Awake craniotomy using dexmedetomidine and scalp blocks: a retrospective cohort study

Craniotomie sur patient éveillé utilisant la dexmédétomidine et des blocs des nerfs du scalp: une étude de cohorte rétrospective

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Abstract

Purpose Anesthetic and surgical considerations for awake craniotomy (AC) include airway patency, patient comfort, and optimization of real-time brain mapping. The purpose of this study is to report our experience of using dexmedetomidine and scalp blocks, without airway intervention, as a means to facilitate and optimize intraoperative brain mapping and brain tumour resection during AC.

Methods We conducted a retrospective cohort study of 55 patients who underwent AC from March 2012 to September 2016. The incidence of critical airway outcomes, perioperative complications, and successful intraoperative mapping was determined. The primary outcome was the incidence of a failed AC anesthetic technique as defined by the need to convert to general anesthesia with a secured airway prior to (or during)

brain mapping and brain tumour resection. Secondary outcomes were the intraoperative incidence of: 1) altered surgical management due to information acquired through real-time brain mapping, 2) interventions to restore airway patency or rescue the airway, 3) hemodynamic instability (> 20% from baseline), 4) nausea and vomiting, 5) new onset neurologic deficits, and 6) seizure activity.

Results There were no anesthesia-related critical events and no patients required airway manipulation or conversion to a general anesthetic. Multimodal language, motor, and sensory assessment with direct cortical electrical stimulation was successfully performed in 100% of cases. In 24% (13/55) of patients, data acquired during intraoperative brain mapping influenced surgical decision-making regarding the extent of tumour resection. Nine (16%) patients had intraoperative seizures.

Conclusions Dexmedetomidine-based anesthesia and scalp block facilitated AC surgery without any requirement for urgent airway intervention or unplanned conversion to a full general anesthetic. This approach can enable physiologic testing before and during tumour resection facilitating real-time surgical decision-making based on intraoperative brain mapping with patients awake thereby minimizing the risk of neurologic deficit and increasing the opportunity for optimal surgical resection.

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Résumé

Objectif Les considérations anesthésiques et chirurgicales faisant envisager une craniotomie sur patient éveillé (CE) sont notamment la perméabilité des voies aériennes, le confort du patient et l'optimisation de la cartographie cérébrale en temps réel. L'objectif de cette étude est de rapporter notre expérience de l'utilisation de la dexmédétomidine et des blocs des nerfs du scalp, sans

intervention sur les voies aériennes, afin de faciliter et d'optimiser la cartographie cérébrale peropératoire et la résection de tumeurs cérébrales au cours d'une CE.

Méthodes Nous avons mené une étude de cohorte rétrospective sur 55 patients ayant subi une CE entre mars 2012 et septembre 2016. L'incidence des événements critiques sur les voies aériennes, des complications périopératoires et des succès de la cartographie cérébrale peropératoire a été déterminée. Le critère d'évaluation principal était l'incidence de l'échec de la technique anesthésique par CE, défini par la nécessité de la convertir en anesthésie générale avec sécurisation des voies aériennes avant (ou pendant) la cartographie cérébrale et la résection de la tumeur. Les critères d'évaluation secondaires étaient des mesures de la survenue peropératoire des événements suivants : 1) modification de la gestion chirurgicale en raison d'informations acquises au cours de la cartographie cérébrale en temps réel, 2) interventions visant à restaurer la perméabilité des voies aériennes ou libérer l'accès aux voies aériennes, 3) instabilité hémodynamique ($> 20\%$ par rapport à l'inclusion), 4) nausées et vomissements, 5) apparition de nouveaux déficits neurologiques, et 6) convulsion.

Résultats Il n'y a pas eu d'événements critiques liés à l'anesthésie et aucun patient n'a nécessité d'intervention sur les voies aériennes ou de conversion à une anesthésie générale. Une évaluation multimodale du langage, de la motricité et de la sensibilité avec stimulation électrique corticale a été réalisée avec succès dans 100 % des cas. Chez 24 % des patients (13/55), les données acquises au cours de la cartographie cérébrale peropératoire ont influencé la prise de décision concernant l'étendue de la résection tumorale. Neuf patients (16 %) ont présenté des convulsions peropératoires.

