



Awake craniotomy and electrophysiological mapping for eloquent area tumours

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ABSTRACT

Objective: An awake craniotomy facilitates radical excision of eloquent area gliomas and ensures neural integrity during the excision. The study describes our experience with 67 consecutive awake craniotomies for the excision of such tumours.

Methods: Sixty-seven patients with gliomas in or adjacent to eloquent areas were included in this study. The patient was awake during the procedure and intraoperative cortical and white matter stimulation was performed to safely maximize the extent of surgical resection.

Results: Of the 883 patients who underwent craniotomies for supratentorial intraaxial tumours during the study period, 84 were chosen for an awake craniotomy. Sixty-seven with a histological diagnosis of glioma were included in this study. There were 55 men and 12 women with a median age of 34.6 years. Forty-two (62.6%) patients had positive localization on cortical stimulation. In 6 (8.9%) patients white matter stimulation was positive, five of whom had responses at the end of a radical excision. In 3 patients who developed a neurological deficit during tumour removal, white matter stimulation was negative and cessation of the surgery did not result in neurological improvement. Sixteen patients (24.6%) had intraoperative neurological deficits at the time of wound closure, 9 (13.4%) of whom had persistent mild neurological deficits at discharge, while the remaining 7 improved to normal. At a mean follow-up of 40.8 months, only 4 (5.9%) of these 9 patients had persistent neurological deficits.

Conclusion: Awake craniotomy for excision of eloquent area gliomas enable accurate mapping of motor and language areas as well as continuous neurological monitoring during tumour removal. Furthermore, positive responses on white matter stimulation indicate close proximity of eloquent cortex and projection fibres. This should alert the surgeon to the possibility of postoperative deficits to change the surgical strategy. Thus the surgeon can resect tumour safely, with the knowledge that he has not damaged neurological function up to that point in time thus maximizing the tumour resection and minimizing neurological deficits.

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1. Introduction

There is lack of class 1 evidence to prove that radical excision of low or high grade gliomas is superior to a partial excision with radiation therapy, however, many retrospective studies have shown benefits in terms of longer recurrence free survival in those patients undergoing more radical surgery [1–5]. An awake craniotomy is a reliable method of ensuring neural integrity during the excision of lesions located within or near eloquent areas. With the patient awake, it is possible to electrically stimulate the cortex to locate functional areas. Furthermore continuous clinical

neurological testing can theoretically detect early deficits in the motor, sensory or language domains during tumour removal. Knowledge that the patient's neurological function is intact up to that point in the surgery will give the surgeon confidence to continue with the excision probably resulting in a more radical excision. These surgeries are done for tumours in or adjacent to highly eloquent cortex and patients are bound to develop deficits if functional cortex is transgressed, retracted or made ischaemic [6]. The incidence of deficits reported by those groups who reserve awake craniotomies for tumours around eloquent cortex varies from 4.5 to 24% [7–9]. Naturally those workers [8] who use awake craniotomies for tumours far from eloquent areas will report low rates of neurological deficits. Haglund et al. [10] assert that tumour resection should be limited to within 1 cm of eloquent cortex as determined by intraoperative stimulation whereas Gil-Robles and

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Duffau [20] are more aggressive and have data to prove that excising tumour up to the very edge of the tumour-eloquent cortex margin results in temporary neurological deficits that recover in 6–12 months. We describe our experience in 67 consecutive awake craniotomies for the excision of tumours located in or around eloquent areas operated over an 8-year period with a view to correlate intraoperative and postoperative deficits with cortical stimulation findings.

2. Methods

The patients were selected for an awake craniotomy if they satisfied the following criteria: (i) parenchymal brain lesions in or adjacent to an eloquent area as seen on the magnetic resonance (MR) image. Eloquent areas were defined as those in or abutting the central lobule, supplementary motor area (SMA), dominant frontal operculum (Broca's area), dominant superior temporal gyrus (Wernicke's area) or dominant angular gyrus. Lesions in the premotor gyrus but extending deep into the white matter were also included [9]; (ii) a cooperative patient; (iii) age above 13 years [11,12]. Preoperatively, patients underwent detailed neurological and neuropsychological assessments and the entire surgical procedure was explained to them. On the day prior to surgery the patient was made familiar with the language testing that would be done intraoperatively. They were also asked to mention if they notice any abnormal sensations or feel an aura of a seizure during electrical stimulation or tumour removal.

