Vascular consequences of operculoinsular corticectomy for refractory epilepsy

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OBJECT Surgery in the insular region is considered challenging because of its vascular relationships, the proximity of functional structures, and its deep location in the sylvian fissure. The authors report the incidence and consequences of ischemic lesions after operculoinsular corticectomy for refractory epilepsy.

METHODS The authors retrospectively reviewed the data of all patients who underwent an insular resection with or without an opercular resection for refractory epilepsy at their center. All patients underwent postoperative MRI, enabling a radiological analysis of the ischemic lesions as a result of the corticectomies. The resections were classified according to the location and extent of the insular corticectomy and the type of operculectomy. Each patient underwent clinical follow-up.

RESULTS Twenty patients underwent surgery. All patients underwent insular corticectomy with or without an operculectomy. Ischemic lesions were identified in 12 patients (60%). In these patients, 11 ischemic lesions (55%) were related to the insular corticectomy, and 1 was related to the associated periinsular resection. The ischemic lesions associated with the insulectomies were typically located in the corona radiata running from the insula to the periventricular region. Nine patients (45%) developed a postoperative neurological deficit, among whom 6 (67%) had an insular corticectomy–related ischemic lesion. All reported neurological deficits were transient. Five patients (25%) had ischemic lesions without neurological deficit.

CONCLUSIONS Operculoinsular corticectomies are associated with ischemic lesions in approximately 60% of patients. However, given that no patient had a definitive postoperative deficit, these ischemic lesions have few clinical consequences. Therefore, this surgical procedure can be considered reasonably safe for the treatment of refractory epilepsy.

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KEY WORDS insula surgery; operculoinsular resection; ischemic lesions; refractory epilepsy

REIL first described the insular lobe in 1809. It is considered as the fifth lobe and is not visible at the surface of the brain because of its deep anatomical position. The insula only becomes visible when the sylvian fissure is opened, as it is covered by the frontal, parietal, and temporal opercula. The insula is triangular and consists mainly of 3 short and 2 long gyri separated by the central insular sulcus. The insular cortex lies superficial to the extreme capsule, claustrum, external capsule, and putamen. Its vascular supply is principally provided by perforating arteries arising from the M₂ segments of the middle cerebral artery (MCA).^{3,8,15,36,38,39}

The function of the insula is not fully characterized, but it appears to be involved in, among other things, somatosensory representation, viscerosensory functions, visceromotor control, autonomic responses, pain processing,

and gustatory and auditory responses.^{3–5,14,15,28,36} Insular seizures may go unrecognized since they can mimic temporal, parietal, and frontal lobe seizures.^{7,20,21,27,29,32,33} As the involvement of the insular cortex is being more and more suspected in some epileptic patients, the structure has been increasingly investigated over the past decade using depth electrodes or combined subdural and depth electrodes through craniotomies with direct insular cortex exposition.^{6,9,21,23,30,37}

Because of its restricted accessibility, vascular relationships, and the proximity of functional structures, the insular region is considered a challenging region in which to perform surgery. Nevertheless, because of microsurgical technical progress, it is possible to perform resective surgery of the insular region with lower morbidity.^{12,24,27,35,41–43}

The aim of our study was to analyze the vascular risk

ABBREVIATIONS ECoG = electrocorticography; EEG = electroencephalography; LLA = lenticulostriate artery; MCA = middle cerebral artery.

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of epilepsy surgery in the insular area. More specifically, we report here on the incidence and consequences of ischemic lesions after operculoincisor corticectomies for refractory epilepsy.

Methods

We retrospectively studied all patients at our center who underwent an insular corticectomy with or without an opercular resection for medically refractory epilepsy from 1998 to 2012, inclusively. Cases with tumors or vascular malformations were excluded. All patients had a postoperative follow-up of at least 6 months.

All patients underwent a comprehensive preoperative investigation that included (for most patients) video-electroencephalography (EEG) monitoring, brain MRI, neuropsychological evaluation, SPECT, PET, magnetoencephalography, and EEG gated functional MRI. The location and extent of the insular and opercular resections were based on this preoperative workup, which was completed with intracranial EEG recordings in most patients.

All patients underwent postoperative MRI within 3 months after surgery, enabling determination of the areas resected and analysis of the ischemic lesions resulting from the corticectomies. Insular resections were classified as anterior, posterior, superior, inferior, or radical. The MRI included coronal T2 and FLAIR sequences. All patients were clinically evaluated during their postopera-

tive hospital stay, 2 months after surgery, and twice a year afterward.

