Conference Proceeding

Intraoperative electrocorticography

Gabriela Alcaraz, Pirjo Manninen

Abstract

Intraoperative electrocorticography (ECoG) is the recording of electrophysiological activity from electrodes placed directly on the exposed surface of brain, during surgery for epilepsy and tumor resection. The ECoG is helpful in defining the seizure onset and spread within the cortical surface and delineation of the interface between epileptogenic zones and functional cortex substance of the brain. Intraoperative ECoG is an invasive procedure, it is performed during surgery mostly commonly during awake craniotomy but at times during general anaesthesia. As most anesthetic agents will affect ECoG, they should be minimized or stopped prior to any recording. Activation of intraoperative epileptiform activity may also be required if there are no spontaneous discharges. The appropriate management of the anesthetic during the time of ECoG is critical for its success. There are limitations and some controversies to all the uses of intraoperative ECoG, thus each center will set their own indications, criteria, and protocols.

Key words: Electrocorticography, epilepsy, neuroanaesthesia

INTRODUCTION

Intraoperative electrocorticography (ECoG) is the recording of electrophysiological activity from electrodes placed directly on the exposed surface of a brain, most commonly during the surgical treatment of epilepsy.[1-4] The first use of intraoperative ECoG recordings was performed by Foerster and Alternberger in 1935. In the late 1930s through the 1950s, Herbert Jasper and Wilder Penfield further developed this technique, using ECoG for localisation of epileptogenic focus during surgical treatment of epilepsy. Epilepsy surgery continues to be a well-established therapeutic intervention for patients with medically refractory seizures, but the success depends on the accurate localisation and complete removal of the epileptogenic zone.[3,4] The epileptogenic zone includes all the areas of brain that generate spontaneous epileptic seizures. Though there is some controversy, ECoG, an invasive technique, still plays an important role in the surgical treatment of patients with epilepsy. The effects of anaesthetic agents on intraoperative ECoG is an important consideration for the anaesthesiologist in caring for these patients.

Department of Anesthesia, Toronto Western Hospital, University Health Network, University of Toronto, Toronto, Ontario, Canada

Address for correspondence:
Dr. Pirjo Manninen, Department of Anesthesia, Toronto Western Hospital, 399 Bathurst Street, Toronto, Ontario M5T 2S8, Canada.
E-mail: pirjo.manninen@uhn.ca

Access this article online	
Quick Response Code:	Website: www.jnaccjournal.org
	DOI: 10.4103/2348-0548.199942

TECHNICAL ASPECTS

Neuronal activity in the brain gives rise to transmembrane currents that generate potentials which are measured in the extracellular medium.[5] The registry of these potentials when recorded from the scalp is referred to as the electroencephalogram (EEG), or as the ECoG when recorded directly from the surface of the cortex.[3,4] With scalp EEG recordings, the presence of high-resistance tissues (skull, meninges, skin) between the current source and the recording electrodes induces a distorting and attenuating effect. Thus, the spatial resolution of the EEG is very low (around 5–9 cm). Epileptiform

How to cite this article: Alcaraz G, Manninen P. Intraoperative electrocorticography. J Neuroanaesthesiol Crit Care 2017;4:S9-12.

discharges must involve the synchronised activity of a significant extension of the cortex (approximately 6 cm^2) to be detected by scalp EEG. Therefore, scalp EEG may be limited in its detection, precise localisation and determination of the extent of epileptogenic zones. As the ECoG records the cortical potentials directly from the surface or by depth electrodes, it bypasses the signal-distorting tissues. The spatial resolution of the recorded field is improved, providing a clearer view of activity that seen on scalp EEG.

