Hemodynamics and Respiratory Stability during Awake Craniotomy: Dexmedetomidine-Based Sedation Versus Propofol-Based Sedation

MOSTAFA M. ELADANY, M.D.*; FATMA M. KHAMIS, M.D.*; MAGDY A. OMERA, M.D.*; AMGAD A. MATAR, M.D.** and HOSSAM M. MOSTAFA, M.D.*

The Departments of Anesthesiology & Intensive Care* and Neuro-Surgery**, Faculty of Medicine, Suez Canal University, Egypt

Abstract

Background: Awake craniotomy aims to remove most of the brain tumour while preserving the eloquent area of the brain, many factors affecting hemodynamics and respiration during awake craniotomy especially sedative drugs: Type and doses.

Aim: To detect hemodynamics and respiratory effects of dexmedetomidine-based sedation and propofol-based sedation in patients undergoing awake craniotomy for resection of brain tumours, which has encroachment on eloquent area of the brain.

Patients and Methods: Randomized, prospective, comparative clinical trial study was carried out on 28 patients of both sexes undergoing elective surgery for brain tumor resection in the routine surgical lists in Suez Canal University Hospital, from January 2012 to April 2014, patients are divided into two equal groups (14 patients): Propofol group and dexmedetomidine group.

Results: There were more hemodynamically stability and respiratory stability among dexmedetomidine group, there were statistically significant difference between the two groups in some time intervals.

Conclusions: Dexmedetomidine provide more hemodynamically stability and less effects on respiration during awake craniotomy.

Key Words: Awake craniotomy – Hemodynamic – Dexmedetomidine – Propofol.

Introduction

SPACE occupying lesions in or adjacent to eloquent areas of the cortex, excision of tumours in the sensory and motor areas in the dominant hemisphere and sensorimotor cortex in either hemisphere that might otherwise be considered inoperable [1]. Tuominen, et al. [2] suggested that awake craniotomy with bipolar cortical stimulation may help to reduce the risk of postoperative impairment following resection of tumors located in or near speech and motor areas also under intra operative magnetic resonance imaging IMRI control.

Generally, anaesthetic aims for awake craniotomy technique are Maintaining patient cooperation, optimal analgesic care, adequate sedation and anxiolysis during the different surgical steps with comfortable positioning plus nausea, vomiting, and seizure prevention and Homeostasis: Safe airways and adequate ventilation; Hemodynamic stability; Normal intracranial pressure [3].

Propofol used to be standard drug for sedation in monitored anesthesia care (MAC) for awake craniotomy [4].

The reported adverse effects of propofol are: Pain on injection [5], Bradycardia [6], Arterial hypotension [7], Bloodstream infection [8], Airway obstruction, Changes in serum lipids [9], Excitation of the CNS [10], including seizures in susceptible patients [11]. Other uncommon complications include hypertriglyceridemia and pancreatitis. High-dose propofol infusions may be associated with the “propofol syndrome” [12].

Dexmedetomidine is a new highly selective α2-agonist with sedative, analgesic, and anesthetic-sparing effects [13]. It does not suppress ventilation. The primary action of α2-adrenergic receptor agonist is the inhibition of norepinephrine release that causes attenuation of excitation in the central nervous system. Small-dose infusion of this drug in healthy volunteers provided sedation that could be easily reversed with verbal stimulation [13].
Patients and Methods

After Approval of the Ethical and Scientific Committee of Faculty of Medicine, Suez Canal University, and obtaining informed written consent from each patient. This study is a randomized, prospective, comparative clinical trial study, was carried out on 28 patients of both sexes undergoing elective surgery for brain tumor resection in the routine surgical lists in Suez Canal University Hospital, Ismailia, Egypt from January 2012 to April 2014.

