Journal of Clinical Anesthesia (2013) **25**, 150–159

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Journal of Clinical Anesthesia

Review Article

A review of scalp blockade for cranial surgery☆

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Received 7 October 2011; revised 30 April 2012; accepted 8 June 2012

Keywords:
Acute pain;
Chronic pain;
Intracranial surgery;
Local anesthetics;
Neuroanesthesia;
Regional anesthesia;
Scalp blockade

Abstract Neural blockade of the scalp may be used as an adjunct to general anesthesia or serve as the principal anesthetic for both intracranial and extracranial procedures. Effective scalp blockade typically requires anesthetizing multiple peripheral nerves; blockade of one or more of these is often used to diagnose and treat conditions such as chronic headache. Improved anatomic knowledge has refined the use of scalp blockade so that directed neural blockade is achieved. The vascularity of the scalp, proximity of arteries supplying the cerebral circulation, use of large volumes of local anesthetic, and presence of intracranial devices or bony defects require attention. The impact of perioperative scalp blockade on acute and chronic pain may offer insight into the benefits of perioperative neural blockade generally.

1. Introduction

Neural blockade of the scalp has a history that extends over several eras of anesthetic and neurosurgical practice. It was first used to permit intracranial surgery when general anesthesia for these procedures had not yet advanced sufficiently to be considered safe [1], later to permit functional mapping during surgery without interference from general anesthesia [2] and, most recently, as an adjunct to general anesthesia to maintain hemodynamic control [3] and provide perioperative analgesia [4-6]. Although generally indicated for intracranial surgery, scalp blockade is useful for extracranial procedures of the scalp [7,8]. Components of scalp blockade also are used to treat chronic headache [9]. A century of experience leaves little doubt as to the efficacy of scalp blockade as a stand-alone technique for intracranial procedures of the anterior and middle cranial fossae. Recent studies have shown clear efficacy when scalp blockade is used as an adjunct to general anesthesia when hemodynamic control is the outcome. A growing but incomplete body of literature suggests a role, perhaps an important one, for scalp blockade or surgical site infiltration with local anesthetics (LA) to improve postoperative analgesia. However, the existing literature does not yet clearly delineate the mixture of patient factors, surgical factors, adjunctive analgesics, and technique of scalp blockade or LA infiltration for which meaningful analgesic benefits are reliably obtained.

2. History

The concept of peripheral nerve block of the scalp was developed in the early 1900s by Harvey Cushing and George

☆ Supported by departmental funding only.

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http://dx.doi.org/10.1016/j.jclinane.2012.06.024

Crile and was motivated by their appreciation of the potential benefits of effective regional anesthesia [10,11]. Progress in this area was limited by the available LA. Cocaine was addictive and toxic to the heart and brain, whereas procaine, apart from its short duration of action, was limited by an increased frequency of allergic reactions [11,12]. Heinrick Braun's addition of epinephrine to LA had the benefit of increasing the duration of the LA effect [13]. Scalp blockade initially was performed to permit execution of intracranial procedures without the need for general anesthesia [1]. With the introduction of endotracheal tubes and the evolution of general anesthesia, the use of scalp blockade declined in frequency. However, it retained an important niche in permitting functional assessment of awake patients during certain procedures [2]. There continues to be a need to perform intracranial surgery and concurrent functional assessments [14-16]. Both the availability of longer-acting LAs and the evolution of the technique for scalp blockade from a field block to selective neural blockade have opened new opportunities for its integration into current practice.

3. Indications

Neural blockade of the scalp is applicable for all supratentorial intracranial procedures. Scalp blockade minimizes the hemodynamic response to surgical stimulus, it may decrease intraoperative anesthetic requirements, and it may reduce postoperative pain and analgesic consumption [3,4]. Although it can provide effective anesthesia for skin incision and craniotomy, it does not provide anesthesia of the dura [17]. As such, for "awake" craniotomy, in addition to scalp blockade, an intravenous (IV) analgesic is often necessary for patient comfort but it must be titrated to permit functional assessment. Scalp blockade also can be used in other subspecialty surgery involving the cranium, including dermatological (photodynamic surgery for actinic keratosis, resection of infiltrating carcinoma of the scalp) and plastic (cranioplasty) surgery [7,8].

