Microsurgery of epileptic foci in the insular region

Clinical article

RAMEZ MALAK, M.D.,1 ALAIN BOUTHILLIER, M.D.,1 LIONEL CARMANT, M.D.,2 PATRICK COSSETTE, M.D.,2 NORMAND GIARD, M.D.,2 JEAN-MARC SAINT-HILAIRE, M.D.,2 DONG BACH NGUYEN, M.D.,2 AND DANG KHOA NGUYEN, M.D.2

Departments of 1Neurosurgery and 2Neurology, Centre Hospitalier de l’Université de Montréal, Notre-Dame Hospital, Montréal, Québec, Canada

Object. The insular region has long been neglected in the investigation and treatment of refractory epilepsy. Surgery in the insular region is rarely performed because of the risk of injury to the opercula, the arteries transiting on the surface of the insula, and the deep structures such as the basal ganglia and the internal capsule. This study was undertaken to report the results of insular surgery using modern microsurgical techniques in patients with epilepsy.

Methods. The authors performed a retrospective study of cases involving patients who underwent surgery for insular lesions associated with epilepsy over the last 10 years. In the majority of patients, intracranial electrodes were implanted with neuronavigation guidance to confirm the localization of the epileptic foci.

Results. Nine patients underwent insular surgery: 7 for refractory epilepsy with no tumor and 2 for tumors associated with seizures. Four of the resections were performed in the left hemisphere. After an average follow-up of 54 months (range 14–122 months), Engel Class IA outcome had been achieved in 6 of 7 cases in the Epilepsy Surgery Group. The remaining patient had an Engel Class III outcome after partial insular resection but later became seizure-free (Engel Class IA) following insular Gamma Knife surgery.

Postoperatively, the majority of patients suffered from minor reversible hemipareses that disappeared completely within a few months. There was no surgical mortality.

Conclusions. Insular surgery is both safe and beneficial when it is well planned and performed with modern microsurgical techniques and good anatomical knowledge. Insulectomy is associated with little permanent morbidity and a high rate of seizure control. To the authors’ knowledge, this is the first series of insulectomies predominantly performed for refractory epilepsy since those performed by Penfield. (DOI: 10.3171/2009.1.JNS08807)

Key Words • cortical stimulation • insula • intractible epilepsy

T he insula is considered the fifth lobe of the brain, hidden deep in the sylvian fissure, covered by the frontal, parietal, and temporal opercula. It has long fascinated anatomists, surgeons, and physiologists because of its enigmatic functions and dangerous access.1,31,44 To get to the insula, the surgeon has to dissect the sylvian fissure, retract highly functional opercular zones (especially on the dominant side), then pass through a wall of sylvian arteries (all the branches of the M2 segment of the middle cerebral artery).50 Furthermore, insular resection is limited by deep-seated structures such as the internal capsule, the basal ganglia, and the lenticulostriate arteries.55,50,51,54 Various roles have been given to the insula, including that of a secondary sensory center, higher autonomic control, and a component of the limbic system. Other reported functions include secondary language and motor control.1,28,31 Yet there was no irreversible neurological deficit attributed to the loss of insular cortex itself following complete resection by Penfield36 or after radical excision of tumors in or adjacent to this area by Yaşargil.61

After the publication of Yaşargil et al.,61 there was a renewed interest in the surgical treatment of insular tumors31,30,32,52,53,63 and cavernomas3,25,49,55 with good results. In addition, recent electrophysiological studies have incriminated the insula as a site of intractable epilepsy.5,16,17,27,28,34,40,42 We report here our series of insular surgery cases, including 7 cases involving patients who underwent epilepsy surgery and 2 cases of insular tumors associated with seizures.