Inclusion criteria of the patients were: (1) Patients aged 21-65 years. (2) Both sexes. (3) Surgical indications involve: Resection of brain functional area metastases, meningiomas, and low grade gliomas which has an encroachment on eloquent area of the brain. The eloquent cortex areas were defined as the motor areas (precentral gyrus) and speech areas (left frontal operculum, angular gyrus and superior temporal gyrus) Localization of the brain tumors were carried preoperatively via CT and MRI with contrast. (4) Patients are ASA I (American Society of Anesthesiologists physical status Grade I) = (normal healthy patients) or ASA II (American Society of Anesthesiologists physical status Grade II) = (patients with mild systemic disease that is well controlled and no functional limitations).

Exclusion criteria of the patients: (1) Refusal of the patient. (2) Any disorder of the cardiovascular, respiratory, hepatic, renal, systems known from history or clinical examination. (3) Patients in whom difficult intubation is expected [Mallampati score (class 3 or 4)], as the patient may need intubation during the operation. (4) Obesity, BMI (Body mass index) greater than 30kg/m². (5) Obstructive sleep apnea syndrome.

During the preoperative visit, the techniques were explained to patients including benefits and complications, data obtained from the patients including: A- Demographic data such as age, sex, ASA status, occupation, marital status, residency and BMI. B- Medical history: (1) Medical disorders as hypertension, diabetes, heart, chest, liver or kidney diseases, (2) Past history of operations or hospitalization, (3) Past anesthetic history with impact on previous airway problems during previous surgery, drug hypersensitivity, any previous post-operative complications that could be attributed to anesthesia, (4) History of convulsions, anticonvulsant or neurological deficits, other neurological symptoms as: Headaches, speech deficit and motor deficit and (5) Smoking. C- Physical examination includes: a- General examination and vital signs (heart rate, blood pressure, respiratory rate and temperature), b- Heart, chest and abdominal examinations, c- Neurological examination, for any neurological deficits or abnormalities (e.g. weakness). D- Anesthetic assessment; Airway assessment and examination of sites of local anesthesia. E- Investigations: Were done as required per case according to the pre-operative anesthesia clinic protocol.

All regular medications especially anticonvulsant were continued till the time of surgery.

IV cannula insertion for the patient by 18G or 20G cannula was performed. Premeditation with ranitidine 50mg iv, half an hour before induction, in the pre-operative holding area.

In the operative theatre standard anesthetic monitoring included an electrocardiogram (ECG), noninvasive arterial blood pressure (NIBP), pulse oximetry, and end-tidal carbon dioxide (ETCO₂) and respiratory rate measured via an oxygen delivery nasal cannula with oxygen supplement delivered at 4 L/min. Central venous catheter, invasive arterial cannula and urinary catheter are optional. Prophylactic antibiotics, ondansetron (4mg), dexamethasone (8mg), and metoclopramide (10mg) were given intravenously before induction of anesthesia to prevent nausea and/or vomiting.

Anesthesia technique was monitored anesthesia care (MAC) technique but could be changed to general anesthesia with laryngeal mask or endotracheal tube, if there is difficulty in maintaining airway of the patient or the patient cannot tolerate any step of the operation.

Local anesthesia was performed for all patients as skull block; performed to six nerves on each side; 40 to 60mL anesthetic volume is used for infiltration (a mixture of lidocaine 0.5% and bupivacaine 0.25%, with epinephrine 1:200 000). (1) Auriculotemporal nerve (mandibular branch of trigeminal nerve): Infiltration over zygomatic process and distal temporal artery; (2) Zygomaticotemporal nerve (zygomatic nerve’s terminal root that originates from maxillary branch of trigeminal nerve): Infiltration from supraorbital margin to posterior part of zygomatic arch; deep and superficial injections are recommended since the area above the temporalis fascia is the most frequently reported site of postoperative pain; (3) Supraorbital nerve (root of frontal nerve which originates from ophthalmic branch of trigeminal nerve): Infiltration from the nasal root to the
midpoint of the eye; (4) Supratrochlear nerve (root of frontoalveolar nerve which originates from ophthalmic branch of trigeminal nerve): Infiltration together with supraorbital nerve; (5) Greater occipital nerve (posterior ramus of C2): infiltration about 2.5 cm lateral to the nuchal’s median line, directly medial to occipital artery; (6) Lesser occipital nerve (anterior branches of C2 and C3): Infiltration 2.5 cm lateral to greater occipital nerve, plus local anesthetic infiltration was applied also to sites of Mayfield headholder pins and sites of skin incision.

