Review Article

Anesthetic considerations for stereotactic electroencephalography implantation

Chakrabarti Rajkalyan, Anurag Tewari[1], Shilpa Rao[2], Rafi Avitsian[3]

Department of Anesthesiology, Newham University Hospital, Barts Health NHS Trust, London, [1]DBS and IONM, Evokes LLC, Mason, [3]Department of of Anesthesiology, Cleveland Clinic Foundation, Cleveland, Ohio, [2]Department of Neuro-Anesthesiology, Yale School of Medicine and Yale-New Haven Hospital, CT, USA

Abstract

The refractory seizures have significant impact on the quality of life and increase long term neurologic and non-neurologic complications. Implantation of Stereotactic Electroencephalography (SEEG) leads is one of the newer surgical techniques intended to localize seizure foci with higher accuracy than the conventional methods. Most of the commonly utilized anesthetic agents depress EEG waveforms affecting intra operative monitoring during these surgeries. Hence, the anesthetic goals include a stable induction and maintenance with agents which have minimal effect on EEG. This article discusses the peri-operative considerations of multiple anti-epileptic medications, recent advances in anesthetic management, and important post-operative concerns.

Keywords: Anesthesia, epilepsy surgery, intra-operative EEG, intra operative monitoring, refractory seizures, SEEG, seizure foci, stereotactic electroencephalography

Introduction

The stereotactic electroencephalography (SEEG) lead implantation is an invasive method of monitoring and localizing seizure foci in patients with drug resistant, focal epilepsies. It allows recording seizures with the aim of achieving three-dimensional analysis of the epileptogenic zone.[1] Though there are several articles on surgical details of SEEG there is paucity of literature discussing the anesthetic challenges related with SEEG.

History of Stereo-Electroencephalography (SEEG)

Du Bois-Reymond first demonstrated the action potential of nerves in 1848 and is also credited for describing the electrical activity of muscle, the first electromyography (EMG). The electrical activity of the brain was described in 1875 by Caton, while Han Berger (in 1928-29) was the first to obtain EEG traces from human brains. The first use of intra-operative EEG was by Foerster and Alternberger in 1935. Herbert Jasper and Wilder Penfield further developed this technique, using electrocorticography (ECoG) for localization and as a surgical treatment of epilepsy. They also achieved mapping of cortical functions by direct electrical stimulation.

Penfield and Jasper[2] were the first to record intra-operative EEG. After the development of stereotactic device by Spigel and Wycis in 1947,[3,4] Talairach and Bancaud[5-7] were first to use SEEG, using Stereotactic technique in 1950. Stereotactic localization of different cortical areas needs a statistically constructed proportional reference system where inter-commissural line, in contrasted ventriculography, acts as the foci or the reference point.[8] Stereotactic and stereoscopic tele-angiography provide an excellent definition of the anatomy

Address for correspondence: Dr. Chakrabarti Rajkalyan, Newham University Hospital, Barts Health NHS Trust, London.
E-mail: drrajkalyan@yahoo.co.in

Access this article online	
Quick Response Code:	Website: www.joacp.org
	DOI: 10.4103/joacp.JOACP_342_18

How to cite this article: Rajkalyan C, Tewari A, Rao S, Avitsian R. Anesthetic considerations for stereotactic electroencephalography implantation. J Anaesthesiol Clin Pharmacol 2019;35:434-40.

of the cerebral gyri and sulci,[9,10] thereby helping in planning avascular paths for placement of electrodes by means of a double grid mounted on a Talairach stereotactic halo.[11] Freehand implantation of depth electrodes has been reported to have acceptable accuracy.[12] A system without a stereotactic halo (known as a frameless system) can be used with the same precision and safety.[13,14] Song *et al*., in 2003, described a method for longitudinal implantation of electrodes in which the system without a stereotactic halo was combined with neuro-navigation guidance using neuro-endoscopy.[15]

In the early 1980s, tomography and digital angiography were used to locate targets. From the second half of the 1980s, magnetic resonance imaging replaced tomography and, in the middle of the 1990s, digital angiography was replaced by magnetic resonance digital angiographic imaging. To simplify the method and improve its accuracy, neuro-navigation systems were also introduced at that time. Guenot[16] and Almeida[17] used the capacity of SEEG to provide critical information that would support or contraindicate surgery.

