cortical origin of spasms. In patients with discrete epileptogenic lesions, the pragmatic approach to surgery should follow the same principles applied to focal epilepsy favoring, whenever possible, unilobar, one-stage resections.

Introduction

Epileptic spasms account for 13–45.5% of new onset epilepsies in infancy and represent the most common manifestation of intractable epilepsy in the first 3 years of life. Although epilepsy surgery is an established treatment option in children with drug-resistant seizures, literature on surgical treatment of drug-resistant spasms is limited to small retrospective series with short follow-ups. In two different cohorts, surgery for infantile spasms was performed in 6% of 543 children operated during calendar year 2004 and in 4.4% of 238 children operated between 1990 and 2011. Epilepsy surgery for spasms poses considerable challenges as the seizure onset zone is difficult to identify and multilobar resections or hemicerebral resections are often performed, with high risk of complications and postsurgical deficits. These factors, in addition to uncertainty concerning developmental outcome, contribute to delaying surgery.

We retrospectively analysed clinical features, imaging, noninvasive and invasive EEG findings, surgical procedures and histopathology of 80 children we operated on for drug-resistant spasms and assessed factors associated with favorable seizure outcome after surgery. We also...
analyzed how intracranial EEG recordings, beyond their role in guiding the surgical strategy in a subgroup of patients, provided relevant insights into the pathophysiology of spasms.

**Methods**

**Patients**

Patients were operated at two Epilepsy Surgery Centers (Children’s Hospital Anna Meyer, Florence and “Claudio Munari” Center, Niguarda Hospital, Milan) between January 1997 and June 2014. Inclusion criteria for this study were: (1) age between 6 months and 15 years at the time of surgery; (2) epileptic spasms being a target for surgery; (3) at least 1 year follow-up after surgery.

We identified 80 patients, evaluated clinical features, imaging, invasive and noninvasive EEG findings, surgical procedures and histopathology (complete information is available in Data S2 Methods and References).

This study was approved by the Pediatric Ethical Committee on Human Experimentation of the Tuscany Region. Informed consent for all procedures was obtained for all patients.

**Results**

**Patients**

(Table 1). There were 43 males and 37 females; mean age at surgery was 5.8 ± 4 years. Spasms were the initial seizures in 46 out of 80 patients (57.5%), while the remaining 34 patients (42.5%) had focal seizures at epilepsy onset. In ten patients (12.5%) spasms were the only seizure type, while in the remaining 70 patients (87.5%) focal seizures were, or had been, also present. Age at seizure onset (all seizures) was 1.3 ± 2 years; age at spasm onset was 1.9 ± 3 years. The mean interval from seizure onset (all seizures) to surgery was 4.5 ± 3 years; the mean interval from spasms onset to surgery was 3.9 ± 3 years.

All patients were drug-resistant. Seizures occurred daily in 76 patients (95%) and weekly in the remaining 4 (5%). Spasms were symmetric in 39 patients (48.8%), asymmetric in 19 (23.8%), and both symmetric and asymmetric in the remaining 22 (27.5%).

Twenty-one patients (26.3%) had hemiparesis and three (3.8%) visual field defects.

**Neuroimaging**

(Table 1). Preoperative magnetic resonance imaging (MRI) was available for all patients. We identified structural abnormalities in 77 patients (96.3%), which were unilateral in 69 (89.6%); 51 unilobar and 18 multilobar or involving an entire hemisphere.

**Scalp video-EEG recordings**

(Table 1). Intercital scalp EEGs were available for analysis in all patients. EEG abnormalities were focal in 56 patients (70%) and diffuse in the remaining 24 (30%). Ictal scalp EEGs recordings of spasms were available for analysis in 70 patients (87.5%). They revealed the EEG counterpart of spasms to be a slow wave with superimposed low voltage fast activity in 60 patients (85.7%) and a slow or sharp wave in the remaining 10 patients (14.3%). In 25 patients (35.7%) ictal activity had an obvious focal predominance.