2.1. Anaesthesia management

Anaesthesia for awake craniotomy starts with developing a rapport with the patient before the patient arrives in the operating room. The patient is comfortably positioned with a neck support, a pillow under the knee, and a warm-air blanket. The electrocardiogram, oxygen saturation and direct arterial pressure are monitored continuously and oxygen (2 L min^{-1}) is administered through a nasal cannula. After securing intravenous access, $50\ \mu\text{g}$ boluses of fentanyl and propofol or dexmedetomidine infusion, titrated to tolerate the circumferential scalp block requiring multiple injections, were given. A mixture of bupivacaine ($1\text{--}1.5\ \text{mg kg}^{-1}$) and lignocaine ($3\text{--}4\ \text{mg kg}^{-1}$), along with epinephrine in a concentration of $5\ \mu\text{g ml}^{-1}$ is used for the block targeted at the nerves of the scalp. The nerves anesthetized are the supraorbital and supratrochlear nerve by infiltrating from the nasal root to the midpupillary line; the zygomaticotemporal and auriculotemporal nerves by infiltration from the supraorbital ridge to the root of the zygoma; the lesser and greater occipital nerves by infiltrating from the mastoid to the external occipital protuberance. The Mayfield clamp pin-sites and the surgical incision are also infiltrated with local anaesthetic as an extra precaution should the scalp block be incomplete. A low dose infusion of propofol ($1\text{--}2\ \text{mg kg h}^{-1}$) is used to sedate the patient during incision and craniotomy. The propofol is discontinued and the dura infiltrated with lignocaine 2% using a 26G hypodermic needle by the surgeon under the microscope. The patient is awakened for cortical stimulation and neurological assessment during tumour removal. Intravenous fluids were restricted and adjusted to replace blood loss as urinary catheters were avoided.

2.2. Cortical stimulation

A bipolar stimulator with the tips 5 mm apart was used for the cortical stimulation (Nicolet Biomedical Inc., Madison, WI, USA). A strip of electrodes was placed over the cortex in close proximity to the stimulation zone to detect after-discharges during stimulation. If after-discharges were detected, the stimulating current

was reduced to avoid generating an intraoperative seizure. Typically, stimulation at a duration of 1 ms began with a current of 1 mamp increasing by increments of 1 mamp to a maximum of 6 mamps until responses were obtained. Sterile numbered tick-ets marked cortical areas of positive response. Contralateral motor movements occurred on stimulating motor areas. Patients reported paresthesiae when the primary sensory area was stimulated.

2.3. Language assessment

Language assessment was done by a neuropsychologist who administered the tests in the patient's native language. If the patient was unable to perform a particular test before surgery, that was excluded from intraoperative testing. Hence there was a preoperative baseline assessment of language function. The following tests were used [9,12].

Naming tasks: Fifty pictures of different objects were shown one by one to the patient from an alphabet book. The pictures were shown randomly and they were trained to name each object as it appeared. Cortical stimulation was applied as soon as they appeared.

Reading and serial counting tasks: We used a set of 20 different sentences. The patients were asked to read slowly and cortical stimulation was also done simultaneously. They were also asked to count numbers from 1 to 50 consecutively and cortical stimulation was performed simultaneously.

Sudden speech arrests occurred on stimulating the language areas during naming, reading or counting tasks. On operating in or near the angular gyrus, calculation and colour naming tasks were administered before and during stimulation. Calculation included single digit addition or subtraction. Colour naming was tested with colour plates of the various colours of the spectrum namely red, orange, yellow, green, blue, indigo, and violet. This initial entire cortical stimulation and brain mapping usually lasted about 10 min.

