Awake craniotomy for removal of intracranial tumors is most challenging procedure. The critical aspect of awake craniotomy is to maintain adequate analgesia and sedation, hemodynamic stability, airway safety, while keeping the patient immobile for duration of surgery, cooperative for neurological testing. The routine blood investigations were normal; the patients did not have any other systemic illnesses.

The aim of this cases report was to present a sedation technique with dexmedetomidine infusion combined with bupivacaine for scalp block for intracranial tumor resection under awake craniotomy. This technique allowed the main surgical steps without the occurrence of major complications, such as psychomotor agitation, hemodynamic changes, and over sedation, without airway manipulation and, mainly, it did not affect the patients’ cognitive evaluation.

MATERIALS AND METHODS: After obtaining institutional ethics board approval and written patient consent five patients of intracranial tumors were enrolled were awake craniotomy. The patients’ details were shown in the table. The routine blood investigations were normal; the patients did not have any other systemic illnesses. All members of the team were briefed in advance so that a calm atmosphere prevails in the operating room. On arrival into operation theatre two 18G IV cannulae were inserted and pantocid 40mg as an acid prophylaxis, midazolam 1mg for anxiolysis, ondansetron 4mg antiemetic, fentanyl 100mcg for analgesia and levetiracetam 500mg for seizure prophylaxis were administered intravenously. Oxygen 3L/min was administered through nasal prongs.

The patient was continuously monitored for ECG, oxygen saturation, noninvasive blood pressure, end tidal CO2 and level of sedation was monitored by bispectral index. The bladder was catheterised because of prolonged urinary retention. Dopamine at 5 mcg/kg/min and midazolam 1 mg for anxiolysis were titrated to maintain sedation. The bladder was catheterised because of prolonged urinary retention. Dopamine at 5 mcg/kg/min and midazolam 1 mg for anxiolysis were titrated to maintain sedation.
duration of procedure and as there is need for mannitol infusion.

Before placing the patient into operative position bolus dose of dexmedetomidine infusion 1mcg/kg administered over 20 minutes, followed by continuous infusion at a rate of 0.2-0.5mcg/minute (titrated BIS index to 70-90). The patient was placed in supine position with head slightly rotated operative side facing up. Care was taken for adequate visibility of the patient to anaesthesiologist during intraoperative testing. Fentanyl 20mcg IV was administered intermittently to provide additional analgesia and patient comfort.

Supraorbital, supratrochlear, zygomaticofacial, auriculotemporal, greater auricular, greater and lesser occipital nerves were blocked using 20 ml of 0.5% bupivacaine. We have limited total dose to 3mg/kg body weight. In Addition 1% lignocaine was infiltrated at the three head holder pin sites and along skin incision line. Adrenaline (5ug/ ml) was added to minimize acute rises in plasma anaesthetic concentration and maximise the duration of the block.[5] 20gms mannitol was infused about 15 min prior to completion of craniotomy. A pad soaked in 1% lignoaine was applied over dura to provide analgesia during dural opening. Once the dura was opened, brain was found to be relaxed and pulsating. For localization of primary motor cortex, our patients were asked to "move your toes/fingers, squeeze the ball" and they are well educated and trained about these tasks in preoperative visits. The procedures have lasted for about 3-4 hr. At the end of the surgery, all the patients except one were fully awake and communicating. One patient was converted to general anesthesia due uncontrolled seizures. All the patients were shifted to the intensive care unit for further observation. The patients' haemodynamic parameters were stable throughout the procedure. The post-operative course was uneventful and discharged after 5 days.

**DISCUSSION:** There is an increase in number of indications in intracranial surgery for the patient to be awake during some or all of the operation. To achieve a goal of increased lesion removal, improved survival benefit,[1] shorter hospitalization time, reduced cost and a decreased incidence of postoperative complications, a sound anatomical knowledge of the nerve blocks and the knowledge to predict intraoperative events is extremely rewarding for the neuroanaesthetist.[6,7]

One of the most important considerations for awake craniotomy is careful patient selection. The patient should cooperate in an unfamiliar and stressful environment for an extended period of time. Infact, the only absolute contraindication for awake technique is an uncooperative patient. A Our anesthesia team in preoperative visit detailed about expected discomforts and level of cooperation expected, potential and tasks that will be performed for motor testing.