Anesthesia for SEEG insertion also evolved gradually alongside the surgical technique but the main goal was always the same, not to interfere with intraoperative EEG monitoring. Thereby, over the anesthesiologists have strived to select appropriate drugs for induction and maintenance. Apart from standard monitoring for general anesthesia, different forms of intraoperative EEG monitoring were tried during these kinds of operations over the years. So far, none of the intraoperative monitoring has been proven to be superior to other.

Indication of Stereo-Electroence Phalography

SEEG is indicated in patients with medically refractory focal epilepsies who are amenable to surgical treatment. Collaboration of the results obtained from noninvasive preoperative investigations, particularly imaging and video-EEG examinations, does not always concur. Hence, invasive techniques like SEEG for recording seizures often must be used. In addition to the general criteria used for non-invasive monitoring,[18-22] additional specific criteria were considered in choosing SEEG instead of other methods of invasive monitoring. These criteria included: 1) The possibility of a deep-seated or difficult-to-cover location of the epileptogenic zone 2) The failure of a previous subdural invasive study to clearly outline the exact location of the seizure-onset zone; 3) The need for extensive bi-hemispheric explorations; and 4) A pre surgical evaluation suggestive of a functional network involvement (for example, the limbic system) in the setting of normal MRI findings.[23]

Surgical Techniques of SEEG Electrode Implantation

Prior to the surgery, a stereo-contrasted volumetric T1-weighted MRI sequence is performed. Images are then transferred to stereotactic neuro-navigation software, where trajectories are calculated. On the day of surgery, while the patient is under general anesthesia, the Leksell stereotactic frame is applied using standard technique.[23] Once the patient has been attached to the angiography table with the frame, stereotactic Dyna CT and 3D digital subtraction angiography are performed in some of the cases. The preoperative MR images, the stereotactic Dyna CT scans, and angiographic images are then digitally processed using a dedicated fusion software. These fused images are then utilized during the implantation to confirm the accuracy of the final position of each electrode and to guarantee the absence of vascular structures along the electrode insertion path. The desired target (s) is/are reached using commercially available depth electrodes with the help of conventional stereotactic technique. The electrode insertion progress is then observed under live fluoroscopic control in a frontal view to determine the straight trajectory of each electrode. Post implantation Dyna CT scans are obtained while the patient is still anesthetized and positioned on the operating table. The reconstructed images are then fused with the MRI data using the previously defined fusion software. The subsequent merged data sets are displayed and reviewed in axial, sagittal, and coronal planes, which allows confirmation that the electrodes have been appropriately placed.

Robotic SEEG placement

This technique involves the positioning of multiple electrodes in the brain executed accurately using the Renishaw neuromate ® surgical robot.[24] Gadolinium-enhanced MRI is used to determine the position of the surface vessels on the brain. Multiple trajectories are then planned on the robot software. The Leksell stereotactic frame is used for the preoperative localization. A reference CT data set is acquired in the frame, which is then fused to the preoperative MRI. The Leksell frame is then fixed to a standard frame holder attached to the robot. Care is taken to immobilize the operating table completely at this stage to avoid inadvertent movement between operating table and robot during the procedure. The robotic arm is now driven to each electrode position followed by puncturing of the skin with a sharp probe and use of a twist drill. An immediate postoperative CT scan is then acquired to compare each actual electrode position in relation to the planned trajectories. The Leksell frame is then disconnected from the robot and removed from the patient.

Pre-operative assessment

Anesthesiologists need to be cognizant of the fact that patients with intractable epilepsy, have usually been on long term, multi anti-epileptic medications. Thus, the effect of different anti-epileptic drugs on pharmacodynamics of anesthetics as well as their effect in combination of perioperative medications should be kept in mind and pre-operative investigations should be ordered accordingly. For example, Valproate can cause thrombocytopenia and platelet dysfunction, so it should preferably be stopped or changed to other medication as soon as the decision of surgery is taken.[25] Effective communication between primary team and anesthesiologist is necessary as modification of anti-epileptic medication could destabilize the patient. Decision to stop anti-epileptic medication on the day of the operation should also be discussed with primary team. In addition, drug interactions between different classes of medications also need to be considered. Anti-platelet medications (Aspirin, Clopidogrel) should be discontinued per guidelines before the surgery as intra cerebral hemorrhage is one of the main complications of this surgery. If the patient is on long term anti-coagulation therapy for Cardiac (Atrial Fibrillation) or some other reason (deep vein thrombosis, pulmonary embolism) the peri-operative anti-coagulation strategy should be discussed with the appropriate team to reach to an agreement.

