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## Semiology of insular lobe seizures

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*« Les premiers seront les derniers (The first shall be the last) »*

The role of the insular lobe in focal epilepsies has had a tumultuous history; it held Penfield's focus in the 1950s [1] and then became a target for pioneering neurosurgeons [2-4] before falling into oblivion from the 1960s to 1980s. At a time when intra-operative electrocorticographic recordings were the only measure of direct anatomic-electrophysiological correlations, defining the epileptogenic area relied upon the identification of abundant, focal interictal activity and the ability to evoke symptoms identical to those of spontaneous seizures during electrical stimulation of the target region [1,5]. These two findings were frequently encountered in the insular lobe of patients in whom a temporal lobectomy had been performed without success. Penfield was the first to identify the similarities in semiology between temporal lobe epilepsy (TLE) and insular lobe seizures and pointed out the risk of misdiagnosis, that is, seizures classically attributed to the temporal lobe in fact originating in the insular lobe [6]. Since then, the epileptology community had become familiar with this concept, and yet, everything would change with the introduction of pre-operative chronic intracranial recordings using stereo-electroencephalography (SEEG).

The use of intracranial electrodes to monitor patients from several days to even multiple weeks provided the opportunity to register not only interictal activity but ictal events, thereby enabling epileptologists to directly examine the seizure activity and its dynamic spatio-temporal development in close association with symptoms. Accordingly, the era of short duration, intra-operative explorations had passed and was replaced by the recording of seizures with chronic intracranial electrode implantations. However, the first electrode implantation techniques did not allow easy access to the insular-opercular complex due to the high surgical risk, whether with subdural grids or SEEG electrodes. This barrier, unfortunately, led the epileptology community to ignore this hidden but potentially important target, and therefore, to quite literally forget the very notion of insular lobe seizures. This was particularly evident in the writing of epilepsy surgery encyclopedias, where insular seizures could not be found in the classical seizure classification [7] and was often given only a few lines of text [8,9]. For decades, the nosography of epilepsy disregarded the insular lobe and acted as if each hemisphere was composed of only four lobes: frontal, temporal, parietal and occipital lobes.

However, the direction of insular lobe seizure took a change again in the early 1990s when progress in the field of brain imaging allowed anatomic-electro-clinical correlations to be established between insular lesions and the semiology of the associated seizures [10]. Meanwhile, as modern SEEG explorations allowed electrodes to safely cross the opercular wall

to reach the insular lobe [11], there was renewed enthusiasm to examine this once neglected origin of seizures.

In 1998, at the San Diego American Epilepsy Society meeting, we presented the SEEG findings from a case series of 20 patients who were suffering from mesial temporal lobe epilepsy (MTLE) and had an implantation within the insular lobe [12]. Three points were highlighted in the conclusion and have stood the test of time:

- Some of the symptoms attributed to temporal lobe seizures are generated by the propagation of mesial temporal discharges to the insular lobe in accordance with the notion of the symptomatic zone proposed by Rosenow and Luders [13].
- Some seizures thought to arise from the mesial temporal lobe in fact have an onset within the insular lobe; this is often unappreciated and can be a source of surgical failures in “TLE”. This result has been confirmed by recent SEEG studies [14,15].
- The insular ictal discharge tends to propagate to the ipsilateral frontal lobe.

Gradually, more and more epilepsy centers began directly exploring the insular lobe using SEEG techniques and embarking on the task of understanding this complex region [16,17,18,19]. From 2000 to 2017, there has been a 17-fold increase in publications regarding insular lobe seizures compared to the previous 50 years (PubMed search based on title for “insular” AND (“seizure” OR “epilepsy”) (The search was limited to English-language publications and studies of humans) returned three publications between 1950-1999 and 52 during 2000-2017 period). The analysis of the anatomico-electro-clinical correlations has progressed significantly with the publications of multiple large case series and has made it possible today to propose a consensual description of the major clinical forms of insular lobe seizures.

