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1. Skull blocks for awake craniotomy anesthetize all of the following nerves EXCEPT:

a. Auriculotemporal nerve
b. C4 cervical nerve root
c. Supratrochlear nerve
d. Branches of the ophthamic division of the trigeminal nerve
e. Lesser occipital nerve
a. Auriculotemporal nerve

The auriculotemporal n. is a branch of the mandibular division (3rd) of the trigeminal nerve and is sensory to the temple.

b. C4 cervical nerve root

The greater occipital nerve arises from C2 and innervates the posterior scalp

c. Supratrochlear nerve

The trigeminal nerve gives off a first division (v1- the ophthalmic division) which branches off into the the frontal nerve which then divides into the supratrochlear and supraorbital nerves. These nerves help provide sensation to the forehead.

Osborne et al, “Scalp block” during craniotomy: a classic technique revisited
JNA, 2010;22(3):187-194
d. branches of the ophthalmic division of the trigeminal nerve

This branch of the trigeminal nerve will ultimately split into the supraorbital and supratrochlear nerves.

e. lesser occipital nerve

The lesser occipital nerve arises from C2 and C3 cervical nerve roots

2. All of the following are true regarding noxious stimuli associated with craniotomy under general anesthesia EXCEPT:

a. Application of the mayfield pin head holder may lead to undesirable increases in ICP
b. Efforts to blunt the hemodynamic response to direct laryngoscopy can help protect patients from dangerous increases in transmural pressure (TMP) and mean arterial pressure (MAP) during craniotomy for aneurysm clipping
c. There is some evidence that skull blocks help to blunt the hemodynamic response to the craniotomy incision
d. Another noxious stimuli is the pain from resection of the brain itself
a. Application of the mayfield pin head holder may lead to undesirable increases in ICP

Certain aspects of craniotomy can induce noxious stimulation, namely laryngoscopy, insertion of cranial pins, incision and periosteal-dural contact. Consequent increases in blood pressure and heart rate can cause further increases in intracranial pressure in patients with brain pathology as well as cause an increased risk of aneurysm rupture in patients with intracranial aneurysms.


*Pinosky et al, The effect of bupivicaine Skull block on the hemodynamic response to craniotomy, A and A, 1996;83:1256-61*
b. Efforts to blunt the Hemodynamic response to direct laryngoscopy can help protect patients from dangerous increases in transmural pressure (TMP) and mean arterial pressure (MAP) during craniotomy for aneurysm clipping.

Controlling blood pressure during induction in patients with intracranial pathology is helpful in limiting increases in ICP. The incidence of aneurysm rupture during induction of general anesthesia (1-2%) has a high mortality and is usually precipitated by a rise in blood pressure after direct laryngoscopy and intubation. Some anesthetic adjuncts successful in preventing the rise in blood pressure include narcotics, β-adrenergic antagonists, intravenous or topical lidocaine, additional barbiturates or propofol, or deepening the inhalational anesthetic.

*Cotrell and Young’s Neuroanesthesia, 5th ed., Ch. 13*  
Saunders, Elsevier, 2010
c. There is some evidence that skull blocks help to blunt the hemodynamic response to the craniotomy incision.

Blocking noxious input to the sensory nerve supply of the scalp helps to prevent the hemodynamic response to pin head holder application and the craniotomy incision. In a double blind prospective study, 21 patients were administered either saline (control) or 0.5% bupivicaine for skull blocks after induction and 5 min. before pin insertion. Significant increases in blood pressure and heart rate were evident in the control group whereas blood pressure remained unchanged in the bupivicaine group.

Osborne et al, “Scalp block” during craniotomy: a classic technique revisited, JNA, 2010


d. Another noxious stimuli is the pain from resection of the brain itself

The uncomfortable parts of the awake craniotomy procedure are the mayfield pin head holder, the craniotomy, and manipulation of the dura. Manipulation of the brain parenchyma is painless due to the lack of nociceptors present there.


3. Regarding post-craniotomy pain, which of the following statements is TRUE?

a. Post-craniotomy pain is not a problem for neurosurgical patients
b. Practitioners are hesitant to administer systemic opioids after craniotomy because of their impact on neurological assessment and respiratory depression.
c. Non-steroidal anti-inflammatory agents are ideal for post op pain in this patient population
d. IM Codeine and Morphine have no benefit in pain after craniotomy but do carry a risk of N and V
e. There is no evidence that regional skull blocks are helpful in reducing post craniotomy pain
At least 60% of patients experience moderate to severe pain after craniotomy. Furthermore, pain intensity during the acute postoperative period predicts later development of chronic pain. Kauer et al published a study of 145 patients seizure free after undergoing anterior temporal lobectomies for epilepsy over a 9 year period. Six percent of patients complained of headache lasting between 2 months and 1 year after surgery and 12% had ongoing headache at 1 year post op. Of these, a quarter had medically uncontrolled headaches while one third still required regular analgesia a year after surgery.


b. Practitioners are hesitant to administer large doses of systemic opioids after craniotomy because of their impact on neurological assessment and respiratory depression.