Group 1 (propofol group): (14 patients)

The patients of this group received bolus fentanyl (0.5-1 \( \gamma \)g/kg) and bolus propofol (0.5-1mg/kg). Then, maintenance with a continuous infusion of propofol (2-5mg/kg/h) by using syringe pump. The target level of sedation is “3” of Observer’s Assessment of Alertness/Sedation Scale during the procedure. During brain functional mapping examination, the infusion of propofol is discontinued to achieve level of sedation of 5 of Observer’s Assessment of Alertness/Sedation Scale.

Patients were placed on the operating table in the correct position for surgery (lateral or supine) with skull fixed using Mayfield headholder, using extra cushions and padding to ensure maximum patient comfort Maintenance IV fluids was consisted of normal saline at the rate of 50 to 100mL/h, and replacing losses as needed.

At any time during the procedure when excessive pain is expected, additional anesthesia was given by increasing the propofol infusion and/or fentanyl bolus (50\( \gamma \)g) plus infiltration of the local anesthetic into the pin sites of skull clamp fixation device, scalp and dura matter. Approximately 10 minutes before neurological testing (verbal, sensory and motor testing), propofol was discontinued. All patients were awake and conversant within 10-20 minutes of stopping the medications.

Functional localization was performed by applying an electric current to the cortical surface with the N 50 modified bipolar instrument (Fisher; Leibinger, Germany). When searching for the primary motor cortex, stimulation was started with a frequency of 20-50 Hertz, pulses of 0.5-1 milliseconds, and a current as low as 2 MA and usually up to 5-7 MA. When searching for sensory cortex or language, stimulation was initiated with the same settings, but the current was increased to 10-15 MA. Mapping and monitoring neurological function was continued in cortical and subcortical areas throughout the surgical procedure.

Language functions was evaluated with standard stimuli and methods used by neurologists and neuropsychologists for the clinical assessment of aphasia. The evaluation included tests of receptive language (comprehension), repetition, and expressive language (fluency). As visual stimuli could not be presented to many patients due to position and draping, the language evaluation used auditory stimuli only. Immediately prior to electrical stimulation mapping, a thorough baseline evaluation was completed against which performance during stimulation could be judged. As the language tasks and stimuli that was used are relatively simple and are performed perfectly by normal adults, any dysfluency, arrest, or error (i.e. paraphasic, perseverative, or inappropriate response) during stimulation mapping was considered significant. When a site tested positive, stimulation was removed until speech returned to baseline, and then was retested at least once to confirm the result.

Motor functions was mapped by exposing and observing the appropriate side of the patient’s body, watching for involuntary muscle activity at the same frequency as the electrical stimulation. Somatosensory functions was mapped using patient self-report of somatosensory phenomena. As with language testing, sites positive for somatosensory function was retested at least once to confirm the reliability of the patient’s self-report.

Mapping and monitoring neurological function was performed throughout the surgical procedure.

After completion of the examination, the predetermined conscious sedation technique was resumed for tumor resection and closure with a propofol infusion (2-5mg/kg/h), a supplemental dose of fentanyl (50\( \gamma \)g).

Group 2 (Dexmedetomidine group): (14 patients)

The patients of this group received a bolus of fentanyl (0.5-1 \( \gamma \)g/kg) and dexmedetomidine infusion as a loading dose (0.5-1 \( \gamma \)g/kg) over 10 minutes and then, maintenance infusion of 0.2 to 0.7 \( \gamma \)g/kg per hour. The low end of this range was started and only increased if the patient reports discomfort. The target level of sedation is “3” of Observer’s Assessment of Alertness/Sedation Scale during the procedure except during brain functional mapping examination, the target level of sedation is “5” of Observer’s Assessment of Alertness / Sedation Scale.