Intraoperative ECoG requires a craniotomy to access the surface of the brain. The platinum–iridium or stainless steel electrodes are then placed directly on the surface or within the substance of the brain.[4] Rigid electrodes with ball-shaped tips known as Medusa or a Montreal frame may be used. These systems require individual placement of about 16–20 electrodes over the cortical region with attachments at the other end to a frame placed on the craniotomy site. These systems allow for the adjustment of the distance between the contacts. More recently, they have been replaced by flexible plastic grids or strip electrodes which are easily and quickly implanted within thin clear soft plastic sheets. The ECoG surface electrode recordings are helpful in defining seizure onset and spread within the cortical surface and delineation of the interface between epileptogenic zones and functional cortex. On the other hand, ECoG recording from deep structures such as the insula and amygdalo-hippocampal complex can be performed by 'depth electrodes'. These electrodes have recording contacts along their whole length and are inserted directly into the deep brain areas. These two systems can be considered complementary and the combination of both provides better three-dimensional ECoG recordings and stimulation data to guide the surgeon in performing more precise and safer resections. Decisions about the type and placement location of the implanted electrodes are usually based on previous non-invasive evaluation studies of the patient regarding the epileptogenic focus of their seizures.

The ECoG recordings are digitalised, displayed and recorded using computerised systems. Background ECoG recording represents basal cortical activity, and usually has large amplitude, ranging between 30 and 50 μV/mm. To ensure adequate capture of epileptiform discharges or activity, the frequency band-pass filters used are between 0.5 and 70 Hz. The waveform pattern of background ECoG varies with the locations of the electrodes, any pre-existing lesion and may be influenced by the presence of anaesthetic and sedative drugs.

APPLICATIONS AND INTERPRETATION

The ECoG has been largely used in epilepsy surgery to identify the location of the epileptogenic focus, map the extent of the irritative (area around the epileptogenic focus) zone, and to assess for the completeness of resection. The ECoG recording of epilepsy usually shows epileptiform potentials, which are sharp, transient and are different from the background activity. During a recording session, it may not be possible capture a spontaneous seizure, but spontaneous interictal epileptiform activities (IEAs) are most frequently seen.[4] These IEAs, also known as interictal spikes, are the recordings that usually arise from the irritative zone during the intervals between clinical seizures. They may be spikes, polyspikes, sharp waves, spikes-and-waves, sharp-and-slow wave complexes and/or any combination. The amplitude of the IEAs correlates directly with the proximity to the epileptogenic focus, thus helping in the identification and guiding the surgical resection of the latter. The IEAs are rarely seen in asymptomatic individuals, hence their presence has a high positive predictive value for the diagnosis of epilepsy.

In addition to the localisation of epileptogenic zones, intracranial electrodes for ECoG may be used during the mapping of eloquent brain function, both in epilepsy and tumour surgery.[6,7] The purpose of the brain mapping is to reliably identify cortical areas and subcortical pathways involved in motor, sensory, language and cognitive function. Such identification is important in neurosurgical oncology, where improvement in survival with greater tumour resection is weighted against functional loss. The distortion of cerebral topography from tumour mass effect, and the possibility of functional reorganisation through plasticity mechanisms, may reduce the reliability of the prediction of cortical sites based on classic anatomical criteria. The simultaneous recording of ECoG is used to identify the presence of spontaneous or stimulation-induced epileptic discharges, so-called 'afterdischarges', that can occur after electrical stimulation of the cortical areas. Confirming the absence of these 'afterdischarges' at the stimulation threshold which is defined as the lowest intensity that produces motor symptoms, helps to validate the cortical functional mapping. The ECoG also helps in the detection of electrographic or non-convulsive seizures and can prevent the development of intraoperative seizures. Another use of ECoG is to define the irritative cortex allowing for maximum resection of the lesion and epileptogenic tissue which improves seizure outcome in patients with brain tumours.[8] The most common complication that may occur during ECoG recording and stimulation for mapping is the onset of seizures. Intraoperatively, these seizures can be treated readily with application of a cold solution on the exposed cortex which then allows for continuation of the mapping.

ANAESTHESIA AND ELECTROCORTICOGRAPHY

Because intraoperative ECoG is an invasive procedure, the placement of the electrodes and monitoring is performed during surgery with anaesthesia. Ideally, pharmacologic agents used for during the ECoG testing phase of the procedure should have minimal depressant effects upon the electrical activity of the brain. Thus, an awake craniotomy is the best and the most commonly used technique, but ECoG can also be performed with general anaesthesia. During the awake craniotomy, analgesia and sedation may be administered with various techniques such as 'conscious sedation' or 'asleep awake asleep'. Whatever techniques and anaesthetic agents are used, the important principle for ECoG recording is to eliminate or minimise the drug effects prior to ECoG to ensure the ability to localise the epileptogenic focus and functional brain mapping, if required.