Concern for elevated arterial CO2 concentrations from opioid-induced respiratory depression causing increased ICP together with sedation, miosis, nausea and vomiting masking the signs of increased ICP are reasons some practitioners hesitate with administration of opiate administration. In a survey of members of the neuroanaesthesia society of Gr. Britain and Ireland, Only three percent said that they would use opioids other than IM codeine postoperatively. Fear regarding opioids in this group stemmed from the risk of respiratory depression and sedation. However, Stoneham et al and others have found no adverse effects from the use of PCA morphine in studies investigating analgesia for post op craniotomy patients.


c. Non-steroidal anti-inflammatory agents are ideal for post op pain in this patient population

While non-steroidal anti-inflammatory drugs may enhance analgesia and reduce postoperative opioid consumption, there is the risk of postoperative hematoma due to their effect on platelet function. In a 5 year retrospective study of 7000 craniotomies, 1.1% of patients returned to the operating room for postoperative surgical evacuation of hematoma. In this group, administration of antiplatelet agents (aspirin and NSAIDS) was the most commonly associated risk factor.

*Palmer, Postoperative haematoma: a five-year survey and identification of avoidable risk factor. NS, 1994;35:1061-5*
d. IM codeine and morphine have no benefit on the pain after craniotomy but do carry a risk of nausea and vomiting

Morphine may provide more consistent and improved analgesia compared to IM codeine, but with increased risk for PONV.

In one study of 30 post-craniotomy patients randomized to receive either IM codeine vs morphine PCA, Stoneham et al noted similarly decreased pain scores in the groups with no difference in the incidence of nausea and vomiting.

In another larger study (N=120) of craniotomy patients (skull base tumors) a morphine PCA with ondansetron group had lower pain scores than a placebo group and a morphine group but the ondansetron did not seem to reduce the incidence of nausea and vomiting.


e. There is no evidence that regional skull blocks are helpful in reducing post craniotomy pain

In a Metaanalysis conducted by Guilfoyle et al., (single or double blind randomized controlled trials comparing pre- or postoperative regional skull block (RSB) against no intervention, systemic analgesia or other local intervention, in adults undergoing craniotomy), the authors concluded that Regional skull block (RSB) was associated with a significant reduction in pain for several hours after craniotomy. They found a significant reduction in pain scores up to 6 to 8 hours postoperatively and subgroup analysis showed RSB had a longer duration of action when performed postoperatively compared with preoperative injection. There were no complications attributable to RSB in any of the included trials.

4. Regarding systemic toxicity from local anesthetic injection, which of the following statements is TRUE?

a. **In order to decrease the risk of systemic toxicity, the recommended maximum dose of Lidocaine 2% is about 300 mg**

b. **Epinephrine hastens absorption of local anesthetic.**

c. **Patient factors like age and hepatic function are not necessary when determining the maximum safe amount of local anesthetic.**

d. **Hypercapnia and acidosis decrease the amount of free LA available to the brain putting patients at risk for seizures.**

e. **Propofol administration to treat systemic local anesthetic systemic toxicity can act as a substitute for lipid emulsion therapy.**
a. In order to decrease the risk of systemic toxicity, the recommended maximum dose of Lidocaine 2% is about 300 mg

Maximum recommended doses for local anesthetics are provided in order to prevent the administration of excessive amounts of local anesthetic which could result in systemic toxicity. While it is helpful to be aware of dose limitations of injection of Lidocaine (300 mg); Lidocaine with epinephrine(500mg) Bupivacaine, without epi (175mg) and with epi (225 mg); it is important to also consider a patient’s age (adjust dose for those <4months or >70 years), patient comorbidities like renal or liver dysfunction, and the site of injection.

b. Epinephrine hastens absorption of local anesthetic

The vasoconstriction provided by epinephrine is helpful not only in prolonging the intended conduction blockade, but this will also help slow absorption of local anesthetics thereby minimizing increases in plasma levels. The recommended concentration is 1:200,000 (5mcg/ml). Epinephrine should not be added to blocks of the digits or penis because of the risk of tissue ischemia.

*Miller’s Anesthesia. 8th ed., Philadelphia, PA: Elsevier Churchill Livingstone; 2015:Ch 57*

c. Patient factors like age and hepatic function are not important when determining the maximum safe amount of local anesthetic.

Immature liver function contributes to lower levels of alpha-1 acid glycoprotein (for binding drugs like lidocaine) in infants (<4 months) resulting in more available drug. This added to decreased clearance of LA in newborns leads to higher more sustained plasma levels and an increased risk of systemic toxicity. Lower doses of local anesthetics, therefore are recommended for this age group.