2.4. Surgical management and intraoperative ultrasonography

Intraoperative ultrasonography (IOUS) located lesions not seen on the surface and defined their margins. The shortest route to the lesion was chosen, the corticectomy or sulcal opening being tailored to the brain mapping and IOUS findings to avoid injury to eloquent cortex. We always began tumour removal farthest from the eloquent cortex and progressively moved towards eloquent cortex. Our policy was to continue with tumour resection either up to the sulcus delimiting the tumour from positively mapped cortex or into the eloquent cortex provided stimulation of the tumour did not elicit responses. In these cases, resection was guided by continuous neurological assessments and surgery was stopped only if neurological deficits occurred or normal cortex/white matter seen. Tumour extending into the eloquent cortex was not removed if positive responses were obtained on stimulation of the tumour. Upon registering the slightest weakness, delay in obeying a command or aphasia, the surgeon ceased to resect tumour or retract brain in that region and attention shifted to another part of the operating field. The patient was allowed to rest and re-tested after a few minutes until improvement noted; tumour removal continued in the new location always under continuous neurologic examinations. The IOUS helped detect residual tumour in the depths of the field or under overhanging edges of brain and further tumour removal continued with monitoring of neurological function and stimulation of white matter. After haemostasis and closure we removed the head clamp and transferred the patient to the intensive care unit for overnight monitoring. Patients were usually discharged on the 3rd postoperative day. All patients had postoperative plain and contrast CT scans on the 7th postoperative day and were referred to the radiation and medical oncologists depending on the histopathology

Table 1
Histological grading of the 67 tumours.

Histological classification	No. of patients
DNET	2
Pilocytic astrocytoma	1
Astrocytoma grade 2	11
Oligodendroglioma grade 2	7
Oligoastrocytoma grade 1	1
Oligoastrocytoma grade 2	14
Anaplastic astrocytoma grade 3	9
Anaplastic oligodendroglioma	9
Anaplastic oligoastrocytoma	5
Anaplastic ependymoma	1
Glioblastoma multiforme	7

report. Over the last year, with improvement in infrastructure, we have started doing hyperacute MRIs within 6–8 h of surgery.

3. Results

3.1. Patient characteristics

During the study period, 2002–2010, 883 patients underwent craniotomies for supratentorial tumours under the authors' supervision. Of these, 84 were chosen for an awake craniotomy based on the criteria mentioned above and of these, 67 with a histological diagnosis of glioma were included in this study (Table 1). There were 55 men and 12 women aged 13–58 years (median 34.6 years) with a mean follow-up of 40.8 months. One patient with an oligodendroglioma had undergone surgery 10 years ago under general anaesthesia and presented with a recurrent tumour.

3.2. Patient cooperation and duration of surgery

All patients tolerated the procedure well and none were converted to general anaesthesia. One patient with a left insular glioma, became restless during the craniotomy requiring deeper sedation that precluded intraoperative language and motor assessment during tumour removal. Surgery was restricted to a partial excision. This patient developed status epilepticus postoperatively and was ventilated for 72 h after which he was extubated and found to have a right hemiparesis that improved by the time of discharge.

3.3. Location of tumour, extent of excision and neurological deficits

Table 2 shows the distribution of the tumours by location, classified by the presence of preoperative deficits and extent of excision.

Language areas: We grouped the 12 patients with tumours around the dominant temporal operculum, posterior part of the inferior frontal gyrus and the angular gyrus into the “language” group. Of these, 6 had preoperative deficits – there were 4 patients with inferior frontal gyrus lesions who had a telegraphic speech

and there were 2 patients with dominant temporal lobe lesions who had paraphasic speech. In addition to these, 3 also had a mild facial weakness. We achieved a radical excision in 8 patients. In 7 of the 12 cases localization by stimulation of eloquent cortex was positive. Although 5 patients had intraoperative speech arrests during tumour excision, only 1 had persistent word finding difficulty at discharge (Table 3).

Insula: In 7 patients with left insular tumours, 2 had preoperative hemiparesis. We were able to locate the speech areas in 6 patients with stimulation. The tumours were radically excised in 2, subtotally in 4 and partially in 1. Intraoperative weakness during excision occurred in 5 that persisted in 4 as a mild weakness at the time of discharge. At follow-up, only 1 patient had persistent weakness.