The results of the intracranial investigation and seizure control have been published elsewhere.^{24,37}

Results

Twenty consecutive patients (11 females and 9 males; average age 33 years, range 16–51 years) who underwent surgery at our institution were enrolled. Seventeen patients had complex partial seizures and 3 had simple partial seizures. Preoperative MRI findings were either normal or revealed abnormalities consistent with focal cortical dysplasia. Fifteen patients underwent intracranial electrode placement with extraoperative long-term recordings to better define the location and extent of the subsequent insular and opercular resection. One patient underwent intraoperative electrocorticography (ECoG) and 4 patients underwent neither intracranial extraoperative recording nor ECoG (Table 1). All patients with intracranial electrode placement underwent MRI the day after the procedure to assess the position of each electrode contact. Based on review of postimplantation MR images, there was no ischemic lesion related to subdural or depth electrode positioning.

Two patients underwent a pure insulectomy. The insulectomies were combined with an opercular resection in 13 patients, a frontoorbital resection in 1 patient, a temporal lobectomy in 3 patients, and a frontal lobectomy in 1

TABLE 1. Patient data

Case No.	Age (yrs)	Sex	Seizure Type	LTIER/ECoG	Side	Surgery	Postop Deficit	IL
1	35	F	CP	LTIER	Rt	Insulect + F operculect	+	+
2	36	F	SP		Rt	Insulect + FPT operculect	+	+
3	16	M	SP	ECoG	Lt	Insulect + F operculect	+	–
4	23	F	CP	LTIER	Rt	Insulect + T lobect	+	+*
5	36	M	SP	LTIER	Lt	Insulect	–	–
6	26	F	CP	LTIER	Rt	Insulect + T lobect	–	–
7	35	F	CP		Rt	Insulect + FP operculect	+	+
8	38	F	CP	LTIER	Lt	Insulect + T operculect	–	+
9	43	M	CP	LTIER	Rt	Insulect + F lobect	+	–
10	35	M	CP	LTIER	Lt	Insulect	+	+
11	51	M	CP	LTIER	Rt	Insulect + T operculect	–	+
12	37	M	CP	LTIER	Rt	Insulect + F operculect	–	+
13	28	F	CP	LTIER	Rt	Insulect + operculect	–	+
14	24	M	CP		Rt	Insulect + F operculect	+	+
15	47	M	CP		Lt	Insulect + FP operculect	–	–
16	25	M	CP	LTIER	Lt	Insulect + T lobect	–	–
17	23	F	CP	LTIER	Rt	Insulect + FPT operculect	+	+
18	38	F	CP	LTIER	Lt	Insulect + FT operculect	–	–
19	35	F	CP	LTIER	Rt	Insulect + PT operculect	–	+
20	32	F	CP	LTIER	Rt	Insulect + FP operculect	+	–

CP = complex partial; F = frontal; FP = frontoparietal; FPT = frontoparietotemporal; FT = frontotemporal; IL = ischemic lesion; insulect = insulectomy; lobect = lobectomy; LTIER = long-term intracranial EEG recording; operculect = operculectomy; SP = simple partial; T = temporal; + = present; – = absent.

* Ischemic lesion related to temporal lobectomy.

FIG. 1. Illustration showing the localization and the extent of the insular cortex resections in the 20 studied patients. The numbers correspond to the case numbers in Table 1. Copyright Patrice Finet. Published with permission.

patient. The operculectomies were performed as follows: 4 frontal, 3 frontoparietal, 2 frontoparietotemporal, 2 temporal, 1 frontotemporal, and 1 parietotemporal. Two techniques of insulectomy were used, often in combination. For the transsylvian approach, the sylvian fissure was microsurgically opened, with preservation of all M_2 , M_3 , and M_4 branches of the MCA. Major veins were preserved most of the time. Neuronavigation could be used when the anatomy of the deep sylvian fissure was difficult to ascertain. A self-retaining retractor was intermittently positioned over the opercula. After the insula was exposed, pial bipolar coagulation between the M_2 branches was performed, followed by microscissor opening of the insular surface. Then, the insular cortex was removed by subpial microsuctioning. To achieve complete cortical removal, perforating arteries from the M_2 branches were usually sacrificed. For the transopercular approach, cortical incisions were made between the M_4 opercular arteries. This was followed by subpial suctioning of the hidden surface of the perisylvian opercula until the periinsular sulcus was reached. After progressively lifting the insular pia, the insular cortex was removed by subpial microsuctioning. Again, most perforating arteries were sacrificed to achieve complete cortex removal. With both approaches, suctioning was never done away from the pia. When performed on the dominant side for language, opercular retraction has to be very delicate and as brief as possible. The exact localization and the extent of resection of the insular cortex were determined precisely for each patient on postoperative MRI (Fig. 1).