## Semiology of insular lobe seizures

The study of insular seizure semiology involves analyzing the presence and evolution of clinical symptoms that appear during the development of an ictal discharge within the insular lobe. Seizures may arise from any part of the insula and frequently spread to adjacent medial opercular regions which share some functional commonalities with the insula. The best example is the painful seizure, where the ictal discharges often involve the posterior insula as well as the medial parietal operculum [20], and such functional commonality is well supported by the

neurophysiological evidence [21]. Therefore, it is often more accurate to describe these as insulo-opercular seizures, rather than strictly insular seizures.

The diversity of symptoms associated with insular lobe seizures is related to the multiple functional roles of the insula and its diffuse connectivity with other lobes [22,23,24]. The insular origin of a broad range of symptoms, including those related to somatosensory, visceral, auditory, vestibular, olfacto-gustatory and language systems, has been confirmed by the direct electrical stimulation study of the insula using SEEG electrodes [25].

After being first reported in 2006, it has been recognized that insular origin seizures can also present with predominant motor manifestations which are linked to the propagation of the insular discharge to the frontal lobe [14,19,26,27], especially towards the fronto-mesial regions [28], and/or possibly to the subcortical structures [29]. Such motor symptoms masquerading as frontal seizures are usually preceded by brief period of insulo-opercular symptoms (Fig. 1). Signs of intense opercular involvement, such as hypersalivation, clonic facial contractions, and strong laryngeal constriction, also help to identify the insular origin of seizures (Fig. 1).

We propose that the semiological features of insular seizures be considered according to the following three distinct levels: general characteristics, major symptoms, and minor symptoms.

### General characteristics

Insular seizures are, for the most part, of the ‘simple partial’ type, i.e., they proceed, at least initially, with the persistence of consciousness and contact, and patients have a preserved capacity to react to the interactions of others.

These seizures are frequently nocturnal and often occur in clusters.

Except in the context of weaning medications during video-EEG recording, secondary generalization is rare.

It is very common to observe a significant delay from initial consultation to diagnosis of insular seizure. Referring doctors instead pursue diagnoses of psychogenic non-epileptic seizures, pure psychiatric conditions, sleep-related disorders or other functional neurological diseases due to poor recognition of this clinical entity.

### Major symptoms

These are clinical signs that are frequently observed in insular seizures. If these following symptoms are identified, then electrode implantation within the insular cortex is mandatory during SEEG evaluation.

### **Insulo-opercular semiology**

Owing to the largely subjective nature of this semiology, careful interview is required for an accurate and thorough assessment of each symptom. Intracortical electrical stimulation through SEEG electrodes is necessary to firmly establish anatomo-semiological correlations.

Somatosensory manifestations are frequently found, in particular when the ictal discharge develops in the posterior insulo-opercular region. Patients often provide diverse descriptions owing to the multisensory nature of this region [22,23]. Migratory/spreading paraesthesias of a 'tingling' type or low intensity electric currents are the most commonly observed subjective sensations, typically involving large cutaneous areas [25,30]. When these symptoms are bilateral and symmetrical or of a heat/cold nature they are even more supportive of an insular origin. Further, they can take on a painful character (Fig. 2a), which is considered pathognomonic of an insular seizure [20,31].

A discomfort referred to the cervico-laryngeal region, ranging from a simple difficulty of swallowing to the terrifying sensation of suffocation by strangulation (Fig. 2b), is very frequently observed [17] and usually points to an origin in the anterior insula [25]. This unpleasant sensation is often accompanied with shortness of breath or a prolonged respiratory pause, sometimes for tens of seconds. Yet this respiratory pause or breath holding can be subtle, only becoming evident towards the end of the seizure when respiration recommences in dramatic gasps for breath. Strong laryngeal constriction seems to reflect an opercular involvement.

