# Intracranial Electrodes (icEEG)

Last updated: July 3, 2019

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Intracranial EEG (icEEG) may be recorded:

1. **Subdural electrodes** – contacts embedded in thin Silastic plates arranged in STRIPS or GRIDS (strip electrodes and grid electrodes differ only in shape);

2. **Depth electrodes s, sEEG (stereoEEG)** - multiple-contact WIRES placed stereotactically into brain substance.

Suggested readings:

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**Diagnostic utility of invasive EEG for epilepsy surgery: Indications, modalities, and techniques**

*Prasanna Jayakar, †Jean Gotman, ‡A. Simon Harvey, §André Palmini, ¶Laura Tassi, #Donald Schomer, ††Francois Duboeuf, **Fabrice Bartolomei, †Alice Yu, ††Pavel Krsek, §§Demitrios Vlais, and ††Philipe Kahane*

*Epilepsia, 57(11):1735–1747, 2016*

doi: 10.1111/epi.13515
INDICATIONS

Used when noninvasive studies fail to adequately delineate epileptogenic zone.

1. **Discordant (incoherent)** EEG localization and MRI findings!!!
2. Epileptogenic zone in or near eloquent cortex – indication for cortical mapping (intraoperative or preoperative).
3. Localization to particular lobe when **imaging has no abnormalities**
4. **Deep lesions** (e.g. hypothalamic hamartoma, periventricular gray heterotopias)
5. **Temporal lobe seizures:**
   a) bilaterally independent
      N.B. most bitemporal epilepsies need icEEG (many turn to be unilateral!)
   b) with **negative imaging** (MRI and FDG-PET)
   c) to distinguish **neocortical** from medial TLE
   d) **extratemporal** lobe-onset seizures with rapid propagation to medial temporal lobe
   e) temporal seizures suspected to extend to extratemporal areas* (so called “temporal plus” epilepsies)

— otherwise, cases of temporal lobe epilepsy with consistent anatomo-electro-clinical findings are usually operated on after noninvasive investigations.

*insulo-opercular complex, temporo-parieto-occipital junction, or to the anterior frontal cortex

- **sensitive**, but provide **limited view** (record only from electrode area) – prior noninvasive studies must provide enough information to ensure appropriate electrode placement.
  N.B. intracranial EEG is most helpful when hypothesis about location of epileptogenic zone is clearly defined on basis of noninvasive data!
- can be used for **chronic recordings** (up to 1 month).
- **cortical heterotopias** may indicate that cortex is malformed – both SEEG and subdural grids may be indicated; same applies to **cortical dysplasias**.

Another group of indications (not related to epilepsy) – **brain mapping** in preparation for surgery

FREQUENCY

- with advent of newer imaging techniques, only 10-35% of surgical candidates require invasive EEG (vs. 50-60% 10-20 years ago).
  cortical resections based on invasive EEG data without MRI abnormality → seizure-free outcome in only 20% patients
INTRACRANIAL ELECTRODES

Joseph R. Smith and Kostas N. Fountas: invasive monitoring is used:
- in anterior mesial temporal resections: 47% with normal MRI, 19% with MTS, and 29% with foreign tissue.
- in extramesial temporal or neocortical (XMT) resections: 88% with normal MRI, 45% with foreign tissue lesions.

USA traditionally has been a subdural grid proponent; now paradigm shifts – from subdural grids in 99% of cases to SEEG in 99% of cases (Dr. Sharan at T. Jefferson).

CONTRAINDICATIONS

- mental retardation or uncooperative patient – will pull leads in EMU.

PREOPERATIVE, PERIOPERATIVE

- continue home AEDs perioperatively; once electrodes are implanted and patient goes to EMU, epileptologists will discontinue AEDs (for activation).
- no dexamethasone – risk of infections.
- no NSAIDs – risk of bleeding.
- check platelet function if patient is on valproic acid.

COUNSELLING

I spent over an hour with the patient discussing preop workup findings, talking about the grid surgery and the time in the EMU with the grids, possibility of needing evacuation of a hematoma, the possibility that she would not have seizures in the prescribed time period of a week.

- patients need to be counseled carefully about the possibility of lengthy studies that require them to be confined to a single room for, in rare instances, weeks at a time.
- intracranial electrodes and head dressing can be heavy and difficult to tolerate for some patients.

EPIDURAL ELECTRODES

- in comparison to scalp EEGs, epidural recordings have improved signal-to-noise ratio (due to reduced volume conduction and amplitude attenuation + eliminated myogenic and kinesigenic scalp artifacts).
- semi-invasive nature = fewer infectious and hemorrhagic complications.
- epidural electrode designs:
  a) ball electrodes
  b) screws - made of titanium and has a shaft to prevent overpenetration; length varies to allow stable placement and to accommodate the varying thickness of the scalp and calvaria; screw head is hexagonal to permit easy placement and removal of the electrodes with a wrench; right-angled EEG monitoring leads can be placed in the screw head.
  c) pegs - composed of mushroom-shaped Silastic elastomer, and the stalk tapers from a diameter of 4.7 mm to a diameter of 0.5 mm; at the stalk base, either stainless steel or platinum tips are used to conduct the electrical current.
  d) strip arrays
SUBDURAL ELECTRODES

- contacts embedded in thin Silastic plates arranged in STRIPS or GRIDS (strip electrodes and grid electrodes differ only in shape);
  - placed through burr holes (strips) or craniotomy (grids).
  - contact cortical surface directly.
  - some surgeons implant in epidural space (e.g. in patient with prior craniotomy - scaring may obliterate subdural space, esp. over eloquent cortex).
  - allows recording of multiple regions and cortical mapping.
  - cortex within depth of sulcus is not sampled adequately!
- STRIPS allow smaller craniotomies to be made and should be used generously over the convexitities and sylvian fissure.
- high-density (5 mm between contacts) grids are indicated only for peri-Rolandic epilepsy.

ELECTRODES

Ad-Tech
http://adtechmedical.com/
Ad-Tech Medical Instrument Corporation, Racine, WI, USA
Catalog >>

- intended for temporary (< 30 days) use with recording, monitoring and stimulation equipment.
- Ad-Tech’s products are natural rubber latex-free.

Characteristics
- composed of the same contact and insulating materials as depth electrodes.
- platinum based disk-shaped electrodes on a silicone based sheet (Silastic) that easily conforms to brain surface.
  - large contacts and Silastic insulation minimize dural pain upon cortical stimulation.
  - platinum (vs. stainless steel) allows safe postop MRI.
- distance between contact centers 10 mm.
- contact diameter 4 mm, exposed contact diameter 2.3 mm.
- tails are 1.5 mm diameter

Strips
- paddle width - 8 mm
- paddle length: 32 mm (2 contacts – only LTM)
I\textsuperscript{NTRACRANIAL} E\textsubscript{LECTRODES} E13 (6)

52 mm (4 contacts)
72-74 mm (6 contacts)
92 mm (8 contacts)
112 mm (10 contacts – only LTM)
132 mm (12 contacts – only LTM)

\textbf{IOM (intra-operative monitoring) strip} – contacts numbered; only 4 or 6 or 8 contacts

\textbf{Cable-attached with 3D style contacts} (5 mm exposure):

\textit{Cross-section view of 3D style contact}
Clover-leaf IOM electrode

- shape stabilizes the electrode contact on the brain.
- stainless steel numbered 3D style contacts (5.0 mm exposure).
**LTM (long-term monitoring) strip** – contacts non-numbered (#1 contact is most distal) with reinforcement;
- has 2 contact version (32 mm paddle)
- 4 contact version has 15 mm spacing between contacts (67 mm paddle length)
INTRACRANIAL ELECTRODES

LTM narrow-body strip – paddle width 6 mm, exposed contact diameter 1.8 mm, only platinum, only 4 or 6 or 8 contacts (paddle length same as regular version):
**LTM “3D” strip** – contacts numbered; **paddle width** 10 mm (2 mm wider than regular version), **exposed contact diameter** 5.0 mm, only **platinum**, only 4 or 6 or 8 **contacts** (paddle 2 mm longer than regular version)

**LTM “L” shaped strip** – only 8 contacts, straight or curved paddle:
INTRACRANIAL ELECTRODES

Reversed:

LTM “T” shaped strip – only 8 contacts, straight paddle:
I
NTRACRANIAL
E
LECTRODES

LTM multi-strip

Dual-sided interhemispheric LTM subdural electrodes
Mirror-imaged contacts on both sides of the electrode:
**Grounding electrode**

NS02R-KS10X-000
**Intracranial Electrodes**

**Grids**
- **single tail** options available (used with the single-tail passing needles - reduces the number of tunnels)
  
N.B. single tail grids have platinum marker in different location.

**Catalog #: FG08A-SP10X-000**

**Catalog #: FG16A-SP10X-000 & FG16A-SS10X-000**

**Catalog #: FG16C-SP10X-000**
INTRACRANIAL ELECTRODES

Catalog #: FG20C-SP10X-000 - $916.00

Catalog #: FG32C-SP10X-000 & FG32C-SS10X-000
INTRACRANIAL ELECTRODES

Platinum marker (typical)

5 mm outer diameter
60 cm tail

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32
33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48
49 50 51 52 53 54 55 56 57 58 59 60 61 62 63 64

1.5 mm

2.1 mm

80 mm

80 mm

80 mm

375 mm
5-mm spacing (8x8):
Catalog #: FG64C-SP05X-000 & FG64C-SS05X-000

35-contact temporal lobe grids
- designed for the temporal and sub-temporal cortex.

Right temporal lobe:
Catalog #: FG35D-SP10X-Z00 - $1,190.00
Left temporal lobe:
Catalog #: FG35E-SP10X-Z00 - $1,190.00

Split grid
Contoured shape (scallops & slits)
- more flexibility to conform to the brain (contact Ad-Tech to inquire if your preferred grid can also be contoured).
- numbered, platinum contacts, 4.0 mm diameter with a 2.3 mm exposure.
- 10 mm contact spacing is standard; additional spacing options available.