All patients underwent MRI between 3 days and 3 months after surgery. These MR images allowed the identification of ischemic lesions in 12 of the 20 studied patients (60%). Among these 12 patients, 11 ischemic lesions (55%) were attributed to the insular corticectomy, while 1 ischemic lesion (5%) was related to the associated resec-

tion (temporal lobectomy). In this latter patient with the ischemic lesion related to the temporal lobectomy, there was no other ischemic lesion.

The insular infarcts were identified mainly on coronal T2 and FLAIR sequences on early MR images (Fig. 2); on T2, FLAIR, and T1 sequences on late MR images (Fig. 3); and/or on diffusion-weighted sequences (Fig. 4) on early MR images. The infarcts were typically located in the corona radiata running from the insula to the periventricular region (Fig. 5). As expected, anterior insular resections were associated with ischemic lesions extending up to the frontal horn of the lateral ventricle, while posterior insular resections were related to ischemic lesions reaching the body or atrium of the lateral ventricle. Inferior and superior insulectomies could present with either an inferior or superior ischemic lesion, respectively. Inferior ischemic lesions typically extended from the insular area, reaching the temporal horn of the lateral ventricle.

FIG. 2. Early postoperative coronal T2-weighted (left) and FLAIR (right) MR images showing insular resection-related ischemic lesions (arrow) located in the corona radiata running from the insula to the periventricular region.

FIG. 3. Late postoperative coronal T1-weighted gadolinium-enhanced MR image revealing an ischemic lesion (arrow) resulting from an insulectomy.

The ischemic lesions were uniformly associated with the different resected insular areas without predilection for a specific region. The risk of developing an ischemic lesion in our series seemed to be the same whether the insular resection was anterior, posterior, inferior, superior, central, or radical. The occurrence of an ischemic lesion was not influenced by whether a cortical dysplasia was identified on MRI.

After surgery, 9 patients (45%) had neurological deficits. One patient had a left superior homonymous quadrantanopia related to a right posterior temporal ischemic lesion following a temporal lobectomy. The same patient and 6 others had a transient hemiparesis with brachiofacial predominance. One patient had a transient increase in preexisting hemiparesis, 1 patient had a transient dysphasia, and 1 patient had a transient hyperesthesia of the hand. Aside from the quadrantanopia, all postoperative deficits were transient and resolved within 1 week after surgery in most of the patients. Among these 9 symptomatic patients, 6 had an ischemic lesion related to the insular resection. Consequently, 5 patients with an ischemic lesion resulting from an insulectomy were asymptomatic. There was no statistical relationship between the identification of an ischemic lesion and the occurrence of a neurological deficit ($p = 0.4$, Fisher exact test). None of the patients with an ischemic lesion resulting from an insulectomy complained of memory loss. No ischemic lesions were detected in the territory of the lateral lenticulostriate arteries (LLAs).

Discussion

In this series of operculoinsular corticectomies, 45% had transient neurological complications (no permanent

FIG. 4. Early postoperative axial diffusion-weighted MR image showing an acute ischemic lesion related to an insular corticectomy.

deficit) and 60% had an ischemic lesion on postoperative MRI. Surgery of the insular area is known to be challenging because of its close relationship with vascular structures.^{8,15,26,36,38–40} Immediate postoperative deficits have been previously reported with incidence rates between 9.2% and 74% in the literature. Comparison in incidence rate of transient or permanent complications is difficult

FIG. 5. Drawing illustrating a typical insulectomy-related ischemic lesion located in the corona radiata running from the insula to the periventricular region. Copyright Patrice Finet. Published with permission.

considering that these series have mostly dealt with patients with tumors or vascular malformations often extending outside the insula both to surrounding lobes and in deeper subcortical structures. In our series, corticectomies were performed in insular cortices without macroscopically visible lesions.