Peri-operative monitoring

Standard monitoring should be established as per guideline of American Society of Anesthesiologists (ASA) and The Association of Anesthetists of Great Britain and Ireland (AAGBI) before induction. Invasive arterial blood pressure monitoring is not required unless patient's clinical history warrants one (e.g. unstable cardiac conditions etc.). Arterial line is normally established after the induction. The neuromuscular blockade may be monitored using a Peripheral nerve stimulator to evaluate the Train of Four ratio.

Anesthetic Goal and Peri-Operative Anesthetic Management

Anesthetic goals are as follows [Table 1].

Table 1: Anesthetic goals for Stereo-Electroencephalography (SEEG)

1	Smooth Induction and emergence
2	Maintain adequate cerebral perfusion pressure
3	To ensure absolute patient immobility
4	To cause least interference with intra-operative EEG monitoring
5	To enhance the chance of seizure detection
6	Treatment of any complications

Smooth induction and emergence

Standard induction with Propofol (2-2.5 mg/Kg), Fentanyl (1-2 mcg/Kg) and Rocuronium (0.6 mg/kg) or Vecuronium (0.1 mg/kg) is routine. If intra-operative EEG monitoring is planned, benzodiazepines are better avoided to decrease their effect on EEG suppression. Infusion of an opioid such as remifentanil (0.08-0.25 mcg/kg/min) from the beginning of the induction is preferred as it facilitates stable induction and blunts the sympathetic response due to laryngoscopy and skull pinning. If remifentanil infusion is not used, then bolus of Propofol or short acting Beta blocker like Esmolol can be used to blunt the sympathetic response. Some centers use Dexmedetomidine infusion for intra-operative maintenance, along with remifentanil, if EEG monitoring is planned. The typical dose is 0.5 mcg/kg/hr. The possible side effects that have been seen are bradycardia with bolus, dry mouth on waking up, and possible hypotension (especially in older and sicker patients).

Emergence needs to be smooth as well. Coughing or bucking on the Endotracheal tube can trigger a sympathetic response with tachycardia and hypertension, both of which are not desirable as it can lead to intra cerebral bleeding. If the patient was on opioid infusion intra-operatively, it needs to be turned off or reduced after the skull pin is removed. Awakening the patient on very low dose remifentanil infusion (0.01-0.02 mc/kg/min) is another option. This technique gives an opportunity to extubate after the patient is fully awake and obeying command without any sympathetic response. If remifentanil infusion is not a part of the anesthetic plan then emergence sympathetic response can be treated with short acting beta blocker Esmolol (1 mg/kg), both Alpha and beta blocker (Labetalol) and/or prophylactic use of IV Lidocaine (1.5-2 mg/Kg).

Maintain adequate cerebral perfusion pressure

The key in neurosurgical cases is to maintain adequate Cerebral Perfusion pressure. CPP is MAP - (ICP or CVP whichever is high). The goal of anesthesia is to maintain a normal CPP (70-90 mm of Hg). Usually monitoring of blood pressure may be required with arterial line insertion (optional) and MAP should be maintained around 70-75 mm of Hg with the help of fluid and vasopressors, if necessary. Neck position is important, as it may impede venous drainage from brain, leading to increase in the ICP and decrease in CPP.

Patient immobility

It is pertinent that "absolute" patient immobility is ensured while inserting the SEEG electrodes.[24] The robotic hand or stereotactic process both uses some fixed reference point of the patient. If the patient moves from the original position,

then the trajectory calculation could be erroneous, and the lead can reach to a wrong position. One of the strategies to achieve this is to initiate a muscle relaxant infusion after the induction. Rocuronium (0.3-0.6 mg/kg/hr) or Cis-atracurium (1-2 mcg/kg/min) can be a good choice for infusion. Patients on antiepileptics may need higher doses or frequent checking of Train of Four.