Patients were placed on the operating table in the correct position for surgery (lateral or supine) using extra cushions and padding to ensure maxi-
Hemodynamic stability in awake craniotomy: dexmedetomidine versus propofol.

At any time during the procedure when excessive pain is expected, additional anesthesia was given by increasing the dexmedetomidine infusion and/or fentanyl bolus (50 µg) plus infiltration of the local anesthetic into the pin sites and scalp. Approximately 10 minutes before neurological testing (verbal, sensory and motor testing), dexmedetomidine was discontinued. Neurology testing (verbal, sensory and motor testing) was performed while surgical resection took place (as mentioned in group 1).

All patients (both groups) received fentanyl 1 µg/kg for postoperative analgesia at the time of skin closure. Postoperatively the patients were monitored in the ICU for any neurological deterioration due to cerebral oedema, intracranial haemorrhage and seizures. Postoperative pain was treated with IV fentanyl. IV ondansetron was given for nausea and vomiting if needed.

Assessment of the two techniques:

Arterial blood pressure, heart rate, oxygen saturation, respiratory rate and ETCO₂ were measured at: T1-Before induction of anesthesia (baseline), T2- After induction, T3-Time of skin incision and surgical stimulus. T4-Time of stoppage of sedation, T5-Time of arousal and functional mapping testing, T6-At the end of the surgery.

Statistical analysis: Collected data were processed using SPSS version 15 (SPSS Inc., Chicago, IL, USA). Quantitative data were expressed as means ± SD while qualitative data were expressed as numbers and percentages (%). Student t-test and ANOVA test were used to test significance of difference for quantitative variables that follow normal distribution and Chi Square was used to test significance of difference for qualitative variables. A probability value (p-value) <0.05 was considered statistically significant.

Results

Table (1) shows the hemodynamic monitoring in the studied groups during follow-up intervals. There was significant lower mean heart rate in dexmedetomidine group than in propofol group at T3, T4 and T6 time intervals (p≤0.01) (Graph 1). There was significant lower MAP in dexmedetomidine group than in propofol group at T3 time interval (p<0.001) (Graph 2).

Table (1): Hemodynamic monitoring in the studied groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Propofol group (n=14)</th>
<th>Dexmedetomidine group (n=14)</th>
<th>t-test</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>84.93 ± 7.216</td>
<td>87.00 ± 6.62</td>
<td>–.64</td>
<td>0.532</td>
</tr>
<tr>
<td>T2</td>
<td>78.00 ± 5.987</td>
<td>88.36 ± 6.61</td>
<td>–1.2</td>
<td>0.252</td>
</tr>
<tr>
<td>T3</td>
<td>88.36 ± 5.982</td>
<td>85.00 ± 5.68</td>
<td>4.81</td>
<td>0.0001**</td>
</tr>
<tr>
<td>T4</td>
<td>78.29 ± 5.823</td>
<td>70.79 ± 5.22</td>
<td>–3.01</td>
<td>0.01**</td>
</tr>
<tr>
<td>T5</td>
<td>77.93 ± 6.391</td>
<td>74.00 ± 6.85</td>
<td>1.69</td>
<td>0.114</td>
</tr>
<tr>
<td>T6</td>
<td>87.14 ± 4.400</td>
<td>76.07 ± 5.73</td>
<td>–4.95</td>
<td>0.0001**</td>
</tr>
<tr>
<td>MAP:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>103.86 ± 6.503</td>
<td>105.64 ± 6.52</td>
<td>–.72</td>
<td>0.484</td>
</tr>
<tr>
<td>T2</td>
<td>95.14 ± 8.132</td>
<td>92.21 ± 5.04</td>
<td>–1.1</td>
<td>0.276</td>
</tr>
<tr>
<td>T3</td>
<td>101.21 ± 6.363</td>
<td>90.29 ± 4.71</td>
<td>4.6</td>
<td>0.0001**</td>
</tr>
<tr>
<td>T4</td>
<td>84.64 ± 6.356</td>
<td>80.29 ± 5.37</td>
<td>–2.0</td>
<td>0.063</td>
</tr>
<tr>
<td>T5</td>
<td>94.00 ± 7.835</td>
<td>91.57 ± 4.60</td>
<td>.93</td>
<td>0.368</td>
</tr>
<tr>
<td>T6</td>
<td>97.36 ± 5.108</td>
<td>89.21 ± 23.88</td>
<td>–1.4</td>
<td>0.198</td>
</tr>
</tbody>
</table>