Conclusions L'anesthésie basée sur la dexmédétomidine et le bloc des nerfs du scalp a facilité l'intervention de CE sans nécessiter aucune intervention urgente sur les voies aériennes ou conversion non prévue en anesthésie généralisée complète. Cette approche autorise la réalisation de tests physiologiques avant et pendant la résection de la tumeur, facilitant la prise de décision chirurgicale en temps réel basée sur la cartographie cérébrale peropératoire chez des patients éveillés, minimisant ainsi le risque de déficit neurologique et augmentant les chances de résection chirurgicale optimale.

The modern practice of awake craniotomy (AC) evolved with the development of sedative agents that facilitated existing local anesthetic techniques.^{1,2} This allowed the precise anatomic localization of neurologic function in patients who are undergoing supratentorial craniotomy, with

most benefit seen predominantly for epilepsy surgery.³ Awake craniotomy with brain mapping has become the gold standard for patients undergoing surgery for tumours near or within eloquent areas of the brain. A wide variety of techniques have been described including the asleep-awake-asleep approach (SAS), monitored anesthesia care, and asleep-awake, awake-awake-awake, and conscious sedation approaches.⁴ Meng *et al.* recently succinctly summarized AC as a pre-awake, awake, and post-awake phase, with and without airway intervention.⁵ The goals include maintaining airway patency, optimizing cerebral perfusion, facilitating real-time brain mapping, minimizing postoperative pain, and allowing rapid recovery and assessment of neurologic function after surgery.⁶

Various anesthesia drugs have been used to achieve these goals, including propofol, remifentanyl, fentanyl, and dexmedetomidine.⁷ The use of dexmedetomidine was first reported for AC surgery in 2001, predominantly as an adjunct to the SAS method, but with an additional benefit of decreased respiratory depression.^{8,9} While a propofol/remifentanyl technique provides satisfactory conscious sedation, recognized drawbacks of this approach include hypoventilation and hypercapnia due to airway obstruction.¹⁰ Dexmedetomidine is a lipophilic imidazole derivative that acts as a selective pre- and post-synaptic α_2 adrenoceptor agonist. It has anxiolytic, sedative, and anesthetic properties. Dexmedetomidine provides sedation that resembles natural sleep without cognitive impairment, making it an excellent anesthetic choice for AC surgery.¹¹ An additional feature of dexmedetomidine making it attractive for AC surgery is its shorter arousal time compared with propofol.¹² It has also been hypothesized that dexmedetomidine has a capacity to maintain the cerebral metabolic rate to cerebral blood flow (CBF) coupling. This results in a decrease in CBF, via an α_{2B} receptor-mediated vasoconstriction, to match the reduced cerebral metabolic requirements during sedation and anesthesia.^{13,14} As research continues to elucidate the effect of dexmedetomidine on cancer cells, characterizing the impact of dexmedetomidine on tumour biology will be an important step in establishing the role of dexmedetomidine in neurooncology.^{15,16}

In 2012, we instituted a standardized anesthetic technique of intravenous dexmedetomidine and scalp nerve blocks for patients undergoing AC surgery. This technique has been previously described by our group for select high-risk neurosurgical patients and is now the primary technique used at our institution.^{17,18} In this present study, we examined whether a dexmedetomidine-based sedation facilitates optimal conditions for AC defined as the avoidance of the need for airway manipulation or conversion to general anesthesia prior to complete brain mapping and brain tumour

resection. Secondary outcomes included the establishment of optimal conditions for successful brain mapping and maximal tumour resection during AC.⁵

Methods

Eligibility and data collection

Following institutional Research Ethics Board approval in November 2016, waiving the need for patient consent, all adults (age ≥ 18 yr) who underwent AC at our institution during the study period from 1 March 2012 to 1 September 2016 were eligible for inclusion. Patients were excluded if their health records were incomplete or if they had a documented refusal of consent to participate in research. The intraoperative records of all patients who underwent AC surgery during the study period were screened for eligibility and, if applicable, data were extracted. Demographic and perioperative data collected included American Society of Anesthesiologists physical status (ASA-PS) classification, age, height, sex, weight, use of preoperative anti-epileptic drugs, tumour characteristics (pathology and location), and amplitude of direct cortical electrical stimulation (DCES), types and doses of anesthetic agents, intraoperative adverse events (airway manipulation or instrumentation, conversion to general anesthesia, nausea, and seizure activity), surgical outcomes (extent of resection and deviation of surgical plan based on preoperative functional magnetic resonance imaging), and select postoperative outcomes (new neurologic deficits, duration of hospital stay).

