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Awake craniotomy

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Introduction

Over the past ten years the resection of brain lesions in awake patients has become an increasingly frequent procedure, even in young patients [1]. The indications have extended from lesions located in eloquent brain areas and epilepsy surgery to less specific procedures with no functional goal, in an attempt to reduce the side-effects of general anaesthesia [2, 3], facilitate early postoperative neurological evaluation, hasten recovery and shorten hospital stay [4]. This technique has several specific objectives and can be achieved through various means [5]. Success requires rigorous patient selection and meticulous preparation. All these aspects will be reviewed in this lecture.

Current indications for awake craniotomy

Classically, an awake craniotomy is indicated when the patient's co-operation is needed during the neurosurgical procedure. Since the introduction of awake craniotomy by Horsley at the end of the 19th century, and its later use by Penfield for the surgical treatment of intractable epilepsy [6, 7], the indications have enlarged and can be classified into four categories [8]. The first category includes procedures necessitating cortical mapping while avoiding any interference from the anaesthesia agents on the electrophysiological recordings. The second category includes the resection of lesions located close to, or in, functionally essential motor, cognitive or sensory cortical areas. The third refers to those procedures necessitating the obliteration or resection of vascular lesions associated with the vascular supply to functionally important areas. The fourth category has no functional goal and encompasses minor intracranial procedures and is undertaken to facilitate faster recovery and earlier discharge [4].

The decision to perform an awake craniotomy depends on surgical and patient considerations (Table 1). The first element in the decision is a surgical dilemma: a generous resection of brain tissue lessens the probability of recurrence and increases survival, but also increases the risk of major functional deficits that may severely affect the patient's quality of life [9]. Although techniques such as intra-operative motor or sensory-evoked responses [10] and neuro-navigation may help preserve neurological function, they do not outperform direct awake functional testing in delineating the tissue to be removed. The risk-benefit balance of awake craniotomy also depends on the risks associated with the patient himself, that may compromise surgery or be life threatening: airway patency and the risk of obstructive apnoea, severity and frequency of seizures, propensity for nausea and vomiting, the intracranial pressure (ICP), propensity for bleeding, the patient's willingness to cooperate, anxiety and discomfort associated with the procedure, and the presence of abnormal movements and cognitive deficits that may impede understanding, communication and co-operation. Pitch and colleagues have recently proposed an algorithm based on patient neuro-anatomical data and language functional mapping, and taking into account the anaesthetic, surgical or neuropsychological risks associated with awake craniotomy, to decide whether a patient should undergo an awake procedure or general anaesthesia [11].

Table 1

Elements influencing the risk-benefit balance of an awake craniotomy

Benefit	Risk
Surgery	
Functional preservation	Recurrence of pathology
Immediate follow-up - earlier recovery - earlier discharge - earlier neurological follow-up	
Electrophysiological recordings - no effect of anaesthesia	
Patient	
Active participation - functional testing	Obstructive apnoea
	Seizures
	Nausea & vomiting
	Bleeding
	Elevated ICP
	Psychological disturbance - anxiety - pain - discomfort - unwillingness to co-operate
	Neurological abnormalities - abnormal movements - dysphasia

Specific objectives of anaesthetic management

In order to optimise the benefits of awake craniotomy, the objectives of anaesthetic management must be threefold [8]: to maximise the patient's co-operation, to preserve general homeostasis, and to limit the interference between anaesthetic agents and the quality of electrophysiological recordings. Patient co-operation requires optimal analgesia, anxiolysis and sedation adapted to surgical events, the prevention of any discomfort related to positioning or the length of the procedure, prevention of nausea and vomiting, as prevention of seizures. Maintenance of homeostasis involves the preservation of a patent airway and adequate ventilation, haemodynamic stability, and brain relaxation. Thorough evaluation and preparation of the patient will guide the choice of the best suited anaesthetic technique.

