

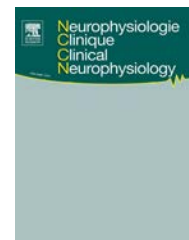


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COMPREHENSIVE REVIEW

Planning and management of SEEG

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KEYWORDS

Epileptogenic zone;
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Stereo-EEG

Summary Stereoelectroencephalography (SEEG) aims to define the epileptogenic zone (EZ), to study its relationship with functional areas and the causal lesion and to evaluate the possibility of surgical therapy. Planning of exploration is based on the validity of the hypotheses developed from electroclinical and imaging correlations. Further investigations can refine the implantation plan (e.g. fluorodeoxyglucose positron emission tomography [FDG-PET], single photon emission computerized tomography [SPECT], magnetoencephalography [MEG] and high resolution electroencephalography [EEG-HR]). The scheme is individualized according to the features of each clinical case, but a general approach can be systematized according to the regions involved (temporal versus extra-temporal), the existence of a lesion, its type and extent. It takes account of the hemispheric dominance for language if this can be determined. In “temporal plus” epilepsies, perisylvian and insular regions are among the key structures to investigate in addition to mesial and neocortical temporal areas. In frontal lobe epilepsies, determining the functional and anatomical organization of seizures (anterior versus posterior,

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mesial versus dorsolateral) allows better targeting of the implantation. Posterior epilepsies tend to have a complex organization leading to multilobar and often bilateral explorations. In lesional cases, it may be useful to implant one or several intralesional electrode(s), except in cases of vascular lesions or cyst. The strategy of implantation can be modified if thermocoagulations are considered. The management of SEEG implies continuous monitoring in a dedicated environment to determine the EZ with optimal safety conditions. This methodology includes spontaneous seizure recordings, low and high frequency stimulations and, if possible, sleep recording. SEEG is applicable in children, even the very young. Specific training of medical and paramedical teams is required.

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Introduction

The aim of SEEG is to determine the location of the epileptogenic zone (EZ) and the propagation pathways in order to perform a surgical treatment. While the concept and definition of EZ has been subject to some controversy according to different schools of epileptology, the present work refers to the definition developed by the founders of the SEEG methodology and is based on neurophysiological criteria [1–6]. The EZ integrates the site of onset and primary organization of ictal discharges and their correlation with clinical expression, interictal electrical abnormalities and anatomical data. Analyzing the spatial-temporal dynamic of ictal events is crucial for defining the EZ. Once established, relationships between the EZ and functional anatomical structures, and feasibility of surgical resection or disconnection have to be evaluated. Thus the strategy of implantation must meet the following objectives: (1) to define the EZ at the neurophysiological level, (2) to study its relations with highly functional areas and the causal lesion if identified and (3) to evaluate the possibilities of surgical treatment. The management of SEEG implies the determination of EZ with optimal security conditions. The training of medical and paramedical teams is included in these recommendations.

Methods

As in other chapters related to SEEG methodology, recommendations concerning the planning and management of the exploration were drawn from the experience of the teams confronted in a consensus meeting and supported by the data from existing literature. We identified references from PubMed with the terms "stereo-EEG", "stereo-electroencephalography", "SEEG", "invasive recordings", "epileptogenic zone", "electrical stimulations", "epilepsy surgery", "partial seizures", "temporal lobe epilepsy", "frontal lobe epilepsy", "parietal lobe epilepsy", "occipital lobe epilepsy", "insular epilepsy", "hippocampal sclerosis", "malformation of cortical development", "focal cortical dysplasia", "cryptogenic epilepsy", "negative MRI" in various associations, without time limit and including articles in

French and English. The final list was established on the basis of the SEEG-specific reference including historical data, with relevance to current practices.

We will successively examine the consensus recommendations concerning (1) pre-implantation assessment (phase 1); (2) planning of implantation according to the presumed location and lateralization of the EZ; (3) particular aspects according to the underlying cause of epilepsy (lesional versus non-lesional); (4) specificities of management in children and (5) training of personnel.

Pre-implantation assessment

Since the first recordings and the development of the methodology by the pioneers of SEEG, widespread improvement of techniques and practices has progressively been adopted, while remaining faithful to the initial concepts. The pre-implantation assessment is currently well codified although resources may vary significantly from one center to another, as evidenced by a recent European survey [7]. Seizure recordings with EEG-video monitoring and high quality MRI constitute the minimum base that is mandatory for planning SEEG. Morphological imaging techniques are regularly improved according to technological advances [8–10]. Protocols dedicated to epilepsy must be available. Other non-invasive techniques have proved to be useful to localize the EZ and to refine the implantation strategy, especially in negative-MRI cases (PET with 18F-fluorodeoxyglucose (FDG) [11–15]; SPECT during the ictal period, compared to interictal examination [16]; MEG [17,18]; and electrical source imaging with EEG-HR [19–22]). The multimodal approach seems the most attractive but requires access to highly specialized and expensive equipment. However, the respective contribution of each tool is not formally established to date. If the patient's age and possibilities of cooperation allow it, performing a neuropsychological assessment and/or functional MRI (fMRI) may be useful at this stage in determining hemispheric dominance for language and anticipating the functional consequences of the surgical resection. However, this point of view was not shared by all teams, especially pediatric teams, since fMRI is not feasible in a number of situations.

Planning of implantation

The implantation project integrates all the data acquired during the non-invasive phase. It is materialized on a scheme elaborated within the medical-surgical multi-disciplinary team. The strategy of implantation depends basically on the validity of the hypotheses developed from anatomico-electro-clinical correlations [1,5,6]. This step implies the elaboration of hypotheses on the EZ and their confrontation with the cerebral space of the patient, taking into account the anatomical data and vascular constraints. It is advised to formulate a main hypothesis to focus the exploration and alternative hypotheses to avoid sampling error. The project also anticipates the surgical approach and the limits of the cortical resection. In some cases, if thermocoagulations are planned, it may be suggested to orient the implantation strategy accordingly [23,24]. The relevance of each implantation should be regularly evaluated in order to optimize the practices.

The number of electrodes mainly depends on the cerebral volume and the number of structures and lobes to be explored according to the initial hypotheses. Each electrode must be justified by clinical, electrophysiological or anatomical arguments. The number of implanted electrodes is not fixed absolutely; this varies most often between 7 and 14 [25]. Apart from special cases (additional exploration or "over-implantation" for thermocoagulations), when only a small number of electrodes (<6) seems necessary, the relevance of the SEEG should be discussed. Conversely, when a large number of electrodes seem necessary (>15), one can wonder about the contribution of a complementary investigation that could reduce their number. This view has been discussed by some experts, who consider that an upper limit in the number of electrodes should not be fixed. However, it must be recalled here that, despite a low risk of complications related to the implantation of SEEG electrodes [25–27], the benefit-risk ratio of each electrode must be considered, especially in highly functional areas.

Electrode nomenclature is not standardized. The choice of alphabetical letters initially chosen by the pioneer team has evolved according to the various centers. There is no simple and easily reproducible reference system; the most commonly used being the name of the cerebral target of the electrode. However, with the increasing use of oblique electrodes, it is necessary to add the entry point on the cortex that complicates the readability of the scheme. At this stage, it is not possible to recommend a specific nomenclature, but it is suitable that it could be clearly explained within each center.

General rules

The implantation scheme is individual and formulated on the basis of electroclinical and imaging data. However, a general approach may be systematized according to lobar involvement and anatomico-functional connectivity, the existence of structural abnormalities and the type of causal lesion. It takes into account hemispheric dominance for language if this can be determined. Multiplication of electrodes in highly functional areas (speech, motor skills) should be avoided unless the question precisely addresses their

early involvement. Lateralized implantation focusing on one hemisphere should be preferred, but if it is necessary to implant contralateral electrodes, these should be placed symmetrically if possible. In contrast, a bilateral and symmetrical exploration, with the same number of electrodes in both hemispheres, is not recommended.

Temporal lobe epilepsies

Semiology of temporal lobe seizures and different types of EZ organization based on SEEG have been widely described [1,28–35]. Cumulative data have allowed limiting the use of SEEG in an increasing number of cases, especially in typical mesial temporal lobe epilepsies (MTLE) related to a structural lesion. Moreover, in some so-called "MRI-negative" cases, it has been suggested that invasive recordings can be avoided if there is strong electroclinical evidence for typical mesial temporal involvement and concordant metabolic abnormalities [36,37]. It should be emphasized that this view obtained a low consensus rate in the current expert working party, especially in dominant hemisphere in which systematic SEEG was still considered mandatory in non-lesional cases. Sampling involves the mesial temporal structures (hippocampus and amygdala), the entorhinal cortex, the middle temporal gyrus (MTG) and basal cortex, the superior temporal gyrus (STG) and, if possible, the temporal pole and the insular cortex.

In the hypothesis of neocortical temporal epilepsy, sampling includes the lateral temporal regions (STG and MTG) in their anterior and posterior parts. It should be noted that orthogonal electrodes also target the hippocampus or para-hippocampal gyrus (PHG) by their mesial contacts. In the case of anterior propagation, it may be appropriate to explore the temporal pole, the inferior temporal gyrus (ITG), the fronto-temporal junction and the anterior insula. Several electrodes can be placed in these regions to ensure better coverage (since a single electrode with few contacts in the STG or MTG would be insufficient). If the propagation is mainly posterior, the sampling will include the temporal planum, the supramarginal gyrus, the posterior insula, the fusiform gyrus and the temporo-occipital junction.