Methods

Patient Cohort

This is a retrospective study of cases involving patients who underwent insular surgery at Notre-Dame Hospital performed by one of the authors (A.B.) in the last 10

Abbreviation used in this paper: SISCOM = subtraction ictal SPECT coregistered to MR imaging.
years. We made a distinction between epilepsy surgery and tumor surgery. In the Epilepsy Surgery Group, patients underwent surgery for control of drug-resistant epilepsy. They were referred and evaluated by our epilepsy multidisciplinary team. They underwent a complete epilepsy workup, including neuropsychological evaluation, video-electroencephalographic monitoring, MR imaging, and SISCOM. Most of these patients also underwent PET and an invasive study with intracranial electrodes. Magnetic resonance imaging and histological examination did not reveal tumors in these cases, but other pathological conditions such as gliosis and cortical dysplasia may have been present. Each case was discussed in a multidisciplinary epilepsy conference among epileptologists, neuroradiologists, neuropsychologists, and the neurosurgeon. The team made recommendations regarding intracranial electrode coverage and the extent of cortical resection. Figures 1–3 present images related to representative cases in this first group.

In the second group, the Tumor Surgery Group, the main objective of surgery was diagnostic confirmation and oncological control of insular tumors, and hopefully, better seizure control. Although these patients presented with epilepsy, original brain imaging studies clearly demonstrated the presence of tumor, and no other workup was necessary to localize the epileptogenic zone.

Operative Techniques

Intracranial electrodes were introduced into the insular cortex either by stereotaxy or after open craniotomy. Stereotactic electrode implantation was performed in 1 patient using the method described by Talairach and Bancaud. For the remaining patients, depth electrodes (Spencer depth electrodes, Ad-Tech Medical Instrument Corp.) were placed in the insular cortex under direct vision after microsurgical opening of the sylvian fissure (Fig. 4). In most cases, 2 depth electrodes were inserted in the insula. The sampled insular area (posterior vs anterior) was determined by noninvasive localization tests. For each electrode, 2 contacts were inserted into the insular cortex, separated by 2 mm. The depth electrodes
Microsurgery of epileptic foci in the insular region

were sutured to subdural electrodes placed on the lateral surface of the hemisphere and to the dura mater. The craniotomy and the site of sylvian fissure dissection were planned with the neuronavigation system. Postimplantation MR imaging was always performed to determine the exact position of the electrodes. In all patients in whom insular electrodes were implanted, subdural electrodes covering the ipsilateral rolandic area, the opercula, and the subfrontal cortex were also implanted. Other cortical areas were sampled based on preoperative studies.

The insulectomy was either performed as part of an insulo-operculectomy, temporal lobe resection plus insulectomy, or as a pure insulectomy. Operculoinsulectomy was performed only when the operculum was part of the epileptogenic focus and only in the nondominant hemisphere. Single (frontal, temporal, or parietal) or multiple opercu-

lectomies were performed based on presurgical evaluation. The opercular cortex was removed first by subpial resection between the branches of the sylvian artery, using ultrasonic aspiration at very low intensity. After completion of operculectomy, subpial resection continued to include the insula. The localization (anterior or posterior) and the extent of insulectomy were based on all the data available. If a lesion such as cortical dysplasia or gliosis was suspected on the basis of MR imaging, the entire abnormal insular cortex was removed. In patients with normal MR imaging findings, we opted to remove the insular cortex corresponding to the ictal focus seen with SISCOM. Surgery was guided by anatomical landmarks—the peri-insular sulci and the deep central insular sulcus—and by neuronavigation. In patients undergoing insulectomy plus temporal lobectomy, the insula was exposed and removed by subpial resection.