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**Table 1.** General characteristics, neuroimaging and electroclinical findings of 80 patients operated on for epileptic spasms.

| N°Pts | 80 |
|-------|----|
| Sex   | 43M/37F |
| Age at seizure onset | 1.3 ± 2 years |
| Age at surgery | 5.8 ± 4 years |
| Epilepsy duration | 4.5 ± 3 years |
| Type of first seizure | 46 Spasms |
| Type of first seizure | 34 Focal sz |
| Spasm semiology | 19 Asymmetric |
| Seizure frequency | 76 Daily |
| Preoperative cognitive level | 23 Borderline or mild ID |
| Preoperative neurological deficits | 21 Hemiparesis |
| Brain MRI | 77 Abnormal (69 unilateral abnormality) |
| Interictal EEG (scalp) | 24 Diffuse or multifocal |
| Ictal EEG (scalp) | 45 Diffuse or multifocal |
| Invasive EEG recordings | 22 SEEG |
| SOZ Topography | 56 Unilobar |
| SOZ vs. MRI visible lesion | 41 SOZ overlapped with lesion |
| F, female; ID, intellectual disability; IQ, cognitive level scores; L, left; M, male; Pts, patients; R, right; SEEG, stereoelectroencephalography; SOZ, seizure onset zone; Sz, seizure; MRI, magnetic resonance imaging. |
Invasive recordings

(Table S1). We performed invasive recordings in 24 patients (30%), due to either nonlocalizing electroclinical data on surface recordings (15 patients), proximity of the epileptogenic lesion to eloquent areas (6 patients) or unrevealing MRI (3 patients).

Twenty-two patients had SEEG (10–15 electrodes with 8–15 contacts each) and two patients had a combined subdural and depth electrodes approach.

During invasive recordings we captured both spasms and focal seizures in 18 patients and only spasms in six.

Intracranial EEGs during spasms demonstrated in 21 children low voltage fast activity, which was either isolated (14 patients), followed (5 patients) or preceded (2 patients) by a slow wave. In three remaining children (including two explored using a combined approach), we observed a slow or sharp wave with superimposed low voltage fast activity. In eight children, focal subclinical fast discharges preceded clinical spasms by several seconds.

Analysis of EEG data revealed that cortical areas involved at spasm onset could either remain constant or vary in subsequent clusters in the same patient (Fig. 1). In all 21 patients in whom MRI or histopathology revealed a lesion, electrodes exploring the structurally abnormal area constantly captured the initial ictal activity at spasm onset (Figs. 1,2).

Figure 1. Stereotactic scheme, Post-implantation and Preoperative MRI, Intracranial Ictal EEG. Patient 7, Table S1. (A) Stereotactic scheme, according to the bicommissural reference system (lateral view), of a right frontal SEEG exploration. Electrodes, labeled by uppercase letters, are indicated with either circles or dotted lines, depending on the orthogonal or oblique trajectory of the implantation. (B) Post-implantation sagittal T1-weighted MRI showing the exact position of each electrode. Electrodes appear as either circles or lines depending on the orthogonal or oblique trajectory of the implantation and are labeled as in the stereotactic scheme. (C) Preoperative axial T1-weighted MRI showing a right frontal anterior area of cortical thickening and abnormal folding consistent with FCD (black arrows). (D,E,F) Intracranial ictal EEG recordings during three clusters of spasms. Electrodes exploring the lesion (i.e. electrodes H, F, R, J and L; black rectangles) are always involved by initial ictal activity at spasm onset. Conversely, no consistent pattern of spread is recognizable. Electrodes are labeled as in the stereotactic scheme and postimplantation MRI. FCD, focal cortical dysplasia; MRI, magnetic resonance imaging.
When comparing different recordings in the same patient or between patients, we could not identify a consistent pattern of ictal spread during spasms. In the 18 children in whom both spasms and focal seizures were recorded, spasms either followed (7 patients), or preceded (6 patients), or occurred independently (11 patients) from focal seizures. In all patients in whom focal seizures were recorded and a lesion was demonstrated by either MRI or histopathology, or both, initial ictal activity always involved the electrodes exploring it. No consistent pattern of spread could be identified when comparing electrographic features of spasms versus focal seizures.

**Identification of the SOZ**

The SOZ had lobar distribution in 56 patients (70%), and was multilobar or involved the whole hemisphere in 24 patients (30%); it overlapped with the MRI visible lesion in 41 patients (53.2%), extended beyond it in 28 patients (36.4%) and was narrower than it in eight patients (10.4%). MRI was unrevealing in the three remaining patients.