Viscero-sensory and viscero-motor manifestations are also frequently observed. They include nausea and epigastric or abdominal heaviness, similar to those described in MTLE, and occasionally vomiting [14,16,17,25,32]. These symptoms may or may not ascend in the chest or abdomen. These visceral symptoms typically suggest an ictal discharge in the anterior insula. In fact, it is difficult to know by means of clinical observation alone whether these symptoms reflect insular seizures propagating to the mesial temporal structures or vice versa; this uncertainty can be attributed to the existence of dense reciprocal connections between the antero-inferior insula and the temporo-mesial limbic regions. This has likely contributed to the misdiagnosis of insular lobe seizure as MTLE and therefore to unexpected surgical failures of apparent MTLE. However, such misdiagnosis is less of an issue today given the contribution

of magnetic resonance imaging, and role of hippocampal sclerosis in localizing the epileptogenic area to the mesial temporal structures. This crucial piece of semiological information was obviously missed by the pioneers.

### **Frontal/pseudo-frontal semiology**

Insular origin hypermotor seizures, often occurring in clusters and with nocturnal predominance [19,26], present with automatic motor behaviors with or without arm elevation (Fig. 2c), which often leads to erroneous suspicion of fronto-mesial seizures [28]. The hypermotor presentation was initially associated with seizures originating from the antero-superior portion of the insula [26], but later studies showed more posterior insular origins can also display this semiology [19,28]. Symptoms range from discreet gestural movements (e.g., brief pedaling, swinging movements along the vertical axis of the body) to sometimes spectacular motor phenomena, characterized by apparent disorderly agitation with choreo-dystonic features (asymmetric tonic posturing, hemiballistic movements, bucco-linguo-facial dyskinesia) or organized behaviors that reflect apparent strong emotional states (i.e., symbolic behaviors). In the vast majority of cases, these often dramatic hyperkinetic activities are not consciously perceived by patients. The automatic affective-behavioral aspects of hypermotor seizures are reminiscent of adaptive behaviors that derive from phylogenetically-old brain structures in association with survival [33], such as those seen in response to impending danger (e.g., fear, aggression, pain, expressions of suffering).

### **Minor symptoms**

These are less frequent, but their presence reinforces the probability of an insular origin of the seizure. The association of multiple minor symptoms reflects the rich functional value of the insulo-opercular region, as has been demonstrated with electrical stimulation through SEEG electrodes [25]. Of note, while these minor symptoms can easily be missed especially in the presence of major disabling symptoms, patients often disclose them following the electrical cortical stimulation in both a qualitatively- and chronologically-precise manner.

### **Auditory hallucinations**

Usually consisting of simple elementary sounds, but may present as complex sounds. They can originate not only from the temporal operculum (Heschl's gyrus) but also from the postero-

inferior portion of the insula [25]. These auditory symptoms are occasionally associated with somatosensory symptoms centered around the ear.

### **Eyelid blinking**

This likely represents a minor symptom of hypermotor seizures [19], and is manifested by palpebro-facial spasm with thinning of the palpebral fissure ipsilateral to the epileptic insulo-opercular region (Fig. 2d). It should not be confused with clonic hemifacial contractions, which equally involve the upper and lower face and indicate the contralateral subcentral opercular region. Co-occurrence of ipsilateral eyelid blinking and contralateral clonic facial contractions is possible. Whether the uncrossed cortico-bulbar projections [34] or ephaptic stimulation of trigeminal/facial nerves [35] can explain these phenomena remains to be clarified.

### **Hypersalivation**

This usually reflects the spread of the ictal discharge to the supra-sylvian opercular region and is sometimes accompanied with dysarthria [17].

### **Gustatory symptoms**

Gustatory symptoms rarely encountered but are of high localizing value according to cortical stimulation data which has shown that these symptoms are evoked by stimulation of the middle portion of insular lobe situated below the sub-central operculum [25,36]. Olfactory symptoms have also been identified in the same insular subregion but are even more rare than the gustatory symptoms [25,36].

### **Vestibular symptoms**

Rarely encountered, vestibular symptoms are generally localized to the posterior part of the insula. When these symptoms are reproduced by electrical stimulation of the insula [25], patients describe hallucinations of body movement in space (translation (e.g., flying/rising, falling), rotation or indefinable sensations of head spinning) rather than sensations of instability or movement of the visual environment.