Fig. 2. Case 7. A: Preoperative coronal MR image showing cortical dysplasia involving the right insula and frontoparietal operculum (arrows). B and C: Intraoperative images obtained before and after resection of the cortical dysplasia. The opercular cortex was removed first by subpial resection between the branches of the sylvian artery. The subpial resection was then continued to include the insula. D: Postoperative MR images showing complete resection of the insulo-opercular cortical dysplasia. The insula was completely resected.
after temporal lobectomy including the superior temporal gyrus. Finally, pure insulectomy was performed after microsurgical dissection of the sylvian fissure. Insular cortex was then removed by resection between sylvian M2 branches. Tumor resection was performed using standard neurosurgical techniques after microdissection of the sylvian fissure. Neuronavigation was used in all cases for planning the craniotomy and localizing the deep structures. Care was taken to preserve the insular vessels and the lenticulostriate arteries.

**Results**

**Patient Characteristics**

Nine patients with seizures underwent insular surgery—7 for epilepsy control and 2 for treatment of tumors. The majority of the patients were female (7 female, 2 male). The patients in the Epilepsy Surgery Group tended to be younger (range 16–36 years, mean 29 years) than those in the Tumor Surgery Group (46 and 59 years, mean 52.5 years). Four of the resections were performed in the left hemisphere.

**Presenting Symptoms**

Symptoms at onset of seizures varied from somatosensory to viscerosensory and/or somatomotor and included painful or nonpainful contralateral paresthesias, throat constriction, tonic posturing, nausea, and déjà vu (Table 1). Only 1 patient in the Epilepsy Surgery Group had a preoperative neurological deficit (left hemiparesis, related to a frontoooperoinsular dysplasia). A preoperative deficit (dysphasia) was present in 1 of the 2 patients in the Tumor Surgery Group (Table 2).

**Epilepsy Surgery Group**

Seven patients were referred for epilepsy surgery. They had suffered from drug-resistant epilepsy for a mean of 19 years (range 7–34 years). Most of the patients had multiple seizures per day, which had a great impact on their quality of life. High-resolution MR imaging findings were normal in 2 patients. In the remaining patients there was MR imaging signal compatible with atrophy, cortical dysplasia, or...
TABLE 1: Demographic and clinical characteristics of patients in the Epilepsy Surgery Group*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Initial Sz Manifestations</th>
<th>Preop Deficit</th>
<th>MRI</th>
<th>EEG</th>
<th>Ictal SPECT</th>
<th>PET</th>
<th>Intracranial Electrode Signal</th>
<th>Resection</th>
<th>Postop Deficit</th>
<th>Epilepsy Control (Engel class)</th>
<th>FU (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22, F</td>
<td>lt hemiparesthesias, throat constriction</td>
<td>no</td>
<td>rt hemisph atrophy, rt hippoc sclerosis</td>
<td>rt diffuse</td>
<td>rt ant temp, rt ant ins</td>
<td>—</td>
<td>rt temp &amp; ins</td>
<td>rt temp &amp; rt ins ant</td>
<td>trans lt hemiparesis, UE 4+/5 (2 mos), quadrantanopia</td>
<td>IA</td>
<td>42</td>
</tr>
<tr>
<td>2</td>
<td>36, M</td>
<td>rt painful hemiparesthesias</td>
<td>no</td>
<td>N</td>
<td>lt front temp</td>
<td>lt ins post, lt cingular</td>
<td>N</td>
<td>lt ins post</td>
<td>lt ins post</td>
<td>none</td>
<td>III†</td>
<td>40</td>
</tr>
<tr>
<td>3</td>
<td>16, M</td>
<td>rt brachiofacial clonic jerking</td>
<td>no</td>
<td>gliosis: lt ins &amp; lt front</td>
<td>lt central parasagittal</td>
<td>lt ins</td>
<td>lt ins front &amp; ins</td>
<td>lt ins &amp; lt front</td>
<td>trans rt hemiparesis, UE 3/5 (2 wks)</td>
<td>IA</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>36, F</td>
<td>tonic posturing</td>
<td>lt hemiparesis</td>
<td>cort dysplasia: rt ins &amp; rt operc</td>
<td>rt front central</td>
<td>rt front inferior</td>
<td>—</td>
<td>—</td>
<td>rt ins &amp; rt operc (front, pariet, temp)</td>
<td>trans incr of hemiparesis (2 mos)</td>
<td>IA</td>
<td>91</td>
</tr>
<tr>
<td>5</td>
<td>35, F</td>
<td>leg weakness fixed gaze, head deviation, pedaling, chewing</td>
<td>no</td>
<td>cort dysplasia: rt ins &amp; rt operc</td>
<td>bilat temp</td>
<td>rt ins, rt front, rt front operc</td>
<td>—</td>
<td>—</td>
<td>rt ins &amp; rt front operc</td>
<td>trans lt hemiparesis 4+/5 (2 mos)</td>
<td>IA</td>
<td>122</td>
</tr>
<tr>
<td>6</td>
<td>26, F</td>
<td>throat &amp; jaw paresthesias, anxiety, déjà vu</td>
<td>no</td>
<td>N</td>
<td>rt centrotemporal</td>
<td>rt temp pole, rt pariet, rt ins</td>
<td>rt temp</td>
<td>rt temp, ins</td>
<td>rt temp, ins</td>
<td>none</td>
<td>IA</td>
<td>21</td>
</tr>
<tr>
<td>7</td>
<td>35, F</td>
<td>nocturnal, tonic posturing</td>
<td>no</td>
<td>cort dysplasia: rt temp</td>
<td>rt front pariet</td>
<td>lt temp</td>
<td>—</td>
<td>rt ins &amp; rt operc (front, pariet)</td>
<td>none</td>
<td>IA</td>
<td>14</td>
<td></td>
</tr>
</tbody>
</table>