Deteriorating nerve conduction together with decreased clearance of local anesthetics are reasons to adjust the limits on maximum doses of local anesthetics in the elderly.

Amides like lidocaine and bupivacaine are metabolized in the liver and metabolites excreted by the kidney. Patients with decreased hepatic blood flow and or decreased enzyme function may have a decreased ability to clear local anesthetics administered. Furthermore these patients oftentimes have associated renal and cardiac disease which can also contribute to significantly elevated local anesthetic levels.


Rosenberg, Maximum recommended doses of local anesthetic: a multifactorial concept, Regional anesth and pain medicine, 2004, 29(6):564-575
d. Hypercapnia and acidosis decrease the amount of free local anesthetics available to the brain putting patients at risk for seizures.

It is critical that practitioners recognize the signs of local anesthetic systemic toxicity which typically involves the central nervous system (CNS) and cardiovascular system. Seizures a manifestation of increased local anesthetic in the brain will lead to hypercapnia and acidosis which will worsen toxicity. Hypercapnia leads to increased local anesthetic delivered to the brain courtesy of increased cerebral blood flow. Lower intracellular pH will increase conversion of local anesthetic from its base form to its less diffusible cation form causing ion trapping and increased CNS toxicity. Immediate attention to the airway and ventilation may help to prevent progression to cardiac arrest.

*Miller’s Anesthesia. 8th ed., Philadelphia, PA: Elsevier Churchill Livingstone; 2015:Ch 36*

e. Propofol administration to treat systemic local anesthetic toxicity can act as a substitute for lipid emulsion therapy.

Once signs and symptoms of local anesthetic (LA) systemic toxicity are recognized, immediate attention to securing the airway in order to limit continued worsening of hypoxia and acidosis is paramount. Treatment with benzodiazepines, thiopental, and/or propofol can help eliminate seizure activity however caution is advised in the face of cardiovascular depression. The early use of Lipid emulsion acts as a “lipid sink” to bind local anesthetic and pull this agent from cardiac tissue thereby facilitating improved hemodynamics.

The recommended dose of lipid emulsion is 1.5ml/kg (LBW) of a 20% lipid emulsion followed by an infusion of 0.25ml/kg/min x10 min after hemodynamic stability is established. An additional bolus and increase in the infusion to 0.5ml/kg/min is recommended for persistent hemodynamic instability. Upper limit (10ml/kg over 30 min).

Propofol's low lipid content (10% lipid content) may not be used as a substitute for lipid emulsion therapy. Furthermore, the large doses of lipid emulsion needed to treat LA systemic toxicity make propofol with its myocardial depressant effect a risky alternative.

5. Risks of skull block may include all of the following EXCEPT:

a. **Intravascular injection**

b. **Nerve injury**

c. **Injection into the subarachnoid space**

d. **Hematoma**

e. **Phrenic nerve injury**
a. Intravascular injection

Intravascular injection can lead to local anesthetic systemic toxicity which will typically occur 15 minutes after injection. Three hundred fifty-four records of craniotomy patients for epilepsy performed under conscious-sedation analgesia were reviewed and two cases of seizures were temporally related to skull block administration. Epinephrine added to local anesthetics for scalp infiltration is helpful for hemostasis in an area with a rich vascular supply, however in Pinosky’s study demonstrating a benefit of skull block in blunting the hemodynamic response to head pinning, bupivacaine 0.5% without a vasoconstrictor was chosen to avoid the risk of hypertension from inadvertent intravascular administration. Based on proximity to the nerves to be blocked, vessels that are most likely to be directly injected include the superficial temporal artery (nearby the auriculotemporal nerve) and the greater occipital artery (nearby greater occipital nerve).


b. Nerve injury

It is important to use anatomic landmarks for locating the nerves to block for successful and safe skull analgesia. The proximity of the facial nerve to nerves injected during “scalp block” (branches of the trigeminal nerve) makes facial weakness/paralysis a potential complication (manifesting as unilateral weakness of the face). However, no cases have been reported in the literature.

c. Injection into the subarachnoid space

Okuda et al reported inadvertent injection of local anesthetic (without epinephrine) into the subarachnoid space during an occipital nerve block to help a patient with occipital headaches whose past surgical history included a retromastoid craniotomy for microvascular decompression which left him with an occipital bone defect. Despite a brief loss of consciousness, hypoventilation and nausea, the patient had no sequelae. A palpable bony defect is probably a relative contraindication to performing a skull block.

d. Hematoma

Before proceeding with skull blocks, be sure clotting abnormalities have been corrected by the neurosurgical team.

e. Phrenic nerve injury

Although the lesser occipital nerve is a branch from the ventral rami of C2 and C3, this nerve is blocked at the level of the occiput which is well above the cervical spine roots C3, 4, and 5 which give rise to the phrenic nerve.