Awake Craniotomy: Controversies, Indications and Techniques in the Surgical Treatment of Temporal Lobe Epilepsy

Ramesh L. Sahjpaul

ABSTRACT: In 1886, Victor Horsley excised an epileptogenic posttraumatic cortical scar in a 23-year-old man under general anaesthesia and discussed his choice of anaesthesia: "I have not employed ether in operations on man, fearing that it would tend to cause cerebral excitement; chloroform, of course, producing on the contrary, well-marked depression." His concerns regarding anaesthesia are reiterated 100 years later as evidenced by the ongoing controversy over the choice of anaesthetic in surgical procedures for epilepsy. The current controversies regarding the necessity for local anaesthesia in temporal lobe epilepsy operations concern the utility of electrocorticography in surgical decision making, its relationship to seizure outcome and the value of intraoperative language mapping in dominant temporal lobe resections. The increasing sophistication of pre-operative investigation and localization of both areas of epileptogenesis and normal brain function and the introduction of minimally invasive surgical techniques and smaller focal resections are changing the indications for local anaesthesia in temporal lobe epilepsy. Thus, indications which were previously absolute are now perhaps relative. This article reviews the current indications for craniotomy under local anaesthesia in the surgical treatment of temporal lobe epilepsy.

RÉSUMÉ: La crâniotomie sous anesthésie locale: indications et techniques dans le traitement chirurgical de l'épilepsie temporale. En 1886, Victor Horsley a excisé une cicatrice corticale post-traumatique épileptogène chez un jeune homme de 23 ans sous anesthésie générale et a discuté du choix de l'anesthésie: "je n'ai pas utilisé l'éther pour les interventions chez l'homme par crainte de causer de l'excitation cérébrale; le chloroforme, bien entendu, produit au contraire une dépression marquée." Ses inquiétudes concernant l'anesthésie sont réitérées 100 ans plus tard comme en témoigne la présente controverse sur le choix de la substance anesthésique pour la chirurgie de l'épilepsie. Les controverses actuelles concernant la nécessité d'une anesthésie locale dans la chirurgie de l'épilepsie temporale concernent l'utilité de l'électrocorticographie comme aide à la décision au cours de la chirurgie, sa relation aux résultats et sa valeur pour la cartographie du langage pendant l'intervention dans la résection d'un lobe temporal dominant. L'investigation préopératoire de plus en plus sophistiquée, la localisation des zones épileptogènes et des zones normales, l'introduction de techniques chirurgicales très peu effractives et les résections focales plus limitées changent les indications pour l'anesthésie locale dans l'épilepsie temporale. Ainsi, les indications qui étaient antérieurement absolues sont probablement relatives. Cet article revoit les indications actuelles de la crâniotomie sous anesthésie locale dans le traitement chirurgical de l'épilepsie temporale.

Can. J. Neurol. Sci. 2000; 27: Suppl. 1 – S55-S63

Awake craniotomy is performed using neurolept analgesia, a term first proposed by De Castro and Mundeleer in 1959 to describe a state of indifference and immobilization produced by the combined administration of the neuroleptic haloperidol and the narcotic analgesic phenoperidine.[1] Following numerous modifications, the combination of droperidol and fentanyl became the most widely used method for producing neurolept analgesia, which is distinguished from neurolept anaesthesia by the preservation of consciousness, although the two terms are often used interchangeably. The technique of awake craniotomy demands, perhaps more so than any other neurosurgical procedure, the utmost of cooperation between the surgical and anaesthesia teams. It is performed using a combination of local anaesthetic blockade of nerves innervating the scalp and dura using long acting anaesthetic agents, intraoperative sedation, and supplemental analgesia. This manuscript reviews the role of

From the Department of Clinical Neurological Sciences, Division of Neurosurgery, London Health Sciences Centre, London, ON, Canada.
Reprint requests to: R. Sahjpaul, Department of Clinical Neurological Sciences, Division of Neurosurgery, London Health Sciences Centre, 339 Windermere Road, London, ON, N6A 5A5 Canada

awake craniotomy in the surgical treatment of temporal lobe epilepsy (TLE) including its advantages and disadvantages, controversy surrounding its use, and describes the surgical technique used at London Health Sciences Centre in over 1200 cases.

HISTORICAL CONSIDERATIONS AND MODERN DILEMMAS

In 1886, Victor Horsley excised an epileptogenic post-traumatic cortical scar in a 23-year-old man under general anaesthesia.[2,3] In his report, Horsley discussed his choice of anaesthesia: "I have not employed ether in operations on man, fearing that it would tend to cause cerebral excitement; chloroform, of course, producing on the contrary well-marked depression." His concerns regarding anaesthesia are reiterated 100 years later as evidenced by the ongoing controversy over the choice of anaesthetic in surgical procedures for epilepsy. Trop et al[4] have summarized subsequent key developments. As early as 1928, Forster in Germany was using ether anaesthesia along with electrical stimulation of the cortex to reproduce the patient's seizure pattern and identify motor areas. Later Penfield extensively used local anaesthesia techniques along with cortical stimulation for epilepsy surgery at the Montreal Neurological Institute. Simultaneous developments in EEG, such as Berger's report on the EEG in man in 1929, Forster's recording of EEG from exposed cortex to identify epileptic foci, and pioneering work by Jasper and Gibbs on the necessity for electro-physiological recording to accurately localize epileptiform activity, played a crucial role in strengthening the argument for operating on patients free from the effects of general anaesthesia; the importance of a fully awake patient at the time of cortical stimulation and recording was felt to be crucial to the success of epilepsy surgery.[5,6] The current controversies regarding the necessity for local anaesthesia in TLE operations concern the utility of electrocorticography (ECoG) in surgical decision making, its relationship to seizure outcome, and the value of intraoperative language mapping in dominant temporal lobe resections. A question yet to be answered (and relevant in these days of budgetary constraints) is whether intraoperative ECoG and decision making is more cost-effective than standardized resections based on (usually lengthier) extraoperative ictal monitoring. Recent advances in neuroimaging such as fMRI and in surgical technology such as image guidance systems have fuelled these controversies. The variability in the choice of anaesthetic for epilepsy surgery is illustrated by the fact that of 46 centres surveyed, 23 used general anaesthesia in all cases, eight used local anaesthesia only when operating within the dominant hemisphere, 13 used local anaesthesia rarely and two used local anaesthesia exclusively.[7]

AWAKE CRANIOTOMY – ADVANTAGES

Advantages of using local anaesthesia for epilepsy surgery include optimization of intraoperative ECoG recordings (free from the effects of general anaesthesia) and the ability to perform accurate intraoperative functional cortical mapping. In addition to assisting in intraoperative clinical decision making, such information also adds to our understanding of neuroanatomy and neurophysiology.