* Ant = anterior; cort = cortical; EEG = electroencephalography; front = frontal; FU = follow-up; hemisph = hemisphere or hemispheric; hippoc = hippocampal; incr = increase; ins = insula; N = normal; operc = opercular; pariet = parietal; post = posterior; Sz = seizure; temp = temporal; trans = transient; UE = upper extremity; — = not recorded.

† After subsequent Gamma Knife surgery, outcome was Engel Class IA.
TABLE 2: Demographic and clinical characteristics of patients in the Tumor Surgery Group

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Aura</th>
<th>Preop Deficit</th>
<th>CT/MRI</th>
<th>Resection</th>
<th>Path Dx</th>
<th>Postop Deficit (duration)</th>
<th>Sx Control (Engel class)</th>
<th>Adjuvant Tx</th>
<th>Postop Survival (cause of death)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>59, F</td>
<td>rt paresthesias</td>
<td>dysphasia</td>
<td>lt ins tumor</td>
<td>total</td>
<td>anaplastic astrocytoma</td>
<td>trans incr of dysphasia (1 mo)</td>
<td>IA</td>
<td>refused</td>
<td>3 mos (sepsis)</td>
</tr>
<tr>
<td>9</td>
<td>46, F</td>
<td>nausea</td>
<td>none</td>
<td>lt ins &amp; front operc</td>
<td>partial</td>
<td>oligo astrocytoma</td>
<td>none</td>
<td>IA</td>
<td>&gt;110 mos†</td>
<td></td>
</tr>
</tbody>
</table>

* Path = pathological; RT = radiotherapy.
† Still alive as of this writing.

Discussion

Hierarchical Background

In 1809, the German anatomist Johann-Christian Reil coined the name insula (die Insel), meaning island, to describe the hidden cortex situated in the depth of the sylvian fissure. Penfield, more than a century later, was the first to perform electrical stimulation and electrocorticography on exposed insula after temporal lobectomy in epileptic patients. Stimulation of 82 separate insular points in 36 awake patients resulted mainly in visceral and sensory responses. Insulotomy was performed if the insula had residual epileptiform activity after completion of temporal lobectomy. No immediate neurological deficit was observed after the first series of 4 insulotomies. Guillaume and colleagues also described the surgical techniques of insular resection for insular epilepsy. Depending on macroscopic observation or abnormal electrocorticographic activity, they performed either frontal or temporal opercular resection to expose the insula.