Primary motor or sensory areas: Of 4 patients, 3 with motor gyrus tumours had preoperative weakness. Localization by stimulation was positive in 3 patients. The tumour in the sensory gyrus was radically excised with no postoperative weakness but with cortical sensory loss. The remaining tumours were biopsied or partially excised.

Supplementary motor area and premotor area: This formed the largest group with 32 cases. Nine patients had mild preoperative weakness. Localization by stimulation was positive in 24. Intraoperative worsening of deficits occurred in 8 cases, while another 2 had SMA weakness in the immediate postoperative period. Three patients had persistent deficits at discharge which completely improved at follow up. The excision was radical in 25, subtotal in 6 and partial in 1.

Prefrontal: There were 9 cases with tumours in front of the premotor gyrus. Only one patient had mild pyramidal signs. In two cases localization by stimulation of the motor gyrus was positive. Eight tumours were radically excised and none developed postoperative deficits.

4. Intraoperative electrophysiological monitoring and intraoperative events

4.1. Cortical and white matter stimulation

In 41 patients (61.1%), the language and/or motor areas were successfully located through intraoperative electrophysiological cortical stimulation. In 7, no response was elicited with electrical stimulation since the tumours were located in the prefrontal area, that is, in the gyrus in front of the premotor gyrus. In these patients radical tumour excision did not produce neurological deficits and white matter stimulation at the end of surgery was negative. In 14 patients (20.8%), electrophysiology failed to elicit responses despite the fact that the tumours were located near eloquent areas. In this group, 3 developed deficits intraoperatively. One was a right-handed patient with a tumour in the left insula, in whom cortical stimulation around the frontal and temporal opercula did not elicit speech arrest or motor responses. We

Table 2

Locations of gliomas in 67 patients operated by awake surgery showing their preoperative deficits, extent of excision, postoperative deficits and whether electrophysiology located motor/sensory or speech areas.

Location	Preoperative deficits		Extent of excision			Transient deficits	Deficits at the time of wound closure	EP+	Total
	Motor	Language	Radical	Subtotal	Partial/biopsy				
Language	3	6	8	4	0	5	2	7	12
Left insula	2	1	2	4	1	5	4	6	7
Primary motor or sensory	3	1	1	4	2	1	1	3	7
SMA/premotor	9	1	25	6	1	8	9	24	32
Prefrontal	1	0	8	1	0	0	0	2	9
Total	18	9	44	19	4	19	16	42	67

“Language” includes Wernicke and Broca’s areas and the dominant angular gyrus EP+ responses obtained with cortical electrical stimulation.

Table 3
Of the 67 patients undergoing awake surgeries, 9 patients had postoperative neurological deficits at discharge. At follow up, only 4 patients had persistent neurological deficits.

Location	Pre operative deficits	Extent of excision	Histopathology	Intraoperative event	Postoperative weakness at discharge	Follow up
Premotor right	Nil	Subtotal	Anaplastic astrocytoma 3	Slowing of movements and proximal muscle weakness	Persistent weakness	Persistent weakness
Insula left	Language dysfunction	Partial	Oligoastrocytoma grade 3	Hemiparesis	CT showed a small haematoma. Power improved after 72 h	Improved
Insula left	Nil	Subtotal	Oligodendroglioma grade 2	Mild facial and upper limb weakness and word naming difficulty	Persistent weakness	Persistent weakness
Insula left	Nil	Subtotal	Oligoastrocytoma grade 2	Temporary hemiparesis	Improved in 1 h	Improved
Insula left	Mild weakness	Subtotal	Oligoastrocytoma grade 3	Hemiparesis	Improved in 48 h	Improved
Motor cortex	Weakness	Biopsy	Anaplastic oligodendroglioma 3	Worsened after the biopsy	Some improvement next day	Persistent weakness
Premotor left	Nil	Radical	Oligoastrocytoma grade 2	Right facial weakness	Persistent weakness	Improved
Premotor left	Nil	Partial	Oligoastrocytoma grade 2	Transient dysarthria and difficulty in naming; probably manipulation of SMA	No deficits	Improved
Premotor left	Mild weakness	Subtotal	Oligoastrocytoma 1	Ankle dorsiflexion weakness; white matter stimulation + SMA motor deficit	Persistent weakness	Improved
Premotor right	Nil	Subtotal	Anaplastic astrocytoma 3	Worsened power	Improved by 3 months	Improved
Premotor right	Mild weakness	Subtotal	Anaplastic astrocytoma 3	Weakness thumb and index finger	Died status epilepticus	Improved
Premotor right	Nil	Radical	Recurrent astrocytoma grade 2	Weakness of left foot and pronator sign	Persistent weakness	Improved
Premotor right	Nil	Radical	Glioblastoma multiforme	Delayed response to commands	SMA syndrome	Improved
Premotor right	No	Radical	Astrocytoma grade II	Delayed response to commands with mild right hemiparesis	SMA syndrome with mild right hemiparesis	Mild right hemiparesis
Left SMA	No	Radical	Recurrent oligoastrocytoma II	Weakness right upper and lower limbs	SMA syndrome	Improved
Left SMA	No	Radical	Astrocytoma grade II			