Intraoperative ECoG

The techniques of local anaesthesia, ECoG, and tailored resections developed by Penfield and Jasper[8] almost 50 years ago are now commonplace.[7] However, anatomically uniform resections such as standardized anterior temporal lobectomy[9] and amygdalohippocampectomy[10] performed under general anaesthesia have also become popular and the necessity for ECoG has been questioned. General anaesthesia using drugs such as barbiturates, benzodiazepines, and volatile anaesthetics risks obscuring intraoperative ECoG spike activity and suppressing epileptiform activity, even at low to moderate drug dosages.[11,12] Propofol also induces dose dependent changes in the ECoG i.e. attenuation or obscuration of epileptiform activity at sedative doses.[13,14] Interestingly, activation by propofol of ECoG epileptiform activity and other neural excitatory phenomena has also been reported.[15,16,17] The utility of ECoG to plan resection is based in part on the premise that variability in the epileptogenic zone between individuals is sufficient to require "tailoring" the resection to the pathophysiology of the individual patient.[18] The argument for local anaesthesia and ECoG may be more valid in neocortical lesional TLE (where "standard" resections are not feasible) than in mesiobasal TLE eg. mesial temporal sclerosis. In the former situation lesionectomy alone provides good seizure control;[19] the controversy involves the need to resect surrounding cortex exhibiting interictal spikes on ECoG. Numerous studies[20-27] have failed to definitively answer this question although most report a trend towards better outcome with lesionectomy plus removal of epileptogenic cortex; this would seem to be an argument supporting the use of local anaesthesia (for optimal quality ECoG) in this situation. In contradistinction, seizure outcome in mesiobasal limbic TLE appears to be no better when comparing tailored resections using local anaesthesia and ECoG with

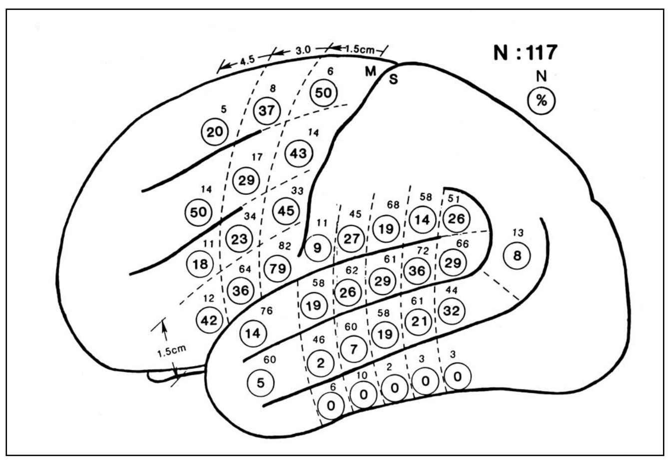
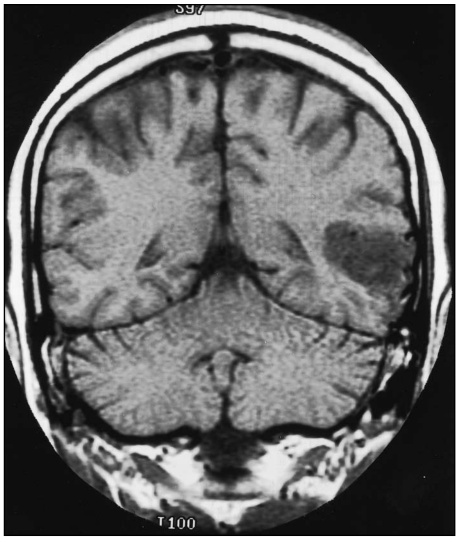


__Figure 1:__ Individual variability in left hemisphere language localization. The upper number in each zone refers to the number of patients with a language site in that zone and the lower number (in the circle) is the percentage of those patients with sites of significant evoked naming errors. Note the finding of language function in the anterior temporal lobe that would be resected in a standard anterior temporal lobectomy under general anaesthesia. (from Ojemann GA, Dodrill C. Verbal memory deficits after left temporal lobectomy for epilepsy: mechanisms and intraoperative prediction. J Neurosurg 1985;62:101-107 with permission.)

A



B

C

Figure 2. *(A) MR head of a left posterior temporal dysembryoplastic neuroepithelial tumour (DNET) causing intractable epilepsy. (B) intraoperative photograph showing left frontotemporoparietal exposure as seen by the surgeon with the patient's forehead to the right and left ear towards the top of the photograph. Electrical stimulation at sites E & G produced speech arrest in expected posterior perisylvian locations. Stimulation at sites H & J (arrows) produced speech arrest in an unexpected location in the basal lateral temporal lobe > 10 cm from the pole. Ictal and interictal abnormalities were concentrated anterior to the vein of Labbé and this area was resected completely (C) with preservation of sites H & J (arrows) without deficit. The patient remains seizure-free without language deficit 36 months later.*

standardized resections under general anaesthesia;[20-23] the argument for intraoperative ECoG becomes more tenuous.