4-tail:
Catalog #: FG64C-SP10X-0C6 - $1,439.00

Single-tail:
**Intracranial Electrodes**

Perpendicular tail - smaller craniotomy (tails also positioned closer together):
Catalog #: FG64C-SP10X-020 - $1,584.00

**PMT**
Catalog >>

**Auragen System (Integra)**
Catalog >>
**INTRACRANIAL ELECTRODES**

### PROCEDURE

**SUBDURAL STRIP ELECTRODES**

- **Dr. Holloway** prefers neuronavigation.
- standard-sized burr hole at desired location (e.g. temporal [for temporal lobes], pterional [for frontal lobes], parasagittal [for medial surface of hemispheres])
- dura opened with cruciate incision.
- do not use foreign bodies (bone wax or Gelfoam) with electrodes - risk of infection if left in place.
- mannitol, hyperventilation are unnecessary.
- keep electrodes covered until insertion when they are rinsed in irrigation solution.
- #3 Penfield dissector is used to depress cortex and guide electrode into subdural space and along its proper trajectory (e.g. inferiorly and anteriorly around temporal tip; second strip may be passed inferiorly and posteriorly toward uncus).
- dura is not closed.
- electrode wires are tunneled with large-bore needle to exit skin 2-3 centimeters from burr hole incision.
- cable-retaining scalp suture is discouraged because this increases risk of breaking electrode cable subcutaneously if pulled violently during seizure.
- wound is closed in layers with Vicryl sutures in deep fascia & subcutaneous layer, staples in skin.
- consider BioPatch around wires at exit sites
- wound is dressed with Telfa and covered by Selofix.
- ABD dressing is placed over wounds, and head is wrapped with Kerlix.

**SUBDURAL GRID ELECTRODES**

- **Dr. Holloway** prefers neuronavigation.
- performed under general anesthesia, unless language or sensorimotor stimulation mapping is required as part of implantation procedure.
- most grids contain 32-64 contacts and require craniotomy.
- craniotomy is centered over suspected epileptogenic focus and should be large enough to sufficiently sample surrounding brain; place lots of bur holes (e.g. 4 for small temporal craniotomy) – to separate dura along all perimeter to minimize cortical bruising.
  e.g. (sub)temporal craniotomy* most importantly should be low enough to reach middle fossa floor; if entered pneumatized bone cells – just wax it; temporal skin incision - L shape starting at zygoma, extending up and then curving posteriorly just above the pinna (Dr. Holloway does not pin patient unless planning navigation to find lesion or place depth electrode during the same operation).

  *see also p. Op300 >>; for incision – see p. E15 >>

- hyperventilation is discouraged unless absolutely necessary; MANNITOL must be given.
- avoid DEXAMETHASONE if leaving leads outside.
- supine position with head rotated 60 degrees.
- dura is opened; careful microdissection of dura from cortex if patient had previous craniotomy! (previous craniotomy might be a contraindication for electrodes over the eloquent cortex due to the risk of damage while dissecting adhesions; H: SEEG); Dr. Spencer denervates dura to minimize postoperative headaches.
- electrode grid is positioned and edge secured to dural edge with 4-0 silk!
for subtemporal position, first inspect the space by gently retracting temporal lobe – make sure there are no bridging vessels!; slide grid into position while **squirting saline** between grid and brain (to hydroplane); if slightest resistance – stop and explore!

- use two grounding needle electrodes into muscle; then connect grid electrode to the cable and pass it to neurology team.
- place heavy lap pad soaked in saline on the grid.
- recording confirms good contact (make sure nobody touches the patient)
  - if grid buckles or does not fit space, contacts may be cut off or Silastic between electrode rows can be sliced to modify contour.
  - several strip electrodes may be added to sample cortex from surrounding areas, such as interhemispheric fissure or basal temporal lobe (subtemporal) or Sylvian fissure or subfrontal.
- leads are brought out through dural incision without depressing grid into cortex.
  - **each lead is tunneled separately** through skin (use 14G Angiocath) for several centimeters without traversing craniotomy wound.
    - N.B. tunnel under temporalis muscle (otherwise, wire will interfere with muscle closure)
  - leads are secured to scalp with purse string sutures and tying around lead in Roman sandal fashion → Dermabond on top.
  - consider BioPatch around wires at exit sites.
- dura is closed as watertight as possible; place patch (**DuraGen**) if defect remains and reinforce with **DuraSeal**.
- **strip ground epidural electrode** is placed inversely, i.e. contacts facing bone.
- bone flap is secured into place, and scalp is closed in layers using Vicryl for galea and staples for skin. Dr. Spencer does not replace bone (or replaces it without reattachment).
- place **long 00 silk stitch** through scalp (away from incision) and place **air knot** with long loose ends – will help secure leads in place during recording.
- standard head dressing is applied (leads exit top of head dressing).
- head is wrapped in a bulky dressing with a chin strap to prevent dislodgement of the electrodes during seizures and the electrodes exiting from one or two sites.

Subdural grid electrode array in place:

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**IMPLANTATION STRATEGIES**

- **regional venous anatomy** may dictate the use of custom-cut grids or subdural strips to avoid sacrificing a critical vein (e.g. location of the vein of Labbé may make grid placement difficult).
**L-shaped Interhemispheric Electrodes**

- special type for recording from *medial surface of hemispheres* - for monitoring of the supplementary motor area and cingulate.
- interhemispheric fissure is entered where the veins permit, and both strips are placed, one directed anteriorly and the other posteriorly.
- frequently, both sides can be accessed from a unilateral approach by making an opening in the falx between the superior and inferior sagittal sinuses.

**Frontotemporal Subdural Strip Study**

- frequently used bilaterally as a survey study for patients with poorly lateralized frontotemporal semiology.
INTRAOPERATIVE MAPPING
- see p. Op300 >>

COMPLICATIONS

**STRIP ELECTRODES**
- morbidity is low and primarily is infection (overall rate ≈ 0.85%).
- other reported complications: accidental extraction of electrodes, cortical contusion.

**GRID ELECTRODES**
- most common complication is infection.
- second most common problem is mass effect from cerebral edema (most common in children, where subdural space is minimal) - grid should be removed immediately.
- subdural hematoma from damage to bridging vein during implantation (EEG shows attenuation and becomes useless).

POSTOP
- see below >>

ELECTRODE REMOVAL
**INTRACRANIAL ELECTRODES**

- once sufficient data are obtained, grid must be removed.
- if resective surgery is planned, grid's relationship to underlying cortex must remain unchanged while reopening craniotomy.
  
  Dr. Spencer sometimes postpones large resections for 4-6 weeks to avoid retraction on mildly edematous brain immediately after grid removal.

- scalp (including electrode leads) is washed with Betadine for 5 minutes.
- electrode leads are stretched slightly and cut with heavy scissors as close to skin edge as possible - cut ends are allowed to retract back below skin surface to minimize contamination.
- scalp again is washed with Betadine.
- bone flap is removed and placed in bowl of antibiotic irrigation solution.
- dura is opened, leaving grid-stabilizing sutures intact to ensure that relationships between electrode and cortical topography are undisturbed.
- once surgeon has extrapolated mapped data to underlying cortex, grid is removed and discarded → appropriate resection is completed.

**DEPTH ELECTRODES**

- multiple-contact wires placed stereotactically.
  - sample deep structures (such as hippocampus and amygdala); can also be used to record activity from the neocortex through which electrodes pass, though the sampling area is quite limited.
  - primary indication – to record from hippocampus (esp. bitemporal abnormalities on surface EEG / MRI).
  - technically also feasible for other deep lesions (e.g. hypothalamic hamartoma, periventricular heterotopia, cortical dysplasia).

**CANNULAS**

**AD-TECH**
Two-Piece Slotted Cannula Kit

Ø = 2.11 mm
L = 240 mm

2 x 2SC-240X Two-Piece Slotted Cannula

1:REF 2SCK1-240X
Used with Ad-Tech Devices

Passing Needle Kit with Obturator

Ø = 13 ga
L = 3.5 in

5 x APN-3.5X Passing Needle with Obturator

1:REF APNK1-3.5X
Used with Ad-Tech Electrodes
**ELECTRODES**

- electrodes are flexible (old ones were rigid).
- 1–1.5 mm in **diameter**, arrays of 4–10 individual **contacts** with **spacing** from 5–10 mm between the center of each.
- material - platinum, though stainless steel, nickel-chromium and gold have all been used; **silver** and **copper** are toxic to brain tissue.
- outer casing is usually polyurethane which houses the individual wires for each contact.
- electrodes can also be configured to allow for the performance of microdialysis (for research applications).
- there are special bolts that can be used for laser guidance (for LITT after SEEG electrode is removed).

**AD-Tech**

For general info – see above >>
ROSA adaptors – see p. Op40 >>

- contacts are made of MRI compatible platinum.
- contact wires are made of platinum, and are individually insulated with Teflon; outer insulation of the electrode shaft is polyurethane impregnated with barium.
- lead securing cap snugs on the bolt.

**Anchor Bolts**

- titanium body (MRI friendly).

**Internal Bolt Diameter:**

LSBK1: 1.5 mm  
LSBK2: 0.99 mm

**Anchor Bolt Cap:**

Diameter: 6 mm  
Length: 8 mm

**Silicone Gasket:**

Diameter: 6.5 mm  
Length: 8.9 mm
**LSBK1-AX**

2.5mm distal opening diameter. 21mm long. Use with SD style depth electrodes.

**Catalog #: LSBK1-AX-05**

<table>
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<th>Catalog #</th>
<th>Description</th>
<th>Distal Opening Diameter “D”</th>
<th>Length “L”</th>
<th>Units</th>
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<tbody>
<tr>
<td>LSBK1-AX-05</td>
<td>Use with SD depth electrodes</td>
<td>2.5 mm</td>
<td>21 mm</td>
<td>3 Per Box</td>
</tr>
<tr>
<td>LSBK1-AX-06</td>
<td>Use with BF, LD or MM depth electrodes</td>
<td>2.5 mm</td>
<td>21 mm</td>
<td>3 Per Box</td>
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**LSBK1-BX**

2.5mm distal opening diameter. 13.3mm long. Use with SD style depth electrodes.

**Catalog #: LSBK1-BX-05**

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<th>Length “L”</th>
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<td>LSBK1-BX-05</td>
<td>Use with SD depth electrodes</td>
<td>2.5 mm</td>
<td>13 mm</td>
<td>3 Per Box</td>
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<tr>
<td>LSBK1-BX-06</td>
<td>Use with BF, LD or MM depth electrodes</td>
<td>2.5 mm</td>
<td>13 mm</td>
<td>3 Per Box</td>
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**LSBK2-AX-04**

.99mm distal opening diameter. 21mm long. Gold Anodized. Use with RD style depth electrodes.