Lessons provided by SEEG have also helped in characterizing more complex EZ organization, leading to the concepts of temporo-perisylvian or temporal "plus" epilepsies [38–52]. Improvement of knowledge of these different entities helps to guide the implantation strategy, depending on the hypotheses that have been elaborated (Fig. 1). In the hypothesis of anterior temporo-perisylvian epilepsy, sampling involves the orbito-frontal cortex, the anterior insula, the precentral opercular cortex and the anterior cingulate gyrus (ACG) in addition to the aforementioned temporal regions. In case of involvement of posterior regions, it will involve the posterior STG (Heschl's gyrus and planum temporale), the posterior insula and the post-central operculum, the temporo-parieto-occipital junction and the posterior cingulate gyrus (PCG). In the hypothesis of bitemporal epilepsy, or in the case of early contralateral spread, one side supposed to be the most involved is sampled as in the implantation scheme described for MTLE, with at least one contralateral electrode (usually in the anterior hippocampus). Other electrodes can be

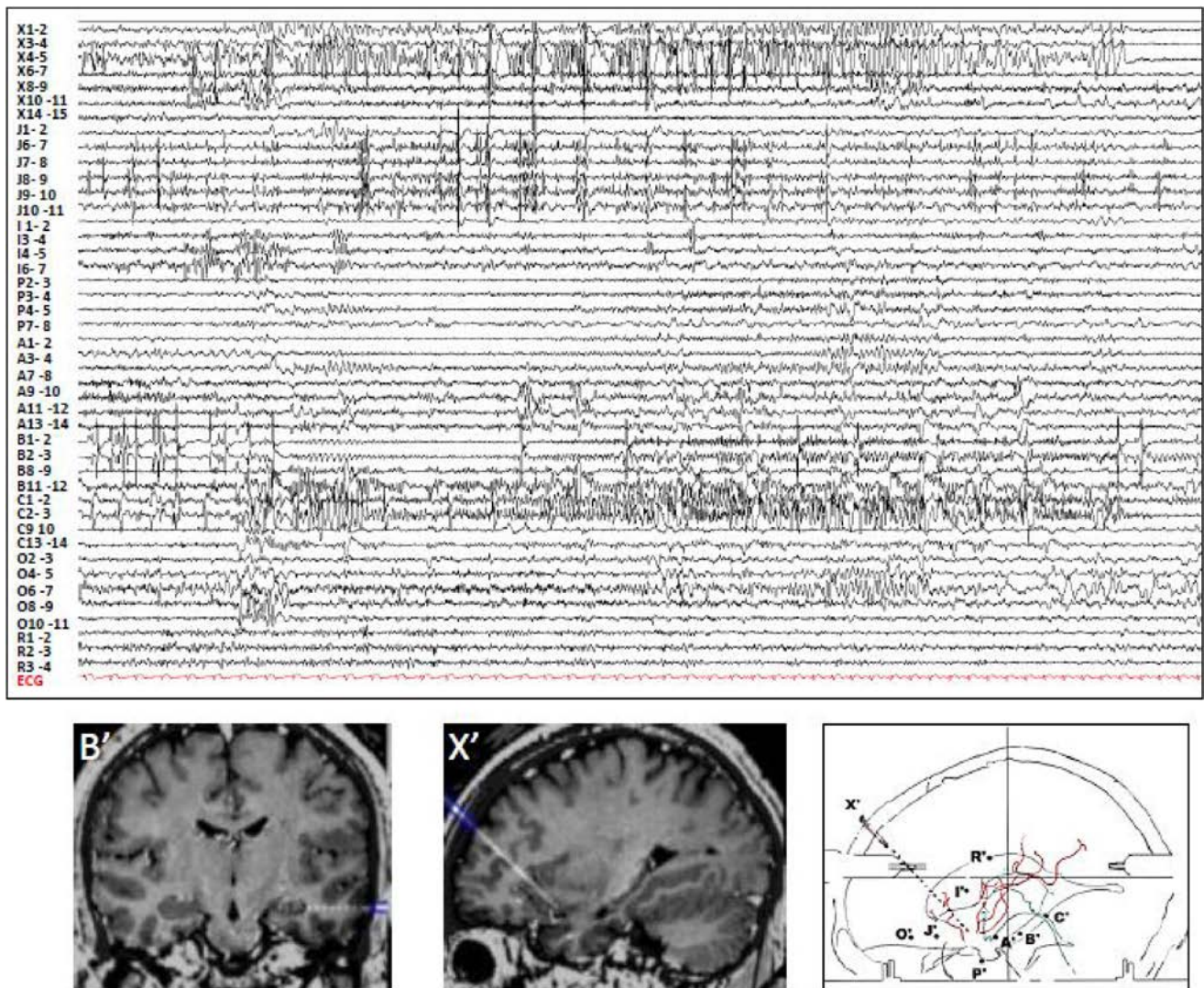


Figure 1 Temporo-perisylvian epilepsy, left hippocampal sclerosis. The sampling involved the left mesial temporal structures (amygdala [A'], anterior and posterior hippocampus [B', C']) and the lateral temporal cortex (lateral contacts of the same electrodes), the temporal pole [P'], the orbitofrontal cortex [O'], the inferior frontal gyrus (lateral contacts of [J'], [I']) and anterior cingulate gyrus (mesial contacts of [J']), the frontal operculum [R'] and the anterior insula (mesial contacts of [X'] and [I']). Ictal discharge during a seizure limited to a subjective manifestation (paresthesia in the right hemibody) illustrates the complex organization of the epileptogenic zone. Note the rapid discharge in the left anterior hippocampus, less visible in the posterior hippocampus, amygdala, temporal pole, orbitofrontal cortex, and the simultaneous discharge within the insula, characterized by a high amplitude spiking activity followed by a depression of activity. Asynchronous spikes are also visible on the inferior frontal gyrus during the discharge. The same organization was observed after seizures including objective manifestations. A left temporal mesial resection was performed, associated with frontal and insular disconnection. The patient has been seizure free for 7 years but reported frequent auras in the early post-operative period.

discussed depending on the electroclinical data (entorhinal cortex, amygdala, posterior hippocampus, temporal pole). There is no decisive argument to justify the choice of one or more contralateral electrodes. As far as possible, bilateral electrodes should be symmetrical.

Frontal lobe epilepsies

Understanding of organization of frontal epilepsies has also greatly benefited from information provided by SEEG. Thus

study of the semiology of frontal lobe seizures described by the Sainte-Anne School has identified different origins and spread pathways with antero-posterior and mesial-lateral systematization. According to this classification, anterior frontal seizures (fronto-polar, orbital, frontal mesial involving the ACG) have been distinguished from posterior frontal seizures, involving the supplementary motor area (SMA) and the motor cortex, and seizures originating from the dorso-lateral regions [53–60]. These data have been enhanced by more contemporary works in the area of sleep epilepsies [61,62], selection of candidates [63], characteristic