Anesthetic technique

Standard monitors were applied, intra-arterial blood pressure monitoring was instituted in all cases, and end-tidal carbon dioxide was monitored via a sampling channel integrated into a facemask or nasal cannula (depending on patient preference). A dexmedetomidine loading dose of $1 \mu\text{g}\cdot\text{kg}^{-1}$ was administered over 15 min and an infusion started at $0.3\text{--}0.4 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$. Scalp nerve blocks were performed using an anatomical approach with 0.375% bupivacaine and epinephrine prior to pin insertion for both perioperative anesthesia and postoperative pain.^{19,20} Midazolam ($0.01\text{--}0.05 \text{ mg}\cdot\text{kg}^{-1}$) and fentanyl ($1 \mu\text{g}\cdot\text{kg}^{-1}$) were administered before performance of the scalp blocks.

The patient was positioned awake and a Mayfield frame was used in all cases with supplemental lidocaine applied to the pin sites prior to insertion. The incision line was infiltrated with 2% lidocaine and additional infiltration of the muscle and topical anesthesia of the dura were

performed by the surgeon as required throughout the case. The dexmedetomidine infusion rate was titrated to achieve a Ramsey sedation score between 2 and 4 until the dura was opened.²¹ Bolus doses of dexmedetomidine ($0.05 \mu\text{g}\cdot\text{kg}^{-1}\text{--}0.1 \mu\text{g}\cdot\text{kg}^{-1}$) were permitted throughout the case and infusion rates were increased as necessary to achieve required levels of sedation. Fentanyl for analgesia during bone flap and dural opening were provided at the discretion of the anesthesiologist; if required, a low-dose remifentanyl infusion was permitted for supplemental analgesia. All infusions were discontinued at dural opening and restarted following neurophysiologic testing on a case-by-case basis. Hydromorphone, for postoperative analgesia, was administered following completion of the “awake” phase.

Neurologic assessment

Patient assessment involved pre- and postoperative neurophysiologic testing using a battery of tests for speech and cognition, including phonemic word fluency using the letters F, A, and S, semantic fluency tests (animal naming),²² the line bisection test,²³ trail making tests A and B,²⁴ the Wechsler Memory Scale-Logical Stories (Anna Thompson story)²⁵ judgement of line orientation,²⁶ digit span, digit symbol, Rey-Osterreith Complex Figure,²⁷ Barthel Index,²⁸ Hospital Anxiety and Depression Scale,²⁹ and Patient Assessment of Own Functioning.³⁰

All patients underwent intraoperative speech and sensorimotor testing, which was dictated by the location of the tumour and of the cortical areas exposed by the craniotomy. Speech testing included number counting, naming, and word-generation tasks. Mapping was performed using bipolar Direct Cortical Electrical Stimulation [DCES with the OCS2 Ojemann Cortical Stimulator (Integra Life Sciences; Plainsborough, NJ, USA)]. Stimulation was performed with 500-msec pulses at 60 Hz with a starting amplitude of 2 mA and peak amplitude of 8mA³¹ (Figure).

Primary and secondary outcomes

The primary outcome was the incidence of failure of the AC anesthetic technique, defined by the need to convert to general anesthesia with a secured airway prior to (or during) brain mapping and brain tumour resection. Secondary outcomes were the incidence of: 1) optimal conditions for successful brain mapping and maximal tumour resection, 2) interventions to restore airway patency or rescue the airway, 3) significant ($> 20\%$ from baseline) hemodynamic instability, 4) nausea and vomiting, 5) new-onset neurologic deficits, and 6) seizure activity.

Figure Correlation between intraoperative neurologic testing (a, b), cortical area exposed at craniotomy (c), and functional magnetic resonance images (d, e)

Statistical analysis

Data distribution was tested for normality using the Shapiro-Wilk test. Continuous data are presented as mean [standard deviation (SD)] when normally distributed or median (interquartile range [IQR]) when not normally distributed; categorical data were presented as proportions. The 95% confidence intervals are reported where appropriate. Fisher's exact test was used to test differences between groups. $P < 0.05$ was considered statistically significant. All statistical analyses were performed using STATA 14 (STATACorp LP, TX, USA).

Results

We identified 56 patients who underwent AC surgery during the study time period. One patient chart was excluded because of an incomplete health record. Fifty-five patients were included in the cohort, including ten patients described previously by Garavaglia *et al.*¹⁷ Demographic

and intraoperative characteristics are summarized in Table 1. Most patients were male (55%), ASA-PS $\geq$ III (99%), and had preoperative seizure activity (64%) requiring anti-epileptic medication. The median [IQR] patient postoperative hospital stay was 2 [1-3] days.