**Catalog #: LSBK2-AX-04**

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<th>Length “L”</th>
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<td>LSBK2-AX-04</td>
<td>Use with RD depth electrodes</td>
<td>0.99 mm</td>
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<td>3 Per Box</td>
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**LSBK2-AX-04**
**Kit for ROSA robot:**

<table>
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<tr>
<td>DDK2-2.4-30X</td>
<td>2.4mm Drill Kit; (2) 30cm drill with stop and (2) wrenches. Sterile, Single-Use Only.</td>
</tr>
<tr>
<td>DDK2-2.4-16X</td>
<td>2.4mm Drill Kit; (2) 16cm drill with stop and (2) wrenches. Sterile, Single-Use Only.</td>
</tr>
<tr>
<td>LSB-PWL-2.4-10N</td>
<td>Placement/Removal Wrench for LSB series (24.5cm). Non-Sterile, Re-Usable.</td>
</tr>
<tr>
<td>OB-20-190X</td>
<td>Obturator, used to make track inside brain - used with RD style anchor bolts. Sterile, Reusable.</td>
</tr>
<tr>
<td>RULER-20N</td>
<td>20cm Channeled Ruler for drill stop &amp; depth electrode measurement. Non-Sterile, Re-Usable.</td>
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</tbody>
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**Slotted cannulas**

<table>
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<th>2-piece slotted Cannula with obturator</th>
<th>Length</th>
<th>Price</th>
</tr>
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<tbody>
<tr>
<td>2SCK1-190X</td>
<td>190 mm</td>
<td>$1042</td>
</tr>
<tr>
<td>2SCK1-240X</td>
<td>240 mm</td>
<td>$1042</td>
</tr>
</tbody>
</table>

**Passing needle**

<table>
<thead>
<tr>
<th>APNK1-3.5X</th>
<th>Length</th>
<th>Price</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.5 in</td>
<td></td>
<td>$96 ($482 box of 5)</td>
</tr>
</tbody>
</table>

**Electrodes**

**Spencer® depth probe**

- **contact spacing** measured from center of contact to center of contact.
- numbered and color coded for identification.
- 19 cm marker.
- stay flange for additional security.

**1.12 mm diameter (SD style)**

2.41 mm contact size:
### INTRACRANIAL ELECTRODES

#### # of contacts  | Contact spacing | Recording area | Catalog #  | Price
---|---|---|---|---
4 contact  | 5 mm  | 17 mm  | SD04R-SP05X-000  |
4 contact  | 10 mm  | 32 mm  | SD04R-SP10X-000  |
6 contact  | 5 mm  | 27 mm  | SD06R-SP05X-000  |
6 contact  | 10 mm  | 52 mm  | SD06R-SP10X-000  |
8 contact  | 5 mm  | 37 mm  | SD08R-SP05X-000  | $343
8 contact  | 10 mm  | 72 mm  | SD08R-SP10X-000  |
10 contact | 5 mm  | 47 mm  | SD10R-SP05X-000  |
10 contact | 10 mm  | 92 mm  | SD10R-SP10X-000  |
12 contact | 5 mm  | 57 mm  | SD12R-SP05X-000  |
14 contact | 5 mm  | 67 mm  | SD14R-SP05X-000  |

#### 1.32 mm contact size:

#### # of contacts  | Contact spacing | Recording area | Catalog #  |
---|---|---|---|
4 contact  | 2.2 mm  | 8 mm  | SD04R-AP58X-000  |
6 contact  | 2.2 mm  | 12 mm  | SD06R-AP58X-000  |
8 contact  | 2.2 mm  | 17 mm  | SD08R-AP58X-000  |

#### 1.96 mm diameter (AD style) – used with STEREOTACTIC DEVICES

#### 1.27 mm contact size:

#### # of contacts  | Contact spacing | Recording area | Catalog #  |
---|---|---|---|
4 contact  | 5 mm  | 16 mm  | AD04R-SP10X-000  |
4 contact  | 10 mm  | 31 mm  | AD04R-SP05X-000  |
6 contact  | 5 mm  | 26 mm  | AD06R-SP05X-000  |
8 contact  | 5 mm  | 36 mm  | AD08R-SP05X-000  |

#### sEEG depth

#### 0.86 mm diameter (RD style) – has stylet, used with STEREOTACTIC DEVICES

#### 2.29 mm contact size:

#### # of contacts  | Contact spacing | Recording area | Catalog #  |
---|---|---|---|
6 contact  | 5 mm  | 27 mm  | RD06R-SP05X-000  |
8 contact  | 4 mm  | 30 mm  | RD08R-SP04X-000  |
8 contact  | 5 mm  | 37 mm  | RD08R-SP05X-000  |
10 contact | 3 mm  | 29 mm  | RD10R-SP03X-000  |
10 contact | 4 mm  | 38 mm  | RD10R-SP04X-000  |
10 contact | 5 mm  | 47 mm  | RD10R-SP05X-000  |
10 contact | 6 mm  | 56 mm  | RD10R-SP06X-000  |
10 contact | 7 mm  | 65 mm  | RD10R-SP07X-000  |
**Behnke fried depth electrodes**
- for simultaneous macro and micro recordings.
- 1.28 mm diameter.
- 8-contact macro recording outer body.
- 8 or 9-contact micro recording bundle.
- contact 1 starts 1.0 mm +/- 0.5 mm from body tip.

8-contact macro depth electrode:

9-contact micro-wire bundle:
(8 recording & 1 reference contact); ends in “standard” pigtail.

**Macro-Micro Depth Electrodes (MM style)**
- one-piece design.
- for simultaneous macro and micro recordings.
- 1.3 mm diameter.
- 80% **platinum**, 20% **iridium** HML insulator.
- 50.8 platinum micron wire.
- allow 6-8 weeks for delivery (made to order).

<table>
<thead>
<tr>
<th>Catalog #</th>
<th>Description</th>
<th># of Macro Contacts</th>
<th># of Micro Contacts</th>
</tr>
</thead>
<tbody>
<tr>
<td>MM16C-SP05X-000</td>
<td>16-contact macro-micro depth</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>MM16D-SP05X-000</td>
<td>16-contact macro-micro depth</td>
<td>8</td>
<td>8</td>
</tr>
</tbody>
</table>

PMT
Catalog >>
- diameter 0.85 mm.
- cranial bolt goes through the same ROSA guiding tube as the drill.
- lead securing cap screws on the bolt.

MICRODEEP SYSTEM (DIXI MEDICAL)
See brochure >>

AURAGEN SYSTEM (INTEGRA)
See brochure >>

Diameter 1.25 mm.

Integra does not have anchors; also Integra does not make any tools that work specifically for the Rosa so as far as providing a solution, that is difficult.
Bill Chiklakis
Sr. Brain Mapping & Stereotactic Specialist
Integra Neurosurgery
978.973.5374t
TRAJECOTORIES

H = hippocampal, Am = amygdala, OF = orbitofrontal, OT = amygdalohippocampal, PC = cingulate, SMA = supplementary sensorimotor area.

Sphenoidal

- placed percutaneously, ideally with **fluoroscopic guidance**, under the zygomatic arch, against inferior surface of greater wing of sphenoid bone, until they rest near the foramen ovale.
- electrodes are directed toward the midportion of the foramen ovale.
- record from basilar and medial temporal lobe regions:
proximity of sphenoidal electrodes to the basal medial aspect of the temporal lobe may increase their sensitivity for detecting mesial temporal discharges - in patients suspected of having mesial temporal lobe epilepsy, the spike amplitude was largest with sphenoidal electrodes; Marks and coworkers found that the large sphenoidal spikes may be associated with extratemporal and extrahippocampal foci, thus raising questions regarding the specificity of sphenoidal electrodes (esp. if placed without fluoroscopic guidance).

**Ad-Tech**
- two needle lengths available - 51 mm & 70 mm long.
- 0.28 mm stainless steel or 0.18 mm platinum wire.
- 30 cm in length; un-insulated 3-4 mm tip.
- 21G (0.84 mm) needle.
Intracranial Electrodes

Foramen Ovale

- for cannulation technique - see p. Op310 >>

- were first developed in 1985 as a semi-invasive alternative to depth electrodes for the evaluation of mesial temporal lobe epilepsy; now used by many epilepsy centers only as an adjunct to standard scalp EEG for the evaluation of temporal lobe epilepsy; rather high false localization rate, thus, experts rely on true depth electrodes.
  
  N.B. percutaneous foramen ovale electrodes may be sufficient to clarify onset laterality but should not be used alone when there is a question of lateral versus mesial or temporal versus extratemporal onsets

- advance under live fluoroscopy (lateral view) to make sure electrode does not go into posterior fossa; don’t push if feel even slightest resistance.
- electrode construct is mounted on a thin stainless steel wire to allow appropriate flexibility to avoid puncturing the pia-arachnoid layer.
- electrode is placed into ambient cistern.
- typically, it is 4- or 6-contact electrode.
- electrode is fixed to cheek skin with adhesive tape, Dermabond.
- may be applied for EEG recordings for up to 3 weeks.
- removal of foramen ovale electrodes does not require anesthesia; transient spasm or dysesthesias in the ipsilateral teeth may be elicited during withdrawal of electrode.

Ad-Tech

- 1.02 mm diameter.
- 2.41 mm long, platinum contacts.
- each set includes two foramen ovale electrodes, two 17 gauge (1.016 mm) introducing needles for placement, and two TECH-ATTACH connector blocks.

4-contact, 5 mm spacing:
**Hippocampal**

- most common SEEG indication - to record from hippocampus; sometimes SEEG records activity up to 70 seconds earlier than subdural contacts (and up to 20% of SEEG-recorded seizures never show up on subdural recordings).
  
  **D. Spencer:**
  - SEEG is 20% more sensitive than subdural strip electrodes in detecting mesial temporal seizures (other experts find that every mesial temporal seizure shows up on subdural recordings);
  - pattern of spread never showed epileptiform activity in the contralateral neocortex prior to the ipsilateral neocortex, thus, the subdural strip electrodes never provided falsely lateralizing data (other experts reported such rare events).

  - some expert concerns that hippocampal (longitudinal) depth electrode placement may affect verbal memory – see p. E25 >>

**Approaches:**

a) **vertex approach** orthogonal to temporal lobe (sample temporal lobe from medial to lateral) via the middle or inferior temporal gyrus.
  - entry 10-14 cm posterior to nasion and 2-3 cm lateral to midline.
  - use **navigation** (e.g. AxiEM – stylet goes through the lumen of electrode or need to use slotted cannula – verify this with AD-TECH).
  - electrodes may enter through the holes / slits in grid electrode (if one is placed).

Typical amygdala and hippocampal explorations:
T1 post-implantation MRI;
In the sagittal slice at the level of the left mesial temporal structures electrodes sampling the amygdala (*) and hippocampus ($).
The coronal slices are parallel to the trajectories of the two electrodes.
b) preferred - **posterior approach** (SPENCER occipital trajectory) - along AP axis of hippocampus (most anterior contact in amygdala, most posterior - in posterior temporo-occipital lobe).