Insular surgery for treatment of epilepsy was abandoned after the publication by Sillvenius et al., showing that the addition of insular resection to temporal lobotomy did not increase epileptic control but significantly increased surgical morbidity. This publication reviewed the cases of all epileptic patients who underwent temporal lobectomy at the Montreal Neurological Institute from 1946 to 1962; 106 patients had residual epileptiform activity in the insula. Of those 106 patients, 58 had total or partial insulotomy and 48 had no insular ablation. The percentage of patients with satisfactory control of epilepsy was similar in the 2 groups (45% in the insulotomy group compared with 42% in the no-insulotomy group); yet the rate of hemiparesis was increased from 3% in the no-insulotomy group to 5.9% in the insulotomy group.
group to 21% in the insulectomy group. This complication, named “manipulation hemiplegia,” was explained by the unavoidable manipulation of the middle cerebral vascular tree when an insular decortication was performed. Only 4 patients (7%) in the insulectomy group had motor deficit at discharge from the hospital, and there is no mention of how many among them had permanent deficit after longer follow-up. Also, the decision to perform insulectomy was based on intraoperative interictal electrocorticography. In our series we relied on ictal recording from implanted deep brain electrodes to define an independent insular epileptic focus. For that reason, we found it unnecessary to perform interictal electrocorticography after insulectomy.

Review of the Literature on Insular Surgery

Enthusiasm for insular surgery returned with the use of microsurgical techniques, which generated a more interventionist attitude regarding insular lesions. In 1992, Yaşargil published his large series of 177 tumors in the paralimbic system, which included 80 tumors in the insula. Postoperatively, 85% of the patients (all groups combined) had minor or no deficits and 92.5% of the patients became seizure-free. Several series summarized in Table 3) and some case reports have been published to date about insular surgery for tumor or cavernoma resection.

On the basis of these studies, we can draw several conclusions. First, lesions in the insula seem to be highly epileptogenic; 202 (76%) of 265 patients with insular tumors had epilepsy at presentation (Table 3). According to Duffau et al.,

insular lesions tend to produce a high rate of medically intractable epilepsy; 60% of cases of epilepsy secondary to low-grade insular glioma are drug-resistant compared with 15% of cases secondary to other cortical gliomas. In a recent study, Chang et al. found that 81% of 332 patients treated surgically for low-grade glioma presented with epilepsy and that in 40% of patients seizures were uncontrolled preoperatively. In a subset of 28 patients with insular tumors, 79% presented with epilepsy, and it was drug-resistant in only 21% of those cases. From our literature review, lesionectomy appears to be very efficient in controlling insular epilepsy. On average, an Engel Class I outcome was obtained in 81% of patients, and 87% of patients who presented with epilepsy had Engel Class I or II at follow-up.

Second, insular surgery is associated with a high rate of transient postoperative deficit. Transient hemiparesis was reported postoperatively in almost all of the case series, with rates ranging from 0 to 50% (Table 3). Analyzing data from the 11 largest series of insular surgeries reported since 1988, we found 48 cases of transient hemiparesis in a total of 288 patients (17%). Incidence of permanent deficit is lower, reported only for 10 patients (3%). Finally, transient dysphasia may occur following insular surgery in the dominant hemisphere. This complication was reported in 24 of 288 patients, with these 24 patients representing 16% of the 148 who underwent surgery in the dominant hemisphere. Only 2 cases of permanent dysphasia occurred (0.7% of all patients or 1.4% of patients who underwent surgery in the dominant hemisphere). Of note, all cases of permanent deficit (paresis or dysphasia) were related to an infarct in the territory of the lenticulostriate arteries.