Graph (1): Mean heart rate (beats/min) in the studied groups. There was significant lower mean heart rate in dexmedetomidine group than in propofol group at T3, T4 and T6 time intervals (p≤0.01).

Graph (2): Mean arterial pressure (MAP; mmHg) in the studied groups. There was significant lower MAP in dexmedetomidine group than in propofol group at T3 time interval (p<0.001).
Table (2) shows the Respiratory rate (RR) and End-tidal CO\textsubscript{2} (ETCO\textsubscript{2}) monitoring in the studied groups during follow-up intervals. There was significant higher mean RR in dexmedetomidine group than in propofol group at T2 time interval ($p<0.05$) (Graph 3).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Propofol group (n=14)</th>
<th>Dexmedetomidine group (n=14)</th>
<th>$t$-test</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory rate</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>T1</td>
<td>14.43</td>
<td>1.91</td>
<td>14.21</td>
<td>1.71</td>
</tr>
<tr>
<td>T2</td>
<td>9.5</td>
<td>1.28</td>
<td>12.28</td>
<td>1.48</td>
</tr>
<tr>
<td>T3</td>
<td>13.64</td>
<td>1.86</td>
<td>14.85</td>
<td>1.65</td>
</tr>
<tr>
<td>T4</td>
<td>14.71</td>
<td>1.48</td>
<td>13.5</td>
<td>2.4</td>
</tr>
<tr>
<td>T5</td>
<td>16.57</td>
<td>1.28</td>
<td>14.5</td>
<td>1.4</td>
</tr>
<tr>
<td>T6</td>
<td>13.78</td>
<td>2.01</td>
<td>13.5</td>
<td>1.5</td>
</tr>
</tbody>
</table>

ETCO\textsubscript{2}: | Mean | SD | Mean | SD |          |          |
| T1 | 30.07 | 2.23 | 30.35 | 2.09 | -0.34 | 0.73 |
| T2 | 35.42 | 3.27 | 32.64 | 2.76 | 2.43 | 0.022 ** |
| T3 | 26.42 | 2.76 | 28.92 | 3.47 | -2.106 | 0.044 * |
| T4 | 32.92 | 2.81 | 30.57 | 2.95 | 2.16 | 0.04 * |
| T5 | 29.17 | 5.08 | 31.07 | 2.97 | -1.23 | 0.22 |
| T6 | 28.21 | 4.09 | 29.78 | 4.88 | -0.92 | 0.36 |

There was significant higher mean ETCO\textsubscript{2} in propofol group than in dexmedetomidine group at T2 and T4 time interval ($p<0.05$) (Graph 3).

There was significant higher mean ETCO\textsubscript{2} in dexmedetomidine group than in propofol group at T2 and T6 time intervals ($p<0.05$) (Graph 4).

Graph (4): Mean end-tidal CO\textsubscript{2} (ETCO\textsubscript{2}, mmHg), in the studied groups. There was significant higher mean ETCO\textsubscript{2} in propofol group than in dexmedetomidine group at T2 and T4 time interval ($p<0.05$). There was significant higher mean ETCO\textsubscript{2} in dexmedetomidine group than in propofol group at T3 time intervals ($p<0.05$).

Table (3) shows the oxygen saturation (SpO\textsubscript{2}) monitoring in the studied groups during follow-up intervals. There was significant higher mean SpO\textsubscript{2} in dexmedetomidine group than in propofol group at T2 and T6 time intervals ($p<0.05$) (Graph 5).