- allows implantation of *amygdala + anterior and posterior hippocampus* using single 6-contact electrode.
- requires stereotaxic frame or frameless platform with adequate clearance at back of head.
- final target point is within *inferior anterior amygdala* (determined on coronal MRI images) - slightly inferior to plane of hippocampus so that electrode will pass through length of hippocampus.
- make sure PCA is avoided.
— hitting brainstem or PCA may be decreased by targeting tip placement in lateral amygdala and lateral hippocampus, making sure occipital burr hole is not too medial, and by targeting of lateral ventricle just posterior to hippocampal tail; helpful step - place rigid guide cannula into ventricle (be sure CSF flows from cannula) and then insert depth electrode into ventricle - electrode will lie in temporal horn adjacent to hippocampus with its tip entering targeted amygdala - quality of ictal recording appears to be as effective as with intraparenchymal electrode.

N.B. intraventricular electrode position is OK.

Song et al. describe a combination of frameless navigation and neuroendoscopy for the placement of longitudinal hippocampal depth electrodes:

- using frameless stereotaxis, a trajectory is developed for introduction of the endoscope into the atrium of the lateral ventricle.
- under endoscopic visualization, the electrode is passed into the temporal horn along the hippocampus, without actual penetration of the tissue.
- plain X-ray is used intraoperatively to verify an appropriate trajectory.
- CSF pulsations do not appear to degrade the recordings.
- because the electrode does not actually traverse tissue, this method offers the advantage that the non-resected hippocampus is not injured.

### STEREOEEG (SEEG)

- individualized stereotactic implantation of intracerebral (depth) electrodes to study the epileptogenic neuronal network in its dynamic (temporal) and 3D (spatial) aspect*, with an optimal time and space correlation with the clinical semiology in order to perform a tailored safe resection.

*main difference from simple depth electrode explorations

- SEEG methodology was developed almost 60 years ago in France (Jean Talairach and Jean Bancaud during the 50s) and it has proven its efficacy and safety over the last 55 years.
  - vs. cortical electrodes on superficial cortex (Wilder Penfield and colleagues at Montreal Neurological Institute)
- the first implantation of intracerebral electrodes for epilepsy was performed on May 3, 1957 in Saint Anne Hospital (Paris, France).

### INDICATIONS

a) data obtained from phase I noninvasive investigations not sufficiently congruent to propose direct surgery (i.e. failure to clearly define the EZ) – see >>

b) epilepsy caused by malformations of cortical development poorly accessible to surgical resection for which SEEG-guided ablation (e.g. RF-TC) might represent a palliative therapeutic option

- SEEG tends to be the gold standard for phase III investigations because of its low complication rate and its high ability to explore deep region in comparison to subdural grid electrodes.

### IMPLANTATION STRATEGY

subtle scalp-EEG signs of seizure onset – see p. D27 >>
Seizure **semiology** is extremely important in diagnosing ictal zone and seizure propagation network – guides SEEG implantation strategy!
Ultimate SEEG goal – delineate EZ.

- implantation strategy is tailored based on preimplantation **hypothesis** that takes into consideration the electro-clinical correlation of the patient's seizures and their relation with a suspected lesion.
  N.B if the preimplantation hypotheses are incorrect, the placement of the depth electrodes will be inadequate and the interpretation of the SEEG recordings will not give access to the definition of the Epileptogenic Zone (**missing electrode** phenomenon; H: a) repeat scalp EEG b) go back to OR and implant crucial electrode).

- **Talairach and Bancaud philosophy:**
  - ictal clinical symptomatology must be viewed as a whole (seizure pattern); seizure symptoms (and more particularly the ‘signal-symptom’), when taken individually, could lead to erroneous interpretation.
  - do not rely on isolated electro-clinical signs, as well as to clusters or evolution of a few signs as identical clinical sign may result, indeed, from the ictal disorganization of cortex regions which, even if different, may have common subcortical projections.
    - e.g. an ascending epigastric sensation, ‘typically’ of (mesio-)temporal lobe origin, may as well translate an ictal involvement of the insular cortex as that of the mesial prefrontal cortex, or be the first manifestation of a discharge within a hypothalamic hamartoma.

- **SEEG electrodes** should sample:
  1. **Anatomic lesion** (if identified, so called “lesional implantation design” vs. “nonlesional design” – needs broader coverage):
     - “spear” it with electrode (one electrode can be directed to “spear” more than one lesion – useful strategy when need to keep the number of electrodes minimal when sampling multiple lesions, e.g. in tuberous sclerosis).
     - “fence” it around with electrodes.
     - even if SEEG shows that lesion is not involved in seizure onset, still resect it if it is easily accessible
  2. More likely structure(s) of **ictal onset**
  3. Early and late **spread regions**
  4. Most common deep nodes (insula, cingulate) and major “highways” (fronto-parietal, cingulo-opercular) – seizures maybe originating and / or propagating there.
    - e.g. insular lesion – implant whole cingulate (if posterior cingulate shows early propagation, avoid surgery – i.e. SEEG is here not to guide surgery but to tell when to avoid surgery)
  5. Interactions with the **functional eloquent** (cognitive, sensorimotor, behavioral, etc.) networks.
    - **there are no standard targets** - the selection of the structures to be explored must be determined individually; this is even true in lesional epilepsies where a SEEG study must clarify the relationships between the lesion itself and the region of seizure generation (the leading principle is to investigate all the cortical areas, the ictal involvement of which is suspected).
    - 3D “conceptualization” of the network nodes upstream and downstream from the hypothesized epileptogenic network is an essential component.
    - focus is not to map lobes or lobules, but epileptogenic networks, which, in general, involve multiple lobes.
    - investigation may include lateral and mesial surfaces of the different lobes, deep-seated cortices as the depths of sulci, insula, posterior areas in the interhemispheric cortical surface.

- sampling happens along the electrode trajectory.
Trajectory is more important than the target or entry point areas.

**Principles of SEEG implantation**

1. Demonstrate that brain regions suspected to be involved in seizure onset and early propagation (the ‘epileptogenic zone’) show the expected ictal pattern.
2. Consider the possibility that above pattern might in fact reflect the propagation of an ictal discharge generated elsewhere.
3. Delineate the border of the ‘epileptogenic zone’ as precisely as possible, in order to perform the minimum cortical resection - this requires the placement of electrodes in brain structures located outside the theoretical limits of the suspected ‘epileptogenic zone’.
4. Assess whether the removal of the cortical areas involved in seizure generation will be possible or not – this requires the investigation of the eloquent areas that are of interest, relatively to the hypothetical ‘epileptogenic zone’, and with respect to the possible boundaries of the planned resection.
5. Evaluate the precise relationships between an anatomical lesion (when present) and the ‘epileptogenic zone’ – this requires to investigate the epileptogenicity of the lesion itself and in any cases of the surrounding cortex, the number of the ‘lesional’ electrodes to use depending on the morphology, extent and anatomical location of the lesional process.

**Technical aspects**

- **trajectories** are checked to ensure that no trajectory collisions are present.
- **entry sites** are examined not to be prohibitively close (less than 6 mm distance) at the skin level.
- avoid forehead (thus, frontal trajectories are steep).
- implantations of > 15 depth electrodes are rare.
- targeting is through **avascular trajectories** – need appropriate imaging: see below >>
  - enter through gyrus crown and avoid crossing sulci (“rivers flow in valleys”)
  - **pial vessel are most dangerous** as they are tethered vs. deep vessels are yielding.
- if planning craniotomy and depth electrodes, Dr. Spencer places depth electrodes first – through dural slits (before opening dura – brain shifts).
  - e.g. if placing orthogonal insular electrodes, accuracy is very important

**IMAGING (PREOP)**

- **CT with contrast** or **CTA** are recommended by experts as some vascular structures might not be noted on contrasted MRI images.
- for high risk implantations (e.g. insular SEEG), recommend **DSA**: 
### SEEG VS. GRIDS

Tailored to individual patient!
- SEEG and subdural electrodes can be combined.
- **SEEG may access any cortical area** (incl. mesial, depth of sulci, insula, postoperative, multilobar, bilateral) but spatial coverage may be incomplete (i.e., unexplored gray matter areas).

  vs. **2/3-3/4 of cortex is inaccessible to subdural electrodes.**

<table>
<thead>
<tr>
<th></th>
<th>SEEG</th>
<th>Subdural grids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial cortex</td>
<td>Fair (limited coverage)</td>
<td>Excellent</td>
</tr>
<tr>
<td>Deep structures</td>
<td>Good</td>
<td>Poor</td>
</tr>
<tr>
<td>Anatomic relationships</td>
<td>Can be challenging</td>
<td>Straightforward</td>
</tr>
<tr>
<td>Cortical mapping</td>
<td>Fair (requires careful planning, good at exploring cortical depths, e.g. perisylvian)</td>
<td>Good (regular contiguous pattern, surface well covered)</td>
</tr>
<tr>
<td></td>
<td>opercula); if need to resect near language areas, better do awake crani</td>
<td>Challenging (can use burholes for strips)</td>
</tr>
<tr>
<td>--------------------------</td>
<td>-------------------------------------------------------------------------</td>
<td>-------------------------------------------</td>
</tr>
<tr>
<td>Bilateral</td>
<td>Straightforward</td>
<td>Poor (displacement, distortion)</td>
</tr>
<tr>
<td>Stereotaxy</td>
<td>Good</td>
<td></td>
</tr>
<tr>
<td>Data</td>
<td>3D</td>
<td>2D</td>
</tr>
<tr>
<td>Seizure capture</td>
<td>Earlier</td>
<td>May capture only late cortical projection of deep EZ</td>
</tr>
<tr>
<td>Complications</td>
<td>1-5%</td>
<td>5-30%</td>
</tr>
<tr>
<td>Cortical violation</td>
<td>Penetrates</td>
<td>No violation</td>
</tr>
<tr>
<td>Brain distortion</td>
<td>None</td>
<td>Common</td>
</tr>
<tr>
<td>Pneumocephalus</td>
<td>None</td>
<td>Common</td>
</tr>
<tr>
<td>CSF leak</td>
<td>Less common</td>
<td>More common</td>
</tr>
<tr>
<td>Previous craniotomy*</td>
<td>Straightforward (must avoid hardware)</td>
<td>Difficult (subdural adhesions)</td>
</tr>
<tr>
<td>Adding additional electrodes (during EMU stay)</td>
<td>Possible</td>
<td>Not practical</td>
</tr>
<tr>
<td>Electrode removal</td>
<td>At bedside</td>
<td>Requires OR</td>
</tr>
<tr>
<td>Subsequent resection</td>
<td>Challenging (performed after removal, must use stereotaxy) but also gives more time for planning**</td>
<td>Straightforward but needs to happen at the same procedure with grid removal (reopen same craniotomy, use electrodes to guide)</td>
</tr>
</tbody>
</table>

*alternative – epidural peg electrodes can be used; another alternative – subdural electrode can be placed in the epidural space, but unless the dura has been denervated, these electrodes cannot be used for stimulation-based mapping.