### **Speech disturbance**

This is also a rarely encountered symptom, the degree of which ranges from complete speech arrest (anarthria/aphemia) to missing words or impaired fluency. No paraphasia occurs.



Electrical stimulation of the insula can reproduce these symptoms regardless of the hemisphere dominance for language [17,25].

Finally, despite the fact that the insula constitutes a part of the autonomic cardiovascular system, an objective correlation between insular seizures and heart rate changes is rarely experienced [25,37]. Other possible manifestations of insular seizures, such as ecstatic [38] and gelastic seizures [39], remain highly anecdotal and thus are out of the scope of this article.

## Conclusions

Insular origin seizures have unique characteristics, allowing them to be identified in a straightforward and timely manner. As such, formulating the hypothesis of an insular epileptogenic zone should occur early and not be reserved for when the seizure cannot be localized to any other lobe. Careful interpretation of anatomo-clinical correlations is therefore essential in both constructing the hypothesis of an insular origin and to plan the appropriate SEEG exploration [40]. Developing an inappropriate SEEG schema risks misidentification of the true epileptogenic area. In particular, it can result in a failure to identify the insular origin of seizures, then misdiagnose as a temporal or frontal lobe epilepsy, and finally lead to an inadequate and non-efficacious corticectomy.

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## Disclosure of interest

The authors declare no conflict of interest concerning this article.

## References

- [1] Penfield W, Faulk ME Jr. The insula; further observations on its function. *Brain* 1955;78:445–70.
- [2] Silfvenius H, Gloor P, Rasmussen T. Evaluation of insular ablation in surgical treatment of temporal lobe epilepsy. *Epilepsia* 1964;5:307–20.
- [3] Guillaume MMJ, Mazars G. Cinq cas de foyers épileptogènes insulaires opérés. *Société Française de Neurologie* 1949;766–9.
- [4] Guillaume MMJ, Mazars G. Technique de résection de l'insula dans les épilepsies insulaires. *Rev Neurol* 1949;81:900–3.
- [5] Penfield W, Jasper H. *Epilepsy and the Functional Anatomy of the Human Brain*. Boston: Little, Brown and Company; 1954.
- [6] Guillaume MMJ, Mazars G, Mazars Y. Indications chirurgicales dans les épilepsies dites "temporales". *Rev Neurol* 1953;88:461–501.
- [7] Williamson D, Engel J, Munari C. Anatomic classification of localization-related epilepsy. In Engel J, Pedley T, eds. *Epilepsy*. Philadelphia: Lippincott-Raven, 1997;2405–26.
- [8] Wieser HG. *Electroclinical features of the psychomotor seizure*. Stuttgart: Gustav Fischer, 1983.
- [9] Lüders HO, Comair YG. *Epilepsy Surgery*. 2nd ed. Lippincott Williams & Wilkins; 2001, p. 1060.
- [10] Roper SN, Lévesque MF, Sutherling WW, Engel J Jr. Surgical treatment of partial epilepsy arising from the insular cortex. Report of two cases. *J Neurosurg* 1993;79:266–9.