The most important source of permanent deficits identified in previous series for oncological or vascular indications has been vascular damage of the LLAs.^{12,17,22,35,38,39,44} In our series, no ischemic lesion was attributed to an LLA interruption. The ischemic lesions typically encountered after insular corticectomies were linked to injuries of long perforating branches of the M₂ segments of the MCA. These branches represent 3%–5% of the perforating branches of the M₂ segments of the MCA and reach as far as the corona radiata.³⁹ Yaşargil et al. have highlighted that these vessels most commonly arise from the posterior M₂ branches.⁴⁴ Coagulation of these long perforating arteries from the M₂ segments of the MCA can result in hemiparesis. The remaining perforating branches of the M₂ segments are short (85%–90%) and medium (10%) in length and supply the insular cortex, the extreme capsule, the claustrum, and the external capsule.³⁹ As the insular cortex and a thin layer of the underlying white matter is removed in insular corticectomies, ischemic lesions related to short and medium branches are not observable. Although perforating branches have been described to arise preferentially from the posterior M₂ branches, we did not find a relationship between ischemic lesion occurrence and location of the insular corticectomy in the anterior or posterior insula. We did not observe a zone of predilection for ischemic lesions after insular corticectomy.

Whether these ischemic lesions related to the insular corticectomies are responsible for the transient symptoms presented by our patients remains uncertain. Indeed, only 6 of the 9 symptomatic patients had an ischemic lesion related to the insular corticectomy and 5 patients with a typical insular corticectomy-related ischemic lesion on MRI were asymptomatic. In addition, most of the symptomatic patients recovered within the 1st postoperative week, which would be unusual after an ischemic stroke.^{16,18,19}

The transient postoperative deficits can be explained by different alternate hypotheses. Removing the insular cortex itself could be a potential explanation for postoperative deficits because of the disturbance of its intrinsic functions. The rapid functional recovery could then be explained by the relatively small volume of resection and the high connectivity of the insula to compensatory surrounding and distant structures.^{1,5,10,11,13,34,35}

Another possible explanation for the transient postoperative deficits is the occurrence of vasospasm of the M₂ branches induced by the surgical dissection along these branches.^{22,44} In our patients, MCA manipulation was minimized by using subpial insular resection. Vasospasm was sometimes observed and was treated with papaverine. One patient had an early postoperative angio-CT scan that revealed the absence of an early postoperative vasospasm.

Opercular retraction is another potential source of postoperative deficits. As demonstrated by Lang et al., a significant retraction of the frontal operculum is required to reach the superior periinsular sulci.²² This retraction

can be responsible for postoperative hemiparesis or dysphasia when surgery is performed on the dominant hemisphere. Frontal opercular retraction results in direct pressure on Broca's area or compression of the M₃ branches of the MCA with frontal ischemia as a consequence. We have not noticed any difference in postoperative deficits between superior or inferior insular corticectomies. Performing an operculectomy on the nondominant side or on the dominant side avoiding Broca's and Wernicke's areas is a method to avoid opercular retraction.¹² In our series, an operculectomy was carried out only when the opercula were suspected to participate in the epileptic focus. We realized an operculectomy in the majority of our studied patients. Awake surgery is another potential way to reduce deficits linked to opercular retraction.^{22,25}

Finally, an early transient postoperative deficit can be due to edema as a result of surgical manipulation, especially since most of the symptoms resolved quickly.³¹

Conclusions

Ischemic lesions and neurological deficits are not uncommon in operculoinisular corticectomies performed for treating epilepsy but do not result in permanent neurological deficits. Hence, microsurgery of the insular cortex should be considered as a viable option for patients suffering from intractable disabling seizures, provided the surgeon has good knowledge of the perisylvian anatomy and vasculature.

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Author Contributions

Conception and design: all authors. Acquisition of data: all authors. Analysis and interpretation of data: Finet. Drafting the article: all authors. Critically revising the article: Nguyen, Bouthillier. Reviewed submitted version of manuscript: Finet, Bouthillier. Approved the final version of the manuscript on behalf of all authors: Finet. Study supervision: Bouthillier.

Supplemental Information

Previous Presentation

Portions of this work were presented as an oral presentation at the Société de Neurochirurgie de la Langue Française (SNCLF) in December 2013.

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