Primary outcomes

All patients included in the study underwent successful AC with DCES for motor or speech function. There was no failure of the approach requiring conversion to general anesthesia with a secured airway. No intraoperative interventions were required to restore airway patency.

Secondary outcomes

The preoperative functional MRI-based surgical plan was often modified using intraoperative information acquired from DCES. Modifications to this plan fell into three categories: 1) identification of the corridor for tumour resection [17/55 (31%)], 2) definition of the limit of

Table 1 Demographic and intraoperative characteristics for all ($n = 55$) patients

Characteristic	$n = 55$
Age, yr	46 (16)
Male, n	30 (55%)
BMI ($\text{kg}\cdot\text{m}^{-2}$)	26 (6)
ASA classification, n	
III	31 (56%)
IV	23 (42%)
Preoperative seizures	35 (64%)
Intraoperative mannitol given, n	3 (5%)
Systolic blood pressure (mmHg)	
Maximum	151 (18)
Minimum	108 (14)
Heart rate ($\text{beats}\cdot\text{min}^{-1}$)	
Maximum	76 (12)
Minimum	54 (8)
Maximum PaCO_2 (mmHg)	41 (8)
Duration in minutes	230 [190 – 309]
Postoperative length of hospital stay	2 [1–3]

Data are represented by as mean (standard deviation), median [interquartile range], or percentage (%) as indicated

ASA = American Society of Anesthesiologists; BMI = body mass index; PaCO_2 = partial pressure of carbon dioxide

resection [31/55 (56%)], and 3) identification of the focus of positive stimulation distant from the corridor or site of tumour resection [6/55 (11%)]. In patients in the third category, our practice was to keep patients awake throughout the phase of tumour resection, often with serial episodes of cortical or subcortical stimulation during physiologic testing. Overall, 17/55 (31%) of patients remained awake throughout the process of tumour resection. The remainder underwent some degree or duration of deep sedation following the brain mapping.

Intraoperative medications administered are described in Table 2. Mean (SD) time from commencement of dexmedetomidine infusion to first cessation for testing was 100 (33) min. The mean (SD) cumulative dose of dexmedetomidine administered prior to initiating

neurophysiologic testing was $1.80 (0.76) \mu\text{g}\cdot\text{kg}^{-1}$. Fentanyl and midazolam were predominantly administered prior to pinning with supplemental fentanyl administered at dural opening as needed. Hydromorphone loading for postoperative analgesia took place following completion of testing in the majority of cases. In 7/55 (13%) of patients, hydromorphone was administered prior to testing in a dose range $0.002\text{--}0.13 \text{ mg}\cdot\text{kg}^{-1}$. Mean (SD) intraoperative hydromorphone dose administered in all patients was $1.2 (0.6) \text{ mg}$.

No significant changes in blood pressure were observed related to dexmedetomidine use. One patient experienced an episode of bradycardia associated with hypotension at dural opening, which responded to bolus glycopyrolate (0.2 mg). At the time of the episode, the infusion dose of dexmedetomidine was $1 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$. There were no nausea and vomiting during the procedure. Only one patient required low-dose supplemental remifentanyl infusion at a rate of $0.005\text{--}0.02 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ for analgesia during the initial period up to dural opening. Emergence agitation or delirium was not evident in any patient and no patient required general anesthesia after the initial pre-awake phase.

Tumour characteristics and surgical outcomes are summarized in Table 3. There were no episodes of generalized seizure. Focal seizures induced by DCS occurred in nine (16%) patients, of which eight (15%) occurred in patients with pre-existing preoperative seizure activity on anti-epileptic medication. All occurred at the time of cortical stimulation. Seizures resolved with direct administration of cold saline alone in seven (13%) patients; two patients required midazolam (0.5 mg) and phenytoin after mapping to control focal seizure activity. None of the events required premature termination of cortical mapping and no seizure resulted in airway intervention or conversion to general anesthesia. There was no association between preoperative antiepileptic use and intraoperative seizures ($P > 0.99$) or between intraoperative phenytoin use and intraoperative seizures ($P = 0.69$). Characteristics of seizure activity are summarized in Table 4. Gross total resection of