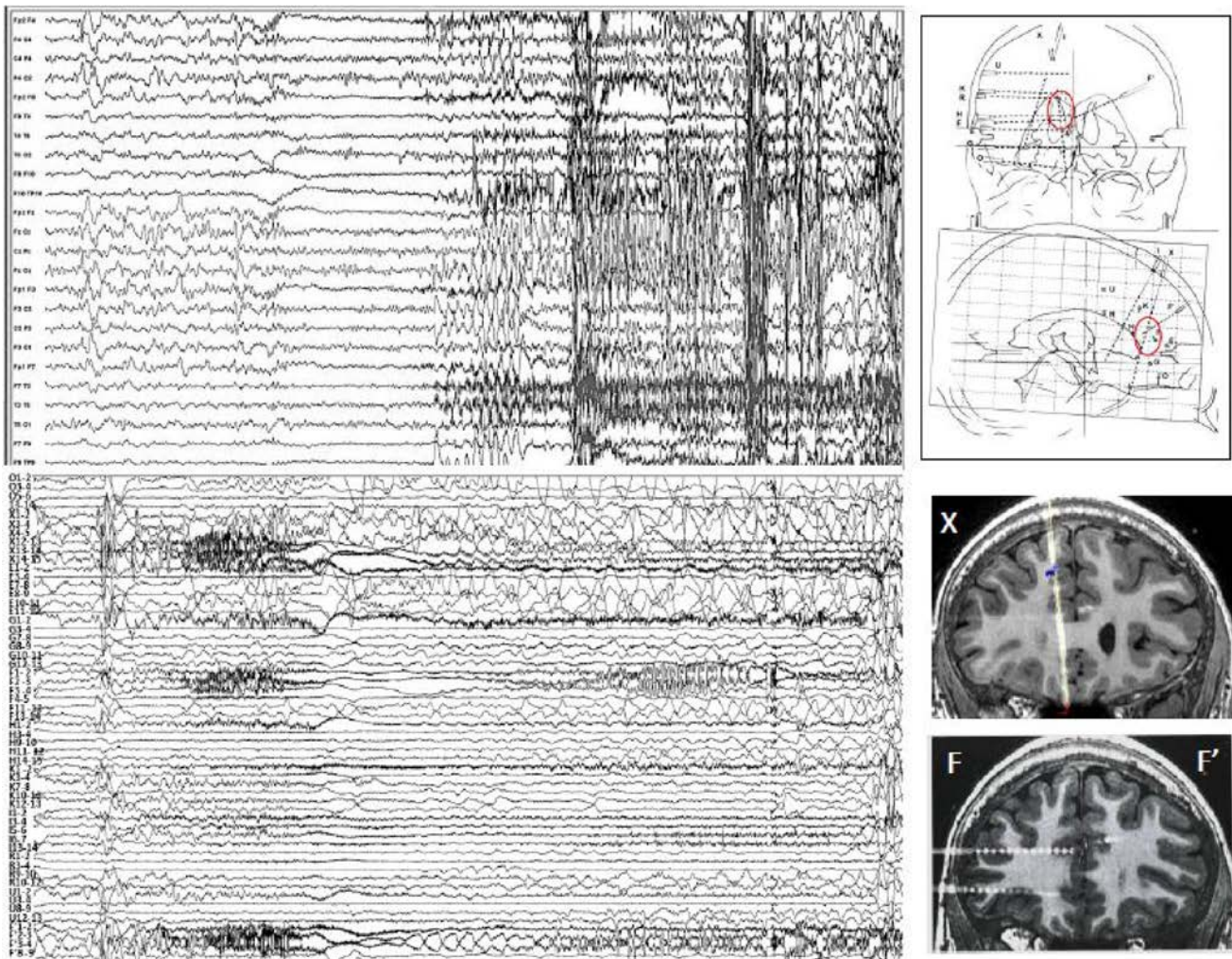


Figure 2 Right frontal sleep related epilepsy, negative MRI case. Comparison between scalp (upper part) and intracranial (lower part) ictal recordings. Ictal semiology was strongly suggestive of frontal mesial involvement without lateralizing features. On scalp EEG, the discharge started during sleep (stage 2) and seemed more rapid on the right frontal region (FP2-F4). FDG-PET demonstrated a focal hypometabolism in the anterior cingulate gyrus (ACG), bilaterally but predominant on the right. The SEEG sampling included the right ACG, which was the main hypothesis with 3 electrodes (supragenual part, area 24, by the mesial part of the electrode [F] and the middle part of [X] which were very closed, infragenual part, areas 25–33, by the mesial part of [G], posterior part, area 32, by the mesial part of [H]; the inferior frontal gyrus was recorded by the lateral part of the same electrodes. Other areas involved the orbitofrontal cortex [O], the anterior insula (mesial part of [I]), the mesial frontal cortex (mesial part of [E], [K], [U]) and the middle frontal gyrus (lateral part of the same electrodes), the superior frontal gyrus (lateral part of [X] and [I]), and the frontal operculum [R]. On the left side, one electrode targeted the ACG, [F'] symmetric to [F]. The ictal discharge was characterized by a low voltage fast activity following a burst of rapid rhythms starting in the ACG ([X12–13 to 14–15, F1–2 to 2–3], red circle on the scheme) and spreading to the connected areas, within the ACG [G1–2, H1–2], the frontal mesial cortex [E1–2, K1–2, U1–2], the insula [I3–4 to 5–6] and the contralateral cingulum [F'1–2]. Note the simultaneous discharge on the right and left sides, which appeared more rapid on the right. A focal right mesial frontal resection was performed, including the cortex sampled by X and F and a focal cortical dysplasia (type 2A) was found by histology. The patient has been seizure free without treatment for 5 years.

manifestations pointing to the frontal lobe such as the so-called “hypermotor” seizures [64] and other motor manifestations [56,65,66], startle epilepsies [67] and striking changes in facial expression with the “chapeau de gendarme” mouth [68]. Anatomic-functional systematization of frontal lobe seizures has thus been proposed [65], in order to refine the exploration of frontal epilepsies (Fig. 2). However, given the volume of the frontal lobe and the role of intra- and interhemispheric connections,

relatively large sampling is usually required. It is therefore necessary to build a solid hypothesis in terms of lateralization and localization before discussing the implantation (anterior versus posterior, mesial versus dorso-lateral areas).

In the hypothesis of mesial anterior frontal epilepsy, sampling involves the orbital region (often requiring a double approach, orthogonal and oblique to target the gyrus rectus), the frontal pole, the ACG with at least 2

electrodes (areas 24 and 32), the superior frontal gyrus (SFG), the anterior insula and often the anterior temporal region (amygdala, temporal pole). The question of bilateral implantation is discussed in case of bilateral anomalies on the scalp EEG and/or doubt regarding the side involved at seizure onset. There is no particular recommendation on the structures to be explored in the contralateral side. The functional regions must be avoided as far as possible. Ideally, the contralateral electrode(s) should be symmetrical with those placed on the ipsilateral side. In the hypothesis of anterior dorso-lateral frontal epilepsy, sampling will include the inferior frontal gyrus (IFG), allowing the location of Broca's area in the dominant hemisphere, the orbital cortex, the lateral frontal cortex (middle and superior frontal gyri) including the pre-motor cortex (areas 8 and 6), the frontal operculum and at least one electrode in the ACG and the insula. The question of bilateral exploration is less frequent than in the previous situation. Oblique electrodes are often useful for exploring the convexity. Exploration of the posterior frontal and central regions was initially limited to clastic lesions within the context of infantile hemiplegia. This allowed description of the complexity of the EZ organization involving highly integrated premotor and sensorimotor systems. It also allowed the study of specific entities such as epilepsy partialis continua, cortical myoclonus and startle epilepsy [56,69,70]. This knowledge was then applied to other etiologies such as developmental tumors and focal cortical dysplasia (FCD) [71,72]. Epilepsies originating from these areas require sampling of primary motor cortex (area 4), premotor areas (area 6) and primary and secondary somatosensory systems (post-central cortex, S2). This implies crossing the central sulcus, which carries a risk of motor deficit in case of hemorrhagic complication. It is justified only when there is a high probability of curative surgery and/or when functional reorganization has been demonstrated beforehand.

Epilepsies of the posterior quadrant

Parietal and occipital epilepsies are less frequent than those previously discussed and the principles of their exploration are based on fewer data, which have been reported more recently [73–78]. The density of the connections with the central, insular and temporal regions on one hand, and with the contralateral side on the other hand, accounts for the clinical polymorphism. These epilepsies often require multilobar and bilateral explorations with particular regard to the pathways of propagation and the implication of functional structures (language, reading, vision, face recognition).

Parietal epilepsies

Epilepsies originating from the post-central region are discussed with central epilepsies and require sampling of the premotor cortex. Involvement of the superior and inferior parietal lobule requires specific sampling particularly of Brodmann's areas 5 and 7, the intra-parietal sulcus, the inferior parietal cortex and the posterior cingulate gyrus, the post-central operculum and the posterior insula. According to electroclinical data, adjacent and connected areas may also be explored such as the central region (in particular

the paracentral lobule), the frontal lobe (premotor cortex), the occipital cortex, the temporo-parietal junction and the temporal lobe. It may be necessary to add electrodes in the contralateral hemisphere.

Occipital epilepsies

Epilepsies originating from the occipital lobe are relatively rare and apart from inaugural subjective manifestations, they frequently present symptoms related to their propagation to adjacent structures [79–81]. Localization with surface EEG and EEG-HR is difficult or even misleading, especially in the case of mesial occipital localization [21], leading to the need for bilateral explorations in occipital epilepsy. The exploration requires a preliminary evaluation of the visual functions: visual field study, visual evoked potentials, diffusion tensor imaging to locate visual fibers, fMRI for vision, reading and face recognition. The strategy of implantation is closely related to the pathways of neurophysiological networks [82–86].

The exploration targets the occipital cortex unilaterally or bilaterally with an electrode located on each side of the calcarine fissure and a basal occipital electrode. The cortex of the occipital pole is difficult to explore with SEEG electrodes; a postero-anterior electrode can be considered but this is rather uncomfortable for the patient. Identification of propagation pathways is crucial. Two main situations are identified, involving either a ventral (occipito-temporal) or dorsal (occipito-parieto-frontal) network. For the ventral network, involved in seizures originating from the infracalcarine area, sampling includes the lingual gyrus, the basal occipito-temporal junction (fusiform gyrus), the anterior calcarine fissure, the cuneus, the posterior hippocampus and the temporo-basal cortex. Contralateral electrodes should be discussed (occipital cortex, lingual gyrus). For the dorsal network, (in seizures originating from the supracalcarine area), sampling involves the cuneus, the parietal lobe (superior and inferior parietal lobule, parieto-occipital sulcus, posterior cingulate gyrus), the lingual gyrus and the temporo-parieto-occipital junction. The need to explore the frontal cortex (frontal eye field) and the contralateral side (cuneus for example) should be discussed by the team.