**there is no min or max time when to perform surgery after SEEG is done; SEEG does not obligate to surgery (SEEG is more like presurgical evaluation tool – it is the whole advantage as intracranial EEG should be diagnostic tool and not to push to have surgery at the end (vs. grids lead to resection almost 100% -it is not fair for diagnostic tool to do that!)

---

### Cases where SEEG is Better

- Widely extended lesion, only a small portion may be epileptogenic
- Multiple possible epileptogenic lesions (nodular heterotopia, tuberous sclerosis)
- Temporal lobe epilepsy: laterality unclear, potential neocortical, or “temporal plus”
- Suspected insular epilepsy
- Investigation after a previous craniotomy
TYPICAL PATTERNS OF COVERAGE

Limbic Network Explorations

- typically, cases of temporal lobe epilepsy with limbic network involvement are operated on after noninvasive investigation only.
- SEEG is indicated if the EZs are suspected to involve extra-temporal areas as well – the SEEG must be wide enough to disclose a preferential spread to:
  a) temporo-insular-anterior perisylvian areas
  b) temporo-insular-orbitofrontal areas
  c) posterior temporal, posterior insular, temporo-basal, parietal, and posterior cingulate areas.

Insular Cortex Explorations

- insula sometimes requires very dense SEEG coverage; however, high vascular risk resulting in an incomplete exploration of the insular cortex.
- two approaches:
  a) orthogonal (transopercular) - through the supra- or infra-sylvian opercula – only point sampling of insula; N.B. accuracy is very important – use formal DSA for vascular anatomy, place electrodes prior to large dural openings (to avoid brain shifts).
  b) parasagittal - by a retro-insular trajectory with a fronto-polar entry point just lateral to SSS – better exploration of the whole insular cortex.
INTRACRANIAL ELECTRODES

- per Dr. Spencer, to cover insula need at least 3 electrodes – 2 orthogonal and 1 parasagittal.
- for nonlesional insula plan to use RNS (laser heat may spread to white matter).

Articles to read:


FRONTAL-PARIETAL NETWORK EXPLORATIONS

- high number of electrodes are required for an adequate coverage of this large region.
- orbito-frontal epilepsy - investigate gyrus rectus, the frontal polar areas, the anterior cingulate gyrus, and temporal pole.
- seizures from the mesial wall of the premotor cortex - investigate rostral and caudal part of the supplementary motor area, the presupplementary motor area, different portions of the cingulate gyrus and sulcus, and the primary motor cortex and mesial, and dorsal-lateral parietal cortex.
- electrodes in rolandic regions are placed when there is a need to define the posterior margin of the resection in frontal network explorations or the anterior margin in parietal-occipital explorations, or when the EZ may be located in or near rolandic cortex.
  — depth electrodes sample the depth of the central sulcus, and the associated descending and ascending white matter fibers.

POSTERIOR QUADRANT NETWORK EXPLORATIONS

- frequent simultaneous involvement of several occipital, parietal, and posterior temporal structures + multidirectional spread to supra and infra sylvian areas.
- placement of electrodes to a single lobe is extremely uncommon.
- cover mesial and dorsal lateral surfaces of the occipital lobes (both infra-calcarine and supra-calcarine areas), in association with posterior temporal, posterior perisylvian, basal temporal-occipital areas, and posterior parietal areas, including the posterior inferior parietal lobule and the posterior precuneus.
- bilateral explorations are generally needed due to rapid contralateral spread of ictal activity.

Electrodes sampling the superior (*) and inferior ($) lips of the left calcarine fissure; a different trajectory has been employed for an additional electrode which encroaches the calcarine fissure with an entry point in the parietal parasagittal cortex (right lower):
INTRACRANIAL ELECTRODES

BRAIN DEVELOPMENTAL ABNORMALITIES

- Individuals with positive imaging that shows brain developmental disorders may have more diffuse brain abnormalities, and the threshold for invasive monitoring must be low (unless all data coherently points to one lesion).
- Subdural grid coverage may not identify the seizure source if it is located in a malformed area of gray matter; therefore, depth electrode coverage of the lesion itself may be required.

PROCEDURE

- Electrode placement is performed with the patient under general anesthesia.
- No need to shave head.
- A-line is not needed.

a) For one electrode may use Vertek arm.
b) **stereotactic frame** – time consuming for multiple electrodes.

c) **STarFix** – very fast but noncustomizable and needs bone fiducials implanted days ahead.

d) **robotized passive tool-holder (ROSA)** – most common approach by experts; see p. Op40 >>

- general anesthesia
- head secured to radiolucent Mayfield holder → O-arm registration.
- occiput is shaved from vertex to inion.
- vertical incision over entrance burr hole.
- cruciate dural incision (if small twist hole is used, dura is cauterized and opened with monopolar electrode).
- occipital lobe is inspected and small adjustments made to avoid puncturing draining vein.
- pia and arachnoid are cauterized.
- specially designed hollow bolt is placed in bone and stereotactically aligned with target point.
- to improve flexible electrode placement accuracy, **rigid guide 14G cannula** is inserted from occipital cortex to point just posterior to hippocampal tail.
- **depth electrode** is introduced through cannula (often can feel subtle “pop” as electrode breaches ependyma) → electrode is advanced gently to target position.
  — if resistance is encountered, do not advance electrode further until all trajectory coordinates are verified by independent operator - recalculating trajectory always is preferable to forcing electrode past resistance.
- stylet and guide cannula are withdrawn (of note, electrode has tendency to back out during stylet and cannula removal, but this may be minimized with practice).
- electrode is anchored by wrapping in grooves of bolt head and secured there by plastic cap filled with liquid Silastic.

The depth electrode shaft is wrapped in the threading of the head of the anchor bolt (arrow). A second anchor bolt (and depth electrode) has been capped with liquid Silastic and a Silastic cap (Ad-Tech):
INTRACRANIAL ELECTRODES

- if cap is not used, then electrode is tunneled to exit site and sutured to skin to prevent dislodgment.
- **occipital subdural strip electrodes** may be added via same burr hole enlarged with Kerrison rongeur.
- after all electrodes are placed, each entry site is covered with antibiotic ointment.
- labels are placed on the distal end of each electrode for later identification in the monitoring suite by the EEG technicians.

**POSTOP**
- patient goes to EMU. see below >>

**ELECTRODE REMOVAL**
- depth electrodes can be removed percutaneously in patient's room (conscious sedation may be necessary for kids only): remove electrode, unscrew bolt (don’t push too hard to engage screwdriver into the bolt – bolt may have become loose over many days, especially in thin squamous temporal bone – easy to push into cranium; if bolt feels loose, use needle driver to unscrew it), place one Monocryl stitch for scalp.
- fishnet-type stockinet cap is placed on the patient’s head to hold the pads in place.
- ≥ 45 degree head up position for the next 24 hours to allow the entry sites to seal off; if they remain dry overnight, the patient is discharged with instructions to keep the head dry for several days. i.e. patient spends at least half a day inpatient after removal of depth electrodes
- resective surgery is planned no sooner than 6 weeks after the electrodes are removed to ensure all entry sites heal properly.

N.B. **brain returns to electrophysiologic baseline** after electrode removal:
  - 3 months – after depth electrodes
  - 4 months – after grid electrodes
INTRACRANIAL ELECTRODES

COMPLICATIONS

SEEG is “blind” procedure – intraop complications difficult to recognize!

- wound infections (0.4-1.8% or 0.03-0.2% per electrode); avoid CSF leaks and tunnel (if feasible); electrodes are not reused due to risk of CJD.
- hemorrhagic complications (0.8-2.9%, or 0.07-0.2% per electrode); one case of lethal IPH reported
- permanent neurological deficit (0.4-1%)
- electrode malfunction
- mortality – one report of lethal IPH, one report of lethal massive brain edema with concomitant hyponatremia.

OUTCOME

SEEG guides further treatment:
- a) conventional surgery
- b) laser ablation
- c) RF ablation

EMU POSTOP

- dispo - over night to ICU, on POD #1 is transferred to EMU; strict head of bed up.
- activity as tolerated but patient is not allowed to get out of bed without 2 people assisting / supervising (in case, seizure happens, - patient may fall and pull electrodes)
- dressing - during first 3 days, CSF may leak around electrode wires – place stitch around lead, Kerlix-ABD dressing is changed as needed using sterile techniques (original Selofix is not changed until electrodes are extracted or staples are removed 1 week postoperatively).
- give antibiotic, e.g. CEFAZOLIN, while intracranial electrodes are in (some experts give antibiotics only for 24 hrs postop); avoid steroids!
- good pain control!!!

IMAGING

- imaging - skull x-rays or CT scan on POD #1 - to confirm electrode positions (basal temporal electrodes are difficult to visualize with CT unless special thin cuts (≤ 2 mm) are taken through middle fossa).
  - MRI-compatible electrodes (platinum, nickel-chromium alloy) are not easily visualized on MRI (or give very large artefacts), nonetheless, some experts prefer postop MRI on every case.
- if sending for MRI, make sure electrode cables do not form a loop.

SOFTWARE

- for electrode visualization and EEG source analysis – available software (FDA-cleared):
  1) CURRY – state of the art
  2) EGI (by Philips)
3) *Osirix MD*

- software may have automated icEEG analysis to help determine EZ.

**EMU STAGES**

1. Recording interictal activity
2. Recording ictal activity, s. spontaneous seizures (SS) and make sure it *habitual* (*look for both – clinical and electrical concordance*
3. Stimulation for brain mapping
4. Stimulation to induce seizures s. direct cortical stimulation induced seizures (DCS-S) – may shorten EMU stay (if not enough spontaneous seizures were captured and DCS-Ss are *habitual*)

Goal - constructing a composite ‘drawing’ of the brain area to be removed (‘what-to-remove area’).