Table 2 Mean (SD) doses of anesthetic and analgesic medications administered

Drug	Prior to Neurophysiologic Testing	Total Intraoperative Dose
Total dexmedetomidine dose ($\text{mg}\cdot\text{kg}^{-1}$)	$1.80 (0.76)$	$2.4 (1.0)$
Bolus dexmedetomidine dose ($\text{mg}\cdot\text{kg}^{-1}$)	$0.80 (0.6)$	$0.13 (0.09)$
Midazolam (mg)/per patient	2 (1)	2.1 (1.3)
Fentanyl (μg)/per patient	101 (65)	106 (71)
Hydromorphone (mg)/per patient	$0.08 (0.25)$	$1.2 (0.6)$

SD = standard deviation

Table 3 Tumour characteristics and surgical outcomes for all ($n = 55$) patients

Characteristic	n (%)
<i>Tumour pathology</i>	
High-grade tumour	25 (46)
Low-grade tumour	29 (53)
Other	1 (2)
<i>Tumour site</i>	
Frontal	34 (62)
Temporal	13 (24)
Parietal	8 (15)
Positive stimulation achieved	45 (82)
Change in surgical plan due to DCES	13 (24)
1) Limit of resection	31 (56)
2) Corridor for resection	17 (31)
3) Resection distant from DCES site	6 (11)
Gross total resection achieved	32 (56)
New postoperative neurologic deficit	3 (5)

DCES = direct cortical electrical stimulation

metastatic lesions, the area of enhancement in high-grade glioma cases, or the area of FLAIR signal abnormality in low-grade gliomas cases was achieved in 32 (56%) patients. Three patients (5%) developed new postoperative neurologic deficits following surgery, one of which was associated with a postoperative hemorrhage within the tumour cavity.

Discussion

We describe our experience of using a predominantly dexmedetomidine-based anesthetic technique in conjunction with scalp blocks to facilitate AC surgery. This technique shows that dexmedetomidine can be used as the primary anxiolytic and sedative drug for AC surgery up to four hours in duration with no adverse respiratory events and providing optimal neurosurgical conditions. In our

cohort, using dexmedetomidine anesthesia, no patient required unplanned airway intervention or conversion to a general anesthesia.

Our reported outcomes are in keeping with the relatively low conversion rates to GA utilizing other anesthetic protocols. Previously published anesthesia failure rates during AC surgery vary, but typically 2–6% of patients require some form of airway manipulation during AC.³² The reasons for airway interventions generally include poor patient selection, an inadequate anesthetic regimen, and intraoperative stimulation-induced seizures. The choice of anesthetic technique influences perioperative adverse events. Goettel *et al.* found no respiratory events when dexmedetomidine/propofol was used for rescue, but 20% with propofol/remifentanyl.³³ Dilmen *et al.* more recently reported moderate-to-severe intraoperative desaturations in almost 20% of cases using a dexmedetomidine-based technique, but with unquantified amounts of remifentanyl or propofol supplementation.³⁴ Our study adds to the evidence base, suggesting that use of dexmedetomidine without propofol can be safely performed with no adverse respiratory events, no conversion to general anesthesia, and successful intraoperative mapping throughout. Lobo *et al.* have suggested that for surgeries with an expected duration exceeding four hours, the SAS technique is more appropriate as patients can cooperate better if their awake phase is preceded by an asleep phase. Concern has also been expressed regarding potential accumulation of dexmedetomidine and delayed return of function in prolonged procedures.³⁵ The median duration of surgery in our study was less than four hours, and there were no deleterious effects on intraoperative mapping or delayed return of function observed.

Awake surgery offers the best approach to optimal tumour removal while minimizing the risk of permanent postoperative neurologic deficits, with both improved quality of life and prolonged survival.³⁶ In a number of our cases, the operative plan was modified based on unexpected findings during stimulation of eloquent brain

Table 4 Incidence of intraoperative seizure activity, classified by anticonvulsant use and tumour pathology

	Stimulation-induced Seizures ($n = 9$)	No Seizures ($n = 46$)	P value
Preoperative anticonvulsant use, n	6 (67%)	29 (63%)	> 0.99
Perioperative phenytoin, n	1 (11%)	11 (24%)	0.67
<i>Tumour pathology, n</i>			0.70
High grade	3 (33%)	23 (50%)	
Low grade	4 (45%)	14 (30%)	
Metastasis	1 (11%)	7 (15%)	
Other	1 (11%)	2 (5%)	

regions within the planned area of tumour resection. In these cases, tumour resection was minimized to optimize intact neurologic function. This finding strongly suggests a benefit to continued patient assessment during tumour resection. The low rate (5%) reported here of new postoperative neurologic deficits following surgery, despite aggressive tumour resection, also compares favorably with other studies. A recent systematic review found a 7% incidence of new focal deficit after AC but 23% after general anesthesia.³⁷ Similarly, Honorato-Cia *et al.* found significantly lower perioperative neurologic deficits using a predominantly dexmedetomidine-based approach (in a dose range of 0.2–1.4 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) compared with other techniques.³⁸ Dilmen *et al.* also reported reduced postoperative neurologic deficits using conscious sedation compared with SAS.³⁴