Intermittent doses of muscle relaxant can also be used but anesthesiologist needs to be vigilant in maintaining muscle paralysis with the help of peripheral nerve stimulator. It should be kept in mind that these patients are often on long term anti-epileptic therapy and often requires higher and frequent dosage of muscle relaxants as their metabolism is enhanced due to hepatic enzyme induction.[26] The chronic use of Phenytoin, Carbamazepine and barbiturates can shorten duration of action of amino-steroid Non-Depolarizing Muscle Relaxants. There are reports on Phenytoin induced Vecuronium resistance.[27]

Effect of anesthetics on intra-operative EEG monitoring

Propofol, thiopental, Isoflurane, sevoflurane and produce inhibition of $GABA_A$ receptors. Each anesthetic drug produces it own signature pattern in the EEG. Anesthetic drugs tend to either excite or depress the EEG, and they follow a pattern summarized by Winters.[28] Most agents produce an initial excitatory stage characterized by desynchronization (possibly loss of inhibitory synaptic function).[28] Amplitude increases as the EEG becomes synchronized, with a predominance of activity in the alpha range [Table 2]. Increasing doses cause progressive slowing until the EEG achieves burst suppression and, finally, electrical silence.[28] Hence titration of the anesthetic drugs should be done by observing the EEG and ensuring that it remains in optimal range (delta-alpha activity).

Intravenous anesthetic agents

Propofol produces dose-dependent depression of the EEG.[29] It can also enhance interictal activity in some patients, with production of burst suppression and electrical silence at high doses.

Etomidate can enhance epileptic activity at low doses (0.1 mg/kg) and may produce seizures in patients with epilepsy as do barbiturates such as methohexital.[28]

The benzodiazepine (BZD) produce frontal beta activity with a decrease in alpha activity at low doses. At higher doses, the BZD produce generalized slowing into the theta and delta range, without burst suppression.[29]

Barbiturates produce mild activation (fast activity) at low doses and a depressant effect leading to burst suppression and electrical silence at higher doses.[29] Of interest is that low-dose methohexital (0.5 mg/kg) has been used to enhance epileptic spike activity during ECoG in surgery to localize and remove seizure foci.[28]

Droperidol has little effect on the EEG when used alone.[29] When combined with fentanyl ("neurolept anesthesia"), droperidol increases EEG alpha activity at low doses.[28] At higher doses, it produces high-amplitude beta and delta activity.

The Ketamine produces high-amplitude theta activity in the EEG, with an accompanying increase in beta activity that appears to represent activation of thalamic and limbic structures. It has been reported to provoke seizure activity in persons with epilepsy but not in normal subjects.[28]

Inhalational agents

Induction with these agents (except desflurane) produces a shift in occipitally dominant alpha rhythm to the frontal region.[29] At anesthetic concentrations, increasing dose produces a reduction in frequency and amplitude, but the degree of depression in relationship to anesthetic depth varies between agents. The use of halogenated inhalational anesthetic agents during monitoring therefore depends on the methods monitored. For monitoring of cortical EEG, halogenated inhalational agents may need to be used in restricted concentrations (or total avoidance) However, if monitoring is done for seizure focus detection, most anesthetic drugs must be avoided since most will suppress seizure activity and prevent detection.

N_2O affects the EEG depending on the other agents being used with it. When used alone, it produces a frontally dominant high frequency (>30 Hz) activity.[29] When used with halogenated inhalational agents, it can be additive or antagonistic depending on the circumstances.[29]

The opioids produce a dose-related decrease in frequency of the EEG until in the delta range, while maintaining amplitude.[29] Some clinicians have found alfentanil useful in enhancing epileptic spikes.[29] Because the opioids do not produce marked suppression of EEG, they frequently are used during electrocorticography (ECoG) in surgery for seizure focus ablation. Remifentanil, a rapidly metabolized opioid, may be well suited for use by infusion, particularly during ECoG.[29] Because opioid anesthesia is often insufficient to produce sedation and lack of awareness, it is usually combined with an inhalational agent (halogenated or N_2O) or sedative drug.