Some aspects of ECoG data obtained under local anaesthesia may be useful in prognostication: Kanazawa et al[29] showed that frequent pre-resection hippocampal spikes correlated with a favourable outcome; Bengzon[30] reported that significantly more patients became seizure-free when interictal spikes were absent on post-resection ECoG; postresection insular spikes do not portend a worse outcome,[31] and have, in fact, been correlated with a favourable outcome;[29] and postresection posterior parahippocampal gyrus interictal spikes do not appear to be associated with an unfavourable outcome.[32] Thus, because the value of postresection ECoG appears to be primarily academic, the technique of terminal general anaesthesia[6] as a means of concluding an operation initiated with the patient awake may be useful if the procedure is prolonged or if the patient is particularly uncomfortable.

Functional mapping

This discussion will be limited to language and memory function, as sensorimotor or other functional mapping is irrelevant in TLE. Language localization has been shown to vary considerably among patients[33] and may be within the confines of a "standard" resection done under general anaesthesia (Figure 1). Ojemann et al[32] found that resections under general anaesthesia were more likely to produce a permanent mild postoperative dysnomia compared to those under local anaesthesia. With the patient awake and after preresection cortical mapping of language, the posterior resection line can be initiated, followed by subcortical resection while continuously monitoring the patient's language function, allowing intraoperative decisions to be made based on this real-time feedback (Figure 2). The surgeon is provided with an element of confidence that if language function is intact at the completion of the posterior cortical and subcortical resection line, it will return to this baseline regardless of any impairment throughout the remainder of the operation or in the postoperative period (barring misadventure). On the other hand, one cannot be certain whether a language deficit following a dominant temporal lobe resection under general anaesthesia will be transient or permanent. Thus, despite advances in fMRI and neuronavigation techniques that allow anatomic localization of brain function under general anaesthesia, real-time feedback regarding the patient's neurologic status remains a distinct advantage of the awake craniotomy procedure.

The value of intraoperative memory mapping is controversial

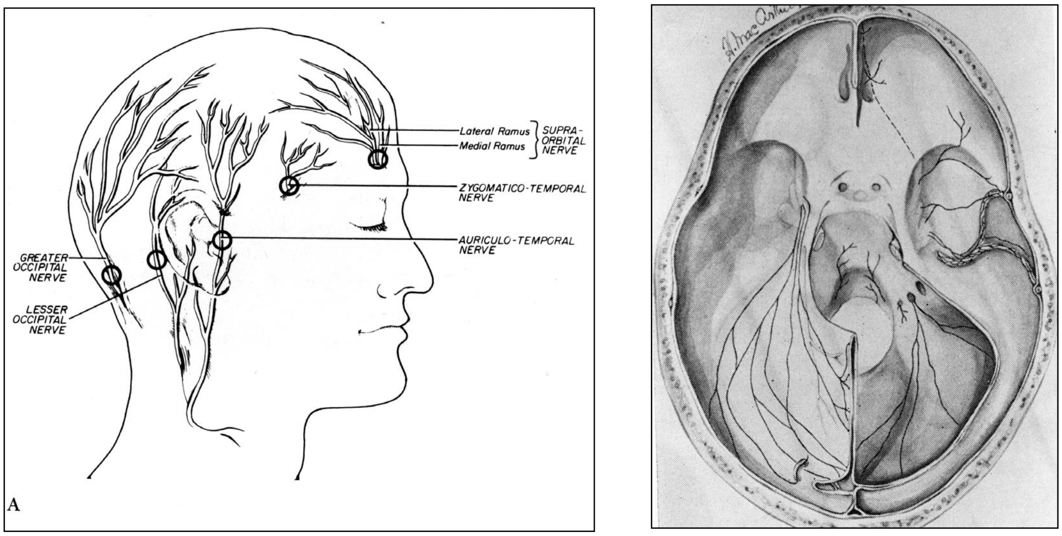


Figure 3. *Cutaneous nerve supply of the scalp (A) and dural innervation (B) as seen from above. Innervation of the tentorium, falx, and basal dura is illustrated. Note the concentration of nerves around the middle meningeal artery and adjacent dural blood vessels, and the anterior to posterior course of the nerves innervating the tentorium. These same nerves then course anteriorly along the falx. Knowledge of the origin and course of these nerves is essential for strategic blockade during surgery. (from Girvin JP. Neurosurgical considerations and general methods for craniotomy under local anesthesia. Internat Anesthesiol Clin 1986;24:89-113; and Feindel W, Penfield W, McNaughton F. The tentorial nerves and localization of intracranial pain in man. Neurology (Minneap) 1960;10:555 with permission.)*

and is summarized by Ojemman et al.[34] This testing is quite time consuming and demanding of the surgical team and the patient, and hence is generally restricted to those selected patients undergoing left temporal resections with very good pre-operative verbal memory and those who "fail" the memory portion of the Wada test with left carotid perfusion. However, the predictive value of intraoperative neocortical and hippocampal stimulation for memory is unclear. For example, stimulation of the left hippocampus through depth electrodes at a current below after discharge threshold rarely evokes disruption of performance on verbal memory measures and even extensive extra-operative lateral temporal cortical stimulation has failed to produce memory deficits.[34]

Other advantages of awake craniotomy

Regular use of the local anaesthesia technique for craniotomy enables neurosurgical and anaesthesia trainees to become thoroughly comfortable with the procedure so that when there is a strong indication for an awake procedure, such as operations in the vicinity of motor, language, or visual cortex, the procedure can be performed with ease, efficiency, and safety. It need not be a "high-stress" situation for either the surgical team or the patient. It is the opinion of the author that a mesiobasal temporal resection is technically the most difficult to perform under local anaesthesia due to the length of the procedure (especially if ECoG and cortical stimulation are performed) and the proximity to pain sensitive structures (tentorium, dura, mesiobasal leptomeninges). If neurosurgical trainees become facile with awake craniotomy techniques in TLE, then they should have no difficulty with awake craniotomy in other locations. Finally, the awake craniotomy procedure may reduce hospital length of stay compared to craniotomy under general anaesthesia,[35] although this issue has not been specifically addressed in TLE.