Neuroleptanalgesia (fentanyl and droperidol) has been the traditional method of sedation because neither drug affects intraoperatively recorded IEAs.[9] The emergence of drugs with shorter duration of action and easier titration of sedation has set aside this practice. After its introduction into anaesthesia, propofol[10,11] became the popular choice for sedation for awake craniotomy for both epilepsy and tumours when ECoG is required, and now dexmedetomidine is also commonly used.[12-14] These agents are usually combined with an opioid.[15-17] Many of the agents used have both pro- and anti-convulsant effects and may activate or suppress IEAs at different doses.[16,17] A summary of the effects and considerations for the most commonly used agents for awake craniotomy is shown in Table 1.

At times, general anaesthesia may be used for the entire surgery. To facilitate ECoG recordings, anaesthesia must be minimised or eliminated, with decreased levels of intravenous and/or volatile agents, even with the known consequent risk of intraoperative awareness. The use of muscle relaxant at the time of ECoG will also help to ensure that the patient does not move. As well, both volatile and intravenous anaesthetics may be used during the asleep phase of the 'asleep awake asleep' techniques. When the patient is awakened, it is important to ensure minimal levels of anaesthetic agents are present during ECoG.

Volatile anaesthetics can modulate neuroexcitability in a dose-dependent manner, manifested most prominently at near burst-suppression doses (1.5 minimum alveolar concentration [MAC]) and being minimal or absent at low doses (0.3 MAC).[18] This property is more prominent with sevoflurane and enflurane, whereas the epileptogenic potential of isoflurane, desflurane and halothane appears to be low. Nitrous oxide (N_2O), either alone or in combination with an inhaled drug (sevoflurane), depresses the interictal spike activity, but this controversial and N_2O is used in many centres routinely during intraoperative ECoG recordings.[19,20]

Activation of intraoperative interictal epileptiform spikes may be required if there are no spontaneous interictal discharges and this occurs more commonly in patients under general anaesthesia.[4] Accuracy of the localisation may also be improved with iatrogenic activation. Agents used for activation have included methohexital, propofol, enflurane, isoflurane, sevoflurane, N_2O, fentanyl, remifentanil and alfentanil.

Table 1: Effects of intravenous agents on the electrocorticography

Drug	Effects on ECoG	Considerations
Benzodiazepines	Potent anticonvulsants Suppress IEAs	Avoid for premedication
Propofol	Pro and anticonvulsant effects Variable responses at all dose ranges: May activate or suppress IEAs Pre-dominantly anticonvulsant activity with sedative doses	Stop infusion 15-20 min before the recording
Dexmedetomidine	Has the least effect on IEAs Low-dose infusion does not interfere with ECoG registry or cortical mapping	Stop or decrease concentration during ECoG Optimal dose 0.2 mcg/kg/h
Opioids Fentanyl, sufentanil, alfentanil, remifentanil Hydromorphone, morphine	No effect on IEAs with low-dose bolus or infusion Provide satisfactory conditions for surgery, ECoG and stimulation testing High bolus doses can cause increase in IEAs No significant proconvulsant activity No effect on ECoG discharges	All synthetic opioids equally good for ECoG Consider reducing dose of remifentanil during ECoG