Table 2: Anesthetic drugs and their effect on the Electroencephalogram

Drug	Effect on EEG Frequency	Effect in EEG Amplitude	Burst Suppression
Isoflurane			Yes, >1.5 MAC
Subanesthetic	Loss of α, ↑ frontal β	↓	
Anesthetic	Frontal 4- to 8-Hz activity	↑	
Increasing dose >1.5 MAC	Diffuse θ and δ → burst suppression→silence	↑→0	
Enflurane			Yes, >1.5 MAC
Subanesthetic	Loss of α, ↑ frontal β	↓	
Anesthetic	Frontal 7- to 12-Hz activity	↑	
Increasing dose >1.5 MAC	Spikes/spike and slow waves→Burst suppression; hypocapnia→Seizures	↑↑	
Halothane			Not seen in clinically useful dose range
Low dose	↑ Frontal 10- to 20-Hz activity	↓	
Moderate dose	Frontal 10- to 15-Hz activity	↑	
Increasing dose >1.5 MAC	Diffuse θ, slowing with increasing dose	↑	
Sevoflurane	Similar to equi-MAC	Similar to equi-MAC	Similar to equi-MAC
Desflurane	Similar to equi-MAC dose of isoflurane	Similar to equi-MAC dose of isoflurane	Yes, >1.2 MAC
Nitrous oxide (alone)	Frontal fast oscillatory activity (>30 Hz)	↑, especially with inspired concentration >50%	No
Barbiturates			Yes, with high doses
Low dose	Fast frontal β activity	Slight ↑	
Moderate dose	Frontal α frequency spindles	↑	
Increasing high dose	Diffuse δ → burst suppression→silence	↑↑↑→ 0	
Etomidate			Yes, with high doses
Low dose	Fast frontal β activity	↓	
Moderate dose	Frontal α frequency	↑	
Increasing high dose	Diffuse δ → burst suppression→silence	↑↑→ 0	
Propofol			Yes, with high doses
Low dose	Loss of α, ↑ frontal β	↓	
Moderate dose	Frontal δ, waxing-waning α	↑	
Increasing high dose	Diffuse δ → burst suppression→silence	↑↑→0	
Ketamine			No
Low dose	Loss of α, ↑ variability	↑↓	
Moderate dose	Frontal rhythmic θ	↑	
High dose	Polymorphic δ, some β	↑↑ (β is low amplitude)	
Benzodiazepines			No
Low dose	Loss of α, increased frontal β activity	↓	
High dose	Frontally dominant δ and θ	↑	
Opiates			No
Low dose	Loss of β, α slows	↔↑	
Moderate dose	Diffuse θ, some δ	↑	
High dose	δ, often synchronized	↑↑	

α = alpha (8-13 Hz) frequency; β = beta (>13 Hz) frequency; δ = delta (Note: Arrows indicate direction of change; number of arrows indicates the magnitude of change. Adapted from Black S, Mahla ME, Cucchiara RF. Neurologic Monitoring. In RD Miller. (ed). Anesthesia. New York: Churchill Livingston, 1994:1323

Muscle relaxants are generally believed to have no effect on the EEG.[29]

So, a balanced anesthetic technique is required for maintenance of anesthesia if intra operative EEG monitoring is planned. It is a delicate compromise between anesthetic depth and optimal condition for EEG monitoring. Maintenance of anesthesia is either done using TIVA or inhalational anesthesia or a combination of both techniques. The combination technique is preferable as it helps to keep the amount of both intravenous and inhalational anesthetic agents down. Inspired concentration of inhalational anesthetic agents often needs to be changed during EEG monitoring.

Techniques to enhance the chance of seizure detection

Since most anesthetics have a depressing effect on brain as well as EEG recording[29] the goal is to cause minimal interference with EEG recording with anesthetic agents and to provide a near awake state for the duration of monitoring. Different techniques

have been tried by different anesthesiologists. In our experience, maintenance is done by remifentanil infusion and volatile anesthetic (Sevoflurane or Isoflurane). Inhalational anesthetic concentration is preferably kept low (0.5-1 MAC). As below 1 MAC of volatile anesthetics, EEG monitoring is not obscured. Concurrent remifentanil infusion which has got a MAC sparing effect helps to prevent awareness. Satisfactory EEG recording is achieved in most of the patients with this technique, although a small number of patients require washing out the volatiles up to 0.2-0.3 MAC for the duration of monitoring. Low dose infusion of Ketamine (10 mg/hr.) has also been tried to enhance the chance of seizure detection for its epileptogenic property.[30] There is no evidence-based data available so far to support the use of Ketamine infusion to augment EEG recording.