Awake craniotomy: complication avoidance and management

Craniotomy under local anaesthesia requires a significant amount of cooperation on the part of the patient. To minimize the risk of intraoperative complications, contraindications for this procedure at the author's centre include developmental delay, lack of maturity, an exaggerated or unacceptable response to pain, and a significant communication barrier. Candidacy for awake craniotomy is assessed on an individual basis after discussion with the patient and/or family. The youngest patient undergoing awake craniotomy for epilepsy at the author's centre was 11 years old (range 11-65 years).[35] Others have also reported on successful anaesthetic management during awake craniotomy in children.[36] A review of 222 patients undergoing awake craniotomy (primarily for TLE) at the author's institution revealed the following complications rates: confusion/disorientation/unwanted head movement (2.3%), severe nausea/vomiting (4.9%), intractable pain (7.7%), minor seizure (6.7%), major seizure affecting course of operation (2.3%), and conversion to general anaesthetic (0.9%).[37] Complications reported in the literature include seizures (16-18%), nausea/vomiting (8-50%), and requirement for general anaesthetic (2-6%).[38-40] The best way to manage intraoperative complications is obviously to avoid them by careful patient selection and meticulous surgical technique, which includes step-by-step anaesthetization of pain-sensitive structures, use of the lowest necessary stimulation currents, and expert neuroanaesthetic

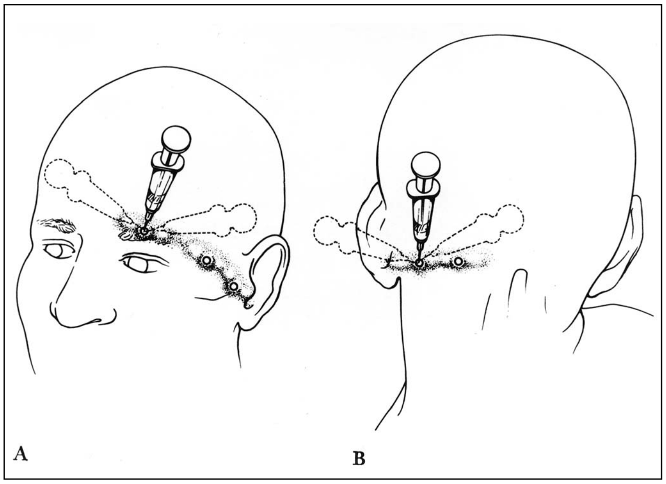


Figure 4. *Regional scalp blockade of supraorbital, auriculotemporal, greater and lesser occipital, and zygomaticotemporal nerves is done at least 20 minutes prior to surgery with approximately 2.5cc bupivacaine 0.5% with epinephrine 1:200,000 solution at each site. (from Girvin JP. Neurosurgical considerations and general methods for craniotomy under local anesthesia. Internat Anesthesiol Clin 1986;24:89-113 with permission.)*

management (see section on Surgical Technique). Avoiding excessive sedation minimizes confusion, disorientation, and unwanted head movement. Nausea and vomiting can be managed with dimenhydrinate and propofol. A spontaneous or induced seizure can usually be aborted with prompt topical application of ice cold saline over the involved cortex at the first symptom/sign of seizure, or with IV propofol. According to proponents of general anaesthesia, excessive pain associated with mesiobasal temporal resections limits the amount of mesial resection (consistently correlated with outcome)[22] that is possible under local anaesthesia. However, this has not been the experience at the author's centre nor that of others.[18]

Surgical technique at the London Health Sciences Centre

The technique used at this centre has been described in detail by Girvin[41] and the following is a brief summary.

Patient selection and preparation

Psychological preparedness is one of the most crucial aspects of operating on patients under local anaesthesia. The patient must have full confidence in the surgical and anaesthesiology teams. Both teams explain the procedure in detail to both the patient and his/her family, usually the day before surgery. A narrated video of the entire procedure is made available to patients should they wish to view it.

Scalp anaesthetization

The cutaneous nerve supply of the scalp and dural innervation are shown in Figure 3. A reverse question mark temporal scalp incision is utilized. A hemiscalp block is performed using approximately 2.5cc bupivacaine 0.5% with epinephrine 1:200,000 at each site. Injection is performed with a 1¼ inch 25 gauge needle. The regional block is done at least 20 minutes (and preferably 1-1.5 hours) prior to skin incision to allow maximal diffusion and infiltration of anaesthetic agent and to reduce potential toxicity. This regional block provides adequate anaesthesia for approximately 6-8 hours (Figure 4). The supraorbital nerve is located by palpation at the supraorbital notch. The greater occipital nerve can be identified by a small depression about 4-5 cm, lateral to the inion and just below the superior nuchal line. The auriculotemporal nerve just anterior to the tragus of the ear is blocked, taking care not to inject into the adjacent artery. Injection below the zygoma can cause transient facial paralysis and should be avoided. The lesser occipital nerve, being more variable than the greater occipital nerve in its course, can be blocked by injection just behind the ear over an anteroposterior distance of approximately 2 cm, with the anterior part of the injection encroaching on the posterior base of the pinna. This is crucial to block any superior twiglets of the great auricular nerve. The final injection is into the subcutaneous

Table: Dosing guidelines in awake craniotomy

	Intermittent Boluses		**Continuous**	
	Initial (µg/kg)	**Supplemental (µg/kg)**	**Infusions (µg/kg/min)**	**Patient-Controlled Sedation**
Neuroleptic				
Droperidol	15-40	15		
Sedative				
Propofol	300-500	300	25-75	Bolus:0.5 mg/kg Lockout: 3 min Basal infusion: 0.5 mg/kg/hr
Opioid				
Fentanyl	0.7	0.35	0.01	
Sufentanil	0.075	0.035	0.0015	
Alfentanil	7.5	3.5	0.5	