**Neuropsychology**

(Table 1). Preoperative cognitive level scores (IQ) were available for all children. We observed borderline
cognitive skills to mild ID in 23 children (28.8%), moderate ID in 22 (27.5%) and severe ID in 18 (22.5%). Cognitive level was normal in 17 patients (21.3%).

**Surgery**

(Table 2). We performed 58 unilobar (72.5%) and 22 multilobar or hemispheric surgical procedures (27.5%).

Resection of the MRI visible lesion was deemed to be complete in 46 (57.5%) patients as was the resection of the SOZ (46 patients). Resection of both the lesion and the SOZ was complete in 33 (41.2%) patients.

Mean follow-up after surgery was 5.7 ± 4.2 years. At last follow-up, 49 patients (61.3%) were in Engel class I, 45 of whom (56.2%) were in Engel class IA category. In 29 patients (36.3%) treatment was withdrawn.

Ten out of 80 patients (12.5%) experienced minor complications after surgery. Six patients (7.5%) required reoperation due to various complications, including sinking of the parietal bone flap after lesionectomy (1), frontal subdural hematoma after lesionectomy + corticectomy (1), hydrocephalus after hemispherotomy (4). There was no surgery-related mortality. Additional information on surgical procedures and postsurgical complications is summarized in Table 2.

**Histopathology**

(Table 2). Histopathology was available for 79 patients and revealed focal cortical dysplasia (FCD) in 37 (46.8%), tumors in 12 (15.2%), gliosis in nine (11.4%), tubers in eight (10.1%), hemimegalencephaly in two (2.5%) and polymicrogyria in one (1.3%). In 10 patients (12.7%) in whom, based on MRI, we suspected FCD, histopathology revealed reactive gliosis. In one patient no tissue specimen was available after hemispherotomy.

**Statistical analysis**

Information on univariate analyses is included in Table S2 and Data S1.

**Outcome I: Engel class IA versus classes IB-IV**

(Table 3). The multivariate logistic models showed that completeness of resection of the SOZ (OR = 0.016, 95% CI: 0.002, 0.122) and of the MRI visible lesion (OR = 0.179, 95% CI: 0.032, 0.999) were both significantly associated with Engel class IA outcome.

The sensitivity analysis confirmed the results of multivariate logistic models.

The multivariate logistic models showed that completeness of resection of the SOZ (OR = 0.010, 95% CI: 0.001, 0.113) was associated with favorable seizure outcome after surgery.

The sensitivity analysis confirmed the results of multivariate logistic models.
Table 3. Multivariate logistic models.

| Outcome class IA vs IIb-IV | Odds ratio | 95% CI   | P-value |
|---------------------------|------------|----------|---------|
| Age at surgery            | 1.383      | 0.994    | 1.926   | 0.055   |
| SOZ lateralization         | 0.196      | 0.030    | 1.265   | 0.087   |
| Unilobar vs Multilobar SOZ| 0.770      | 0.124    | 4.782   | 0.779   |
| Age at seizure onset      | 0.847      | 0.414    | 1.733   | 0.649   |
| Age at spasm onset        | 0.898      | 0.496    | 1.624   | 0.721   |
| Need of Invasive recordings| 0.851      | 0.122    | 5.921   | 0.871   |
| Complet res. MRI visible lesion| 0.179   | 0.032    | 0.998   | 0.050   |
| Complet res. SOZ          | 0.016      | 0.002    | 0.122   | 0.000   |
| Cognitive_0 (normal) ref. | 1.000      |          |         |         |
| Cognitive_1 (mild ID)     | 0.422      | 0.040    | 4.505   | 0.475   |
| Cognitive_2 (moderate ID)| 0.738      | 0.064    | 8.498   | 0.807   |
| Cognitive_3 (severe ID)   | 6.850      | 0.566    | 82.959  | 0.131   |
| _cons                     | 11.530     | 0.760    | 174.810 | 0.078   |