Treatment of Complications

Apart from general complication of anesthesia this procedure has some unique complications. Most important complications are seizure during emergence and intra-cerebral hemorrhage which may manifest as inadequate awakening or seizure. Post -operative seizure is treated by bolus of midazolam or propofol. If the seizure is intractable even after treatment with midazolam and or propofol, other anti-epileptic medications are used. Airway should be protected, and re-intubation may be necessary if the patient is extubated at this point. The post-operative CT scan is useful if patient fails to wake up fully or started having intractable post-operative seizure.

Post-operative complications (Morbidity)

The morbidity rate reported by centers that use depth electrodes ranges from 1% to 5%.[13,14,16,31-35] Hemorrhage and/or infection are the most commonly seen perioperative morbidities. Limiting the number of electrodes to those that are necessary reduces the number of times that electrodes are passed through the brain and thus reduces the risk of hemorrhage. Some institutions use intravenous or oral antibiotics for their patients during the electrode implantation period. However, there are no convincing data to support this practice.[36] Others have not used antibiotics.[37] The risk of a cerebrospinal fluid fistula is reduced by ensuring an electrode exit point through a counter-opening in the skin, several centimeters from the electrode entry point in the cranium, with purse stitches around the exit. The great majority of infections are successfully treated by removing the electrode, together with the use of intravenous antibiotics. Cerebritis and abscesses are extremely rare.[38] Two cases of Creutzfeldt- Jakob disease have been reported,[39] and it is therefore important to avoid reuse of electrodes. Although extremely rare, there are reports from some series regarding patients who died because of implantation of depth electrodes.[34]

Anesthesia for extraction of electrodes

Implanted electrodes are removed when EEG from depth electrodes are obtained and exact 3-dimensional area of seizure focus is mapped. Both general anesthesia and sedation technique used successfully for electrode extraction. The choice of anesthesia depends on the anesthesiologist and patient profile (difficult airway, BMI, Systemic disease).

Future trend

Therapeutic use of depth electrode i.e. thermo-coagulation has been described in the literature.[40,41] It is also being used for deep cerebral stimulation, for example in relation to sub thalamic nuclei or mammillothalamic tract.[42,43] Depth electrodes may be coupled to probes for micro dialysis.[44] Therefore, there is an enormous future potential for increasing the applications of depth electrodes and SEEG, both for diagnostic use and for therapeutic use.[45]

Conclusion

The stereotactic electroencephalography lead placement is an evolving technique in diagnostic epilepsy surgery. Anesthesiologist also need to keep pace for providing anesthesia for this surgery keeping the basic principles of neuro-anesthesiology in place. This article is the first effort to see this procedure from an anesthetic point of view. Anesthesia for epilepsy surgery can be quite challenging, especially for the diagnostic and monitoring part. It is a fine balance between maintenance of a balanced anesthesia and to provide an adequate environment for seizure monitoring.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Centeno RS, Yacubian EM, Caboclo LO, Júnior HC, Cavalheiro S. Intracranial depth electrodes implantation in the era of image-guided surgery. Arq Neuropsiquiatr 2011;69:693-8.
2. Penfield W, Jasper H. Epilepsy and the Functional Anatomy of the Human Brain. Little Boston: Brown and Co; 1954. pp. 363-5.
3. Spiegel LA, Wycis HT, Marks M, Lee AI. Stereotaxic apparatus for operations on the human brain. Science 1947;106:349-50.
4. Spiegel LA, Wycis HT. Thalamic recordings in man with special reference to seizure discharges. Electroencephalogr Clin Neurophysiol 1950;2:23-39.
5. Talairach L, Bancaud J. Stereotaxic approach to epilepsy. Arch Neurol Psychiatry 1951;65:272-90.
6. Talairach J, Bancaud J. Stereotaxic approach to epilepsy. Methodology of anatomo-functional stereotaxic investigations. Progr Neurol Surg 1973;5:297-354.
7. Talairach J, Bancaud J, Szikla G, Bonis A, Geier S,