Pre-operative patient evaluation and preparation

In addition to standard pre-anaesthetic evaluation, attention should be paid to registering elements that may influence the risk-benefit balance of the procedure (Table 2). Good oxygenation and carbon dioxide elimination must be maintained throughout the procedure despite periods of 'unprotected' spontaneous ventilation and careful evaluation to determine the risks of obstructive apnoea is mandatory. Examination of the upper airway and a search for signs of possible difficult intubation including neck stiffness, large tongue, Mallampati score, prognathia, and retrognathia must be performed. A quick and simple test, the STOP-BANG score, may be used to screen for obstructive sleep apnoea and determine the need for more detailed evaluation [12].

Table 2

Specific pre-operative evaluation prior to awake craniotomy

Upper airway
Difficult intubation signs (mouth opening, Mallampati score, large tongue, cervical stiffness, teeth, neck diameter, prognathia, retrognathia) Obstructive apnoea (obesity, sleep apnoea)
Seizures
Current treatment Plasma levels Type, number and frequency of episodes
Nausea and vomiting
Previous history following anaesthesia History of motion sickness
ICP
Type and size of lesion Indirect neuro-imaging signs and clinical signs of elevated ICP:
Bleeding
Type and localisation of lesion Current medications (anti-platelets, non steroidal anti-inflammatory medications) Personal and family past history
Patient collaboration
Anxiety Neurological problems (dysphasia, abnormal movements)

It may be very difficult to manage seizures during the procedure. The adequacy of treatment and plasma levels of medication, as well as the occurrence and frequency of seizures in normal daily life, must be reviewed. A history of nausea and vomiting following previous anaesthesia and a history of motion sickness should lead to the use of prophylactic therapy. Brain relaxation may be difficult to achieve in a spontaneously breathing and sedated patient, particularly if the ICP is high before the start of the procedure. An indirect estimation of ICP is necessary, based on clinical signs or neuro-imaging. The risk of bleeding should be evaluated using the past medical history and the patient's family history, and by registering current medications that may facilitate bleeding, such as anti-platelet agents.

Finally, such a stressful procedure necessitates psychological preparation of the patient by the surgeon and the anaesthesiologist. The reasons for performing the procedure awake must be explained, as well as the sequence of events, the expected length and possible discomfort, the associated risks and the techniques used to limit inconvenience. The patient should be reassured and trained in the functional tests to be used during surgery [13]. Such preparation helps to improve patient tolerance and general satisfaction with the procedure [14]. Premedication may be helpful in reducing anxiety but may lead to respiratory depression and impede co-operation. Practice varies between teams, ranging from small doses of benzodiazepines [15] or clonidine [16] to the use of no pre-medication [17]. Atropine-like drugs are sometimes used but may produce discomfort from a very dry mouth. Corticosteroids reduce brain oedema and prevent nausea and may be given with 5-HT_3 antagonists or metoclopramide to reduce the incidence of nausea and vomiting along with ranitidine [18]. Anti-epileptic medication should be given on the morning of surgery to prevent seizures [19].

Intra-operative anaesthetic management

The main challenge of intra-operative anaesthetic management during awake craniotomy is the need to rapidly adjust the level of sedation and analgesia according to the sequence of surgical events, while ensuring haemodynamic stability and adequate ventilation.

Anaesthetic agents (Table 3)

For flexibility short-acting agents are preferred. The combination of propofol and remifentanil is often chosen [15, 16, 20-22], although no advantage of remifentanil over longer-acting opioids has been demonstrated in terms of intra-operative complications or patient satisfaction [23]. Propofol is easy to titrate but may induce respiratory depression, particularly if used in conjunction with opioids [24]. At subanaesthetic concentrations it may induce motor restlessness and abnormal movements [25]. Target-controlled infusions, which allow accurate control of plasma or effect-site concentration, are the preferred mode of administration. Tight control can be facilitated by the use of depth of anaesthesia monitors [15, 26]. Additional advantages of propofol are its potential anti-emetic properties and short duration of any changes it induces in electrocorticographic recordings [27, 28]. Neurolept derivatives should not be used because of a higher incidence of seizures [28] and, when used in combination with opioids, they may precipitate respiratory depression [25].