**Recording (ECoG s. icEEG)**

- recordings are begun the next day of electrode implantation.
- leads are connected to an isolation box to prevent any inadvertent current from entering the patient.
- impedances are checked with a small current in the vicinity of 10 nA.
- isolation box is then connected to a multichannel amplifier and a recording system.
- due to potential for infection, duration of recording period is limited to 7-10 days (rarely, up to 2 weeks).
- if more electrode contacts exist than EEG recording channels, sample as wide area of cortex as possible, leaving some contacts out; after first seizure, involved regions become more apparent and contacts from uninvolved cortex may be dropped in favor of those closer to ictal onset.
- **first** record without stopping AEDs – may give enough information; if not, after 1-2 days consider activation - weaning AED one at the time.
  - METHOHEXITAL (Brevital®) may be given to try to provoke a seizure: observe for ↓ fast activity in suspected focus.
  - particularly with grid placement, the operation itself may cause a period of decreased seizure activity, which may prolong studies in EMU.
- **goal** – to capture adequate number (at least 3) of spontaneous electrographic seizures using video-EEG (to be sure they originate at the same contacts each time).
  - if the case is complex (e.g., independent bilateral anteromesial temporal foci, or more than one seizure focus suspected based on two clinically different seizure types), at least twice as many seizures may need to be recorded.
  - after the recordings are completed, the patient is placed back on therapeutic doses of AEDs.
- **goal** is to determine:
  1) **ictal onset zone** - the first clear ictal electrical change that: 1) occurs prior to the clinical onset of the seizure, and (2) that it manifests by a fast synchronizing discharge (low-voltage fast activity or recruiting fast discharge of spikes).
    - N.B. lack of one of these two criteria implies an incorrect SEEG investigation (“**missing electrode**” phenomenon – electrodes are recording arriving seizure activity but electrodes are not at ictal onset zone)!
  2) how much of the cortex contiguous to the site of origin is recruited into action to produce a clinical seizure (Bancaud and Talairach term – “*the primary organization of the epileptic seizures*”), i.e. the spatial extent of seizure discharges at the moment where the first clinical sign(s) occurs.
    - N.B. 1) and 2) constitute the epileptogenic zone
INTRACRANIAL ELECTRODES

INTERPRETATION

- usually ECoG is easier to read than scalp EEG because “everything looks much more obvious”.
- SEEG data is acquired using a referential montage, with a reference electrode in the white matter
- tissue adjacent to structural lesion may contain epileptogenic focus, while lesion tissue is relatively silent or may show slow-wave activity.
- most important are ictal data (esp. during ictal onset); seizures within seconds to minutes after another seizure are disregarded as potentially misleading.
- three most common patterns that signal seizure onset zone:
  - voltage attenuation
  - high-frequency oscillations (HFO), esp. fast ripples - delineate the SOZ independently with better specificity, albeit with less sensitivity, than spikes*. see below >>
  - high-amplitude spike and wave (or sharp and slow-wave) discharge followed by attenuation or high-frequency rhythmic discharges.
    *ripples increase preictally and with reductions in AED levels, while spikes are more prominent postictally.
- postictal areas of major SEEG attenuation and/or suppression of background activity may also have localizing feature (esp. in frontal region).
- interictal findings:
  - lesional zone - brain area with abnormal slow-wave activity (or, in some cases, by a major alteration of background activity or by an electrical silence) - presume an underlying macroscopic alteration of the neural tissue – consider for surgical planning (even if MRI is negative)
  - irritative zone – structures involved by spike activity – surgeon should not ignore the extent of interictal spikes (whether they are focal or not), especially, if spikes do not disappear at seizure onset (Lüders and colleagues named that as a “potential seizure onset zone”).
- signs of precise localization: discharge confined to 1-2 contacts (esp. with voltage reversal between two contacts).
- signs of imprecise localization (consider additional implantations):
  - simultaneous involvement of multiple contacts - indicates regional onset (secondary activation of recording sites).
  - simultaneous involvement of two distant contacts - indicates distant onset (secondary activation of contacts)
- caution when interpreting electrical activity at the edge of grids or the end of strips - source just beyond the area of coverage may appear to originate from the edge contacts.

Multifocal discharges
N.B. patients with electro-clinical evidence of different seizure types are excluded from surgery after the non-invasive phase of the evaluation, i.e. SEEG is not indicated.
- occasionally, SEEG may establish that different seizure types have a link in between (i.e. SEEG may demonstrate that the different seizure types, although multifocal, are in fact part of a widely extended area which can be safely removed).

Depth electrode recording of focal seizure onset in right hippocampus:
Epileptiform discharges originating at subtemporal electrodes 5 and 9:
Seizure originating at subtemporal electrodes 2 and 6:

Seizure activity slows down:

Seizure eventually terminates with postictal suppression:
HIGH-FREQUENCY OSCILLATIONS (HFO)

- spontaneous fast oscillatory EEG events:
  - Ripples (80-250 Hz) - normal expression of normal physiological behavior (thought to reflect summated excitatory postsynaptic potentials)
  - Fast ripples (250-500 Hz) - proposed to be biomarkers of epileptogenic areas (summated action potentials from synchronously bursting neurons) but some cognitive processes are associated with fast ripple band activity.

- detected during slow-wave sleep with intracranial EEG or acute intraoperative ECoG.
- seizure-free outcome could be expected in patients in whom the surgical planning includes the majority of interictal HFO-generating brain tissue, while a poor seizure outcome could be expected in patients in whom only a few such areas were planned to be resected.
  
  N.B. some patients become seizure-free without removal of the majority of HFO-generating tissue!
- Cochrane review concluded that the evidence for effective use of ictal HFOs for epilepsy surgery decision-making is rather poor.


BRAIN MAPPING IN EMU

For intraoperative mapping – see p. Op300 >>
Speech Mapping - see p. Op300 >>

- determines areas of eloquent cortex that should not be encroached upon at time of operation.
INTRACRANIAL ELECTRODES

- some experts prefer it over intraoperative mapping (patient has clear sensorium, no time pressure)
- usually done after enough seizures are captured in EMU because cortical mapping stimulation causes suppression (↓ chances of seizures), i.e. mapping stimulation works as AED (principle of RNS)!
- mapping is done 24 hours after loading with AEDs.

**Depth electrode**

See above >>

**Grid electrode**

- two machines available for stimulation:
  1) Green
  2) Grass S12
  3) Ojemann
- technique requires **testing protocol** (appropriate to cortical region investigated) and **neuropsychologist trained** in cortical testing.
- **video-taping** permits recording of electrographic, objective, and subjective phenomena.
- **bipolar (vs. monopolar) stimulation** requires lower currents but both (bipolar and monopolar) are safe procedures and give similar eloquent cortex mapping.
- **stimulation parameters:** two-channel stimulator with isolation and current control circuits provides balanced square wave bipolar **stimuli of high frequency** (50 Hz), delivered for 2-5 sec (most experts say 2 sec is enough), with 0.5 ms duration per phase (i.e. 1 msec pulse width); start at 1 mA and increase in 1 mA steps (up to 14 mA) until clinical subjective or objective responses or afterdischarges were obtained.
  - some experts add **low-frequency** (1 Hz, pulse width 1–3 msec, delivered for 30-40 s) and stimulations with stepwise increasing intensities up to 3 mA.
  - in tumor surgery (as opposed to epilepsy surgery), intraoperative stimulation uses lower current so to maximize tumor resection (but risk of false negative responses).
- **threshold** is established **for afterdischarge** (highest threshold is found at site of major pathology); thresholds are generally in range of 4-6 mA (if thresholds are > 6 mA or when there is inability to establish threshold for afterdischarge in amygdala or hippocampus, there is high correlation with localization of pathological focus).

  Afterdischarge may lead to false* positive response or even may lead to seizure!

  *i.e. while stimulating, monitor adjacent EEG activity to ensure that disruption of neurologic function is directly correlative to stimulation and not to focal afterdischarge.

  Afterdischarge is “all or none” phenomenon and is produced once a critical number of neurons are depolarized.

- **amount of current** needed (i.e. threshold) to produce effect (response, afterdischarge) varies among patients and among cortical regions (use enough current to produce reliable effects without painful spread to dura or nearby cortical vessel).

  N.B. there is a significant variability of stimulation thresholds among patients and no reliable factors (electrode location or patient characteristic [such as age, epilepsy duration]) to predict the threshold
- if mapping stimulus **induces seizures**, stimulate again (often aborts seizure, but have Ativan 2 mg IV ready at bedside).
- goal:
  1) reproducing entirely or in part the spontaneous (habitual) clinical ictal manifestations – include stimulated area in surgical plan.
  2) functional cortical and subcortical **mapping**.
— for primary sensorimotor functions low-frequency stimulations are preferred (positive responses may be obtained from stimulations both in grey and white matter, thus allowing to map critical pathways extensively).
— speech and visual areas are mapped using a combination of low and high-frequency stimulations.
— low-frequency stimulations are usually adequate in inducing subjective acoustic changes, the effect of high frequencies resulting often unpleasant for the patient.

• it is crucial to distinguish between patients with early ictal involvement of highly eloquent regions, who should be excluded from surgery, and those with later spread of the discharge to these structures, who can be operated on with limited surgical risks and with predictable benefit on seizures.
• results are charted:

\[ \text{SEEG-GUIDED RF-TC} \]

In addition to passive recording aimed at localizing the epileptogenic zone, SEEG electrodes can be used to produce focal lesions of the epileptogenic zone by the use of a radiofrequency generator connected to the electrode contacts.

\[ \text{HISTORY} \]

• a first paper reporting an effect of RF-TC on epilepsy was published in 1965
• from the 1970s to the 1990s, stereotactic lesioning was largely developed as an alternative to conventional surgery.
• due to the rather disappointing results of this technique in mesial temporal lobe epilepsy (MTLE) compared to those of conventional surgery, the technique has been almost totally abandoned.
  Important: these early results have been obtained from single lesion procedures guided by noninvasive investigations.
• in 2004, Marc Guénot group decided to use SEEG to guide RF-TC.

\[ \text{INDICATIONS} \]
INTRACRANIAL ELECTRODES

patients who undergo diagnostic SEEG and are diagnosed with:

a) focal epileptic zone poorly accessible to safe surgery (e.g., periventricular heterotopia or insular ictal onset zone) or located very close to cortical areas with a high functional value (motor, language, or visual primary areas) - palliative* procedure

*SEEG-guided RF-TC can be curative in some patients (esp. with gray matter nodular heterotopy-related epilepsy)

b) epilepsy related to a large unilateral epileptogenic network (multifocal ictal onsets) - network functional disruption by ablating multiple, anatomically distant network nodes; chances of seizure-freedom is low, however, significant improvement can occur; may need to repeat RF with new SEEG data about the possible modification of the epileptic network that can help to define the optimal target.

c) epilepsy eligible for conventional surgery – optional* procedure

*in some centers, SEEG-guided RF-TC is offered to most patients undergoing SEEG monitoring; as for patients, who are eligible for surgery, even transient relief from seizures after placement of coagulations in the area of scheduled resection may be of some prognostic value for post-resection seizure outcome, suggesting that the EZ has been correctly identified.

- if patient is not a candidate for SEEG, laser ablation may be a better option.
- no age restrictions.
- most favorable etiology – nodular neuronal heterotopy

KEY FEATURES

- advantages of SEEG-guided RF-TC:
  1) the lesioning can be targeted on the seizure-onset zone delineated by SEEG.
  2) multiple lesions can be performed between adjacent contacts of the SEEG electrodes.
  3) the electrodes used to perform the thermocoagulation are those implanted for the SEEG – no additional surgical risk related to electrodes implantation per se.
  4) lesioning is preceded by a functional mapping through bipolar contact stimulation, thereby preventing/predicting the postlesion neurologic deficit.
  5) procedure does not require anesthesia.