Vedrenne C. Approche nouvelle de la neurochirugie de l'epilepsie. Méthodologiesterotaxique et resultatsthérapeutiques. Neurochirurgie 1974;20(Suppl 1):S1-240.
8. Talairach J, Tournoux J. Co-Planar Stereotaxic Atlas of the Human Brain: 3-Dimensional Proportional System: An Approach to Cerebral Imaging. Stuttgart-New York: Georg Thieme Verlag; 1988.
9. Szikla G, Bouvier G. Hori T. *In vivo* localization of brain sulci by arteriography: A stereotactic anatomoradiological study. Brain Res 1975;95:497-502.
10. Szikla U, Bouvier G, Hori J, Petrov V. Angiography of the Human Brain Cortex. Berlin-Heidelberg-New York: Springer; 1977. p. 273.
11. Talairach J, Szikla G. Application of stereotactic concepts to the surgery of epilepsy. Acta Neurochir Suppl (Wien) 1980;30:35-54.
12. Davies KG, Philips BLB, Hermann BP. MRI confirmation of accuracy of freehand placement of mesial temporal lobe depth electrodes in the investigation of intractable epilepsy. Br J Neurosurg 1996;10:175-8.
13. Mehta AD, Labar D, Dean A, Harden C, Hosain S, Pak J, *et al.* Frameless stereotactic placement of depth electrodes in epilepsy surgery. J Neurosurg 2005;102:1040-5.
14. Murphy MA, O'Brien TJ, Cook MJ. Insertion of depth electrodes with or without subdural grids using frameless stereotactic guidance systems-technique and outcome. Br J Neurosurg 2002;16:119-25.
15. Song JK, Abou-Khalil, Konrad PE. Intraventricular monitoring for temporal lobe epilepsy: Report on technique and initial results in eight patients. J Neurol Neurosurg Psychiatry 2003;74:561-5.
16. Guenot M, Isnard J, Ryvlin P, Fischer C, Ostrowsky K, Mauguiere F, *et al*. Neurophysiological monitoring for epilepsy surgery: The Talairach SEEG method. Stereoelectroencephalography. Indications, results, complications and therapeutic applications in a series of 100 consecutive cases. Stereotact Funct Neurosurg 2001;77:29-32.
17. De Almeida AN, Olivier A, Quesney F, Dubeau F, Savard G, Andermann F. Efficacy of and morbidity associated with stereoelectroencephalography using computerized tomography or magnetic resonance imaging-guided electrode implantation. J Neurosurg 2006;104:483-7.
18. Cossu M, Cardinale F, Castana L, Nobili L, Sartori I, Lo Russo G. Stereo-EEG in children. Childs Nerv Syst 2006;22:766-78.
19. Cossu M, Cardinale F, Colombo N, Mai R, Nobili L, Sartori I, *et al*. Stereoelectroencephalography in the presurgical evaluation of children with drug-resistant focal epilepsy. J Neurosurg 2005;103(4 Suppl):333-43.
20. Jayakar P. Invasive EEG monitoring in children: When, where, and what? J Clin Neurophysiol 1999;16:408-18.
21. Najm IM, Bingaman WE, Lüders HO. The use of subdural grids in the management of focal malformations due to abnormal cortical development. Neurosurg Clin N Am 2002;13:87-92, viii-ix.
22. Widdess-Walsh P, Jeha L, Nair D, Kotagal P, Bingaman W, Najm I. Subdural electrode analysis in focal cortical dysplasia: Predictors of surgical outcome. Neurology 2007;69:660-7.
23. Gonzalez-Martinez J, Mullin J, Vadera S, Bulacio J, Hughes G, Jones S, *et al*. Stereotactic placement of depth electrodes in medically intractable epilepsy. J Neurosurg 2014;120:639-44.
24. Abhinav K, Prakash S, Sandeman DR. Use of robot-guided stereotactic placement of intracerebral electrodes for investigation of focal epilepsy: Initial experience in the UK. Br J Neurosurg 2013;27:704-5.
25. Barr RD, Copeland SA, Stockwell ML, Morris N, Kelton JC. Valproic acid and immune thrombocytopenia. Arch Dis Child 1982;57:681-4.
26. Naguib M, Lien CA. Pharmacology of muscle relaxants and their antagonists. In: Miller RD, editor. Miller's Anesthesia. 7th ed. Churchill Livingstone; London, UK 2009. p. 887.