Table 3

Advantages and disadvantages of anaesthetic agents used for awake craniotomy

Agents	Advantages	Disadvantages
Propofol	- Rapid onset and offset - No prolonged effect on electrophysiological recordings - Anti-emetic properties	- Respiratory depression (with opioids) - Motor restlessness and abnormal movements (sub-anaesthetic concentrations)
Remifentanil	- Short acting: rapid onset and offset - Good analgesia	- Respiratory depression
Neuroleptic agents	- No respiratory depression	- Seizures - Respiratory depression (with opioids) - Prolonged QT interval
Dexmedetomidine	- Good analgesia - Anxiolysis - Sedation but easily arousable - Few interferences with electrophysiological recordings - Suitable pharmacokinetic profile	- Hypotension - Bradycardia
Clonidine	- Same as dexmedetomidine	- Prolonged elimination
Ketamine	- Analgesia - No respiratory depression	- Psychotropic effects - ICP
Inhaled compounds		- Not easy to use in spontaneously breathing patients

Some anaesthetists prefer to use longer-acting agents with less risk of respiratory depression. Among these the α_2-agonists offer good analgesia, anxiolysis, and sedation while keeping the patient easily arousable [29]. Clonidine can be used but dexmedetomidine has a better pharmacokinetic profile and has been successfully used by several authors [30, 31], including in adolescent [32] and paediatric patients [33], and allows accurate mapping of epileptic foci [34].

Volatile anaesthetic agents are not suitable for the management of awake craniotomy, since they are not easily administrated in patients whose ventilation is not controlled. The psychotropic effects of ketamine, as well as its effect on ICP, preclude its use at anaesthetic concentrations in this situation. However, small doses in association with other medications might allow patients to benefit from its excellent anti-nociceptive properties with little respiratory depression. This technique still needs to be evaluated.

Anaesthetic technique

The most difficult aspect of an awake craniotomy is the contrast between the awake phases of functional testing and electrophysiological recordings where any device used to assist the upper airway may be uncomfortable, and phases where anaesthesia is usually deepened to ensure patient comfort. These deep phases may require upper airway control and artificial ventilation. To overcome this problem, several anaesthetic techniques have been proposed. Monitored conscious sedation is the most frequently used. Spontaneous ventilation is maintained throughout the procedure, while supplementary oxygen is given through a facemask [15, 17, 22]. This non-invasive technique is comfortable for the patient, but there may be a high risk of obstructive apnoea in susceptible patients. There are several well tolerated devices which may help prevent obstructive apnoea such as naso-pharyngeal cannula or intra-oral devices used to advance the mandible [35]. A simple oral (Mayo) cannula is usually not well tolerated. Other authors have used a laryngeal mask airway [19, 36] or non-invasive positive pressure ventilation through a nasal mask [37, 38].

Another option is the asleep-awake-asleep technique: the first part of the procedure is conducted under general anaesthesia and controlled mechanical ventilation; spontaneous ventilation is then allowed during the awake phase of functional or electrophysiological testing, and general anaesthesia is induced again along with controlled mechanical ventilation for the third phase. Controlled ventilation may be provided using a laryngeal mask airway [39], or a tracheal tube inserted in the awake patient under fiberoptic control, removed during the awake phase, and re-inserted using the same technique for the second asleep phase [40]. In this situation local anaesthesia of the upper airway is mandatory. A variant to this approach consists of a two-phase technique, the asleep-awake technique, which overcomes the problem of controlling patient ventilation once testing has been completed.

Analgesia must be optimal during the whole procedure to maintain patient tolerance. Besides intravenous analgesia, local or regional analgesia is of great help. A scalp block using epinephrine containing local anaesthetic agent solutions is mandatory. Doses of ropivacaine or levobupivacaine up to 3.6 and 2.5 mg kg^{-1}, respectively, have been demonstrated to be safe [41, 42]. However, a more selective approach, with blockade of six nerves on each side of the scalp (the auriculotemporal, supratrochlear, zygomaticotemporal, auriculotemporal, and greater and lesser temporal nerves) [43] or a superficial cervical plexus block, may help reduce local anaesthetic dose and improve efficacy. Gebhard and colleagues have proposed selective blocks of the arm or leg to prevent involuntary movements during the procedure [44].