An important component of scalp blockade includes block of the greater occipital nerve. This technique has been used in headache clinics for decades. It has utility for patients with occipital neuralgia, migraine, cluster headache, and post-concussive headache [9]. Positive data from randomized, double-blinded studies support its use in occipital neuralgia and cluster headache [18,19]. More recently, the short-term benefit of greater occipital nerve block has evolved to the implantation of an occipital nerve stimulator for chronic migraine [20]. As with the greater occipital nerve, the third occipital nerve can be a generator of chronic cephalgia, particularly after whiplash-induced injury to the C2-C3 zygapophysial joint [21]. The third occipital nerve has a cutaneous distribution that projects to the lower portion of the scalp.

4. Technique

A field block, obtained by haphazard ring blockade of the scalp, may cause the administration of an unnecessarily high volume of concentrated LA. Girvin [14] first described an anatomically directed scalp blockade technique in 1986 for awake patients during intracranial surgery. The procedure involves injection of LA to block the divisions of V1 (supratrochlear and supraorbital nerves), V2 (zygomaticotemporal nerve), V3 (auriculotemporal nerve), and branches derived from C2 and C3 (posterior branch of the great auricular, lesser occipital, greater occipital, and third occipital nerves) (Fig. 1).

The supratrochlear and supraorbital nerves are anesthetized as they emerge along the superior orbital rim with 1 mL of LA for each nerve, using a 23 or 25-gauge needle introduced perpendicular to the skin. In an anatomical study, the supratrochlear nerve emerged from the orbital rim an average 1.6 cm lateral to midline and 0.7 cm below the supraorbital margin, while the supraorbital nerve emerged

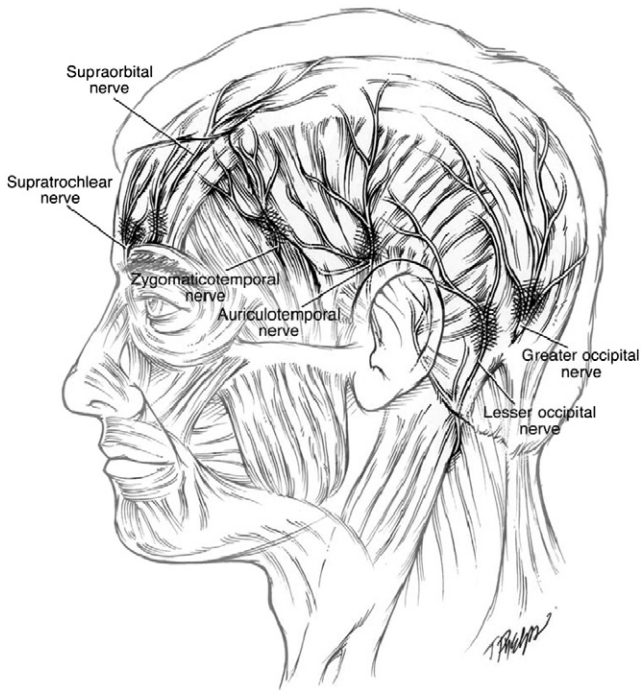

Fig. 1 Anatomical basis for scalp blockade. A scalp block sufficient for awake craniotomy or as an adjunct to general anesthesia can be performed with injections at the crosshatched regions. Special mention is made of the auriculotemporal nerve, which may be blocked earlier in its course than what is shown above, specifically 1.5 cm anterior to the tragus, as described by Pinosky et al [3]. Pinosky et al call for a deep fascial injection, but a superficial injection at this location is sufficient to block the nerve, while a deep injection unnecessarily risks facial nerve block. The zygomaticotemporal nerve may be anesthetized anywhere along its course from its emergence 1 cm posterior to the lateral canthus. (Adapted with permission from Gottschalk A, Yaster M. *Neurocrit Care* 2009;10:387–402 [31]).

from the supraorbital foramen 2.9 cm lateral to midline and 0.5 cm below the supraorbital margin [22]. The supraorbital nerve is blocked simply by palpation and injection at the supraorbital notch. The supratrochlear nerve is anesthetized with a second injection 1–1.5 cm medial to the supraorbtial notch.

The zygomaticotemporal nerve arises between the supraorbital and auriculotemporal nerves with its foramen located on the anterior wall of the temporal fossa behind the lateral orbital rim at the level of the lateral canthus [23,24]. Deep and superficial planes should be injected as the nerve may branch extensively. Blockade of this nerve may be helpful for frame placement, awake intracranial surgery, and especially for surgery in which the temporalis muscle is retracted or raised from the cranium. The nerve may be anesthetized by palpating the lateral orbital rim at the level of the frontozygomatic suture. The index finger is left in the depression of the posterior lateral aspect of the lateral orbital rim and the needle introduced approximately 1 cm posterior to the suture. The needle should be "walked down" the concave wall of the lateral orbital rim until it reaches the level of the lateral canthus. Two mL of LA generally is recommended for an effective block [24].