- [11] Salado AL, Koessler L, De Mijolla G, Schmitt E, Vignal JP, Civit T, Tyvaert L, Jonas J, Maillard L, Colnat-Coulbois S. sEEG is a safe procedure for a comprehensive anatomic exploration of the insula: A retrospective study of 108 procedures representing 254 transopercular insular electrodes. *Oper Neurosurg* 2018;14:1–8.
- [12] Isnard J, Ryvlin P, Guénot M, Ostrowsky K, Fischer C, Sindou M, Mauguère F. Role of the insular cortex in temporolimbic seizures : a SEEG study. AES. San Diego, 6-9 December 1998.
- [13] Rosenow F, Lüders H. Presurgical evaluation of epilepsy. *Brain* 2001;124:1683–700.
- [14] Nguyen DK, Nguyen DB, Malak R, Leroux JM, Carmant L, Saint-Hilaire JM, Giard N, Cossette P, Bouthillier A. Revisiting the role of the insula in refractory partial epilepsy. *Epilepsia* 2009;50:510–20.
- [15] Barba C, Minotti L, Job AS, Kahane P. The Insula in Temporal Plus Epilepsy. *J Clin Neurophysiol* 2017;34:324–27.
- [16] Isnard J, Guénot M, Ostrowsky K, Sindou M, Mauguère F. The role of the insular cortex in temporal lobe epilepsy. *Ann Neurol* 2000;48:614–23.
- [17] Isnard J, Guénot M, Sindou M, Mauguère F. Clinical manifestations of insular lobe seizures: a stereo-electroencephalographic study. *Epilepsia* 2004;45:1079–90.
- [18] Afif A, Chabardes S, Minotti L, Kahane P, Hoffmann D. Safety and usefulness of insular depth electrodes implanted via an oblique approach in patients with epilepsy. *Neurosurgery* 2008;62:471–9.
- [19] Proserpio P, Cossu M, Francione S, Tassi L, Mai R, Didato G, Castana L, Cardinale F, Sartori I, Gozzo F, Citterio A, Schiariti M, Lo Russo G, Nobili L. Insular-opercular seizures manifesting with sleep-related paroxysmal motor behaviors: a stereo-EEG study. *Epilepsia* 2011;52:1781–91.
- [20] Montavont A, Mauguère F, Mazzola L, Garcia-Larrea L, Catenoix H, Ryvlin P, Isnard

- J. On the origin of painful somatosensory seizures. *Neurology* 2015;84:594–601.
- [21] Garcia-Larrea L, Mauguière F. Pain syndromes and the parietal lobe. In: *The Parietal Lobe*, Vallar G. and Coslett, HB, Eds; *Handbook of Clinical Neurology*, Philadelphia: Elsevier; 2018, p. 207–223.
- [22] Augustine JR. Circuitry and functional aspects of the insular lobe in primates including humans. *Brain Res Brain Res Rev* 1996;22:229–44.
- [23] Nieuwenhuys R: The insular cortex: a review. *Prog Brain Res* 2012;195:123–63.
- [24] Almashaikhi T, Rheims S, Jung J, Ostrowsky-Coste K, Montavont A, De Bellescize J, Arzimanoglou A, Keo Kosal P, Guénot M, Bertrand O, Ryvlin P. Functional connectivity of insular efferences. *Hum Brain Mapp* 2014;35:5279–94.
- [25] Mazzola L, Mauguière F, Isnard J. Electrical Stimulations of the Human Insula: Their Contribution to the Ictal Semiology of Insular Seizures. *J Clin Neurophysiol* 2017;34:307–31.
- [26] Ryvlin P, Minotti L, Demarquay G, Hirsch E, Arzimanoglou A, Hoffman D, Guénot M, Picard F, Rheims S, Kahane P. Nocturnal hypermotor seizures, suggesting frontal lobe epilepsy, can originate in the insula. *Epilepsia* 2006;47:755–65.
- [27] Döbesberger J, Ortler M, Unterberger I, Walser G, Falkenstetter T, Bodner T, Benke T, Bale R, Fiegele T, Donnemiller E, Gotwald T, Trinka E. Successful surgical treatment of insular epilepsy with nocturnal hypermotor seizures. *Epilepsia* 2008;49:159–62.
- [28] Hagiwara K, Jung J, Bouet R, Abdallah C, Guénot M, Garcia-Larrea L, Mauguière F, Rheims S, Isnard J. How can we explain the frontal presentation of insular lobe epilepsy? The impact of non-linear analysis of insular seizures. *Clin Neurophysiol* 2017;128:780–91.
- [29] Ghaziri J, Tucholka A, Girard G, Boucher O, Houde JC, Descoteaux M, Obaid S, Gilbert G, Rouleau I, Nguyen DK. Subcortical structural connectivity of insular subregions. *Sci*

Rep 2018;8:8596.