From Craen RA, Herrick I. Seizure surgery: general considerations and specific problems associated with awake craniotomy. Anesthesiol Clin North Amer 1997;15:655-672 with permission

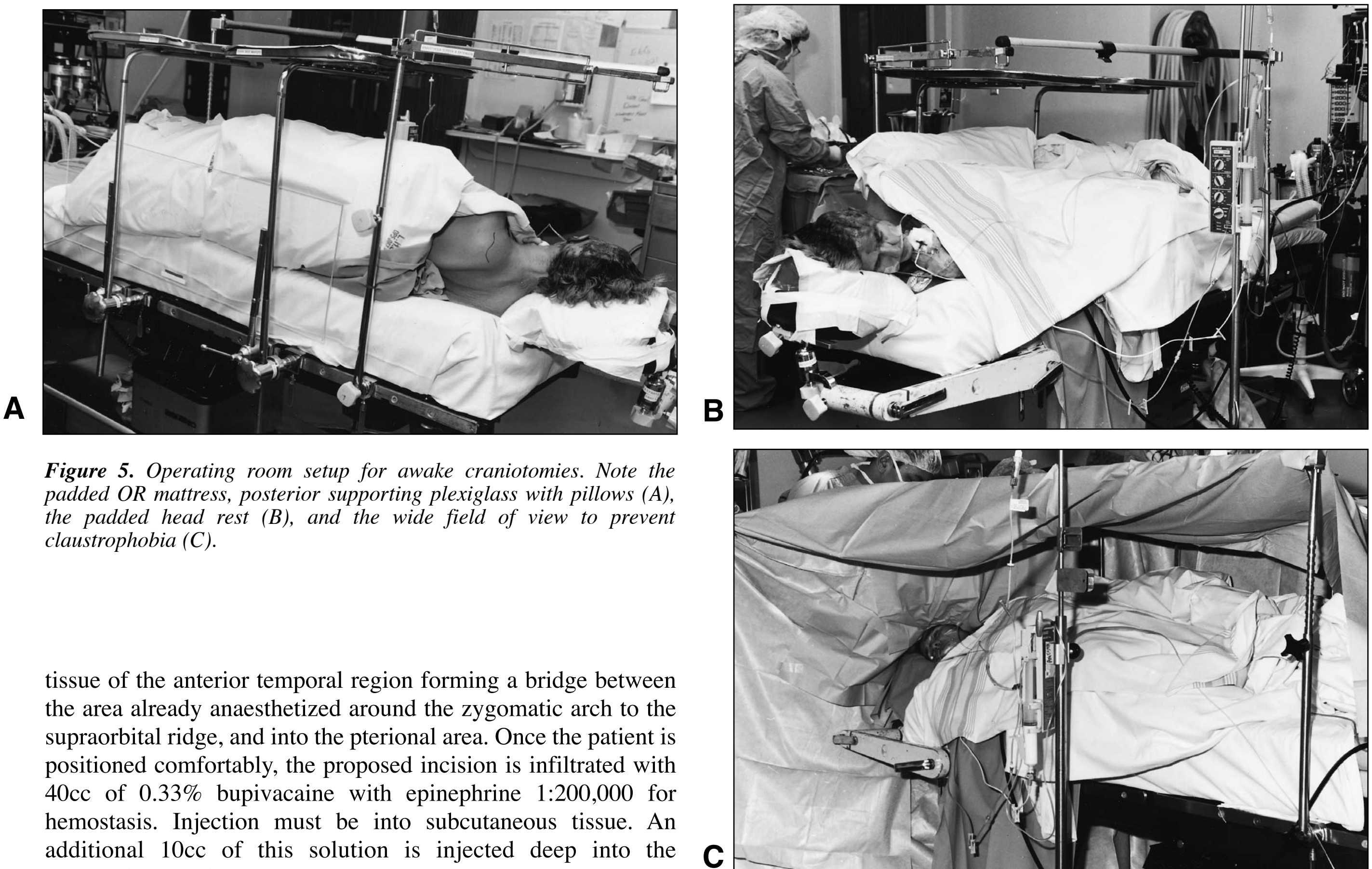


Figure 5. Operating room setup for awake craniotomies. Note the padded OR mattress, posterior supporting plexiglass with pillows (A), the padded head rest (B), and the wide field of view to prevent claustrophobia (C).

tissue of the anterior temporal region forming a bridge between the area already anaesthetized around the zygomatic arch to the supraorbital ridge, and into the pterional area. Once the patient is positioned comfortably, the proposed incision is infiltrated with 40cc of 0.33% bupivacaine with epinephrine 1:200,000 for hemostasis. Injection must be into subcutaneous tissue. An additional 10cc of this solution is injected deep into the temporalis muscle through to the bone. Using this technique and the drug concentrations and volumes indicated, a total bupivacaine dose of 225mg is administered, which is considered a safe total single dose (with epinephrine). The total dose may be adjusted up or down depending on patient size (we have often used up to 250mg bupivacaine without untoward effect).

Patient positioning and anaesthetic monitoring

Figure 5 demonstrates the operating room (OR) set up used at this centre for awake craniotomies. The key features are a soft foam head rest, a padded OR mattress, posterior supporting pillows held in place by plexiglass attached to the OR table, and a pillow between the patient's legs. Once draped, the patient has a large cone of view to allow greatest visibility to reduce claustrophobia. We always place a sign on the OR door that reads "Discretion Please – Patient Awake" to discourage nonessential personnel from entering the OR and to remind personnel that the patient is conscious and care should be taken regarding comments that might be made. Anaesthetic management consists of large bore IV access, oxygen by nasal prongs, noninvasive blood pressure monitoring, and pulse oximetery. Intravenous propofol and fentanyl/remifentanyl are the drugs of choice for patient sedation and analgesia at the author's centre[18] but other combinations such as propofol, midazolam and fentanyl or sufentanil are also effective.[35,38] For details regarding anaesthetic management, the reader is referred to reference 17 and the Table. Urinary catheterization is optional. Male patients can void with a urinal or failing that, can be catheterized in the lateral position. Generally, most patients can tolerate six to seven hours without