Graph (5): Mean pulse oxygen saturation (SpO\textsubscript{2}; percentage) in the studied groups. There was significant higher mean SpO\textsubscript{2} in dexmedetomidine group than in propofol group at T2 and T6 time intervals ($p<0.05$).
Hemodynamic Stability in Awake Craniotomy

Discussion

Propofol, a γ-aminobutyric acid A (GABAA) agonist, the medication commonly used for conscious sedation in awake craniotomy, has the advantages of rapid onset, antiemetic and antiepileptic properties [14]. Propofol was considered the drug of choice for awake craniotomy using MAC technique, however, numerous studies have demonstrated the some shortcomings of propofol, including long arousal time, incidence of intra-operative complications/complaints and interference with the monitoring of intra-operative mapping such as electrocorticographic recording [15,16].

Dexmedetomidine, a highly selective agonist of α2-adrenergic receptors, produces dose-titrated sedation, which resembles natural sleep, decreases anxiety, pain, and cerebral blood flow [14]. These properties make dexmedetomidine a potentially advantageous sedative agent in awake craniotomy [17,18].

To compare the efficacy and safety of dexmedetomidine versus propofol, this randomized, prospective, comparative, clinical trial study was conducted on 28 patients of both genders undergoing elective surgery for brain tumor resection at Suez Canal University Hospital in the routine surgical lists. The patients were randomly assigned into one of two groups; propofol group or dexmedetomidine group.

The major site of noradrenergic innervation in the brain with the highest concentration of presynaptic α2-adrenergic receptors is the locus ceruleus, which is responsible for arousal, sleep, and anxiety. Dexmedetomidine acts at the locus ceruleus areas but does not involve the GABA receptors. Consequently, dexmedetomidine provides a sedation that resembles natural sleep without cognitive impairment [18]. As a result, despite the 120-minute elimination half-life of dexmedetomidine, patients may be easily awakened by verbal stimulation without having to stop the drug infusion [19].

In patients of dexmedetomidine group, our results documented that there were hemodynamic stability (significant lower mean heart rate and MAP and higher mean SpO₂ in dexmedetomidine group than in propofol group). Dexmedetomidine can then act on presynaptic α2 receptors and negative feedback regulation of the release of adrenaline. This results in a role similar to the peripheral ganglion blocker, giving it sympatholytic properties that provide hemodynamic stability and reduced surgically induced anxiety and agitation [14,18].

The usage of propofol for conscious sedation in awake craniotomy has several disadvantages as well. Transfusion of propofol can result in bradycardia and hypotension, as found in our study, which is consistent with previous studies [15]. Bradycardia and hypotension triggered by propofol were dose dependent and had no remarkable detrimental effect on cardiac function. Both conditions were readily treated with vasoactive agents [20].

There was significant lower mean heart rate in dexmedetomidine group than in propofol group at T3, T4 and T6 time intervals (p<0.01). There was significant lower MAP in dexmedetomidine group than in propofol group at T3 time interval (p<0.001). There was significant higher mean RR in dexmedetomidine group than in propofol group at T2 time interval (p<0.05) and there was significant higher mean RR in propofol group than in dexmedetomidine group at T5 time interval (p<0.05).

There was significant higher mean ETCO₂ in propofol group than in dexmedetomidine group at T2 and T4 time interval (p<0.05). There was significant higher mean ETCO₂ in dexmedetomidine group than in propofol group at T3 time intervals (p<0.05). There was significant higher mean SpO₂ in dexmedetomidine group than in propofol group at T2 and T6 time intervals (p<0.05).

Additionally, administration of propofol produced respiratory depression and obstruction (two patients in our study).

Meanwhile, no patient in dexmedetomidine group had any complain of hypotension or respiratory obstruction.

The patients of dexmedetomidine group (n=14) received bolus of fentanyl (0.5-1 γ g/kg) and dexmedetomidine infusion as a loading dose (1 γ g/kg) over 10 minutes and then maintenance infusion of 0.2 to 0.7γ g/kg per hour. The low end of this range was started and only was increased if the patient reports discomfort.