Blockade of the auriculotemporal nerve is possible at several levels. For low temporal intracranial surgery, it should be blocked 1.5 cm anterior to the tragus with infiltration of LA superficially (as deep injection may unnecessarily anesthetize the facial nerve). Otherwise, the auriculotemporal nerve may be effectively blocked 1–1.5 cm anterior to the superior border of the pinna, obviating any risk of facial nerve blockade [25]. The posterior branch of the great auricular nerve is blocked 1.5 cm posterior to the pinna at the level of the tragus. Targeting this nerve is not absolutely necessary for routine scalp blockade, as the sensory contribution is minimal. However, blockade may be beneficial for surgery centered near the mastoid process (ie, acoustic neuroma resection, particularly for the translabyrinthine approach). One mL of LA should be sufficient for blockade of each of the aforementioned nerves.

Finally, the lesser and greater occipital nerves are blocked along the superior nuchal line between the external occipital protuberance and mastoid process. Linear injection of the middle third of the superior nuchal ridge with 5 mL of LA will anesthetize both the lesser and greater occipital nerves. The greater occipital nerve runs alongside the occipital artery, which can be inadvertently punctured with attempted blockade of this nerve. On average, the greater and lesser occipital nerves lie along the nuchal ridge 4 cm and 7 cm from the external occipital protuberance, respectively [26].

The third occipital nerve lies an average of only 3 mm (range, 0–4 mm) lateral to the external occipital protuberance and communicates with the greater occipital nerve, which lies lateral to it [26]. It innervates the lower portion of the posterior aspect of the cranium. This distribution of innervation is unlikely to be important for incisional pain for most supratentorial procedures. However, its contribution to infratentorial procedures is likely much greater. In addition, the initiation of its subcutaneous course 6 cm below the external occipital protuberance and just lateral to midline suggests a site amenable to blockade. In headache clinics, blockade of the third occipital nerve is performed at the root level during fluoroscopic or ultrasonic guidance [21,27].

Finally, any pin sites outside of the intended anesthetic region defined for a given patient may be infiltrated with LA. The effect of pin site injection by itself [28] and in combination with scalp infiltration [29] improved pain scores for up to 48 hours.

Although not traditionally considered a component of scalp blockade, unilateral or bilateral superficial cervical plexus blockade can be considered for infratentorial and occipital procedures [30]. The superficial cervical plexus is formed by the ventral primary rami of C2-C4 and innervates the anterolateral portion of the neck. The rami emerge as 4 distinct nerves along the posterior border of the sternocleidomastoid. The second root usually gives rise to the lesser occipital nerve before it continues to unite with a portion of the third root to form the greater auricular nerve and transverse cervical nerves. The sensory distribution covered by this plexus also renders it a suitable anesthetic in patients for whom surgical access to the region of the carotid bifurcation is contemplated (ie, for carotid endarterectomy or extracranial control of the carotid). It is blocked by an injection at the midpoint of the line connecting the mastoid process and Chassaignac's tubercle (C6). Injection should be made with a fan technique 2–3 cm superior and inferior to this point. The injection should be subcutaneous and behind the sternocleidomastoid muscle, and care should be taken not to exceed 1–2 cm in depth.

Although the dura is innervated by the same cranial and spinal nerves that provide extracranial innervation, it is important to stress that dural innervation is more complex and less accessible to blockade [17]. This situation is the basis for the response to noxious stimuli during disruption of the dura in "awake" patients. Moreover, surgical disruption and inflammation of the dura may lead to postoperative cephalgia despite effective scalp blockade. This intracranially derived pain will be referred to the corresponding somatic neural distribution [31]. In general, neural branches follow adjacent arterial and venous structures. If it becomes necessary, the neurosurgeon performs careful LA injections between dural leaflets, guided by the arterial and venous structures of the relevant portion of the dura.