While other studies have previously shown dexmedetomidine to be effective for AC surgery, the use of supplementary propofol—by either infusion or bolus—was most often used.³⁹

The use of midazolam in AC is controversial, as it can be associated with emergence agitation and delirium when transitioning to the awake phase.⁶ This was not noted in our cohort where 52 (95%) patients received midazolam perioperatively. A mean (SD) dose of 2 mg (1) was administered prior to neurophysiologic testing. In animal studies, midazolam has been shown to interact synergistically with dexmedetomidine resulting in a dose-sparing effect.⁴⁰ The ability to use lower doses of dexmedetomidine to achieve sedation may have some benefits in reducing the side effects of dexmedetomidine and did not interfere with neurocognitive testing in this cohort. A recent study by Suero Molina *et al.* comparing dexmedetomidine and remifentanyl to an SAS technique for AC found that dexmedetomidine did result in better quality and reliable neurologic testing upon cessation.⁴¹

One of the main criticisms of using dexmedetomidine is the potential for blunting of the carbon dioxide (CO_2) response curve,⁴² and some anesthesiologists advocate for advanced airways for the pre-awake phase to minimize this risk. This is an important consideration for those patients with a “tight brain” and increased surgical difficulty. Despite this, hypercapnia, due to either obstruction or hypoventilation, was not found in this cohort. Indeed, the mean (SD) maximal partial pressure of carbon dioxide was very near physiologic levels of 40 mmHg,⁸ suggesting that the central physiologic responsiveness to CO_2 was maintained. In addition, favorable brain tension was reported in the vast majority of cases in this study. Previous reports of dose-related cardiovascular effects have described episodes of bradycardia of up to 27% during AC surgery,⁴³ but within this cohort we found no clinically

significant hypotension and only one episode of bradycardia with a surgical etiology requiring treatment.

Surgical failure during AC is defined as incomplete intraoperative awake monitoring of brain function during tumour resection, typically reported at approximately 2%.⁴⁴ Nonetheless, even with a continuously awake (i.e., awake-awake-awake) approach, there is still an inherent failure rate (approximately 2%) due to seizure activity.⁴⁵ Seizures have historically been the major cause of aborted AC surgery with reported incidences in the range of 3–22%. Nevertheless, phenytoin use is also associated with communication and AC failure, with no evidence of reducing perioperative seizure activity.⁴⁶ The seizure rate in our cohort was comparable to previous studies, and all seizures were triggered by cortical stimulation with most terminated with the application of cold saline.

Our study has some limitations. The retrospective nature of this study introduces several risks of bias and missing data. The surgical team provided information on alterations to the surgical plan following retrospective assessment of patient charts, which may have introduced recall bias. In addition, total doses of local anesthetics used by the surgical team were inconsistently recorded and could not be reported. In addition, formal pain and discomfort scores were not recorded for all patients. As AC surgery is relatively uncommon, there were insufficient data available prior to 1 March 2012 to make a comparison with non-dexmedetomidine anesthesia in our convenience sample.

Conclusions

Awake craniotomy for tumour resection using a dexmedetomidine-based anesthetic and scalp blocks resulted in no airway complications or conversion to general anesthesia. The anxiolytic and analgesic properties of dexmedetomidine enabled patients to remain awake and be surveilled during tumour resection. Further evaluations of clinical outcomes associated with this approach could include the duration of disease-free survival, time to tumour recurrence, and overall quality of life.

Conflict of interest The authors have no conflicts of interest to disclose.

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Author contributions Niamh McAuliffe was involved in the study design, ethics approval, data analysis, and manuscript preparation. Stuart Nicholson was involved in the data acquisition and data analysis. Andrea Rigamonti and Marco Garavaglia were involved in the manuscript preparation. Gregory M.T. Hare was involved in the study design and manuscript preparation. Michael Cusimano was involved in the data acquisition and manuscript preparation. Iryna

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