Invasive Investigation and Insulectomy

For epilepsy surgery in the absence of a lesion, confirmation of epileptic focus within the insula was obtained using implanted depth electrode recordings. There are 2 methods of implanting insular electrodes: stereotactically or though open craniotomy. The stereotactic method, as used in our first patient as well as by Isnard et al., requires image fusion of data from MR imaging and cerebral angiography in the stereotactic guidance system to minimize damage to Sylvian veins and arteries during implantation. The open craniotomy approach, as used for most of our patients, has the advantage of simultaneous insertion of subdural electrodes for large cortical coverage of the lateral hemisphere. In addition, cerebral angiography is not needed in this procedure because the Sylvian fissure is opened and the electrodes are placed under direct vision. Furthermore, this technique increases the certainty that the electrodes are actually in the insular cortex and not in the operculum. Depth electrodes are required because subdural electrodes placed in the Sylvian fissure are unable to differentiate insular from opercular epileptic origin. Once identified, the insular epileptogenic zone is removed either by subpial resection after removal of the affected operculum, or by resection between Sylvian M1 branches after opening of the Sylvian fissure. This technique respects the major Sylvian branches; however, we could not spare the perforating vessels of the insular branches.

Complete seizure control (Engel Class I) was accomplished in 8 (89%) of the 9 patients in our series (6 [86%] of 7 in the Epilepsy Surgery Group). In the single patient who had epilepsy and in whom complete seizure control was not obtained by insular surgery, seizures recurred after a 7-week seizure-free period and were eventually controlled by Gamma Knife surgery. Thus, 100% of our patients who underwent insulectomy have benefited from complete seizure control.

Postoperative Deficits

Our series of patients differs slightly from the other reported series as the patients in the other series were being treated mainly for oncological or vascular conditions, whereas most of our patients had no macroscopically apparent lesions. In our hands, no permanent paresis occurred, but 3 (33%) of 9 patients experienced new-onset transient hemiparesis postoperatively. In 1 other patient, preoperative hemiparesis worsened transiently. Thus 4 (57%) of 7 patients in the Epilepsy Surgery Group experienced either new-onset or worsened transient hemiparesis. This relatively high incidence of transient hemiparesis could be explained by the fact that normal-appearing cortex was being removed as opposed to removing a mass lesion that displaces vascular structures. With respect to language deficits following insular surgery in the dominant hemisphere, 1 of 2 patients in the Tumor Surgery Group (both of whom underwent dominant-hemisphere surgery) demonstrated a transient deterioration of preoperative dysphasia while none of the 2 in the Epilepsy Surgery Group who underwent dominant-hemisphere insular surgery showed postoperative dysphasia.