5. Risks and controversies

As with any type of neural blockade, the risks of LA toxicity due to systemic absorption, direct intravascular injection, or inadvertent administration directly to a specific site must be considered. Systemic plasma concentrations of LAs increase rapidly after scalp blockade, with signs of LA

toxicity presenting during the first 15 minutes after injection [32,33]. Consistent with the vascularity of the scalp, the rate of rise of serum LA concentration is faster than during other types of neural blockade [34-36]. Clinically, complications due to the toxic effects of LA absorption are rare. However, reaching the threshold of systemic LA toxicity results in seizures, or hemodynamic instability leading to cardiac arrest. Among patients undergoing awake craniotomy for intractable epilepsy, two cases of seizures that were temporally related to LA injection during scalp blockade were reported [37]. In contrast to the toxicity observed from systemic absorption, the effects of direct intravascular or intrathecal injection are seen hyperacutely after only a few milliliters of LA have been administered. In the case of direct intravascular injection, high concentration of LA in the central nervous system (CNS) likely results from retrograde flow of the anesthetic to an artery supplying intracranial structures. Vessels that are most likely to be directly injected include the superficial temporal and occipital arteries.

In a rare case, LA was inadvertently introduced into the subarachnoid space of a patient who had an occipital bone defect after a microvascular decompression; the patient was undergoing lesser occipital nerve blockade for occipital headache. The patient lost consciousness but returned to normal after two hours [38]. Injection into shunts and reservoirs used for the intraventricular administration of medication similarly must be avoided. Finally, the anesthesiologist must be mindful of the potential damage to nerves by intraneural injection or local pressure from the LA wheal.

Bupivacaine, ropivacaine, and levobupivacaine are long-acting LAs suitable for use in scalp blockade. Ropivacaine and levobupivacaine have a better safety profile than bupivacaine. Specifically, in experimental protocols, the therapeutic window of levobupivacaine and ropivacine was greater than with bupivacaine, showing altered thresholds for cardiovascular and neurologic toxicity [39,40]. In healthy human volunteers, moreover, the heart was affected at lower doses and serum concentrations of bupivacaine than with ropivacaine [41]. As reflected in Table 1 and Table 2, however, bupivacaine has been studied more widely in scalp blockade than the other two LAs.

Regardless of which LA is used, injection at multiple sites and selectively blocking peripheral nerves of the scalp should help prevent absorption-related toxicity, since as little as 10 mL of LA per side of the cranium is needed. Additional economy with respect to LA volume is achieved by blocking only the nerves relevant to the anticipated surgical incision (ie, great auricular nerve can often be omitted). Selective blockade with minimal volume, as opposed to ring blockade, allows for the use of maximal LA concentration, which may

Table 1 Summary of randomized controlled trials of scalp blockade and local anesthetic infiltration on hemodynamic responses during and after intracranial surgery

Study	Surgery type	Intervention (pts per group)	Timing	Findings
Bloomfeld et al [29]	Supratent	*Infiltration*: 0.25% bupiv/1:200K epinephr vs saline/1:200K epinephr (18 pts/group). Pin sites injected before fixation	Preincis & postsurg	HR higher in saline group at dural & skin closure. No effect on postop hemodynamics. VAS lower in the bupiv group at PACU admission but NS ($P = 0.06$) at 1 hr.
El-Dawlatly et al [28]	Supratent	*Infiltration of pin insertion sites*: 0.25% bupiv vs saline.	Preincis pre-pin placement	VAS lower at 2, 4, 36, 48 hrs. No diffs in hemodynamic parameters.
Geze et al [50]	Intracranl	1) *Scalp block* w/0.5% bupiv, 2) *infiltration at pin insertion sites* with 0.5% bupiv vs 3) IV opioid analgesics (control).	Preincis	Scalp block blunted hemodynamic response to pinning vs other 2 groups.
Hartley et al [52]	Intracranl (pediatr)	*Infiltration*: 1) 0.125% bupiv/1:400K epinephr, 2) 0.25% bupiv/epinephr, 3) epinephr only (control).	Preincis	Both bupiv infiltration groups blunted hemodymamic responses from skin incision to dural reflection.
Lee et al [49]	Supratent	*Scalp block*: 0.25% bupiv vs saline controls.	Preincis	20% increase in HR or MAP triggered intervention: 2.5 mg/kg thiopental & 2 μg/kg fentanyl. Two of 8 pts in scalp block group & 8 of 8 control group pts required the intervention.
Mohammadi et al [51]	Intracranl	*Infiltration*: 0.25% bupiv vs saline controls.	Preincis	Decrease in average MAP, HR from incision to dural opening in bupiv group.
Pinosky et al [3]	Intracranl	*Scalp block*: 0.5% bupiv vs saline controls given 5 min before cranial fixation.	Preincis	With cranial fixation, significant increases noted in SBP 40±6 mmHg, DBP 30±5 mmHg, MAP 32±6 mmHg, and HR 22±5 bpm in controls; no changes in bupiv group.