Souter et al. (2007) suggested administration of a bolus of 0.3 γ g/kg dexmedetomidine then infusion 0.2 to 0.5γ g/kg/h allows successful sedation for awake craniotomy with epileptic foci and motor mapping.

Bustillo et al. (2002) found that, in conjunction with midazolam, dexmedetomidine infusion for sedation significantly prevented neurologic and cognitive testing at infusion rate recommended by manufacturer (0.2-0.7 γ g/kg/h). The case reported
by Bekker et al. (2001) showed that, in conjunction with sevoflurane, they had to reduce the dexmedetomidine infused concentration to half of the lowest infusion rate recommended (0.1 g/kg/h) to perform cognitive testing.

In Shen et al. (2013) study, dexmedetomidine was administered 1.0 μg/kg followed by a maintenance dose of 0.2 to 0.7 g/kg/h.

Conclusion:
Then we concluded that either dexmedetomidine or propofol can be effectively and safely administered for conscious sedation in awake craniotomy. More hemodynamically stability and less impact on respiration suggest that dexmedetomidine might be a more suitable sedative agent in awake craniotomy.

References
المملص السريري

قدمت هذه الدراسة رؤى جراحات الدم وانبعاث الكهرباء في البدائل المتذكرة، وذلك لذالك بدور السريع في المج في المج تحت تأثير مخبر مضاعف ومتعدد الملاحظات- مع البقاء على المريض مستمتفتًا - لتشمل العديد من جراحات الدم خصوصاً أسلوب البيولوجيا في مختلف المراكز الطبية فيها مثل مراكز الكهرباء والأنفية الرئيسية لجراحة الأوعية مع بقاء المريض مستمتفتاً هو تسهيل تحليلات كهرباء قلب الدم أثناء العملية ورسم الخرائط البشرية لتحديد دقيق للملاحظات الدم التي تتحكيم في وظائف الحركة والكلام. وتم هذه الجراحات باستمرار من أسلوب في التخدير وأن كانت كلما تتفق في الاعتياد على المخدر الموضعي والمسكنات والمهنوات.

هدف البحث: مقارنة أسلوبين لدراسة التخدير لذالك هذه النوعية من الجراحات من حيث التفاعل على استقرار العلامات الحيوية للمريض والاستقرار البدني. الأسلوب الأول المعتد على عملي تريبيوفيتر والاسلوب الثاني المعتد على حذر الدعاء سكسيتوميدين.

أسلوب البحث: تم عمل مقارنة بين الأسلوبين في الدراسة لدى المرضى الذين خضعوا لجراحة لازالة أورام الدم السري الذئبي في القيادة من المراكز الطبية ويتراوح عمرهم من 20-75 عاماً حيث تم تقسيم 28 مريض ألي مجموعة عشوائية إلى 14 مريض تم استخدام التهوية المعتد على عقار الدوالي بيفووال و24 مريض تم استخدام التهوية المعتد على عقار الدوالي بيفووال، وفي الـ 28 مريض تم مقارنة العلامات الحيوية ومعدلات التنفس في ستة أوقات تمت التغيرات الأكبر.

النتائج: أظهرت التحليل الإحصائي استقرار العلامات الحيوية لكل المجموعتين وجود دقة عالية لدراسة إحصائية في بعض أوقات العملية لصالح عقار الدوالي بيفووال، وأظهرت التحليل الإحصائي استقرار معدلات التنفس في المرضى لصالح عقار الدوالي بيفووال، مقابلة عقار الدوالي بيفوال.

الخلاصة: أظهرت الدراسة ثبات العلامات الحيوية والتنفس بشكل أفضل في حالة عقار الدوالي بيفووال مقارنة عقار الدوالي بيفوال مما يجعل عقار الدوالي بيفووال مناسب لحالات جراحات الدم مع البقاء على المريض مستمتفتًا أثناء الجراحة.