Different hypotheses could explain the high reported rate of transient hemiparesis and dysphasia following insu-
<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Cases</th>
<th>Type &amp; Localization of Lesion</th>
<th>No. on Lt Side (%)</th>
<th>Surgical Techniques</th>
<th>Extent of Resection</th>
<th>Presentation</th>
<th>Epilepsy Control</th>
<th>New Postop Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yaşargil, 1988</td>
<td>25</td>
<td>AVM, 2 cavernomas</td>
<td>10 (40)</td>
<td>careful opening of sylvian fissure, dissection of sylvian branches, coag of feeders</td>
<td>complete</td>
<td>epilepsy 17 (68), SAH 15 (61), hemiparesis 2 (8)</td>
<td>Engel I: 13/17 (74)</td>
<td>mod temp hemiparesis 5 (20)</td>
</tr>
<tr>
<td>Bertalanffy et al., 1991</td>
<td>6 cavernomas</td>
<td>2 (33)</td>
<td>SSEP, avoidance of removal of perilesional gliosis</td>
<td>NS</td>
<td>epilepsy 2 (33), hemiparesis 2 (33), hemianopsia 2 (33)</td>
<td>Engel II: 2/2 (100)</td>
<td>infarct causing perm paresis &amp; dysphasia 1 (16), trans paresis 1 (16)</td>
<td></td>
</tr>
<tr>
<td>Yaşargil et al., 1992 &amp; Yaşargil, 1996</td>
<td>80 gliomas (56% gr); 35 I, 45 MC</td>
<td>42 (53) wide sylvian opening, dynamic retraction, LLA dissection, papaverine</td>
<td>radical presumed</td>
<td>epilepsy 62 (78), dysphasia 21 (27), sensorimotor 21 (26), neuropsych 28 (35)</td>
<td>Engel I: 74/80 (93); Engel II: 6/80 (7)</td>
<td>mod hemiparesis 10 (12.5); trans 8, perm 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zentner et al., 1996</td>
<td>30 gliomas (50% high gr); 5 I, 9 IO, 16 MC</td>
<td>15 (50) operc tumor resected 1st</td>
<td>T 17%, ST 70%, P 13%</td>
<td>epilepsy 19 (63), focal deficit (dysphasia, paresis) 9 (30), “psychogenic” 2 (7)</td>
<td>Engel I–III: 17/19 (89)</td>
<td>paresis: trans 4 (13), perm 2 (6); dysphasia: trans 3 (10), perm 2 (6); reduced KPS: trans 7 (23), perm 3 (13)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vanaclocha et al., 1997</td>
<td>23 gliomas (30% high gr)</td>
<td>16 (70) awake surg, cortical mapping, wide sylvian opening, US, histol exam of borders</td>
<td>T 87%, ST 13%</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>trans paresis 4 (17) (avg 6 mos) trans dysphasia 1 (4) (1 wk)</td>
<td></td>
</tr>
<tr>
<td>Heffez, 1997</td>
<td>10 vascular lesions, 2 benign gliomas; 9 I, 1 IO</td>
<td>7 (70) stereotaxy, US, Ltd sylvian dissection, SSEP</td>
<td>NS</td>
<td>hemorrhage 4 (40), epilepsy 4 (40), dysphasia 3 (30), sensorimotor 7 (70)</td>
<td>no impr</td>
<td>no new deficits, impr paresis 2/3 (67)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lang et al., 2001</td>
<td>22 gliomas (45% high gr); 8 I, 6 IO, 8 MC</td>
<td>8 (36) stereotaxy, US, cort mapping, awake surg for dominant side, operc tumor resected 1st</td>
<td>T 46%, ST 27%, P 27%</td>
<td>epilepsy 14 (64), dysphasia 4 (18), paresis 8 (32)</td>
<td>NS</td>
<td>paresis: trans 4 (18), perm 2 (9); transient dysphasia: 6 (27%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meyer et al., 2001</td>
<td>11 gliomas (45% high gr); 11 I</td>
<td>5 (45) awake surg, stereotaxy cort &amp; subcort mapping (rptd neurol exam)</td>
<td>T 27%, ST 45%, P 27%</td>
<td>NS</td>
<td>NS</td>
<td>mRS 0 (asymptomatic): 4 (36), mRS 1 (minor Sx): 4 (36), mRS 6 (died of tumor progr): 3 (28)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duffau et al., 2002 &amp; 2006</td>
<td>42 low-gr gliomas; 7 I, 10 IO, 25 MC</td>
<td>12 (29) awake craniotomy, cort mapping US, stereotaxis, preop CTA, lesionectomy plus</td>
<td>T or ST 74%, P 26%</td>
<td>epilepsy 42 (100), dysphasia 8 (19), sensorimotor 8 (19), visceral sensory 8 (19), auditory 4 (9)</td>
<td>Engel I: 34/42 (81); Engel II: 4/42 (9); Engel III: 4/42 (9)</td>
<td>paresis: trans 21 (50), perm 3 (7); dysphasia: trans 10 (24), athymormia** 7 (17)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tirakotai et al., 2003</td>
<td>8 cavernomas; 8 I</td>
<td>3 (40) stereotaxy, US, distal sylvian opening, gliosis resection</td>
<td>T 100%</td>
<td>epilepsy 2 (25), paresis 3 (38)</td>
<td>Engel I: 1/2 (50); Engel II: 1/2 (50)</td>
<td>no new deficits, impr paresis 2/3 (67)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(continued)
Microsurgery of epileptic foci in the insular region