| Outcome class I vs II-IV  | Odds ratio | 95% CI   | P-value |
|--------------------------|------------|----------|---------|
| Age at surgery            | 1.209      | 0.881    | 1.660   | 0.240   |
| SOZ lateralization         | 0.089      | 0.008    | 0.969   | 0.047   |
| Unilobar vs Multilobar SOZ| 0.967      | 0.082    | 11.453  | 0.979   |
| Age at seizure onset      | 0.482      | 0.117    | 1.983   | 0.312   |
| Age at spasm onset        | 0.795      | 0.357    | 1.770   | 0.574   |
| Neurological signs        | 3.893      | 0.468    | 32.404  | 0.209   |
| Need for Invasive recordings| 1.182      | 0.095    | 14.696  | 0.897   |
| Complet res. MRI visible lesion| 2.219  | 0.262    | 18.759  | 0.464   |
| Complet. res. SOZ         | 0.001      | 0.000    | 0.058   | 0.001   |
| Cognitive_0 (normal) ref. | 1.000      |          |         |         |
| Cognitive_1 (mild ID)     | 0.112      | 0.004    | 2.970   | 0.191   |
| Cognitive_2 (moderate ID)| 0.310      | 0.017    | 5.769   | 0.433   |
| Cognitive_3 (severe ID)   | 0.537      | 0.027    | 10.854  | 0.685   |
| Spasm semiology_0 (sym) ref| 1.000      |          |         |         |
| Spasm semiology_1. asym   | 0.168      | 0.010    | 2.818   | 0.215   |
| Spasm semiology_2: sym+asym| 11.984    | 0.560    | 256.558 | 0.112   |
| _cons                     | 34.326     | 0.920    | 1280.798| 0.056   |

| Population average logistic model for longitudinal seizure outcome | Odds ratio | 95% CI   | P-value |
|-----------------------------------------------------------------|------------|----------|---------|
| Age at surgery                                                  | 1.216      | 1.060    | 1.394   | 0.005   |
| SOZ lateralization                                              | 0.718      | 0.286    | 1.801   | 0.480   |
| Unilobar vs Multilobar SOZ                                      | 2.271      | 0.824    | 6.258   | 0.113   |
| 6 months FU ref                                                 | 1.000      |          |         |         |
| 1 Year FU                                                      | 1.144      | 0.443    | 2.955   | 0.782   |
| 2 Years FU                                                     | 1.512      | 0.574    | 3.987   | 0.403   |
| 5 Years FU                                                     | 1.489      | 0.508    | 4.371   | 0.468   |
| >5 's FU                                                       | 1.450      | 0.370    | 5.677   | 0.594   |
| Complet. res. SOZ                                              | 0.019      | 0.005    | 0.067   | 0.000   |
| SOZ superimp to lesion_0 ref                                   | 1.000      |          |         |         |
| SOZ within lesion_1 (less ext)                                 | 3.414      | 0.795    | 14.671  | 0.099   |
| SOZ extend. beyond lesion_2                                     | 4.994      | 1.803    | 13.830  | 0.002   |

(Continued)
Outcome III: Post-surgery AED withdrawal versus continuing treatment

(Table 3). The multivariate logistic models showed a significant association between completeness of the resection of the SOZ and AED withdrawal after surgery (OR = 0.37, 95% CI: 0.074, 0.762).

The sensitivity analysis confirmed the results of the multivariate logistic models.

Longitudinal outcome

(Table 3). Engel class I outcome in relation to follow-up duration was achieved in 67.5% of the 80 patients with at least 1 year follow-up, in 62.9% of 70 patients with 2 years, in 59.6% of 52 patients with 5 years and in 58.1% of 31 patients with >5 years.

The population average logistic models showed that completeness of resection of the SOZ (OR = 0.019, 95% CI: 0.005, 0.067) and later age at seizure onset (OR = 0.424, 95% CI: 0.278, 0.645) were associated with Engel class I outcome after surgery. Conversely, older age at surgery (OR = 1.216, 95% CI: 1.060, 1.394), a SOZ extending beyond the MRI visible lesion (OR = 4.994, 95% CI: 1.803,13.830) and co-occurrence of symmetric and asymmetric spasms (OR = 8.510, 95% CI: 2.530, 28.625) were associated with unfavorable seizure outcome. Comparing subgroups of children of similar ages at seizure onset but operated on 1 year apart revealed unfavorable seizure outcome in those operated later (OR = 1.216, 95% CI: 1.060, 1.394).

There was no significant effect of duration of follow-up on the risk of seizure recurrence.