Specific monitoring

Besides standard anaesthetic monitoring, invasive monitoring of arterial blood pressure is recommended. End-tidal carbon dioxide measurement is easy when the upper airway is maintained with a tracheal tube or laryngeal mask airway. A trend in the end-tidal carbon dioxide can be obtained when sampled through a face mask and will allow detection of obstructive apnoea.

Patient positioning

Because it determines patient tolerance to the procedure, positioning on the operating table must be optimal. The mattress should be comfortable, and, ideally, be designed to prevent bedsores. The knees should be supported, as well as the arms and shoulders. The position of the head should be checked after placement of the pin holder to ensure comfort and ease of spontaneous ventilation. Bladder catheterisation is also mandatory. The temperature should be monitored and warming blankets used accordingly.

Complications and outcome

When the procedure is well planned, complications are rare (Table 4). The most frequent intra-operative complications are obstructive apnoea, nausea and vomiting, seizures, and loss of patient co-operation. Other complications include episodes of raised blood pressure, bradycardia (associated with the trigeminocardiac reflex) and air embolism. These complications may compromise the procedure and require conversion to general anaesthesia. Experienced anaesthesiologists are less prone to encounter complications than inexperienced ones [16].

Table 4

Complications of awake craniotomy

Complication	Consequences	Prevention
Obstructive apnoea & hypoventilation	Hypoxaemia Hypercarbia Increased ICP	Pre-operative evaluation Tight titration of sedation Expired CO_2 monitoring Head positioning – access to airway Non-invasive positive pressure ventilation devices Asleep-awake-asleep technique
Nausea & vomiting	Movement Inhalation Increased ICP	Pre-operative evaluation Psychological preparation Corticosteriods 5-HT_3 antagonists Metoclopramide Limited use of opioids Propofol
Seizures	Movement Increased ICP Compromised ventilation	Pre-operative evaluation Pre-operative prevention Single electrical stimulations
Loss of patient co-operation	Movement Agitation Functional testing compromised	Psychological preparation Tight titration of sedation Good analgesia Good positioning Procedure as short as possible
Hypertension & tachycardia	Bleeding Increased ICP	Optimal analgesia Vasodilators or β-blockers
Trigeminocardiac reflex	Bradycardia	Topical anaesthesia of the dura Atropine
Air embolism	Hypoxaemia Haemodynamic instability	Head positioning Optimal intravascular fluid loading
Bleeding	Increased ICP Anaemia Surgical difficulty	Screening for bleeding disorders Avoid other complications

Obstructive apnoea and hypoventilation are among the main risk factors to be considered in the anaesthetic management. Hypoxemia and hypercarbia may result, with deleterious consequences on ICP. Prevention begins during the pre-operative evaluation of the risk of obstructive apnoea. If the risk is high, the use of non-invasive positive pressure ventilation devices may be an option, along with the use of an asleep-awake-asleep anaesthetic technique. Spontaneous ventilation through a face mask will require monitoring of expired carbon dioxide and tight control of the level of sedation. Caution should also be paid to head positioning to facilitate ventilation and permit access at any time [18].

Nausea and vomiting have been reported to occur in as many as 8% of patients [45]. Prevention includes good psychological preparation to reduce stress, prophylactic administration of 5-HT_3 antagonists or metoclopramide in high risk patients, corticosteroid administration, use of propofol, and limitation of opioid use. Partial or generalised seizures are more frequent during treatment of intractable epilepsy. Prevention also begins during the pre-operative evaluation of patients. At that time, anti-epileptic medication should be instituted if not already done and the dosage should be optimised. Electrical cortical stimulation, when needed, should be single stimulations as opposed to trains of stimulations which may be associated with an incidence of seizures as high as 20% [10]. Epilepsy may lead to ventilation problems, either during the seizure itself or during the postictal period.