From the above discussion, it becomes clear that microsurgical techniques aimed at sylvian fissure opening and preservation of the lenticulostriate arteries are central to surgery in the insular region. Modern techniques such as ultrasound and neuronavigation can be helpful in localizing the lesion but have some limitations (for example, brain shift) and should never replace meticulous dissection based on the well-described microanatomy of the insula.

Operative Technique and Complication Avoidance

When a pure total insulectomy is required, however, we tend to perform more tailored distal sylvian splitting guided by neuronavigation and ultrasound. The advantage of this approach is to limit the risk of extensive sylvian dissection. This is particularly useful for the insertion of insular electrodes, when partial exposure of the insula is needed. This can be achieved with limited retraction. When a pure total insulectomy is required, however, we believe that a wide splitting of the sylvian fissure provides a better approach. In our series, however, it must be noted
that because the opercula was often part of the epileptic focus, most total insulectomies were not performed through the sylvian fissure but rather consisted of subpial resection of the insula as part of operculoinsulectomy.

Some authors have used awake surgery for dominant-side lesions; the main purpose is to monitor language functions within the insula. Unfortunately, most cases of permanent damage are due to vascular compromise that cannot be prevented by this type of monitoring. Furthermore, awake surgery has additional limitations such as patient discomfort, risk of intraoperative seizures, and limiting the extent of tumor resection. For these reasons, many surgeons have not adopted this technique and have preferred to “stay within the tumor” as a guide for the resection.

In some cases we decided to remove the insula plus other cortical structures (opercula or temporal lobe) because they were incriminated as independent epileptogenic foci, either because of an abnormal MR imaging signal (cortical atrophy or cortical dysplasia) or because of spontaneous epileptic activity recorded on intracranial electrodes. In this particular situation removing the temporal lobe or the opercula (only in the nondominant hemisphere) gave us direct access to the insula with no need to open the sylvian fissure. This situation was also described for insular tumors that are localized both in the insula and the opercula. Some authors prefer to start by resecting the opercular tumor first to avoid opening the sylvian fissure. In contrast, Yaşargil et al. suggest that we should start by resecting the insular tumor and then attack the opercular part of the tumor. The latter approach has the advantage of identifying the sylvian branches early during the dissection.

Conclusions

Although initial attempts to resect the insula were associated with a high rate of complications, notably hemiparesis and dysphasia, a better knowledge of the cortical and vascular anatomy of the insula in addition to the use of intraoperative neuronavigation, allows surgery of the insula with acceptable risks for the benefit of seizure control. To our knowledge, this is the first series of insulectomy predominantly performed for refractory epilepsy since Penfield.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Acknowledgments

The authors thank Drs. M. G. Yaşargil and J. G. Villemure for critical review of the article, Dr. D. Lacerte for active participation in the first case, and Dr. T. Nguyen for the illustration.

References


R. Malak et al.
Microsurgery of epileptic foci in the insular region


---


 Portions of this work were presented in abstract form at the Canadian Congress of Neurological Sciences meeting Ottawa, Canada, in 2005 and were also presented at the Congress of Neurological Surgeons meeting in Orlando, Florida, in September 2008.

 Please include this information when citing this paper: published online February 27, 2009; DOI: 10.3171/2009.1.JNS08807.

 Address correspondence to: Alain Bouthillier, M.D., Service de neurochirurgie, Centre Hospitalier de l’Université de Montréal, Notre-Dame Hospital, 1560, rue Sherbrooke Est, Montréal, Québec, Canada H2L 4M1. email: alain.bouthillier@umontreal.ca.