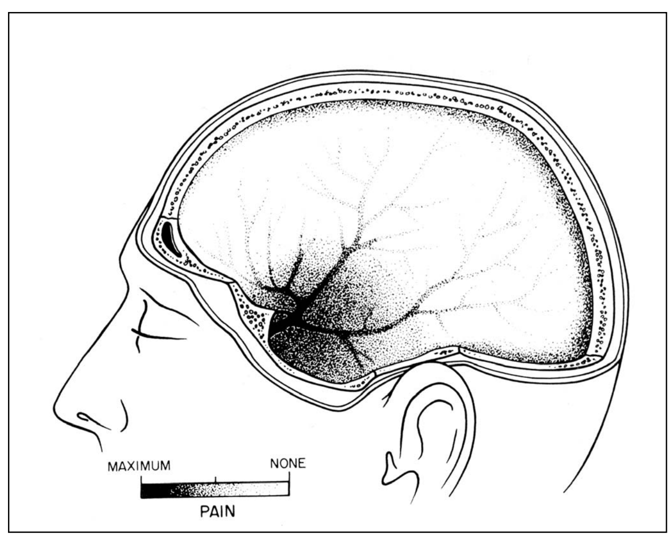


Figure 6. The pain-sensitive areas in the dura are shown. A small temporal craniectomy to expose and block dural nerves in this area is crucial to the rest of the procedure. (from Girvin JP. Neurosurgical considerations and general methods for craniotomy under local anesthesia. Internat Anesthesiol Clin 1986;24:89-113, with permission.)

voiding provided fluid administration is not aggressive. Surgical drapes are sewn in place around the scalp to discourage movement of the drapes and contamination of the operative field, and also to provide an added barrier against a patient who might reach up into the operative field if confused (extremely rare in our experience). Although some centres have placed awake patients in a 3-pin head rest,[35] we have never immobilized the head. Others have also reported satisfaction with leaving the patient's head free during surgery.[38,42,43] With careful attention to surgical technique and step by step infiltration of potentially painful structures throughout the procedure, discomfort can be kept to a minimum and unacceptable patient movement is uncommon (see section on Awake Craniotomy: Complication Avoidance and Management). Patients do appreciate the opportunity to shift their head, if ever so slightly, during surgery to increase their comfort. We often ask patients if they wish to adjust the head position slightly while holding onto the array post screwed into the skull adjacent to the bone-flap edge.

Craniotomy

With an adequate scalp block, temporalis infiltration, and hemostatic infiltration, the skin incision and temporalis fascial incision can be made without discomfort and minimal blood loss. The skin edges are packed with peroxide gauze for further hemostasis. As much of the temporalis muscle as possible is reflected anteriorly. If monopolar cautery is used during this stage, copious irrigation is necessary because of the intense heat generated (which causes considerable pain). Traditionally the craniotomy has been raised using a hand perforator and Gigli saw, but a power craniotome can be used provided appropriate precautions are taken prior to raising the bone flap. The first crucial step is performing a small temporal craniectomy to expose meningeal nerves entering the skull through the foramen spinosum and accompanying the middle meningeal artery to supply the dura (Figure 6). Using a small tuberculin syringe with a bent needle, a solution of 1% xylocaine with 0.25% bupivacaine is injected into the dural leaflets around the middle meningeal vessels which are usually clearly visible, effectively anaesthetizing the convexity dura. The bone flap can then be raised with ease. The patient should be warned of any clicking, biting, or drilling of bone before it occurs. A few dural nerves just above the zygoma may also need to be infiltrated. This is the most painful stage in the exposure of the brain and extreme caution must be taken to minimize discomfort lest the patient lose confidence at an early stage in the procedure, making it difficult to continue under local anaesthesia. The dura is opened in a cruciate fashion and the brain exposed.

Pre-resection ECoG

Retraction of the temporal lobe for placement of electrodes should be done with caution as this often causes discomfort. The tentorium should not be touched and bridging veins should not be manipulated as this translates into traction on the basal dura and tentorium which causes discomfort. Depth electrodes are placed 2.5 cm, and 4 cm from the temporal pole to correspond to the amygdala and hippocampus.

Cortical mapping

The parameters of stimulation are not critical and vary from centre to centre. We have used both constant voltage (extraoperatively) and constant current (intraoperatively) methods. We utilize a monopolar stimulator (manufactured on-site) at a frequency of 50 Hz, train duration 1-3 s, pulse duration 0.5 ms. A bipolar stimulator is preferred by some.[18,43] The least amount of current necessary to produce a response should be used. Stimulations are separated both temporally and spatially. Posterior speech localization requires high stimulus strengths, possibly five to six times that required for rolandic stimulation. Typical responses include speech hesitation or arrest, impairment of serial counting or naming, or anomia. Absence of a response should not be taken to mean the absence of function[18,35] and in such situations resection should proceed with extreme caution while maintaining constant vigilance over language function. There are many different language testing paradigms, but we have found simple counting (especially reverse counting) and object naming to be quite adequate. If there is any doubt, reading can be added. We typically engage the patient in conversation while the posterior resection line is being established.

After discharge thresholds

In discussions of the choice of the appropriate stimulation thresholds for the elicitation of neocortical function in the human brain, there is usually reference made to after discharge thresholds (ADT). It is a well known fact that any electrical stimulation of mammalian cortex can give rise to after discharges (AD), ie. local cortical discharges which outlast the actual period of stimulation. While these discharges usually disappear with a time course of a fraction of a second or very few seconds, some may linger, summate, and/or progress to reach epileptogenic thresholds. Because of this, the ideal is to keep the current of stimulation below the ADT. With the foregoing in mind, ADTs were utilized in order to determine a sub-AD threshold for cortical stimulation. However, the variability in the ADT from one site to another, and even within the same site was sufficient that the only proper way to determine the true ADT was psychophysically. The latter, requiring the use of a series of stimulation strengths, with intervening periods of non-stimulation, was such that 20 to 30 minutes was ideally required for the determination of the ADT at any given point. With the number of points utilized in stimulation, this became totally impractical. For this reason it has not been used at this centre in the last two decades.