- disadvantages of SEEG-guided RF-TC:
  1) invasive
  2) cannot monitor temperature
  3) cannot monitor lesioning in real time
  4) lesion is small (6x8 mm around electrode contact)

  SEEG is limited by sampling area
  SEEG-RFTC is limited by lesion size

TARGET SELECTION

Inclusion criteria for contact pair to be used for RF-TC

A. Involvement in the onset of the ictal discharge

a) selected adjacent contacts must be located in cortical areas showing either a low-amplitude fast activity pattern or recruiting and periodic fast discharge of spikes at the onset of the seizures (prior to clinical onset).

b) interictal paroxysmal activities (spikes, polyspikes, spike-and-wave, or polspike-and-wave complexes) can obviously help to locate the ictal onset zone (especially in case of dysplasia) but are insufficient, if isolated, to be considered as a SEEG-guided RF-TC target.
B. Intralesional location
C. Induction of habitual ictal clinical phenomena by electrical stimulation

Exclusion criteria for contact pair
A. If the selected contacts sample a functionally critical area, as documented by SEEG functional mapping (e.g. movement, speech, vision).
B. Proximity of vascular structures (< 2-4 mm from selected contacts – usually SEEG electrode is already in place).

Target cortex, not white matter!

**PROCEDURE**
- done at the end of SEEG explorations
- postimplantation MRI
- no steroids
- patient is awake - each selected site for SEEG-guided RF-TC is first tested by low- and high-frequency bipolar electric stimulations between two adjacent contacts (see above); site is validated as a RF-TC target if no neurologic side-effect occurred during stimulation (during the video-SEEG recording and neurological testing session)
  - N.B. sometimes postprocedural deficit may be unexpected – due to edema larger than stimulated region; usually such deficit is transient

Protocol for lesioning
RF-TC is delivered between two contiguous contacts located in a target: 40-50 V / 75-120 mA = 6 W is used for 10–60 s, which causes an increase of the local temperature up to 78°–82°C.
- alternatively, power is progressively increased from 1.5 W up to 8.32 W within 60 s; current intensity (usually around 25 mA) is variable according to impedance → lesion between two contacts is an ovoid with a long axis 6 mm and a maximal thickness of 3.5 mm (in some studies, up to 7 mm diameter)
  a) in animal studies, unfixed parameters when power increases in a few seconds (vs. using fixed parameters) until the power delivered spontaneously collapsed* produces largest lesions;
  *why power collapses: thermocoagulation causes coagulation necrosis around the electrode contacts → proportional raise of tissular impedance; from a certain power strength, this raise of tissular impedance becomes so elevated that it finally prevents the power from being delivered by the RF generator.
  - N.B. it is impossible to unintentionally create lesions of excessive size
- in animal studies, use of contiguous contacts on electrodes leads to lesions with a higher volume than those produced with noncontiguous* ones
  *long distance between noncontiguous contacts does not allow the production of a confluent lesion; it produces only 2 smaller separate spherical lesions, each of them centered on an electrode contact.

Visual aspect of multiple radiofrequency thermocoagulation performed on adjacent contacts (A) and separated contacts (B):
• in animal studies, the size of a lesion resulting from RF-TC never kept growing over 30 seconds of the procedure (vs. in monopolar RF, lesioning takes 60 seconds)
• in animal studies, SEEG electrodes are not damaged by RF-TC - no concern about a possible alteration of the brain-electrode interface, which could have led to a risk of adhesion between brain tissue and electrodes.
• SEEG electrodes do not have any thermocouples, making it impossible to monitor temperature during the procedure.
• RF-TC through SEEG electrodes uses bipolar method (vs. conventional RF uses monopolar method) – can create lesions with much sharper limits; conventional monopolar RF electrodes are larger and create larger lesions.
• lesion generators:
  a) Radionics model RFG-3 (Radionics Medical Products, Burlington, MA, U.S.A.)
  b) Inomed Medizintechnik GmbH (Emmendingen, Germany)
- electrodes manufactured by Dixi (Dixi Medical, Besancon, France): 5 to 18 contacts, contact length is 2 mm and diameter 0.8 mm (intercontact spacing - 1.5 mm).
POSTPROCEDURE

- SEEG recordings are performed after the procedure to evaluate how interictal epileptiform activity was influenced by RFTC → SEEG electrodes removed 24 h after RFTC.
- patient is discharged after 24 hrs.
- MRI 3-6 months postprocedurally; when multiple RF-TCs are carried out on the same electrode track, this results in confluent lesion:
INTRACRANIAL ELECTRODES


SIDE EFFECTS

- neurological deficits (may be transient due to edema when multiple lesions are concentrated within a limited brain volume)
- fleeting local pain during coagulation of regions contiguous to the tentorium or to the cavernous sinus.
- habitual seizure may occur during the coagulation procedure (in ≈ 10% of patients)

OUTCOMES

- reported rate of seizure freedom provided by SEEG-guided RF-TC is from 2.5 to 18% of patients
  N.B. invasive SEEG monitoring is reserved for more complex cases of drug-resistant epilepsies, which have lower chances of seizure control even after resective surgery
<table>
<thead>
<tr>
<th>Study</th>
<th>Number of patients</th>
<th>Seizure free</th>
<th>Responders</th>
<th>Permanent neurological deficits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dimova 2017</td>
<td>23</td>
<td>4.4%</td>
<td>35%</td>
<td>4.4%</td>
</tr>
<tr>
<td>Bourdillon 2017</td>
<td>162</td>
<td>7%</td>
<td>48%</td>
<td>1.2%</td>
</tr>
<tr>
<td>Cossu 2015</td>
<td>89</td>
<td>18%*</td>
<td>28%</td>
<td>2.2%</td>
</tr>
<tr>
<td>Catenoix 2015</td>
<td>14</td>
<td>43%**</td>
<td>64%</td>
<td></td>
</tr>
</tbody>
</table>

*disproportionally large number of nodular heterotopias treated
**all patients had malformation of cortical development; 2 patients had seizure recurrence after 5 and 6 years (caution tapering AEDs)

- 41-49% of patients in whom conventional surgery was excluded benefited from RFTC
- in case of seizures recurrence, **RFTC can be repeated** but with uncertain outcome (in Catenoix 2015 series, only 1 of the 3 patients in whom the procedure was repeated became seizure free during 60 months).

**Predictors of favorable outcome after RF-TC**

1) positive MRI
2) MCD or nodular heterotopias
3) number of intralesional RFTC sites
   - some centers implant more SEEG electrodes (than needed just for mapping) in the areas of malformations of cortical development to maximize RFTC efficiency
   - N.B. for RFTC to be successful, need to implant electrodes densely (often during additional procedure)
4) (sub)continuous interictal epileptiform discharges at RFTC sites
5) focal low-voltage fast activity at seizure onset at RFTC sites (87.5% of patients were responders to RFTC in Catenoix 2015 study)
6) induction of seizures similar to spontaneous seizures by high-frequency electric stimulation at the RFTC target sites (in Catenoix 2015 series, all such patients were responders)
7) occipital location

**Alternatives to SEEG-guided RF-TC**

1. VNS (30–63% reduction of > 50% of seizures)
2. DBS of the anterior nucleus of thalamus (54% reduction of > 50% of seizures)
3. SRS
4. MRI-guided FUS
5. MRI-guided laser thermal ablation
   a) it is not possible to obtain an intracranial recording within the same stereotactic procedure in order to target the epileptogenic zone.
   b) multiple lesions in separate sites request the use of several probes, each of them being highly costly.
   c) size of the laser lesion can be larger than that achievable with RF-TC – useful in patients with transient improvement after RF-TC in whom laser might allow increase of the size of the lesion.

SEEG-guided RF-TC like no other stereotactic modality can record, stimulate, and ablate!
the aim of this study - to provide extensive data about efficacy and safety of SEEG-guided RF-TC.

162 consecutive patients with drug-resistant focal epilepsy treated at Pierre Wertheimer Neurological and Neurosurgical Hospital, Lyon University, France over 10 years (01/2003 to 12/2013).

the mean age was 28 ± 12 (SD) years (range 4–59); the mean disease duration was 24 ± 11 (SD) years (range 4–58).

55 patients had temporal lobe epilepsy; 107 had extra-temporal lobe epilepsy (frontal lobe in 46 patients, in the parietal lobe in 18, in the insular cortex in 8, and in the occipital lobe in 6); 22 patients had multifocal epilepsy; in 5 patients the origin remained unknown.

MRI characteristics were classified into four groups: MRI negative (55 patients), hippocampal sclerosis (26 patients), focal cortical dysplasia (44 patients), and other lesions.

61 / 162 patients were eligible to conventional epilepsy surgery

SEEG-guided RF-TC was performed:
   a) when the epileptic zone was accessible to RF-TC (procedure aimed at avoiding a conventional epilepsy surgery)
   b) when the epileptic network, although unilateral, was widely distributed or included eloquent or deep and inaccessible areas (palliative therapeutic option when surgery was not feasible).

follow-up and safety data were collected prospectively.

the outcome was evaluated at 2 months; if RF-TC did not provide results equivalent to those expected after a conventional surgery, including transient improvements, epilepsy surgery or Gamma-knife or repeat RF-TC was performed within the first year following SEEG (i.e. > 2 months after RF-TC) in 50 (27%) patients:
Patients undergoing SEEG-guided RF-TC at the end of the phase II investigation
N=162

First RF-TC outcome at 2 months
N=162

Second procedure
<1 year after RF-TC
N=50
• Surgery (n=47)
• Gamma-knife (n=1)
• 2nd RF-TC (n=2)

First RF-TC outcome at 12 months
N=112

N.B. 27% of patients benefited from another procedure within the year following RF-TC

Primary outcome - seizure freedom at 2 and 12 months after SEEG-guided RF-TC:
25% of patients were seizure-free at 2 months
7% of patients were seizure-free at 12 months cf. with VNS

Secondary outcomes:
• the responders' rate (patients with at least 50% decrease in seizure frequency in comparison with the 3-month period preceding SEEG):
  67% of responders at 2 months
  48% of responders at 12 months
  SEEG-guided RF-TC improves (decrease in seizure frequency ≥ 50%) epilepsy in
  50% of patients cf. with VNS

• long-term follow-up:
  58% of responders maintained their status during the long-term follow-up;
  the mean delay of epileptic status worsening was 3.5 years (from 2 to 6 years);
  at 10 years, 13% of patients remained responders. cf. with VNS

Kaplan-Meier analysis of patient responders at 1 year:
Factors affecting efficiency
- traditional factors known to be important affecting the results of epilepsy surgery (MRI data, epilepsy duration) as well as the number of coagulations during SEEG-guided RF-TC (directly related to the lesion volume) did not affect efficiency at 12 months.
- the seizure outcome was significantly better when the SEEG-guided RF-TC involved the occipital region - odds ratio 6.8237 to be a responder at 12 months (95% CI 1.4–65.5, p = 0.007).
  - hypothesis - probably due to the functional high risk of conventional surgery in this region, which leads to undertaking SEEG RF-TC on restricted epileptogenic zones.
- DNET/ganglioglioma (n = 6 patients): 83% seizure-free and 100% responders at 12 months.