Discussion

In this series, 61.3% of 80 children surgically treated for drug-resistant epileptic spasms were in Engel class I at last follow-up (mean duration of follow-up after surgery 5.7 ± 4.2 years, range: 1–18 years). Initial reports on surgery for spasms included a few selected observations demonstrating that removal of a causative lesion could be successful.15,16 Subsequent series reported average rates of seizure freedom after surgery ranging from 46% to 72%.5,8–10,13,14,17,18 However, a comparison between our own and previous series is hampered by considerable differences in inclusion criteria, presurgical protocols and surgical approaches.

In our series, 70% of patients underwent surgery without invasive recordings (‘one-stage surgery’) and 72.5% underwent unilobar removal/disconnections. In previous series most patients with spasms achieving seizure freedom received multilobar or hemispheric resections/disconnections.6–8,17 Our results suggest that unilobar procedures, which carry lower rates of complications and postsurgical deficits compared to multilobar or hemispheric resections/disconnections,6,8,13,19 are effective in selected patients.

We did not observe a significant increased risk of relapse with longer follow-ups, which is in contrast with Jonas et al.,8 figures indicating seizure freedom rates after surgery to drop from 62.5% at 6 months to 44% at 5 years. However, in our study, the lower percentage of drug-free patients at last follow-up might have partially masked surgical failures.20 In other previous series, small sample size13,18,21,22 or short follow-ups8 prevented adopting longitudinal population average logistic models.

In our series, completeness of resection of the SOZ and of the MRI visible lesion were the main factors determining favorable seizure outcome, as assessed by multivariate logistic models and sensitivity analysis. Two previous studies on surgical treatment of spasms provided indications that complete removal of the SOZ and of the area of cortical dysplasia generating a ‘leading spike’ were important determinants of favorable seizure outcome.10,21 However, due to the small sample size, in neither study could multivariate analysis be performed.

In a large series from Detroit,6 detection of an MRI visible lesion was associated per se, with seizure freedom, while the removal of the SOZ or of the lesion, or both, were not analysed as prognostic factors with respect to seizure outcome in multivariate analysis. The different

| Odds ratio | 95% CI        | P-value |
|------------|---------------|---------|
| Age at seizure onset | 0.424 | 0.278 | 0.645 | 0.000 |
| Spam semiology_0 (sym) ref | 1.000 |       |       |       |
| Spam semiology_1: asym | 1.210 | 0.367 | 3.989 | 0.755 |
| Spam semiology_2: sym+asym | 8.510 | 2.530 | 28.625 | 0.001 |
| _cons | 0.327 | 0.090 | 1.190 | 0.090 |

Variables with P ≤ 0.05 are indicated in bold.

AED, antiepileptic drugs; Asym, asymmetric; Complet, completeness; Extend, extending; ID, intellectual disability; MRI, magnetic resonance imaging; Ref, reference; Res, resection; SOZ, seizure onset zone; Superimp, superimposed; Sym, symmetric.

Table 3. Continued.
statistical inferences obtained in the Detroit and our own series might also be a reflection of us having included only patients operated on for active spasms, while 43% of the Detroit patients included in the multivariate analysis had no longer spasms at the time of surgery.

Using univariate models we identified several weak unfavorable prognostic factors for seizure freedom, including the need for invasive recordings, severe intellectual disability, hemiparesis, visual field defects and the co-occurrence of asymmetric and symmetric spasms. These findings are not unexpected. Invasive recordings are often necessary in complex cases in whom the chances of obtaining complete resection of the SOZ and, consequently, of achieving seizure freedom, are low. Specifically, we used SEEG in presumed symptomatic cases, in patients whose lesions were close to eloquent areas and when scalp EEG was nonlocalizing. These circumstances make surgical approach more complex as they limit the possibility of accurately planning the resection area and achieving completeness of resection. The need for invasive recordings has progressively decreased over the years and achieving completeness of resection. The need for invasive recordings has progressively decreased over the years and will undoubtedly continue to do so, thanks to advances in functional neuroimaging and improved understanding of prognostic factors.

Concerning the other results of univariate analyses, we confirmed neurological signs, severe intellectual disability and coexistence of symmetric and asymmetric spasms to be unfavorable prognostic factors for long-term outcome as they are often related to widespread epileptogenicity or diffuse lesions. We also found earlier age at seizure onset and longer duration of epilepsy before surgery to be accompanied by lower rates of freedom from disabling seizures after surgery, thus suggesting that early intervention is advised in children with spasms.