Insular epilepsies

Seizures primarily arising from the insula have been recently characterized. Semiology is often misleading and may mimic the symptomatology of frontal or temporal seizures. Better knowledge of specific subjective manifestations makes it easier to recognize these. Technical progress has progressively allowed sampling of the insula under good safety conditions and through identifying the anatomic-functional characteristics [47,87–95]. SEEG is particularly effective in demonstrating an insular origin of seizures (Fig. 3). The technique of implantation and the choice of trajectories depend on the experience of the teams and the vascular constraints. The close proximity of the insular cortex with arborization of the middle cerebral artery implies that vascular safety (distance between trajectory and vessel) is a priority. Only the antero-inferior angle is difficult to implant due to the vascular risk. To explore the insula, orthogonal

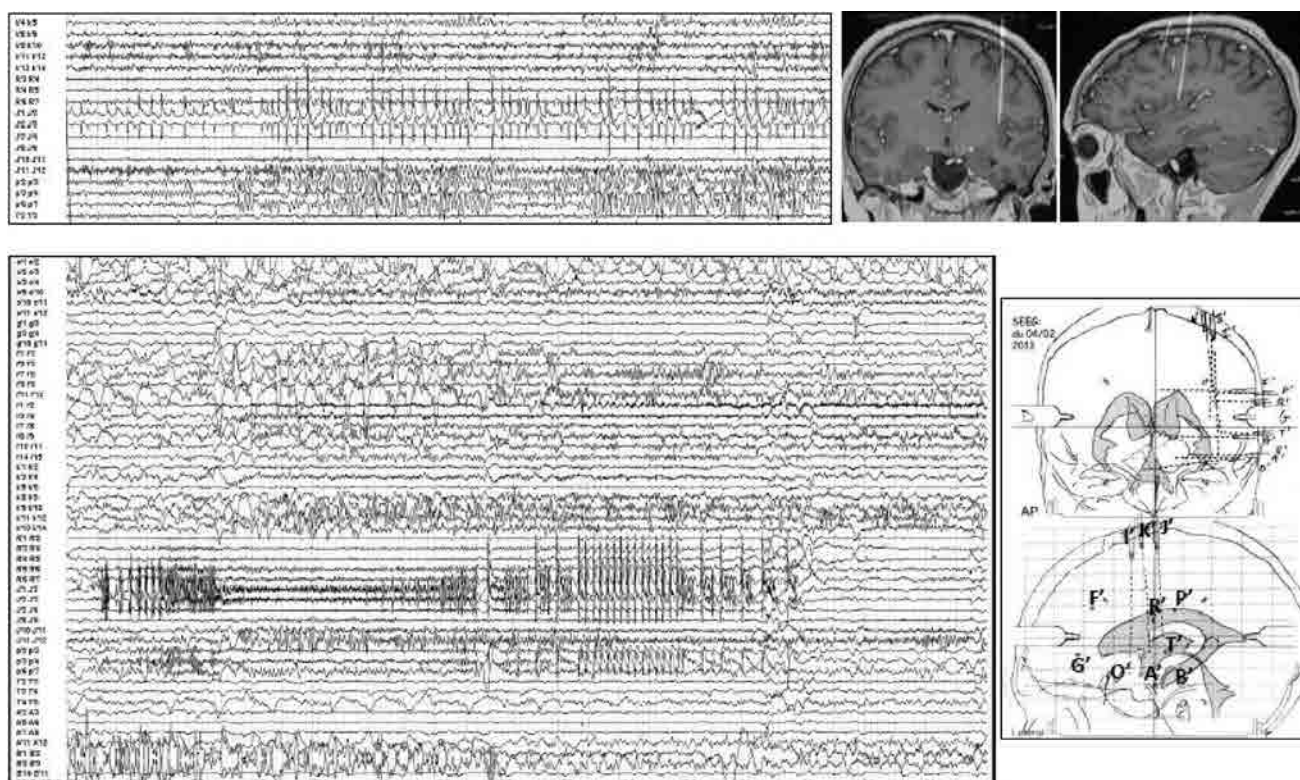


Figure 3 Insular epilepsy, negative MRI case. According to the electroclinical semiology, sampling involved the left insulo-perisylvian region with 6 electrodes in the insula (3 oblique [I', J', K'] and 3 orthogonal [R', P', T'] also covering by the lateral contacts the frontal, parietal and temporal opercula. Other electrodes in frontal lobe sampled the orbitofrontal [O'] and anterior cingulate cortex (mesial part of [G']), the inferior frontal gyrus (lateral part of [G']) and middle frontal gyrus [F']. In temporal lobe, two additional electrodes were placed (amygdala [A'] and hippocampus [B']), both of them recording the middle temporal gyrus by the lateral contacts). In the upper part, see the subcontinuous rhythmic spiking activity recorded interictally, well-localized on two contacts of the electrode [J'], highly suggestive of focal cortical dysplasia. Note the normal activity of closed electrodes [P', K']. In the lower part, ictal discharge started on the same area, with a burst of high amplitude spikes and rapid rhythms followed by a low voltage fast activity diffusing to adjacent electrodes [I', P', R']. The patient has been seizure free for 3 years after focal insular thermocoagulations.

and oblique electrodes can be used. Orthogonal implantation has the advantage of simultaneously investigating the insular cortex and the operculum, which is often crucial in this type of epilepsy. However, it has the disadvantage of recording only a limited volume of the insular cortex. On the contrary, an oblique implantation, along the insular cortex, allows investigation of a larger insular cortical surface, but cannot record the activity of the operculum. If necessary, both orthogonal and oblique approaches can be combined. The insular exploration will be coupled with the exploration of other adjacent regions, frontal, parietal or temporal, depending on the clinical symptomatology. It should be noted that the insular ictal discharges may display early spread to the contralateral side or may be synchronous on both hemispheres, with the risk of false lateralization. Relevant clinical or imaging features are then helpful for the choice of hemisphere.

Lesional epilepsies

SEEG planning for lesional epilepsies mostly depends on the causal lesion. Its specific contribution should be discussed

according to information provided by previous correlations between SEEG data and non-invasive tools. Given the progress of morphological and functional imaging, identifying the limits of a tumor or a dysplastic lesion is rarely a primary goal. On the other hand, the question of the relationship between lesion and EZ is often raised, especially in brain injury and diffuse forms of malformations of cortical development. It should be kept in mind that the EZ and the structural lesion are rarely perfectly contiguous except in the particular case of FCD type 2 [96–99] and some developmental tumors [100,101]. Moreover, a significant number of so-called cryptogenic epilepsies (negative-MRI cases) are in fact of lesional origin, and mainly related to FCD [12,102–104].

Generally, whatever the location, the above-described scheme is modified by adding one or more lesional and perilesional electrodes if possible. In a temporal neocortical location, it is advised to include the mesial temporal structures. Intralesional, perilesional and distant areas sampling is particularly adequate for complex or diffuse cortical developmental malformations such as heterotopia and polymicrogyria with or without schizencephaly [105–111]. The need to record intralesional activity does not apply

to clastic, post-anoxic, post-traumatic or post-encephalitic lesions, in which it is preferable to explore the perilesional cortex rather than necrotic tissues or cystic areas. Moreover, for vascular lesions, it is not recommended to place an intralesional electrode due to the hemorrhagic risk. However, it is possible to record the perilesional cortex in cavernomas [112].

Management of SEEG

The risk-benefit ratio of SEEG should be evaluated by an experienced multidisciplinary team and the discussion noted in the patient's chart. The procedure is managed by a medical and paramedical team specifically trained to invasive EEG monitoring. Information must be given to the patient and his (her) legal guardians if he (she) is a minor or under guardianship.

Patient care

Close monitoring of the patient and the intracranial recordings by the medical and paramedical team in a dedicated environment is mandatory throughout the duration of the SEEG. Continuous monitoring (involving a specialized nurse or an EEG technician near the patient) is preferable but may be discontinuous according to the equipment and staffing of each center. The minimum duration of each recording is not fixed, depending on the clinical situation (frequent or rare seizures, cooperation and behavior of the patient, risk of major seizures or agitation, etc.). The presence of a relative may be sought in some patients, especially in children and patients with agitation during seizures. The purpose of the monitoring is threefold: (1) seizure detection and description; (2) patient protection against the consequences of seizures (such as trauma, secondary generalization and hypoxia); (3) protection of the technical equipment in the case of marked agitation. Clinical monitoring includes the patient's general condition (in particular searching for infectious signs and complication of bed-rest), neurological state (e.g. consciousness, headache, focal deficit, autonomic disorders) and psychological examination (e.g. anxiety, insomnia, agitation). The repair of the head bandage depends on its condition and the prescription of the neurosurgeon. In addition to clinical symptoms, change of the background activity on recordings with the occurrence of focal slow waves or depression of the electrical activity is highly suggestive of the formation of a hematoma and a CT scanner must be urgently performed.

Conditions for defining the EZ

Recording at least one spontaneous seizure reproducing the known semiology is recommended, in order to define the EZ appropriately. Recording several seizures may be required to check their homogeneity or when the patient reports several types of seizures. Neurophysiological techniques for anticipating seizures may be useful to optimize the recording conditions [113], but no method is currently operational. The exploration is usually conducted under progressive drug withdrawal except in the case of frequent

seizures. Anti-epileptic drug reduction is adapted to the patient's condition and assessed at least once a day.