Loss of patient co-operation is associated with agitation, restlessness, and movement. It greatly compromises functional testing. Good psychological preparation, adequate titration of sedation, good analgesia, comfortable positioning on the table and efforts to keep the procedure as short as possible all help. Hypertensive and tachycardic episodes are more frequent during awake craniotomy than during general anaesthesia [46]. They can be prevented by adequate analgesia and, if required, vasodilators or β-blocker agents. Severe bradycardia may occur at the time of dura mater stimulation through the trigeminocardiac reflex and topical anaesthesia of the dura may help prevent this. If encountered, removal of the stimulus and administration of atropine should be considered.

Air embolism may occur during awake craniotomy as in any other neurosurgical procedure [47]. Prevention and treatment of this event is no different. Bleeding may be increased by hypertensive episodes and any cause of increased cerebral venous pressure, such as obstructive apnoea. It may cause an increase in ICP and brain herniation, surgical difficulty, and anaemia. Pre-operative screening for bleeding disorders is necessary, as well as precautions to maintain cerebral venous pressure at an acceptable level.

Studies comparing outcome between awake and general anaesthesia craniotomies are scarce. Most series report satisfactory results of awake procedures in terms of tumour cytoreduction, neurological improvement or the incidence of postoperative neurological deficit, wound complications, haematomas, and death [3]. To our knowledge, only one team has performed a prospective comparison of resection of lesions located in eloquent cortical areas [48], with better results in the general anaesthesia group than in the awake group.

Conclusions

The anaesthetic management of awake craniotomy is challenging. Success is the result of a well prepared teamwork, and necessitates the intervention of skilled individuals. Throughout the procedure, complications must be anticipated and managed according to pre-defined guidelines. Choosing one or the other therapeutic option may not be easy with respect to the evaluation of patient co-operation capacity. Furthermore, there is a need for prospective randomized clinical trials to improve the safety and efficacy of this technique, as well as to validate it in comparison with more conventional procedures.

Key Learning Points

- Awake craniotomy has become a common procedure; its indications have increased.
- The main objectives of anaesthetic management are the facilitation of the patient's co-operation, preservation of general homeostasis, and limitation of possible interferences between anaesthetic agents and electrophysiological recordings.
- Careful patient selection and preparation is mandatory. The operating room team must be composed of skilled people.
- Choosing an anaesthetic technique depends on several factors including the risks of obstructive apnoea, seizures, and nausea and vomiting, as well as on the patient's ability to co-operate, and the site of the lesion. The main challenge of intra-operative anaesthetic management is ability to rapidly adjust the level of sedation and analgesia according to the sequence of surgical events, while ensuring haemodynamic stability and adequate ventilation.
- Throughout the procedure, complications must be anticipated and managed according to well prepared plans.
- Prospective randomised clinical studies are needed to evaluate awake craniotomy in comparison with craniotomy under general anaesthesia