There is considerable variation in the anesthetic management of the awake craniotomy in different institutions. Propofol sedation, commonly in combination with a shorter acting opioid such as fentanyl, or remifentanil, is an effective and popular technique during awake craniotomy, achieving a high degree of patient satisfaction and acceptance. A blinded, prospective, randomized study while comparing the efficacy of dexmedetomidine versus propofol-remifentanil based sedation in patients undergoing awake craniotomy for resection of tumors, hypothesised that the efficacy of performing intra-operative brain mapping is identical between dexmedetomidine and the propofol-remifentanil based sedation.[8]

In our patients, combination of the dexmedetomidine infusion and the scalp block with bupivacaine provided effective sedation, analgesia and allowed full excision of the tumor, without damage or deficits for the patient who had early mobilization and was discharged uneventfully.[9]

Dexmedetomidine (highly specific α2-adrenoceptor agonist) has potential application during awake craniotomy for tumor resection because they provide analgesia and sedation that is easily reversed with verbal stimulation. There is no risk of respiratory depression.[9] The primary action is excitation inhibition in the central nervous system. It is a cerebral vasoconstrictor by stimulating α2b receptors in the cerebral blood vessels with no effect on CMRO2. Dexmedetomidine inhibits the cerebral vasodilatation induced by hypercapnia, thus avoiding increased intracranial pressure and brain bulging. Dexmedetomidine also has anticonvulsant effects that might be helpful during epilepsy surgery or tumor resection. The analgesic effect of dexmedetomidine consistently reduces opioid administration.[10] In a case report by Basavaraj G Kallapur and Raghavendra Bhosale, by administration of dexmedetomidine 1–0.5 mcg/kg/h and propofol 60 mg/h, achieved adequate sedation, analgesia and fully cooperative patient to perform cognitive tests successfully once the infusions were stopped 10 min prior.[10]

In our case series the level of sedation during Dexmedetomidine infusion was monitored by BIS index, maintained BIS score between 70-90 by increasing level of sedating during positioning and head pin fixation and reducing it during testing for functional area intraoperatively.

Patient tolerance of an awake craniotomy relies on effective analgesia of the surgical field, and cannot rely on sedation or anaesthesia alone. In our case scalp block combined with local infiltration provided adequate analgesia, haemodynamic stability and decreased the stress response to painful stimuli.

Complications of awake craniotomy include seizures, cerebral edema, nausea and vomiting, decreased level of consciousness, neurological deficit, pain, and loss of patient cooperation.[1,11] Airway management is uneventful during awake craniotomy under sedation in our reported cases. However, over sedation inevitably runs the risk of apnoea and airway obstruction. Airway obstruction (incidence 0% to 20%) with oxygen desaturation (0-28%) may result in brain swelling because of elevated levels of Paco2.[12]
Equipment for emergency airway control should be available throughout awake craniotomy.
Seizures may occur unexpectedly (0% to 24%) due to decreased levels of anticonvulsants or local anesthetic toxicity. Most of the seizures can be resolved by irrigation of the surgical field with cold saline or administration of propofol.[13] An antiepileptic prophylaxis may be helpful to prevent intraoperative seizures. However, one of reported cases developed uncontrolled seizures in spite of prophylactic measures.

During the procedure precautions were taken to prevent shivering by using warm infusions and blanket.[1] Dexmedetomidine can cause hypotension and bradycardia. However the patients in our case series were hemodynamically stable.

CONCLUSIONS: In our case series, infusion of dexmedetomidine 0.2-0.5 mcg/kg/h and scalp block, has achieved adequate sedation and analgesia during awake craniotomy. By monitoring depth of anesthesia with BIS, we could achieve adequate sedation without airway compromise. Our patients were fully co-operative for cognitive testing. We conclude that dexmedetomidine as an adjunct to scalp block is useful during awake craniotomy for tumour resection.

REFERENCES:

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Table 1: Patient details of case series
Case 1: BIS Monitoring (Spacelab workstation with inbuilt BIS module)

Case 2: Positioning for awake craniotomy