Surgery following a SEEG-guided RF-TC - Predictive value of SEEG-guided RF-TC outcome at 2 months
- classical approach of the Franco-Italian school is to perform resective surgery some weeks after SEEG (vs. other teams perform surgery at the time of RF ablation)
- 91 patients underwent conventional epilepsy surgery after SEEG-guided RF-TC (5 patients had surgery even if they were seizure-free after SEEG-guided RF-TC; reason - the SEEG data suggested that the epileptogenic network largely exceeded the volume of the RF-TC lesion; it is no longer a practice at author’s institution):

<table>
<thead>
<tr>
<th></th>
<th>Favorable surgery outcome</th>
<th>Unfavorable surgery outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favorable initial RF-TC outcome</td>
<td>41</td>
<td>3</td>
</tr>
<tr>
<td>Unfavorable initial RF-TC outcome</td>
<td>26</td>
<td>21</td>
</tr>
</tbody>
</table>

- 93% of patients who were responders at 2 months after an SEEG-guided RF-TC, became Engel's class I or II after conventional epilepsy surgery
  
  *SEEG-guided RF-TC effect is a predictor (positive predictive value 93%) of outcome after conventional cortectomy in patients eligible for surgery*
Hypothesis – SEEG was on target, just RF lesion might have been too small. Expect good surgery result.

- being nonresponder appears to be unreliable indicator for predicting the outcome of surgery (negative predictive value 40%).

Hypothesis – SEEG electrode might have been placed off epileptogenic zone due to the wrong hypothesis on epileptogenic focus localization so that one electrode only (often located at the margin of the exploration field) was usable for RF-TC. Need a second, modified SEEG for further exploration

Side effects: 2 patients (1.1%) of permanent deficit (ankle palsy and hand palsy)* and 2.4% of transient side effects.

SEEG-guided RF-TC is a safe procedure (1.1% of permanent deficit)

*those were expected in 2 patients with a catastrophic evolution of their epilepsy and RF-TC was carried out despite motor symptoms provoked by test stimulation (with patient agreement).

- no patient showed any increase in seizure frequency.

- case series of 89 patients (49 male and 40 female; age range 2–49 years) who had SEEG and RF-TC in 2008-2013
  a) 46 cases (52%) were MRI-negative
  b) mean duration of epilepsy was 15.4 years (range 1–43 years)
  c) 22 patients (25%) were not eligible for resective surgery: EZ that involved highly eloquent areas (4) or nonlocalizing SEEG results (13) or patient refusal (5).

- number of implanted SEEG electrodes per pt 13.9 ± 2.6 (4–21)
- average of 10.6 (range 1–33) coagulations are placed per patient.
- mean volume of single RF-TC lesions (between 2 contiguous contacts), as measured on postcoagulation MR images, was 85.4 ± 31.1 mm³
- outcome:
  28.1% were significantly improved
  18% became seizure-free
• predictors of favorable outcome:
  1) nodular heterotopy (p = 0.0001)
  2) positive MRI (not significant)
  3) hippocampal sclerosis (not significant)
  4) patient's age (p = 0.02885)
  5) number of intralesional TC sites (p = 0.0271)

• severe permanent neurological deficits in 2 patients:
  1) an unexpected complex neuropsychological syndrome (severe impairments of reading, writing and calculation); patient is not seizure free, although a substantial decrease in seizure has occurred
  2) an anticipated permanent motor deficit (dense right hemiparesis); remained seizure-free for 3 years

Radiofrequency thermocoagulation of the seizure-onset zone during stereoelectroencephalography

*Petia Dimova, ††Luca de Palma, †Anne-Sophie Job-Chapron, †Lorella Minotti, §Dominique Hoffmann, and †Philippe Kahane

*Epilepsia, 58(3):381–392, 2017

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• retrospective review of 23 patients (aged 6–53 years) treated with SEEG-guided RF-TC (first patient treated in 2000).
• epilepsy started from 3 to 44 years before SEEG procedure
• 15 patients (65%) had negative MRI findings.
• 3 patients had undergone surgery previously without significant improvement.
• 10 patients were considered noneligible for resective surgery after SEEG:
  7 - involvement of eloquent cortex
  1 - bilateral SOZ
  2 - undefined SOZ (sz not recorded on SEEG) – ablation was guided based on interictal discharges and electrical stimulation results
in 12 patients ablated epileptogenic region was very close to eloquent cortices.

• Insertion of the electrodes was guided by a robotic arm (Neuromate, ISS, France) that was connected to the Talairach stereotactic frame.

• 7 to 17 (mean 13) multicontact SEEG electrodes were implanted per patient.

• in 9 patients (the 2000–2006 period), anatomic localization of the electrode contacts was identified on the postoperative teleradiography (frontal and lateral view) matched with Talairach and Tournoux stereotaxic atlas; after 2006, contacts were directly visualized on postimplantation MRI.

• 2-11 RFTCs per patient (mean 5).

• mean follow-up of 32 months (range 2–119)

• outcome:
  - 35% of patients were responders (≥ 50% decrease of seizure frequency) but only 1 patient (4.4%) became seizure-free
  - 6 of the 10 patients who were considered nonoperable, benefited from RFTC (2 had a ≥ 80% decrease of their seizure frequency).
  - 2 of 8 patients considered good candidates for curative surgery were sufficiently improved to defer surgery, meaning that a small subset of patients may avoid open surgery.
  - 65% of patients (n=15) did not benefit from RF-TC; 9 underwent surgery (surgery was curative in 5; with no statistically significant correlation with the presence or absence of an initial seizure-free period post-RFTC)

• presence of an MRI lesion was the only significant predictor of a positive outcome: 75% positive outcome in MRI+ group vs. 13.3% positive outcome in MRI- group

• permanent side effects: 1 patient (4.4%) had a permanent right thumb hypoesthesia.

• temporary adverse effects (without longterm deficits): intraventricular bleeding (1 pt), a transient increase of habitual seizures (2 pts), IPH (1 pt)

SEEG study (letters refer to the electrodes that sampled the right insular cortex. OF, orbitofrontal cortex; DL, dorsolateral frontal cortex; OP, suprasylvian opercular cortex; DL, dorsolateral frontal cortex; MT, mesiotemporal structures; T1, first temporal gyrus; T2, second temporal gyrus).

Interictal spikes and spike-and-waves are continuously recorded over a 4–5–6 contacts of electrode S and contacts 8–9 of electrode Y that are located in the superior part of the anterior long gyrus of the right insula.

Ictal discharges start at the same location, and are initiated by repetitive burst of polyspikes followed by low voltage fast activity.

Note the complete disappearance of spiking activity after the nine RFTC applied over the contacts S3–S4–S5–S6 and Y6–Y7–Y8–Y9–Y10:
Seizures Outcome After Stereoelectroencephalography-Guided Thermocoagulations in Malformations of Cortical Development Poorly Accessible to Surgical Resection

BACKGROUND: Radiofrequency thermocoagulation (RFTC) guided by stereoelectroencephalography (SEEG) has proved to be a safe palliative method to reduce seizure frequency in patients with drug-resistant partial epilepsy. In malformation of cortical development (MCD), increasing the number of implanted electrodes over that needed for mapping of the epileptogenic zone could help to maximize RFTC efficiency.

OBJECTIVE: To evaluate the benefit of SEEG-guided RFTC in 14 patients suffering from drug-resistant epilepsy related to MCD located in functional cortical areas or in regions poorly accessible to surgery.

METHODS: Ten men and 4 women were treated by RFTC. Thermolesions were produced by applying a 50-V, 120-mA current for 10 to 30 seconds within the epileptogenic zone as identified by the SEEG investigation.

RESULTS: An average of 25.8 ± 17.5 thermolesions were made per procedure. The median follow-up after the procedure was 41.7 months. Sixty-four percent of the patients experienced a long-term decrease in seizure frequency of >50%, of whom 6 (43%) presented long-lasting freedom from seizure. When a focal low-voltage fast activity was present at seizure onset on SEEG recordings, 87.5% of patients were responders or seizure free. All of the patients in whom electric stimulation reproduced spontaneous seizures were responders.

CONCLUSION: Our results show the good benefit-risk ratio of the SEEG-guided procedure for patients suffering from MCD in whom surgery is risky. This study identifies 2 factors, focal low-voltage, high-frequency activity at seizure onset and lowered epileptogenic threshold in the coagulated area, that could be predictive of a favorable seizure outcome after RFTC.

KEY WORDS: Epilepsy, Epilepsy surgery, Malformation cortical of development, Radiofrequency thermocoagulations, SEEG

Outcomes:
- 64% of the patients experienced a long-term decrease in seizure frequency of >50%
- 43% presented long-lasting freedom from seizure
- Of note, initially, almost all patients were responders or seizure free (94%), but this initial benefit disappeared in the first 6 months after RFTC in 35%

- Predictors of favorable outcomes

14 patients (17 to 59 years of age, mean duration of epilepsy 33.6 years, range 17-59 years) with drug-resistant epilepsy related to inoperable* malformation of cortical development (MCD):
- 9 cases of focal cortical dysplasia, 4 cases of nodular heterotopia, and 1 case of bilateral perisylvian pachygyria
  *located in functional cortical areas or in regions poorly accessible to surgery; with modern awake mapping it might be operable in some expert hands

- Number of intraloesional implanted electrodes was optimized* by use of a maximal number of electrodes targeting the lesion to achieve an optimal volume of thermolesion!
  *implanted more electrodes than necessary for mapping, so that electrodes covered the whole extent of the MRI lesion in all patients

- Average of 25.8 ± 17.5 thermolesions were made per procedure.
- The minimal follow-up was 1 year, the median follow-up was 41.7 months.
focal low-voltage fast activity - if present at seizure onset on SEEG recordings, 87.5% of patients were responders.
electric stimulation reproducing spontaneous seizures – all patients were responders.
• there was no correlation between responders' rate and any of the other factors tested (number of coagulations, cause or topography of the malformation)
• no permanent adverse effects

**BIBLIOGRAPHY** for ch. “Epilepsy and Seizures” → follow this [LINK](#)
Additional references for “SEEG”:
2. Lüders “Textbook Of Epilepsy Surgery” 1st ed. (2008), chapters 73, 103-104