proceeded with tumour removal, however, the patient developed a comprehension difficulty during excision of the superior half of the tumour – surgery was therefore terminated at a subtotal excision. The second was a patient with an oligodendroglioma in the dominant frontal operculum in whom the speech areas could not be identified with cortical stimulation and in whom the intraoperative development of cortical dysarthria resulted in a partial excision. The third case, a patient with a recurrent left SMA oligoastrocytoma grade III, had no motor responses to cortical stimulation. During tumour removal there were no events, however, immediately after a radical excision he was slow to respond to commands and white matter stimulation elicited gross movements of the right lower limb. Postoperatively he had an SMA syndrome with a mild motor aphasia that improved by 2 weeks.

4.2. Intraoperative neurological deficits

Table 3 shows the details in 9 patients (13.4%) who had persistent neurological deficits at discharge. In 6 of these patients, white matter stimulation was positive, 5 of whom had responses at the end of a radical excision when mild weakness or slowness to respond had already developed. In the other patient white matter stimulation elicited movement in the lower limb following which he had delayed responses to commands and his foot tapping became slow. This improved after a few minutes; however, we terminated the surgery at a subtotal excision. The remaining 3 patients developed a neurological deficit during tumour removal, white matter stimulation was negative and cessation of the surgery did not result in neurological improvement. Surgery did not proceed to radical excision in these patients. Postoperatively, 9 of the 16 patients had persistent mild neurological deficits at 1

week, while the remaining 7 improved to normal. However on follow-up, 5 more patients improved while 4 had persistent neurological deficits.

4.3. Morbidity and mortality

Intraoperative focal motor seizures occurred in 3 patients (4.4%) that ceased with cold saline irrigation and anticonvulsants. These patients had no further seizures intraoperatively or postoperatively. However, another 2 patients (2.9%) developed postoperative seizures 3 and 6 h after surgery that progressed to status epilepticus – neither had intraoperative seizures or postoperative hematomas. Curiously, both patients were operated upon the same day and had received higher doses of intravenous propofol as they became restless during the surgery. Both required ventilation and intravenous midazolam to control the seizures; one recovered with no residual deficits however the other patient expired due to status epilepticus and intractable brain swelling – the only mortality in the series. Two patients (2.9%) developed wound infection requiring antibiotics.

5. Discussion

5.1. Strategies for minimizing postoperative neurological deficits

Most authors reserve awake craniotomies for tumours around eloquent brain areas, however, a few recommend its usage for all supratentorial intraaxial tumours [8]. A detailed study of the pre-operative MR imaging determines the precise relationship between the tumour and functional areas of the brain [13,14]. When gliomas infiltrate peritumoural eloquent cortex, it is likely that the patient will have preoperative deficits that are prone to worsen if the

surgeon, however skilled, enters that gyrus to excise the tumour [15]. The traditional teaching that internal decompression of a tumour reduces the risk of new neurological deficits is not always true because both high and low-grade gliomas have been reported to contain functional tissue, including some tumours that consist principally of functional tissue [8]. Awake craniotomy with electrocortical stimulation mapping and subcortical white matter stimulation delineates a functional map of the brain surface [6–8,10,16,17] for motor and language functions. Following this, tumour resection that does not transgress language cortex and subcortical functional structures appears to result in a low incidence of postoperative deficits [6]. Our policy is to continue with tumour resection either up to the sulcus delimiting the tumour from positively mapped cortex or into the eloquent cortex; provided cortical stimulation of the tumour did not elicit responses.