References

1. Klimek M, Verbrugge SJ, Roubos S, et al. Awake craniotomy for glioblastoma in a 9-year-old child. Anaesthesia 2004; 59: 607-9.
2. Manninen PH, Tan TK. Postoperative nausea and vomiting after craniotomy for tumor surgery: a comparison between awake craniotomy and general anesthesia. J Clin Anesth 2002; 14: 279-83.
3. Serletis D, Bernstein M. Prospective study of awake craniotomy used routinely and nonselectively for supratentorial tumors. J Neurosurg 2007; 107: 1-6.
4. Blanshard HJ, Chung F, Manninen PH, Taylor MD, Bernstein M. Awake craniotomy for removal of intracranial tumor: considerations for early discharge. Anesth Analg 2001; 92: 89-94.
5. Bonhomme V, Born JD, Hans P. [Anaesthetic management of awake craniotomy]. Ann Fr Anesth Reanim 2004; 23: 389-94.
6. Conte V, Baratta P, Tomaselli P, et al. Awake neurosurgery: an update. Minerva Anestesiol 2008; 74: 289-92.
7. Bulsara KR, Johnson J, Villavicencio AT. Improvements in brain tumor surgery: the modern history of awake craniotomies. Neurosurg Focus 2005; 18: e5.
8. Hans P, Hans P, Bonhomme V. Anesthetic management for neurosurgery in awake patients. Minerva Anestesiol 2006; 72: 1-7.
9. Lanier WL. Brain tumor resection in the awake patient. Mayo Clin Proc 2001; 76: 670-2.
10. Maertens dN, Born JD, Hans P, Remacle JM, Delwaide PJ. Intraoperative localisation of the primary motor cortex using single electrical stimuli. J Neurol Neurosurg Psychiatry 1996; 60: 442-4.
11. Picht T, Kombos T, Gramm HJ, Brock M, Suess O. Multimodal protocol for awake craniotomy in language cortex tumour surgery. Acta Neurochir (Wien) 2006; 148: 127-37.
12. Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology 2008; 108: 812-21.
13. Signorelli F, Guyotat J, Isnard J, et al. The value of cortical stimulation applied to the surgery of malignant gliomas in language areas. Neurol Sci 2001; 22: 3-10.
14. Whittle IR, Midgley S, Georges H, Pringle AM, Taylor R. Patient perceptions of “awake” brain tumour surgery. Acta Neurochir (Wien) 2005; 147: 275-7.
15. Hans P, Bonhomme V, Born JD, et al. Target-controlled infusion of propofol and remifentanil combined with bispectral index monitoring for awake craniotomy. Anaesthesia 2000; 55: 255-9.
16. Berkenstadt H, Perel A, Hadani M, Unofrievich I, Ram Z. Monitored anesthesia care using remifentanil and propofol for awake craniotomy. J Neurosurg Anesthesiol 2001; 13: 246-9.
17. Gignac E, Manninen PH, Gelb AW. Comparison of fentanyl, sufentanil and alfentanil during awake craniotomy for epilepsy. Can J Anaesth 1993; 40: 421-4.
18. Tobias JD, Jimenez DF. Anaesthetic management during awake craniotomy in a 12-year-old boy. Paediatr Anaesth 1997; 7: 341-4.
19. Sarang A, Dinsmore J. Anaesthesia for awake craniotomy--evolution of a technique that facilitates awake neurological testing. Br J Anaesth 2003; 90: 161-5.