ECoG=Electrocorticography, IEAs=Interictal epileptiform activities

CONCLUSION

The use of ECoG, which is an invasive electrophysiological technique of direct recording of the cortical potentials, is helpful to further delineate the regions of epileptogenic activity in patients during epilepsy surgery. The appropriate management of the anaesthetic during the time of ECoG is critical for its success. There are limitations and some controversies to all the uses of intraoperative ECoG. Each surgical centre will set their own indications, criteria and protocols.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Engel J Jr. Clinical neurophysiology, neuroimaging, and the surgical treatment of epilepsy. Review. Curr Opin Neurol Neurosurg 1993;6:240-9.
2. Enatsu R, Mikuni N. Invasive evaluations for epilepsy surgery: A review of the literature. Neurol Med Chir (Tokyo) 2016;56:221-7.
3. Tripathi M, Garg A, Gaikwad S, Bal CS, Chitra S, Prasad K, *et al.* Intra-operative electrocorticography in lesional epilepsy. Epilepsy Res 2010;89:133-41.
4. Chui J, Manninen P, Valiante T, Venkatraghavan L. The anesthetic considerations of intraoperative electrocorticography during epilepsy surgery. Anesth Analg 2013;117:479-86.
5. Buzsáki G, Anastassiou CA, Koch C. The origin of extracellular fields and currents – EEG, ECoG, LFP and spikes. Nat Rev Neurosci 2012;13:407-20.
6. Sanai N, Berger MS. Intraoperative stimulation techniques for functional pathway preservation and glioma resection. Neurosurg Focus 2010;28:E1.
7. Szelényi A, Bello L, Duffau H, Fava E, Feigl GC, Galanda M, *et al.* Intraoperative electrical stimulation in awake craniotomy: Methodological aspects of current practice. Neurosurg Focus 2010;28:E7.
8. Templer JW, Gavvala JR, Tate MC, Schuele SU. Reexamining the value of intraoperative electrocorticography during awake craniotomy. World Neurosurg 2016;91:655.
9. Archer DP, McKenna JM, Morin L, Ravussin P. Conscious-sedation analgesia during craniotomy for intractable epilepsy: A review of 354 consecutive cases. Can J Anaesth 1988;35:338-44.
10. Herrick IA, Craen RA, Gelb AW, McLachlan RS, Girvin JP, Parrent AG, *et al.* Propofol sedation during awake craniotomy for seizures: Electrocorticographic and epileptogenic effects. Anesth Analg 1997;84:1280-4.
11. Samra SK, Sneyd JR, Ross DA, Henry TR. Effects of propofol sedation on seizures and intracranially recorded epileptiform activity in patients with partial epilepsy. Anesthesiology 1995;82:843-51.
12. Souter MJ, Rozet I, Ojemann JG, Souter KJ, Holmes MD, Lee L, *et al.* Dexmedetomidine sedation during awake craniotomy for seizure resection: Effects on electrocorticography. J Neurosurg Anesthesiol 2007;19:38-44.
13. Ard JL Jr., Bekker AY, Doyle WK. Dexmedetomidine in awake craniotomy: A technical note. Surg Neurol 2005;63:114-6.
14. Talke P, Stapelfeldt C, Garcia P. Dexmedetomidine does not reduce epileptiform discharges in adults with epilepsy. J Neurosurg Anesthesiol 2007;19:195-9.
15. Gignac E, Manninen PH, Gelb AW. Comparison of fentanyl, sufentanil and alfentanil during awake craniotomy for epilepsy. Can J Anaesth 1993;40(5 Pt 1):421-4.
16. Modica PA, Tempelhoff R, White PF. Pro- and anticonvulsant effects of anesthetics (Part I). Anesth Analg 1990;70:303-15.
17. Modica PA, Tempelhoff R, White PF. Pro- and anticonvulsant effects of anesthetics (Part II). Anesth Analg 1990;70:433-44.
18. Watts AD, Herrick IA, McLachlan RS, Craen RA, Gelb AW. The effect of sevoflurane and isoflurane anesthesia on interictal spike activity among patients with refractory epilepsy. Anesth Analg 1999;89:1275-81.
19. Hosain S, Nagarajan L, Fraser R, Van Poznak A, Labar D. Effects of nitrous oxide on electrocorticography during epilepsy surgery. Electroencephalogr Clin Neurophysiol 1997;102:340-2.
20. Kurita N, Kawaguchi M, Hoshida T, Nakase H, Sakaki T, Furuya H. Effects of nitrous oxide on spike activity on electrocorticogram under sevoflurane anesthesia in epileptic patients. J Neurosurg Anesthesiol 2005;17:199-202.