27. Platt PR, Thackray NM. Phenytoin induced resistance to vecuronium. Anaesth Intensive Care 1993;21:185-91.
28. Rampil IJ. Electroencephalogram. In: Albin MA, editor. Textbook of Neuroanesthesia with Neurosurgical and Neuroscience Perspectives. New York: McGraw-Hill; 1997. p. 193-220.
29. Sloan TB. Anesthetic effects on electrophysiologic recording. J Clin Neurophysiol 1998;15:217-26.
30. Chakrabarti R, Avitsian R, Tewari A, Pal R. Anesthesia for Stereotactic Electroencephalograph Monitoring: A Case Series. J Neurosurg Anesthesiol 2014;26(4):456.
31. Cossu M, Chabardès S, Hoffman D, Lo Russo G. Presurgical evaluation of intractable epilepsy using stereoelectroencephalography methodology: Principles, technique and morbidity. Neurochirurgie 2008;54:367-73.
32. So N, Gloor P, Quesney LF, Jones-Gotman M, Olivier A, Andermann F. Depth electrode investigations in patients with bitemporal epileptiform abnormalities. Ann Neurol 1989;25:423-31.
33. Sansur CA, Frysinger RC, Pouratian N, Fu KM, Bittl M, Oskouian RJ, *et al*. Incidence of symptomatic hemorrhage after stereotactic electrode placement. J Neurosurg 2007;107:998-1003.
34. Cahan LD, Sutherling W, McCullough MA, Rausch R, Engel J Jr, Crandall PH. Review of the 20-year UCLA experience with surgery for epilepsy. Cleve Clin Q 1984;51:313-8.
35. Munari C. Depth electrode implantation at Höpital Sainte Anne, Paris. In: Engel JJ, editor. Surgical Treatment of the Epilepsies. New York: Raven Press; 1987. p. 583-8.
36. Wyler AR, Walker G, Somes G. The morbidity of long term monitoring using subdural strip electrodes. J Neurosurg 1991;74:734-7.
37. Olivier A, Marchand E, Peters T, Tyler J. Depth implantation at the montreal neurological institute and hospital. In: Engel J Jr, editor. Surgical Treatment of the Epilepsies. New York: Raven Press; 1987. p. 595-601.
38. Espinosa J, Olivier A, Andermann F, Quesney F, Dubeau F, Savard G. Morbidity of chronic recording with intracraniaI depth electrodes in 170 patients. Stereotact Funct Neurosurg 1994;63:63-5.
39. Wieser HG, Schwarz U, Blättler T, Bernoulli C, Sitzler M, Stoeck K, *et al*. Serial EEG findings in sporadic and iatrogenic Creutzfeldt-Jakob disease. Clin Neurophysiol 2004;115:2467-78.
40. Guenot M, Isnard J, Ryvlin P, Fischer C, Mauguière F, Sindou M. SEEG-guided RF thermocoagulation of epileptic foci: Feasibility, safety, and preliminary results. Epilepsia 2004;45:1368-74.
41. Kameyama S, Murakami H, Masuda H, Sugiyama I. Minimally invasive magnetic resonance imaging-guided stereotactic radiofrequency thermocoagulation for epileptogenic hypothalamic hamartomas. Neurosurgery 2009;65:438-49.
42. Dinner DS, Neme S, Nair D, Montgomery EB Jr, Baker KB, Rezai A, *et al*. EEG and evoked potential recording from the subthalamic nucleus for deep brain stimulation of intractable epilepsy. Clin Neurophysiol 2002;113:1391-402.
43. Khan S, Wright I, Javed S, Sharples P, Jardine P, Carter M, *et al*. High frequency stimulation of the mamillothalamic tract for the treatment of resistant seizures associated with hypothalamic hamartoma. Epilepsia 2009;50:1608-11.
44. Cavus I, Kasoff WS, Cassaday MP, Jacob R, Gueorguieva R, Sherwin RS, *et al*. Extracellular metabolites in the cortex and hippocampus of epileptic patients. Ann Neurol 2005;57:226-35.
45. Jones SE, Zhang M, Avitsian R, Bhattacharyya P, Bulacio J, Cendes F, *et al*. Functional magnetic resonance imaging networks induced by intracranial stimulation may help defining the epileptogenic zone. Brain Connect 2014;4:286-98.