Interestingly, the current practice of ADT at other centres does not follow these principles.[18,34] In addition, there is no convincing data to suggest that language outcome is statistically clinically superior after resections guided by ADT. Such a study would be very difficult to do retrospectively because of the numerous confounding variables, and patient matching would be practically impossible. A prospective trial would be more feasible but the number of patients required to show a significant difference in language outcome with the use of ADT compared to resection done without ADT would certainly be very large. Nevertheless, this is an area of potential future research in terms of a prospective trial and is under consideration at this centre.

Resection

Once the posterior resection line has been determined (based on cortical stimulation responses +/- gyral anatomy) the neocortical resection can begin. The anterior temporal lobe is removed en bloc using subpial technique. Manipulation of middle cerebral vessels can be painful and any contact should be avoided during superior temporal gyrus resection, as should contact with, or coagulation of the dura in the temporal fossa, because this is quite painful. Once the neocortical resection is completed the mesial structures can be removed en bloc taking care to avoid traction on the mesial leptomeninges or tentorial edge; if there is discomfort at this stage, (usually a poorly localized retroorbital headache resistant to IV analgesia), injection of xylocaine along the tentorium or into the region of the gasserian ganglion usually suffices. In most cases of mesial temporal sclerosis the posterior hippocampus should be resected to at least the level of the cerebral peduncle which corresponds to a specimen length of approximately 3 to 4 cm in anterior-posterior dimensions. In a comfortable patient, supplemental IV analgesia/sedation can be kept to a minimum, avoiding potential confusion or disorientation and associated movement, thus allowing use of the operating microscope if necessary. A postresection ECoG can then be obtained if desired.

Closure of craniotomy

Closure of the craniotomy is relatively comfortable for the patient but supplemental analgesia and sedation can also be administered by the anaesthetist. If timed appropriately, the patient is usually awake and alert enough to help themselves transfer from the OR table to the stretcher as soon as the head dressing is applied.

Patient satisfaction with awake craniotomy

We have not critically addressed this issue, but Danks et al,[38] in their small series of 21 consecutive patients, reported that all patients were entirely comfortable with the experience and there were no indications of adverse psychological sequelae of the event. Our experience certainly supports this.

Image guided surgery and awake craniotomy

Use of frameless stereotaxy in surgery for temporal lobe epilepsy under general anaesthesia has been described.[44] Image guided surgery systems have now been modified to facilitate their application in awake craniotomy. The Stealth System Treatment Guidance Platform (Medtronic, Sofamor Danek) utilizes a dynamic reference array which can be attached to the cranium to allow continuous optical tracking of registered surgical instruments regardless of head position. With further technical refinement such surgical adjuncts will undoubtedly become commonplace and invaluable in intraoperative lesion localization and resections.

Conclusions

Advantages of awake craniotomy for the surgical treatment of TLE include superior quality ECoG (which may be more helpful in neocortical rather than limbic TLE), optimal functional language mapping, and the ability to continuously monitor the patient's neurological function during the resection. Indirect academic benefits include the opportunity to compile neuroanatomical and neurophysiological data and the training of neurosurgical and anaesthesia personnel in the technique of awake craniotomy. Disadvantages and complications such as patient movement, intractable pain, nausea, vomiting, and seizures can be minimized by careful patient selection and strict attention to surgical and anaesthetic detail.

Acknowledgements

The author is indebted to Dr. John P. Girvin for his ongoing mentorship and invaluable assistance in the preparation of this manuscript.

References

1. De Castro J, Mundeleer P. Anesthesie Sans Barbituriques: La Neuroleptanalgesie Anesthesie Analegsie 1959;16:1022.
2. Horsley V. Brain Surgery. Br Med J 1886;2:670.
3. Taylor DC. One hundred years of epilepsy surgery: Sir Victor Horsley's contributions. In: Engel J Jr, ed. Surgical Treatment of the Epilepsies. New York: Raven Press, 1987;11.
4. Trop D, Olivier A, Dubeau F, Jones-Gotman M. Seizure surgery. Anesthetic, neurologic, neurosurgical, and neurobehavioral considerations. In: Albin MS, ed. Textbook of Neuroanesthesia with Neurological and Neurosciences Perspectives, New York: McGraw-Hill, 1997; 643-696.
5. Penfield W. Combined regional and general anesthesia for craniotomy and cortical exploration. Part I. Neurosurgical considerations. Curr Res Anesth Analg 1954; 33:145-155.
6. Pasquet A. Combined regional and general anesthesia for craniotomy and cortical exploration. Part II. Anesthetic considerations. Curr Res Anesth Analg 1954; 33:156-164.
7. Spencer DD, Ojemann GA. Overview of therapeutic procedures. In: Engel J Jr. ed. Surgical Treatment of the Epilepsies. New York: Raven Press, 1987:455.
8. Penfield W, Jasper H. Epilepsy and the Functional Anatomy of the Human Brain. Boston: Little, Brown, 1954;519-538.
9. Falconer M. Anterior temporal lobectomy for epilepsy. In: Logue V, ed. Operative Surgery. Vol 14: Neurosurgery. London: Butterworth, 1971;142-149.
10. Wieser H, Yasargil M. Selective amygdalo hippocampectomy as a surgical treatment of mesiobasal limbic epilepsy. Surg Neurol 1982;17:445-447.
11. Herrick I. Seizure activity and anesthetic agents and adjuvants. In: Albin MS, ed. Textbook of Neuroanesthesia with Neurological and Neurosciences Perspectives, New York: McGraw-Hill, 1997;625-642.
12. Modica P, Tempelhoff R, White P. Pro- and anticonvulsant effects of anesthetics (Parts I and II). Anesth Analg 1990;70:303-315 and 433-444.
13. Drummond J, Irague-Madoz V, Alksne J, Kalkman C. Masking epileptiform activity by propofol during seizure surgery. Anesthesiology 1992;76:652-654.
14. Rampil I, Lopez C, Laxer K, Barbaro N. Propofol sedation may disrupt interictal epileptiform activity from a seizure focus. Anesth Analg 1993;77:1071-1073.
15. Orser B. Propofol-induced neuroexcitation and receptor desensitization. Can J Anesth 1994;41:366-371.
16. Smith M, Smith S, Scott C, Harkness W. Activation of the electrocorticogram by propofol during surgery for epilepsy. Brit J Anesth 1996;76:499-502.
17. Craen RA, Herrick I. Seizure surgery: general considerations and specific problems associated with awake craniotomy. Anesthesiol Clin North Amer 1997;15:655-672.
18. Ojemann GA. Intraoperative tailoring of temporal lobe resections. In: Engel J Jr. ed. Surgical Treatment of the Epilepsies. New York: Raven Press, 1987;481-488.
19. Fried I, Cascino G. Lesional surgery. In: Engel J Jr. ed. Surgical Treatment of the Epilepsies. New York: Raven Press, 1987;501-509.
20. Hermann B, Wyler A. Comparative effects of dominant temporal lobectomy under general or local anesthesia: language outcome. Epilepsia 1988;29:668.
21. Hermann B, Wyler A. Effects of anterior temporal lobectomy on language function: a controlled study. Ann Neurol 1988;23:585-588.
22. Dodrill CB, Wilkus RJ, Ojemann GA, et al. Multidisciplinary