5.2. Electrophysiological mapping

Our experience with direct cortical stimulation suggests that within the group of patients with tumours around eloquent areas, tumour excision proceeding up to positively identified cortex without leaving a margin places patients at a higher risk of developing postoperative deficits that are usually temporary. About 23.8% of patients developed postoperative deficits which were improved to 13.4% at the time of discharge. This further improved to 5.9% at follow-up. Furthermore, positive white matter stimulation during excision of the deeper portions of the tumour indicates the close proximity of projection fibres and therefore a high chance of postoperative deficits. Those in whom the resection stopped at the sulcus delimiting tumour from positively mapped cortex tended not to have postoperative deficits.

There appears to be two schools of thought regarding the method of dealing with the eloquent cortex–tumour interface. One group [18] prefer to limit the resection margin to more than 1 cm away from the nearest eloquent area as identified by direct cortical stimulation. These authors found that there was an improvement in language deficits when they left such a margin at surgery. In particular, they noted new language dysfunction in 13% of patients when the resection margins were <1 cm away from the nearest language site. This supports the argument that because of the infiltrating nature of gliomas, the periphery of the tumour is likely to occupy or merge imperceptibly with functional tissue [19].

On the contrary, Gil-Robles and Duffau [20] on the basis of cortical stimulation and imaging data question this practice. They proposed that gliomas in functional areas may be resected without leaving any margin between the tumour and positively mapped cortex. They elaborate on the principles of this strategy as being due to preservation of two layers of pia between the tumour and the eloquent cortex when there is a sulcus delimiting the tumour from eloquent cortex. However, when the tumour transgressed the sulcus into the positively mapped cortex, they continued the resection up to the edge of the positive stimulation site. They were able to achieve this successfully by moving the bipolar cortical stimulator 1–2 mm along the gyrus thus improving the spatial resolution of the mapping. In addition, they performed continuous neurological testing of language and motor functions that provided an online feedback. They emphasize the importance of preserving subcortical fibres. Using this strategy, >90% of their patients developed immediate post-surgical deficits that subsequently improved in 97% of them at 3 months. These authors go on to argue that leaving a 1 cm margin of tumour all around results in a very large residue. In actual practice, the 1 cm margin proposed by Haglund et al. [10] is probably restricted to the actual margin with eloquent cortex and not in other areas far away from functional cortex. Thus the residual volume would not be as large as predicted by Gil-Robles and Duffau [20]. From our experience, we feel that when a tumour is

completely delimited by a sulcus separating the tumour from functional cortex, an awake craniotomy with continuous neurological monitoring and white matter stimulation provides a safe environment for a radical excision without a tumour margin. With regard to the more difficult situation when there is no sulcal limit between positive cortex and tumour, we rely on direct cortical stimulation. If stimulating the tumour does not produce responses, we advocate a careful removal and cessation of surgery at the earliest sign of a deficit. These deficits, as shown in 16 of our patients, improved at follow-up persisting in only 4 (5.9%) of them. This is in keeping with the findings of Gil-Robles and Duffau [20]. On the other hand, if there is any positive response from the tumour, we would advocate leaving this tumour behind. In one of our patients a biopsy of tumour located within positively mapped motor cortex resulted in a permanent motor deficit. If a patient has a good outcome after excision of a glioma that on radiology appears to be within the motor cortex, it probably means that the motor cortex has been shifted due to the mass effect and the surgeon has remained within the confines of the tumour.