In this series, invasive recordings demonstrated that for both spasms and focal seizures, electrodes exploring the structurally abnormal area constantly captured the initial ictal electrographic changes, thus suggesting a common mechanism of generation of these two seizure types. In the ten patients who only had spasms, the surgical strategy we adopted was the same as for children also experiencing focal seizures, as they exhibited MRI visible lesions and EEG findings clearly pointing out to the structural abnormality. Two of the ten children who had only spasms underwent SEEG, during which electrodes exploring the structurally abnormal area recorded the initial ictal activity at spasm onset.

Using longitudinal population average logistic models, we observed that, in order to achieve postoperative drug withdrawal, complete resection of the SOZ was a stronger favorable prognostic factor than the mere resection of the MRI visible lesion. An SOZ extending beyond the lesional area was an unfavorable prognostic factor for Engel class I outcome. As a whole, our statistical results suggest that a careful neurophysiological definition of the SOZ for spasms increases the chances to obtain seizure freedom.

During SEEG, we observed spasms to be regularly accompanied by low voltage fast activity, either isolated or intermingled with a slow or sharp wave, as previously reported. An ictal pattern consisting of fast activity, preceded or not by a ‘leading spike’, was described using electrocorticography. In our study, the high number of SEEG electrodes we used helped carrying out a dynamic analysis of the spatio-temporal spread of the ictal discharge from the inner to the outer aspects of the explored areas and precisely localizing initial ictal activity at spasm onset, even when ictal discharges were diffuse on scalp recordings.

Pathophysiology of spasms remains to be fully elucidated. Earliest studies suggested brainstem dysfunction as a trigger for both spasms and the hypsarrhythmic EEG. Based on PET studies showing focal cortical hypometabolism and electroclinical demonstrations of co-occurring partial seizures and spasms, abnormal functional interactions between brainstem and a focal or diffuse cortical abnormality were hypothesized. A predominant role of either the cortical abnormality or the subcortical structures has always been a divisive argument. Uncertainties on pathophysiology have prompted caution in proposing surgery as a viable therapeutic approach for spasms, with subsequent limited surgical referral rates. However, in many cases, spasms have a focal cortical origin as suggested by invasive EEG recordings as well as the favorable outcome following removal of discrete epileptogenic lesions.

Our study has limitations, especially due to its retrospective design, etiologic heterogeneity, the inclusion of children with both spasms and focal seizures and the limited sampling SEEG allows. Certainly, selection of surgical candidates leaves many conditions with epileptic spasms that may have a completely different pathophysiology, especially genetic ones, outside the scope of our study. However, through analysis a large series we confirm that surgery is an effective treatment option for a selected population of children with drug-resistant spasms and suggest that extended resections and invasive recordings may not be needed to achieve seizure freedom. Major determinants of favorable outcome after surgery, that is, completeness of resection of the MRI visible lesion and of the SOZ and shorter duration of epilepsy, are similar to those reported for symptomatic focal epilepsy. Despite the complex pathophysiology of spasms and the difficulties in identifying the SOZ, in patients with a discrete epileptogenic lesion the pragmatic approach to surgery should follow the same principles applied to focal epilepsy, favoring unilobar, one-stage resections, thus
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reducing the risk of facing major surgical complications and postoperative deficits.

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Author Contributions

Carmen Barba: study design, invasive EEG and clinical data analysis and drafting of the manuscript. Roberto Mai: invasive EEG and clinical data analysis and revision of the manuscript. Laura Grisotto: statistical analyses and revision of the manuscript. Francesca Gozzo: clinical data analysis. Simona Pellacani: clinical data analysis. Laura Tassi: EEG data analysis and revision of the manuscript. Stefano Francione: EEG data analysis and revision of the manuscript. Flavio Giordano: surgical data analysis and revision of the manuscript. Francesco Cardinale: surgical data analysis. Renzo Guerrini: study concept, clinical data analysis and drafting of the manuscript.

Conflicts Of Interest

The authors report no disclosures relevant to the publication.

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Supporting Information

Additional Supporting Information may be found online in the supporting information tab for this article:

Table S1. Invasive recordings: Initial ictal activity at onset of spasms and of focal seizures and pattern of ictal spread.
Table S2. Logistic models: Univariate analyses.
Data S1. Supplementary results.
Data S2. Supplementary methods.