prediction of seizure relief from cortical resection surgery. Ann Neurol 1986;20:2-12.

23. Wyler AR, Hermann BP. Comparative results of temporal lobectomy under local or general anesthesia: seizure outcome. J Epilepsy 1988;1:121-125.
24. Awad IA, Rosenfeld J, Ahl J, et al. Intractable epilepsy and structural lesions of the brain: mapping, resection strategies, and seizure outcome. Epilepsia 1991;32:179-186.
25. Berger MS, Ghatan S, Haglund MM, et al. Low grade gliomas associated with intractable epilepsy: seizure outcome utilizing electrocorticography during tumor resection. J Neurosurg. 1993; 79(1):62-69.
26. Rasmussen T. Surgery of epilepsy associated with brain tumors. In: Purpura DP, Penry JK, Walter RD eds. Neurosurgical Management of the Epilepsies. New York: Raven Press; 1975:227-239. (Advances in Neurology; vol. 8.)
27. Wyllie E, Luders H, Morris HH, et al. Clinical outcome after complete or partial cortical resection for intractable epilepsy. Neurology 1987;37:1634-1641.
28. Yeh H, Kashiwagi S, Tew JM, Berger TS. Surgical management of epilepsy associated with cerebral arteriovenous malformations. J Neurosurg 1990;72:216-223.
29. Kanazawa O, Blume WT, Girvin JP. Significance of spikes at temporal lobe electrocorticography. Epilepsia 1996;37:50-55.
30. Bengzon A, Rasmussen T, Gloor P, et al. Prognostic factors in the surgical treatment of temporal lobe epilepsies. Neurology 1968;18:717-731.
31. Rasmussen T. Surgical treatment of patients with complex partial seizures. Adv Neurol 1975;11:415-449.
32. Ojemann L, Ojemann G, Baugh-Bookman C. What is the optimal extent of the medial resection in anterior temporal lobe epilepsy? Epilepsia 1986;27:636.
33. Ojemann GA, Dodrill C. Verbal memory deficits after left temporal lobectomy for epilepsy: mechanisms and intraoperative prediction. J Neurosurg 1985;62:101-107.
34. Ojemann GA, Sutherling WW, Lesser RP, et al. Cortical stimulation. In: Engel J Jr ed. Surgical Treatment of the Epilepsies. New York: Raven Press, 1987; 399-414.
35. Taylor MD, Bernstein M. Awake craniotomy with brain mapping as the routine surgical approach to treating patients with supratentorial intraaxial tumors. J Neurosurg 1999;1:35-41.
36. Tobias JF, Jimenez DF. Anaesthetic management during awake craniotomy in a 12-year-old boy. Paediatr Anaesth 1997;7(4): 341-344.
37. Sahjpaul RL, Girvin JP. Craniotomy under neurolept anaesthesia – indications and techniques (abstract). Can J Neurol Sci 1998; (25) (Suppl. 2)
38. Danks RA, Rogers M, Aglio LS, et al. Patient tolerance of craniotomy performed with the patient under local anaesthesia and monitored conscious sedation. Neurosurg 1998;42(1):28-36.
39. Archer DA, McKenna JMA, Morin L, et al. Conscious-sedation analgesia during craniotomy for intractable epilepsy: a review of 354 consecutive cases. Can J Anaesth 1988;35:338-344.
40. Gignac E, Manninen PH, Gelb AW. Comparison of fentanyl, sufentanil, and alfentanil during awake craniotomy for epilepsy. Can J Anaesth 1993;40:421-424.
41. Girvin JP. Neurosurgical considerations and general methods for craniotomy under local anesthesia. Internat Anesthesiol Clin 1986;24:89-113.
42. Black PMcL, Ronner SF. Cortical mapping for defining the limits of tumor resection. Neurosurg 1987;20:914-919.
43. Berger MS. Lesions in functional (“eloquent”) cortex and subcortical white matter. Clin Neurosurg 1993;41:444-463.
44. Olivier A, Olonso-Vanegas M, Comeau R, et al. Image guided surgery of epilepsy. Neurosurg Clin NA 1996;7(2):229-243.
45. Feindel W, Penfield W, McNaughton F. The tentorial nerves and localization of intracranial pain in man. Neurology (Minneap) 1960;10:555.