5.3. Negative electrophysiological mapping

Only 3 (14.2%) out of 21 patients with negative mapping had deficits. The negative mapping was probably not due to technical reasons since we changed the stimulating electrodes and their connections whenever a negative response was obtained. If no positive response is obtained on stimulation, the tumour is far away from an eloquent area or the craniotomy was too small to expose eloquent cortex. This is the explanation for our prefrontal tumours ($n=9$) in whom no positive response was obtained and in whom no postoperative deficit occurred. The possible reasons for occurrence of deficits with negative mapping apart from technical errors include transgression of white matter fibres at the depth or a vascular injury. If similar motor responses are obtained at the end of resection using the same current as that used at the onset of resection it is likely that the patient will recover full motor function, even if there is an immediate postoperative deficit [6]. This is also the experience of Kim et al. [7] who found that 21% of patients in whom eloquent areas were positively identified, experienced worsening of neurological deficits at 1 month compared to 9% in patients in whom eloquent areas were not identified. Sanai et al. [21] published their series of 250 patients who underwent surgery for gliomas with language mapping. Language areas were positively identified in only 58% of the patients due to small tailored craniotomies and barely 1.6% had a persistent language deficit at 6 months. Thus extensive craniotomies exposing large areas of cortex to map all functional areas is not necessary as tailored craniotomies with negative mapping permit aggressive safe resection as long as continuous neurological monitoring is performed [22].

5.4. Intravenous anaesthetics and neurological deficits

In one of our patients with a left frontal opercular oligodendroglioma, we located the Broca's area through cortical stimulation and did a radical excision of the tumour. During tumour removal he experienced no speech or motor difficulties. For the wound closure we administered an intravenous propofol infusion and discovered that while he was partially sedated he developed a weakness in the right upper limb. The intraoperative ultrasound showed no tumour bed haematoma nor did we witness any seizures. Following the closure the propofol infusion was stopped. The weakness persisted until he reached the recovery room 20 min later when the power was documented to be normal. This phenomenon has been described by Thal et al. [23] who documented worsening of pre-existing neurological deficits during induction of general anaesthesia on the administration of sedatives, midazolam or

fentanyl to patients undergoing surgery for brain tumours or carotid artery disease. Although they did not study the nature of deficits postoperatively on recovery from anaesthesia they suggest that transient postoperative neurological deficits may be drug related as seen in our patient whose weakness improved soon after stopping the propofol. The potential mechanisms stated are altered drug uptake, distribution and metabolism in diseased or abnormal areas of the brain. Also neurons in and around abnormal brain may be more sensitive to sedatives [23].

5.5. Postoperative deficits in SMA gliomas and insular gliomas

The commonest cause of a postoperative deficit is 'aggressive' resection of tumour tissue and manipulation of peritumoural brain [15]. The commonest instance of this problem is mutism and hemiplegia that usually recovers following resection of tumours of the dominant supplementary motor area (SMA). Deficits may be due to the characteristics of the area of resection (e.g. SMA), functioning brain in the brain tumour margins [15], post surgical oedema and haemodynamic changes. Only 3 (9.3%) of 32 patients with SMA gliomas developed postoperative neurological deficits which improved to normal at follow up.

Resection of insular gliomas is a challenge to neurosurgeons due to close proximity of neural and vascular structures. Postoperative deficits are usually due to ischaemia owing to manipulation/damage to the lateral lenticulostriate arteries or excessive retraction of the frontal operculae [24,25]. In our series, only 1 out of 7 patients with left sided insular tumours developed a new permanent postoperative deficit. Thus dominant hemisphere insular gliomas should have an awake craniotomy to improve outcome [26].

5.6. Complications of awake craniotomy

Seizures are the commonest complication of an awake craniotomy that may be provoked by cortical stimulation [7,8,15]. Three of our patients had intraoperative and 2 had postoperative status epilepticus leading to death in 1 patient. We had no systemic infections in this series, probably related to avoidance of endotracheal intubation, urethral catheterisation and the patients have a shorter hospital stay [8,9,15].

6. Conclusions

Awake craniotomy with electrocortical stimulation for eloquent area tumours facilitates removal of a large tumour volume with good functional outcome particularly in dominant hemisphere insular and SMA gliomas. Continuous intraoperative monitoring of motor and language functions is mandatory to prevent neurological deficits with a prompt change in surgical strategy.

Conflict of interest

The authors do not report any conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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