Subclinical ictal discharges (or subclinical seizures) may have the significance of a spontaneous seizure with usual clinical manifestations and must be considered useful data for defining the EZ [114], particularly in FCD [98]. However, electroclinical correlations remain privileged in order to achieve an optimal EZ definition and the value of subclinical discharges when electroclinical seizures are not obtained was a matter of debate for the current working party, with a low consensus rate. In the same way, the reproduction of a usual seizure by low-frequency stimulation can be accepted to define the EZ under specific conditions [98,115–118]. This situation is mainly discussed in seizures of hippocampal origin or in FCD type 2, provided that the electrode is strictly intralesional. The elicited seizure must reproduce the usual clinical and EEG characteristics of spontaneous seizures. The electrical pattern of the discharge (amplitude, duration, propagation) and its correlations with the habitual subjective and objective manifestations are decisive for analyzing seizures triggered by stimulations. Thus the clinical signs must appear before the propagation of the electrical discharge to the structures connected with the stimulated site. In this condition, the induced seizure is considered pertinent to define the EZ ("true positive"). In contrast, if the seizure begins following an after-discharge with the recruitment of a local or remote network, its value in defining the EZ is more questionable. These criteria are also used to study the value of high-frequency stimulations [116,117,119–122]. While high-frequency stimulations are more likely to result in seizures than low-frequency stimulations, they are also more likely to result in in habitual seizures (especially with secondary generalization), which may therefore be unhelpful or misleading in defining the EZ ("false positives"). For high-frequency stimulation, intensity has also to be considered: the lower the intensity, the higher the significance of the elicited seizure. However, electrically-induced seizures (including low-frequency stimulations) were considered by some experts less reliable than spontaneous seizures for the definition of EZ and therefore their value obtained a low consensus rate.

Sleep-related epilepsies

Night recordings are particularly useful in seizures occurring predominantly during sleep [50,61,62,123,124]. They are helpful whenever safety conditions are met, with the need for continuous monitoring. Otherwise, spontaneous sleep during day or after sleep deprivation can be obtained. Sleep induction with melatonin or amitriptyline (0.5 mg/kg intramuscularly) can also be discussed. Amitriptyline combines the inductive effects of sleep and depression of the epileptogenic threshold. Activation of interictal abnormalities and facilitation of seizure occurrence has been shown in different types of epilepsies [125]. During SEEG usual seizures may be obtained after amitriptyline in about 1/3 of the cases (personal data). It is preferable to perform this injection before the withdrawal of anti-epileptic treatment to avoid the risk of secondary generalization. It should be noted, however, that even spontaneous sleep can increase the diffusion of interictal abnormalities and the speed of

propagation of ictal discharges. In the case of diffuse or widespread interictal spikes recorded during the wakefulness and/or sleep, intravenous injection of benzodiazepines at the end of SEEG (diazepam: 10 mg or clonazepam: 1 mg) may contribute to spatially restrict the irritative zone, particularly in FCD Type 2 [98,103]. It also may help to limit the widespread of the EZ in case of highly frequent seizures. This pharmacological approach remains limited to some teams and therefore has a low consensus rate.

Duration of SEEG

The duration of SEEG should be limited to the time required to obtain relevant information on the organization of the EZ and to reach a decision on surgical treatment. The average duration varies between 1 and 2 weeks. In some cases, it can be extended to 3 weeks, but beyond this time the risk-benefit ratio (particularly infectious) should be justified. If it is unfeasible to define the EZ after an initial exploration, in particular because of inadequate or insufficient sampling, a second SEEG may be proposed. However, this must be strongly argued with a new discussion of risk-benefit ratios.

Particularities in children

The SEEG methodology is applicable in children and is well-tolerated [126–131]. In very young children (< 3 years), data are more limited but it has been shown that SEEG can help to propose relatively focal curative interventions [130]. However, this practice requires pediatric experience and a dedicated environment. Continuous monitoring is mandatory because of the large number of seizures usually presented at this age and the possibility of carrying out the shortest explorations. Continuous parental presence is required. Before 2 years, technical problems limit its practice, especially the thickness of the bone (> 2 mm) conditioning the use of SEEG electrodes.

Team training

The specificity of the SEEG methodology implies specific training of the medical (neurologists, neurophysiologists, neurosurgeons) and paramedical (nurses and EEG technicians) teams. The multiplication of centers in national (French) and European terms has benefited a growing number of patients [26,132]. Its advantages in terms of precision and safety have also led to rapid expansion in use by North American teams [25,133,134] as well as other countries around the world. The current expansion and development of SEEG must imply the most rigorous conditions of its good clinical practice. The labeling of reference centers for epilepsy surgery including the practice of SEEG is underway in France. It follows the methodology of the High Authority of Health (HAS) describing a reference framework for center evaluation. Patient recruitment and number of procedures performed each year, human resources (neurologists-epileptologists, neurosurgeons, neuropsychologists, neuroradiologists, neuropathologists with specific expertise in epileptology) are taken into account. Multidisciplinary approach is considered

including continuous EEG-SEEG-video monitoring, advanced morphological and functional imaging, organization of care, academic expertise, links with patient associations and therapeutic education and rehabilitation.

As there is no qualification specifically dedicated to SEEG practice, it is essential that each practitioner can justify training and experience validated both in epileptology and in neurophysiological techniques including intra-cerebral recordings. Participation in national and international SEEG schools is encouraged. Hands-on clinical training with experienced teams is advised. It must involve active participation at all stages of the SEEG including interpretation (represented by a period of 6-12 months and/or at least 10 SEEG). It must concern at least the neurologist and the neurosurgeon wishing to develop the methodology in their center. EEG nurses and technicians must also receive specific training.

Conclusions

SEEG is an effective and safe methodology to define the EZ with the goal of proposing curative surgery in drug-resistant partial epilepsy. As an integrated method rather than a simple technique, it takes account of all the clinical, neurophysiological and anatomo-functional data in order to achieve accurate localization of the EZ. The approach is individual but can be systematized according to the location and the cause of epilepsy. It can be performed in very young children. It requires rigorous training to ensure optimal results and safety of the exploration.

Disclosure of interest

The authors declare that they have no competing interest.

References

- [1] Bancaud J. Surgery of epilepsy based on stereotactic investigations – the plan of the SEEG investigation. *Acta Neurochir Suppl* 1980;30:25–34.
- [2] Chauvel P, Buser P, Badier J, Liegeois-Chauvel C, Marquis P, Bancaud J. La zone épileptogène chez l'homme : représentation des événements intercritiques par cartes spatio-temporelles. *Rev Neurol* 1987;143:443–50.
- [3] Kahane P, Landré E. The epileptogenic zone. *Neurochirurgie* 2008;54:265–71.
- [4] Kahane P, Landré E, Minotti L, Francione S, Ryvlin P. The Bancaud and Talairach view on the epileptogenic zone: a working hypothesis. *Epileptic Disord* 2006;8(suppl 2):S16–26.
- [5] Munari C, Bancaud J. The role of stereo-electroencephalography (SEEG) in the evaluation of partial epileptic patients. In: *The epilepsies*. London: Butterworths; 1987. p. 267–306.
- [6] Talairach J, Bancaud J, Szikla G, Bonis A, Geier S, Védrenne C. Approche nouvelle de la neurochirurgie de l'épilepsie. *Méthodologie stéréotaxique et résultats thérapeutiques*. *Neurochirurgie* 1974;20(suppl 1):1–249.
- [7] Kobulashvili T, Höfler J, Dobesberger J, Ernst F, Ryvlin P, Cross J, et al. Current practices in long term video-EEG monitoring services: a survey among partners of the E-PILEPSY pilot network of reference for refractory epilepsy and epilepsy surgery. *Seizure* 2016;38:38–45.