- [30] Mazzola L, Isnard J, Mauguière F. Somatosensory and pain responses to stimulation of the second somatosensory area (SII) in humans. A comparison with SI and insular responses. *Cereb Cortex* 2006;16:960–8.
- [31] Isnard J, Magnin M, Jung J, Mauguière F, Garcia-Larrea L. Does the insula tell our brain that we are in pain? *Pain* 2011;152:946–51.
- [32] Catenoix H, Isnard J, Guénot M, Petit J, Remy C, Mauguière F. The role of the anterior insular cortex in ictal vomiting: a stereotactic electroencephalography study. *Epilepsy Behav* 2008;13:560–3.
- [33] Tassinari CA, Cantalupo G, Högl B, Cortelli P, Tassi L, Francione S, Nobili L, Meletti S, Rubboli G, Gardella E. Neuroethological approach to frontolimbic epileptic seizures and parasomnias: The same central pattern generators for the same behaviours. *Rev Neurol* 2009;165:762–8.
- [34] Mankall EL. Brainstem. In: *Gray's Clinical Neuroanatomy: The Anatomic Basis for Clinical Neuroscience*, Philadelphia: Elsevier; 2011, p. 157–84.
- [35] Begum T, Ikeda A, Matsushashi M, Mikuni N, Miyamoto S, Hashimoto N, Nagamine T, Fukuyama H, Shibasaki H. Ipsilateral facial sensory and motor responses to basal fronto-temporal cortical stimulation: evidence suggesting direct activation of cranial nerves. *Epilepsy Res* 2006;71:216–22.
- [36] Mazzola L, Royet JP, Catenoix H, Montavont A, Isnard J, Mauguière F. Gustatory and olfactory responses to stimulation of the human insula. *Ann Neurol* 2017;82:360–70.
- [37] Chouchou F, Bouet R, Pichot V, Catenoix H, Mauguière F, Jung J. The neural bases of ictal tachycardia in temporal lobe seizures. *Clin Neurophysiol* 2017;128:1810–19.
- [38] Gschwind M, Picard F. Ecstatic Epileptic Seizures: A Glimpse into the Multiple Roles of the Insula. *Front Behav Neurosci* 2016;10:21.

- [39] Tran TP, Truong VT, Wilk M, Tayah T, Bouthillier A, Mohamed I, Nguyen DK. Different localizations underlying cortical gelastic epilepsy: case series and review of literature. *Epilepsy Behav* 2014;35:34–41.
- [40] Gras-Combe G, Minotti L, Hoffmann D, Krainik A, Kahane P, Chabardes S. Surgery for nontumoral insular epilepsy explored by stereoelectroencephalography. *Neurosurgery* 2016;79:578–88.

## Figure legends

**Fig. 1.** The two semiological patterns of insular origin seizures: (i) insulo-perisylvian semiology followed by signs of significant opercular involvement, (ii) frontal/pseudo-frontal semiology with predominant hyperkinetic symptoms. Major symptoms are indicated by red frames. The frontal/pseudo-frontal symptoms can be preceded by or accompanied with the insulo-perisylvian symptoms (dotted arrow).

**Fig. 2.** Examples of objective symptoms in insular lobe seizures. (a) left-sided painful sensation with grimacing. (b) dyspnea due to cervico-laryngeal discomfort or laryngeal constriction. (c) hyperkinetic seizure with tonic elevation of both upper limbs. (d) left (ipsilateral) eye blinking (arrow) during a left insular seizure.

# INSULAR SEIZURE



## Insulo-perisylvian

Somatosensory  
Cervico-laryngeal discomfort

Visceral/Viscero-psychic  
Auditory  
Vestibular  
Gustatory  
Speech disturbance



## Opercular

Hypersalivation  
Clonic facial contraction  
Laryngeal constriction



## Frontal/Pseudo-frontal

Hyperkinesia  
- Gestural  
- Choreo-dystonic  
- Symbolic-behavioral

Arm elevation  
Ipsilateral eyelid blinking





a



b



c



d