20. Keifer JC, Dentchev D, Little K, et al. A retrospective analysis of a remifentanil/propofol general anesthetic for craniotomy before awake functional brain mapping. Anesth Analg 2005; 101: 502-8.
21. Lobo F, Beiras A. Propofol and remifentanil effect-site concentrations estimated by pharmacokinetic simulation and bispectral index monitoring during craniotomy with intraoperative awakening for brain tumor resection. J Neurosurg Anesthesiol 2007; 19: 183-9.
22. Johnson KB, Egan TD. Remifentanil and propofol combination for awake craniotomy: case report wit h pharmacokinetic simulations. J Neurosurg Anesthesiol 1998; 10: 25-9.
23. Manninen PH, Balki M, Lukitto K, Bernstein M. Patient satisfaction with awake craniotomy for tumor surgery: a comparison of remifentanil and fentanyl in conjunction with propofol. Anesth Analg 2006; 102: 237-42.
24. Herrick IA, Craen RA, Gelb AW, et al. Propofol sedation during awake craniotomy for seizures: patient-controlled administration versus neurolept analgesia. Anesth Analg 1997; 84: 1285-91.
25. Frost EA, Booij LH. Anesthesia in the patient for awake craniotomy. Curr Opin Anaesthesiol 2007; 20: 331-5.
26. Rath GP, Prabhakar H. Spectral entropy monitoring in a patient undergoing awake craniotomy. J Neurosurg Anesthesiol 2007; 19: 144.
27. Soriano SG, Eldredge EA, Wang FK, et al. The effect of propofol on intraoperative electrocorticography and cortical stimulation during awake craniotomies in children. Paediatr Anaesth 2000; 10: 29-34.
28. Herrick IA, Craen RA, Gelb AW, et al. Propofol sedation during awake craniotomy for seizures: electrocorticographic and epileptogenic effects. Anesth Analg 1997; 84: 1280-4.
29. Bhana N, Goa KL, McClellan KJ. Dexmedetomidine. Drugs 2000; 59: 263-8.
30. Mack PF, Perrine K, Kobylarz E, Schwartz TH, Lien CA. Dexmedetomidine and neurocognitive testing in awake craniotomy. J Neurosurg Anesthesiol 2004; 16: 20-5.
31. Moore TA, Markert JM, Knowlton RC. Dexmedetomidine as rescue drug during awake craniotomy for cortical motor mapping and tumor resection. Anesth Analg 2006; 102: 1556-8.
32. Everett LL, van R, I, Warner MH, Set al. Use of dexmedetomidine in awake craniotomy in adolescents: report of two cases. Paediatr Anaesth 2006; 16: 338-42.
33. Ard J, Doyle W, Bekker A. Awake craniotomy with dexmedetomidine in pediatric patients. J Neurosurg Anesthesiol 2003; 15: 263-6.
34. Souter MJ, Rozet I, Ojemann JG, et al. Dexmedetomidine sedation during awake craniotomy for seizure resection: effects on electrocorticography. J Neurosurg Anesthesiol 2007; 19: 38-44.
35. Kuna ST, Woodson LC, Solanki DR, et al. Effect of progressive mandibular advancement on pharyngeal airway size in anesthetized adults. Anesthesiology 2008; 109: 605-12.
36. Tongier WK, Joshi GP, Landers DF, Mickey B. Use of the laryngeal mask airway during awake craniotomy for tumor resection. J Clin Anesth 2000; 12: 592-4.
37. Yamamoto F, Kato R, Sato J, Nishino T. Anaesthesia for awake craniotomy with non-invasive positive pressure ventilation. Br J Anaesth 2003; 90: 382-5.
38. Huncke T, Chan J, Doyle W, Kim J, Bekker A. The use of continuous positive airway pressure during an awake craniotomy in a patient with obstructive sleep apnea. J Clin Anesth 2008; 20: 297-9.
39. Fukaya C, Katayama Y, Yoshino A, et al. Intraoperative wake-up procedure with propofol and laryngeal mask for optimal excision of brain tumour in eloquent areas. J Clin Neurosci 2001; 8: 253-5.
40. Huncke K, Van de WB, Fried I and Rubinstein EH. The asleep-awake-asleep anesthetic technique for intraoperative language mapping. Neurosurgery 1998; 42: 1312-6.
41. Costello TG, Cormack JR, Hoy C, et al. Plasma ropivacaine levels following scalp block for awake craniotomy. J Neurosurg Anesthesiol 2004; 16: 147-50.
42. Costello TG, Cormack JR, Mather LE, et al. Plasma levobupivacaine concentrations following scalp block in patients undergoing awake craniotomy. Br J Anaesth 2005; 94: 848-51.
43. Costello TG, Cormack JR. Anaesthesia for awake craniotomy: a modern approach. J Clin Neurosci 2004; 11: 16-9.
44. Gebhard RE, Berry J, Maggio WW, Gollas A, Chelly JE. The successful use of regional anesthesia to prevent involuntary movements in a patient undergoing awake craniotomy. Anesth Analg 2000; 91: 1230-1.
45. 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.
46. Skucas AP, Artru AA. Anesthetic complications of awake craniotomies for epilepsy surgery. Anesth Analg 2006; 102: 882-7.
47. Balki M, Manninen PH, McGuire GP, El-Beheiry H, Bernstein M. Venous air embolism during awake craniotomy in a supine patient. Can J Anaesth 2003; 50: 835-8.
48. Gupta DK, Chandra PS, Ojha BK, et al. Awake craniotomy versus surgery under general anesthesia for resection of intrinsic lesions of eloquent cortex--a prospective randomised study. Clin Neurol Neurosurg 2007; 109: 335-43.