- [8] Commission on Neuroimaging of the International League Against Epilepsy. Guidelines for neuroimaging evaluation of patients with uncontrolled epilepsy considered for surgery. *Epilepsia* 1998;39:1375–6.
- [9] Duncan J, Winston G, Koeppe M, Ourselin S. Brain imaging in the assessment for epilepsy surgery. *Lancet Neurol* 2016;15:420–33.
- [10] Gaillard W, Cross J, Duncan J, Stefan H, Theodore W. Epilepsy imaging study guideline criteria: commentary on diagnostic testing study guidelines and practice parameters. *Epilepsia* 2011;52:1750–6.
- [11] Chassoux F, Chiron C. [Positron emission tomography. Which indications? Which benefits?]. *Neurochirurgie* 2008;54:219–25.
- [12] Chassoux F, Rodrigo S, Semah F, Beuvon F, Landre E, Devaux B, et al. FDG-PET improves surgical outcome in negative MRI Taylor-type focal cortical dysplasias. *Neurology* 2010;75:2168–75.
- [13] Guedj E, Bonini F, Gavaret M, Trébuchon A, Aubert S, Bouckine M, et al. FDG-PET in different subtypes of temporal lobe epilepsy: SEEG validation and predictive value. *Epilepsia* 2015;56:414–21.
- [14] Mayoral M, Marti-Fuster B, Carreño M, Carrasco J, Bargalló N, Donaire A, et al. Seizure-onset zone localization by statistical parametric mapping in visually normal (18) F-FDG PET studies. *Epilepsia* 2016;57:1236–44.
- [15] Rathore C, Dickson J, Teotónio R, Ell P, Duncan J. The utility of 18F-fluorodeoxyglucose PET (FDG PET) in epilepsy surgery. *Epilepsy Res* 2014;108:1306–14.
- [16] Sulc V, Stykel S, Hanson D, Brinkmann B, Jones D, Holmes D, et al. Statistical SPECT processing in MRI-negative epilepsy surgery. *Neurology* 2014;82:932–9.
- [17] Jung J, Bouet R, Delpuech C, Ryvlin P, Isnard J, Guenot M, et al. The value of magnetoencephalography for seizure-onset zone localization in magnetic resonance imaging-negative partial epilepsy. *Brain* 2013;136:3176–86.
- [18] Knowlton R, Razdan S, Limdi N, Elgavish R, Killen J, Blount J, et al. Effect of epilepsy magnetic source imaging on intracranial electrode placement. *Ann Neurol* 2009;65:716–23.
- [19] Brodbeck V, Spinelli L, Lascano A, Wissmeier M, Vargas M, Vulliemoz S, et al. Electroencephalographic source imaging: a prospective study of 152 operated epileptic patients. *Brain* 2011;134:2887–97.
- [20] Gavaret M, Maillard L, Jung J. High-resolution EEG. (HR-EEG) and magnetoencephalography (MEG). *Neurophysiol Clin* 2015;45:105–11.
- [21] Gavaret M, Trébuchon A, Bartolomei F, Marquis P, McGonigal A, Wendling F, et al. Source localization of scalp-EEG interictal spikes in posterior cortex epilepsies investigated by HR-EEG and SEEG. *Epilepsia* 2009;50:276–89.
- [22] Rikir E, Koessler L, Gavaret M, Bartolomei F, Colnat-Coulbois S, Vignal J, et al. Electrical source imaging in cortical malformation-related epilepsy: a prospective EEG-SEEG concordance study. *Epilepsia* 2014;55:918–32.
- [23] Catenox H, Mauguière F, Montavont A, Ryvlin P, Guenot M, Isnard J. Seizures outcome after stereoelectroencephalography-guided thermocoagulations in malformations of cortical development poorly accessible to surgical resection. *Neurosurgery* 2015;77:9–14.
- [24] Guénot M, Isnard J, Catenox H, Mauguière F, Sindou M. SEEG-guided RF-thermocoagulation of epileptic foci: a therapeutic alternative for drug-resistant non-operable partial epilepsies. *Adv Tech Stand Neurosurg* 2011;36:61–78.
- [25] Mullin J, Shriver M, Alomar S, Najm I, Bulacio J, Chauvel P, et al. Is SEEG safe? A systematic review and meta-analysis of stereo-electroencephalography-related complications. *Epilepsia* 2016;57:386–401.
- [26] Cardinale F, Cossu M, Castana L, Casaceli G, Schiariti M, Miserocchi A, et al. Stereoelectroencephalography: surgical methodology, safety, and stereotactic application accuracy in 500 procedures. *Neurosurgery* 2013;72:353–66.
- [27] Mathon B, Clemenceau S, Hasboun D, Habert M, Belaid A, Nguyen-Michel V, et al. Safety profile of intracranial electrode implantation for video-EEG recordings in drug-resistant focal epilepsy. *J Neurol* 2015;262:2699–712.
- [28] Adam C, Clemenceau S, Semah F, Hasboun D, Samson S, Aboujaoude N, et al. Variability of presentation in medial temporal epilepsy: a study of 30 operated cases. *Acta Neurol Scand* 1996;94:1–11.
- [29] Bancaud J. [Clinical symptomatology of epileptic seizures of temporal origin]. *Rev Neurol* 1987;143:392–400.
- [30] Bancaud J, Brunet-Bourgin F, Chauvel P, Halgren E. Anatomical origin of *déjà vu* and vivid “memories” in human temporal lobe epilepsy. *Brain* 1994;117:71–90.
- [31] Bartolomei F, Chauvel P, Wendling F. Epileptogenicity of brain structures in human temporal epilepsy: a quantified study from intracerebral EEG. *Brain* 2008;131:1818–30.
- [32] Bartolomei F, Khalil M, Wendling F, Sontheimer A, Régis J, Ranjeva J-P, et al. Entorhinal cortex involvement in human mesial temporal lobe epilepsy: an electrophysiologic and volumetric study. *Epilepsia* 2005;46:677–87.
- [33] Chabardès S, Kahane P, Minotti L, Tassi L, Grand S, Hoffmann D, et al. The temporopolar cortex plays a pivotal role in temporal lobe seizures. *Brain* 2005;128:1818–31.
- [34] Maillard L, Vignal J, Gavaret M, Guye M, Biraben A, McGonigal A, et al. Semiologic and electrophysiologic correlations in temporal lobe seizure subtypes. *Epilepsia* 2004;45:1590–9.
- [35] Vignal J, Maillard L, McGonigal A, Chauvel P. The dreamy state: hallucinations of autobiographic memory evoked by temporal lobe stimulations and seizures. *Brain* 2007;130:88–99.
- [36] Carne R, O'Brien T, Kilpatrick C, MacGregor L, Hicks R, Murphy M, et al. MRI-negative PET-positive temporal lobe epilepsy: a distinct surgically remediable syndrome. *Brain* 2004;127:2276–85.
- [37] LoPinto-Khoury C, Sperling M, Skidmore C, Nei M, Evans J, Sharan A, et al. Surgical outcome in PET-positive, MRI-negative patients with temporal lobe epilepsy. *Epilepsia* 2012;53:342–8.
- [38] Barba C, Barbati G, Minotti L, Hoffmann D, Kahane P. Ictal clinical and scalp-EEG findings differentiating temporal lobe epilepsies from temporal “plus” epilepsies. *Brain* 2007;130:1957–67.
- [39] Barba C, Rheims S, Minotti L, Guénot M, Hoffmann D, Chabardès S, et al. Temporal plus epilepsy is a major determinant of temporal lobe surgery failures. *Brain* 2016;139:444–51.
- [40] Bartolomei F, Cosandier-Rimele D, McGonigal A, Aubert S, Régis J, Gavaret M, et al. From mesial temporal lobe to temporoparietal seizures: a quantified study of temporal lobe seizure networks. *Epilepsia* 2010;51:2147–58.
- [41] Blauwblomme T, David O, Minotti L, Job A, Chassagnon S, Hoffmann D, et al. Prognostic value of insular lobe involvement in temporal lobe epilepsy: a stereoelectroencephalographic study. *Epilepsia* 2013;54:1658–67.
- [42] Catenox H, Guénot M, Isnard J, Fischer C, Mauguière F, Ryvlin P. Intracranial EEG. study of seizure-associated nose wiping. *Neurology* 2004;63:1127–9.
- [43] Catenox H, Magnin M, Guénot M, Isnard J, Mauguière F, Ryvlin P. Hippocampal-orbitofrontal connectivity in human: an electrical stimulation study. *Clin Neurophysiol* 2005;116:1779–84.
- [44] Chassoux F, Artiges E, Semah F, Desarnaud S, Laurent A, Landre E, et al. Determinants of brain metabolism changes in mesial temporal lobe epilepsy. *Epilepsia* 2016;57:907–19.

- [45] Chassoux F, Semah F, Bouilleret V, Landre E, Devaux B, Turak B, et al. Metabolic changes and electro-clinical patterns in mesio-temporal lobe epilepsy: a correlative study. *Brain* 2004;127:164–74.
- [46] Hauser-Hauw C, Bancaud J. Gustatory hallucinations in epileptic seizures: electrophysiological, clinical, and anatomical correlates. *Brain* 1987;110:339–59.
- [47] Isnard J, Guénot M, Ostrowsky K, Sindou M, Mauguière F. The role of insular cortex in temporal lobe epilepsy. *Ann Neurol* 2000;48:614–23.
- [48] Kahane P, Bartolomei F. Temporal lobe epilepsy and hippocampal sclerosis: lessons from depth EEG recordings. *Epilepsia* 2010;51(Suppl 1):59–62.
- [49] Memarian N, Madsen S, Macey P, Fried I, Engel J, Thompson P, et al. Ictal depth EEG and MRI structural evidence for two different epileptogenic networks in mesial temporal lobe epilepsy. *Plos One* 2015, <http://dx.doi.org/10.1371/journal.pone.0123588>.
- [50] Nobili L, Cossu M, Mai R, Tassi L, Cardinale F, Castana L, et al. Sleep-related hyperkinetic seizures of temporal lobe origin. *Neurology* 2004;62:482–5.
- [51] Rusu V, Chassoux F, Landré E, Bouilleret V, Nataf F, Devaux B, et al. Dystonic posturing in seizures of mesial temporal origin. *Neurology* 2005;65:1612–9.
- [52] Vaugier L, Aubert S, McGonigal A, Trébuchon A, Guye M, Gavaret M, et al. Neural networks underlying hyperkinetic seizures of “temporal lobe” origin. *Epilepsy Res* 2009;86:200–8.
- [53] Bancaud J, Talairach J. Clinical semiology of frontal lobe seizures. *Adv Neurol* 1992;57:3–58.
- [54] Bancaud J, Talairach J, Morel P, Bresson M, Bonis A, Geier S, et al. “Generalized” epileptic seizures elicited by electrical stimulation of the frontal lobe in man. *EEG clin Neurophysiol* 1974;37:275–82.
- [55] Chauvel P, Kliemann F, Vignal J, Chodkiewicz J, Talairach J, Bancaud J. The clinical signs and symptoms of frontal lobe epilepsy. Phenomenology and classification. *Adv Neurol* 1995;66:115–25.
- [56] Chauvel P, Trottier S, Vignal J, Bancaud J. Somatomotor seizures of frontal lobe origin. *Adv Neurol* 1992;57:185–232.
- [57] Munari C, Bancaud J. Electroclinical symptomatology of partial seizures of orbital frontal origin. *Adv Neurol* 1992;57:257–65.
- [58] Munari C, Giallonardo A, Brunet P, Broglin D, Bancaud J. Stereotactic investigations in frontal lobe epilepsies. *Acta Neurochir Suppl* 1989;46:9–12.
- [59] Talairach J, Bancaud J, Bonis A, Szikla G, Trottier S, Vignal J, et al. Surgical therapy for frontal epilepsies. *Adv Neurol* 1992;57:707–32.
- [60] Talairach J, Bancaud J, Geier S, Bordas-Ferrer M, Bonis A, Szikla G, et al. The cingulate gyrus and human behaviour. *Electroencephalogr Clin Neurophysiol* 1973;34:45–52.
- [61] Nobili L, Francione S, Mai R, Cardinale F, Castana L, Tassi L, et al. Surgical treatment of drug-resistant nocturnal frontal lobe epilepsy. *Brain* 2007;130:561–73.
- [62] Nobili L, Francione S, Mai R, Tassi L, Cardinale F, Castana L, et al. Nocturnal frontal lobe epilepsy: intracerebral recordings of paroxysmal motor attacks with increasing complexity. *Sleep* 2003;26:883–6.
- [63] Trottier S, Landré E, Biraben A, Chassoux F, Pasnicu A, Scarambin J, et al. [On the best strategies on the best results for surgery of frontal epilepsy]. *Neurochirurgie* 2008;54:388–98.
- [64] Rheims S, Ryvlin P, Scherer C, Minotti L, Hoffmann D, Guenot M, et al. Analysis of clinical patterns and underlying epileptogenic zones of hypermotor seizures. *Epilepsia* 2008;49:2030–40.
- [65] Bonini F, McGonigal A, Trébuchon A, Gavaret M, Bartolomei F, Giusiano B, et al. Frontal lobe seizures: from clinical semiology to localization. *Epilepsia* 2014;55:264–77.
- [66] Chassagnon S, Minotti L, Kremer S, Hoffmann D, Kahane P. Somatosensory, motor and reaching/grasping responses to direct electrical stimulation of the human cingulate motor areas. *J Neurosurg* 2008;109:593–604.
- [67] Job A, De Palma L, Principe A, Hoffmann D, Minotti L, Chabardès S, et al. The pivotal role of the supplementary motor area in startle epilepsy as demonstrated by SEEG epileptogenicity map. *Epilepsia* 2014;55:85–8.
- [68] Souirti Z, Landré E, Mellerio C, Devaux B, Chassoux F. Neural network underlying ictal pouting (“chapeau de gendarme”) in frontal lobe epilepsy. *Epilepsy Behav* 2014;37:249–57.
- [69] Chassoux F, Devaux B, Landré E, Chodkiewicz J, Talairach J, Chauvel P. Postoperative motor deficits and recovery after cortical resections. *Adv Neurol* 1999;21:189–99.
- [70] Vignal J, Biraben A, Chauvel P, Reutens D. Reflex partial seizures of sensorimotor cortex (including cortical reflex myoclonus and startle epilepsy). *Adv Neurol* 1998;75:207–26.
- [71] Devaux B, Chassoux F, Landré E, Turak B, Dumas-Duport C, Chagot D, et al. Chronic intractable epilepsy associated with a tumor located in the central area. *Stereotact Funct Neurosurg* 1997;69:229–38.
- [72] Marnet D, Devaux B, Chassoux F, Landré E, Mann M, Turak B, et al. Surgical resection of focal cortical dysplasias in the central region. *Neurochirurgie* 2008;54:399–408.
- [73] Balestrini S, Francione S, Mai R, Castana L, Casaceli G, Marino D, et al. Multimodal responses induced by cortical stimulation of the parietal lobe: a stereo-electroencephalography study. *Brain* 2015;138:2596–607.
- [74] Bartolomei F, Gavaret M, Hewett R, Valton L, Aubert S, Régis J, et al. Neural networks underlying parietal lobe seizures: a quantified study from intracerebral recordings. *Epilepsy Res* 2011;93:164–76.
- [75] Francione S, Liava A, Mai R, Nobili L, Sartori I, Tassi L, et al. Drug-resistant parietal epilepsy: polymorphic ictal semiology does not preclude good post-surgical outcome. *Epileptic Disord* 2015;17:32–46.
- [76] Liava A, Mai R, Tassi L, Cossu M, Sartori I, Nobili L, et al. Paediatric epilepsy surgery in the posterior cortex: a study of 62 cases. *Epileptic Disord* 2014;141:141–64.
- [77] Montavont A, Kahane P, Catenoix H, Ostrowsky-Coste K, Isnard J, Guénot M, et al. Hypermotor seizures in lateral and mesial parietal epilepsy. *Epilepsy Behav* 2013;28:408–12.
- [78] Salanova V, Andermann F, Rasmussen T, Olivier A, Quesney L. Parietal lobe epilepsy. Clinical manifestations and outcome in 82 patients treated surgically between 1929 and 1988. *Brain* 1995;118:607–27.
- [79] Palmieri A, Andermann F, Dubeau F, Gloor P, Olivier A, Quesney L, et al. Occipitotemporal epilepsies: evaluation of selected patients requiring depth electrodes studies and rationale for surgical approaches. *Epilepsia* 1993;34:84–96.
- [80] Salanova V, Andermann F, Olivier A, Rasmussen T, Quesney L. Occipital lobe epilepsy: electroclinical manifestations, electrocorticography, cortical stimulation and outcome in 42 patients treated between 1930 and 1991. *Brain* 1992;115:1655–80.
- [81] Takeda A, Bancaud J, Talairach J, Bonis A, Bordas-Ferrer M. Concerning epileptic attacks of occipital origin. *Electroencephalogr Clin Neurophysiol* 1970;28:647–8.
- [82] Hamamé C, Vidal J, Perrone-Bertolotti M, Ossandón T, Jerbi K, Kahane P, et al. Functional selectivity in the human occipitotemporal cortex during natural vision: evidence from combined intracranial EEG and eye-tracking. *Neuroimage* 2014;95:276–86.
- [83] Jonas J, Frismand S, Vignal J, Colnat-Coulbois S, Koessler L, Vespignani H, et al. Right hemispheric dominance of visual

- phenomena evoked by intracerebral stimulation of the human visual cortex. *Hum Brain Mapp* 2014;35:3360–71.
- [84] Jonas J, Maillard L, Frismand S, Colnat-Coulbois S, Vespignani H, Rossion B, et al. Self-face hallucination evoked by electrical stimulation of the human brain. *Neurology* 2014;83:336–8.
- [85] Lachaux J, George N, Tallon-Baudry C, Martinerie J, Hugueville L, Minotti L, et al. The many faces of the gamma band response to complex visual stimuli. *Neuroimage* 2005;25:491–501.
- [86] Vidal J, Perrone-Bertolotti M, Kahane P, Lachaux J. Intracranial spectral amplitude dynamics of perceptual suppression in fronto-insular, occipito-temporal, and primary visual cortex. *Front Psychol* 2015;5, <http://dx.doi.org/10.3389/fpsyg.2014.01545>.
- [87] Afif A, Minotti L, Kahane P, Hoffmann D. Anatomofunctional organization of the insular cortex: a study using intracerebral stimulation in epileptic patients. *Epilepsia* 2010;51:2305–15.
- [88] Catenoix H, Isnard J, Guénot M, Petit J, Remy C, Mauguière F. The role of the anterior insular cortex in ictal vomiting: a stereotactic encephalography study. *Epilepsy Behav* 2008;13:560–3.
- [89] Catenoix H, Mauguière F, Guénot M, Isnard J, Ryvlin P. Recording the insula during ictal asystole. *Int J Cardiol* 2013;169:e28–30.
- [90] Dylgjeri S, Taussig D, Chipaux M, Lebas A, Fohlen M, Bulteau C, et al. Insular and insulo-opercular epilepsy in childhood: an SEEG study. *Seizure* 2014;23:300–8.
- [91] Isnard J, Guénot M, Sindou M, Mauguière F. Clinical manifestations of insular lobe seizures: a stereoelectroencephalographic study. *Epilepsia* 2004;45:1079–90.
- [92] Mazzola L, Isnard J, Peyron R, Guénot M, Mauguière F. Somatotopic organization of pain responses to direct electrical stimulation of the human insular cortex. *Pain* 2009;146:99–104.
- [93] Montavont A, Mauguière F, Mazzola L, Garcia-Larrea L, Catenoix H, Ryvlin P, et al. On the origin of painful somatosensory seizures. *Neurology* 2015;84:594–601.
- [94] Proserpio P, Cossu M, Francione S, Tassi L, Mai R, Didato G, et al. Insular-opercular seizures manifesting with sleep-related paroxysmal motor behaviors: a stereo-EEG study. *Epilepsia* 2011;52:1781–91.
- [95] Ryvlin P, Minotti L, Demarquay G, Hirsch E, Arzimanoglou A, Hoffmann D, et al. Nocturnal hypermotor seizures, suggesting frontal lobe epilepsy, can originate in the insula. *Epilepsia* 2006;47:755–65.
- [96] Aubert S, Wendling F, Régis J, McGonigal A, Figarella-Branger D, Peragut J, et al. Local and remote epileptogenicity in focal cortical dysplasias and neurodevelopmental tumours. *Brain* 2009;132:3072–86.
- [97] Chassoux F. Malformations of cortical development: which strategy is best? *Neurochirurgie* 2008;54:272–81.
- [98] Chassoux F, Devaux B, Landré E, Turak B, Nataf F, Varlet P, et al. Stereoelectroencephalography in focal cortical dysplasia: a 3D approach to delineating the dysplastic cortex. *Brain* 2000;123:1733–51.
- [99] Tassi L, Colombo N, Garbelli R, Francione S, Lo Russo G, Mai R, et al. Focal cortical dysplasia: neuropathological subtypes, EEG, neuroimaging and surgical outcome. *Brain* 2002;125:1719–32.
- [100] Chassoux F, Landré E, Mellerio C, Laschet J, Devaux B, Dumas-Duport C. Dysembryoplastic neuroepithelial tumors: epileptogenicity related to histologic subtypes. *Clin Neurophysiol* 2013;124:1068–78.
- [101] Chassoux F, Rodrigo S, Mellerio C, Landré E, Miquel C, Turak B, et al. Dysembryoplastic neuroepithelial tumors. An MRI-based scheme for epilepsy surgery. *Neurology* 2012;79:1699–707.
- [102] Chassoux F. Stereo-EEG: the Sainte-Anne experience in focal cortical dysplasias. *Epileptic Disord* 2003;5(suppl 2):S95–103.
- [103] Chassoux F, Landre E, Mellerio C, Turak B, Mann M, Dumas-Duport C, et al., Type II. focal cortical dysplasia: electroclinical phenotype and surgical outcome related to imaging. *Epilepsia* 2012;53:349–58.
- [104] McGonigal A, Bartolomei F, Régis J, Gavaret MG, Trébuchon-Da Fonseca M. Stereoelectroencephalography in presurgical assessment of MRI-negative epilepsy. *Brain* 2007;130:3169–83.
- [105] Chassoux F, Landre E, Rodrigo S, Beuvon F, Turak B, Semah F, et al. Intralesional recordings and epileptogenic zone in focal polymicrogyria. *Epilepsia* 2008;49:51–64.
- [106] Mai R, Tassi L, Cossu M, Francione S, Lo Russo G, Garbelli R, et al. A neuropathological, stereo-EEG, and MRI study of subcortical band heterotopia. *Neurology* 2003;60:1834–8.
- [107] Maillard L, Koessler L, Colnat-Coulbois S, Vignal JP, Louis-Dorr V, Marie PY, et al. Combined SEEG and source localisation study of temporal lobe schizencephaly and polymicrogyria. *Clin Neurophysiol* 2009;120:1628–36.
- [108] Ramantani G, Koessler L, Colnat-Coulbois S, Vignal J-P, Isnard J, Catenoix H, et al. Intracranial evaluation of the epileptogenic zone in regional infrasyllian polymicrogyria. *Epilepsia* 2013;54:296–304.
- [109] Scherer C, Schuele S, Minotti L, Chabardes S, Hoffmann D, Kahane P. Intrinsic epileptogenicity of an isolated periventricular nodular heterotopia. *Neurology* 2005;65:495–6.
- [110] Tassi L, Colombo N, Mai MC, Francione R, Lo Russo S, et al. Electroclinical MRI and neuropathological study of 10 patients with nodular heterotopia, with surgical outcomes. *Brain* 2005;128:321–37.
- [111] Valton L, Guye M, McGonigal A, Marquis P, Wendling F, Régis J, et al. Functional interactions in brain networks underlying epileptic seizures in bilateral diffuse periventricular heterotopia. *Clin Neurophysiol* 2008;119:212–23.
- [112] Sevy A, Gavaret M, Trebuchon A, Vaugier L, Wendling F, Carron R, et al. Beyond the lesion: the epileptogenic networks around cavernous angiomas. *Epilepsy Res* 2014;108:701–8.
- [113] Navarro V, Martinerie J, Le Van Quyen M, Clemenceau S, Adam C, Baulac M, et al. Seizure anticipation in human neocortical partial epilepsy. *Brain* 2002;125:640–55.
- [114] Bancaud J, Ribet M, Chagot D. Origine comparée des paroxysmes de pointes « infra-cliniques » et des crises spontanées dans l'épilepsie. *Rev FEEG Neurophysiol* 1975;5:63–6.
- [115] Kahane P, Tassi L, Francione S, Hoffmann D, Lo Russo G, Munari C. Electroclinical manifestations elicited by intracerebral electric stimulation “shocks” in temporal lobe epilepsy. *Neurophysiol Clin* 1993;23:305–26.
- [116] Kovac S, Kahane P, Diehl B. Seizures induced by direct electrical cortical stimulation – Mechanisms and clinical considerations. *Clin Neurophysiol* 2016;127:31–9.
- [117] Landré E, Turak B, Toussaint D, Trottier S. Intérêt des stimulations électriques intracérébrales en stéréoelectroencéphalographie dans les épilepsies partielles. *Epilepsies* 2004;16:213–25.
- [118] Munari C, Kahane P, Tassi L, Francione S, Hoffmann D, Lo Russo G, et al. Intracerebral low frequency electrical stimulation: a new tool for the definition of the “epileptogenic area”? *Acta neurochir* 1993;58:181–5.
- [119] Adam C, Hasboun D, Clemenceau S, Dupont S, Baulac M, Hazemann P. Fast contralateral propagation of after-discharges induced by stimulation of medial temporal lobe. *J Clin Neurophysiol* 2004;21:399–403.
- [120] Bernier G, Richer F, Giard N, Bouvier G, Mercier M, Turmel A, et al. Electrical stimulation of the human brain in epilepsy. *Epilepsia* 1990;31:513–20.

- [121] Chauvel P, Landré E, Trottier S, Vignal J, Biraben A, Devaux B, et al. Electrical stimulation with intracerebral electrodes to evoke seizures. *Adv Neurol* 1993;63:115–21.
- [122] Wieser H, Bancaud J, Talairach J, Bonis A, Szikla G. Comparative value of spontaneous and chemically and electrically induced seizures in establishing the lateralization of temporal lobe seizures. *Epilepsia* 1979;20:47–59.
- [123] Gibbs S, Proserpio P, Terzaghi M, Pigorini A, Sarasso S, Lo Russo G, et al. Sleep-related epileptic behaviors and non-REM-related parasomnias: Insights from stereo-EEG. *Sleep Med Rev* 2016;25:4–20.
- [124] Nobili L, Cardinale F, Magliola U, Cicolin A, Didato G, Bramerio M, et al. Taylor's focal cortical dysplasia increases the risk of sleep-related epilepsy. *Epilepsia* 2009;50:2599–604.
- [125] Munari C, Andreoli A, Frattarelli M, Casaroli D. Activation with amitriptyline: electroclinical comments on 120 epileptic patients. *Rev Electroencephalogr Neurophysiol Clin* 1977;7:194–7.
- [126] Cossu M, Schiarit M, Francione S, Fuschillo DG, Nobili F, Cardinale L, et al. Stereoelectroencephalography in the presurgical evaluation of focal epilepsy in infancy and early childhood. *J Neurosurg Pediatrics* 2012;9:290–300.
- [127] Dorfmueller G, Ferrand-Sorbets S, Fohlen M, Bulteau C, Archambaud F, Delalande O, et al. Outcome of surgery in children with focal cortical dysplasia younger than 5 years explored by stereo-electroencephalography. *Childs Nerv Syst* 2014;30:1875–83.
- [128] Francione S, Vigliano P, Tassi L, Cardinale F, Mai R, Lo Russo G, et al. Surgery for drug resistant partial epilepsy in children with focal cortical dysplasia: anatomical-clinical correlations and neurophysiological data in 10 patients. *J Neurol Neurosurg Psychiatry* 2003;74:1493–501.
- [129] Gonzalez-Martinez J, Lachhwani D. Stereoelectroencephalography in children with cortical dysplasia: technique and results. *Childs Nerv Syst* 2014;30:1853–7.
- [130] Taussig D, Chipaux M, Lebas A, Fohlen M, Bulteau C, Ternier J, et al. Stereoelectroencephalography (SEEG) in 65 children: an effective and safe diagnostic method for pre-surgical diagnosis, independent of age. *Epileptic Disord* 2014;16:280–95.
- [131] Taussig D, Dorfmueller G, Fohlen M, Jalin C, Bulteau C, Ferrand-Sorbets S, et al. Invasive explorations in children younger than 3 years. *Seizure* 2012;21(8):631–8.
- [132] Devaux B, Chassoux F, Guenot M, Haegelen C, Bartolomei F, Rougier A, et al. Epilepsy surgery in France. Evaluation of activity. *Neurochirurgie* 2008;54:453–65.
- [133] Gonzalez-Martinez J, Bulacio J, Alexopoulos A, Jehi L, Bingaman W, Najm I. Stereoelectroencephalography in the "difficult to localize" refractory focal epilepsy: early experience from a North American epilepsy center. *Epilepsia* 2013;54:323–30.
- [134] Serletis D, Bulacio J, Bingaman W, Najm I, González-Martínez J. The stereotactic approach for mapping epileptic networks: a prospective study of 200 patients. *J Neurosurg* 2014;121:1239–46.