Supratent = supratentorial, bupiv = bupivacaine, preincis = preincision, preincisional, HR = heart rate, epinephr = epinephrine, postsurg = postsurgery, postsurgical, postop = postoperative, VAS = visual analog scale, PACU = Postanesthesia Care Unit, NS = nonsignificant, pre-pin = before pin insertion, diffs = differences, intracranl = intracranial, IV = intravenous, pediatr = pediatric, MAP = mean arterial pressure, SBP = systolic blood pressure, DPB = diastolic blood pressure.

benefit postoperative analgesia (Tables 1 and 2). At our institution, for adult scalp blockade we generally use 0.5% bupivacaine, as it is economical and readily available.

Lidocaine is rarely used alone for scalp blockade due to its shorter duration of action. However, its more rapid onset may be valuable for treating dural pain or during frame placement in awake patients. Some practitioners combine bupivacaine or ropivacaine with lidocaine to capitalize on both the shorter onset of lidocaine and the longer duration of action of the other LA. Some caution must be exercised with this practice, as combining LAs lowers the effective concentration of both drugs in the mixture. Local anesthetic concentration may be important, as it appears that a potency of 0.5% bupivacaine or greater consistently benefits patients in studies of scalp blockade for acute postoperative pain [4,6,42]. However, no studies have directly compared lower with higher concentrations of LA for scalp blockade, so that strong conclusions cannot be made. Consistent with this speculation, in one study where scalp blockade was performed with lidocaine and bupivacaine, no differences were observed in postoperative pain or opioid consumption compared with controls [43].

Another issue to consider is whether to add epinephrine to the LA. Five studies have used epinephrine in concentrations of 1:200,000 or 1:400,000 (Tables 1 and 2). In two safety and efficacy studies of LA with epinephrine during awake craniotomy, it was unclear whether epinephrine actually slowed the absorption of LA, since epinephrine was used in all patients [33,44]. Ropivacaine with and without epinephrine has been used for brachial plexus blockade, with little demonstrated difference in serum levels [45,46]. Despite the use of epinephrine, a rapid rise in LA serum level was observed (peaks $\leq$ 15 min) but did not result in any cardiovascular or CNS toxicities in either study [33,44]. Serum levels also peaked substantially faster than the times observed in studies of epinephrine-supplemented LA during other regional techniques (epidural, intercostal, and axillary nerve blockade) [34-36]. Interestingly, more recently epinephrine was implicated in eliciting *hypotension* shortly after scalp infiltration, although the mechanism was unclear [47]. Epinephrine does play a role in incisional scalp infiltration, as it provides the added benefit of hemostasis.

In their review of scalp blockade, Osborn and Sebeo [48] postulated the *potential* complications including inadvertent intra-arterial injection with retrograde flow to the internal carotid artery leading to respiratory arrest, complications due to coagulopathy leading to hematoma formation, and the possibility of infection. They noted that case reports of these complications, as well as permanent facial nerve paralysis, do not exist in the literature. Regardless, one must be cognizant of a real risk of temporary facial nerve paralysis on the order of hours if blockade is performed haphazardly in the region of the zygomaticotemporal and auriculotemporal nerves. If the deficit is isolated and ipsilateral to the side of the craniotomy, the surgical procedure generally is not the cause. However, in the setting of bilateral scalp blockade, temporary seventh nerve palsy may generate confusion and lead to unnecessary imaging. It should not be surprising that there are no reports of permanent nerve injury in the literature, as the nerves of the scalp are all superficial terminal branches. Overall, scalp blockade appears to be a very safe procedure if one adheres to the fundamental techniques of injecting LA with careful identification of the anatomical sites.

Neurosurgery has advanced with the widespread use of intraoperative image guidance. These imaging techniques require placement of fiducials preoperatively to permit registration of the patient's intraoperative position in space with the stored image. The LA wheal produced while performing scalp blockade has the potential to alter the position of these markers. Therefore, appropriate care must be exercised when performing scalp blockade prior to registration. Alternately, cranial fixation and registration may take place before scalp blockade. Unfortunately, waiting until after cranial fixation and registration prevents the use of scalp blockade for attenuating the often considerable hemodynamic response to cranial fixation. To prevent this response, administration of systemic opioids with or without a general anesthetic such as propofol (Diprivan) would be needed in lieu of scalp blockade [49]. However, scalp blockade performed after image registration and specification of the incision makes it possible to target only the nerves subserving the surgical site.

6. Intraoperative hemodynamics

Scalp blockade drew attention in the 1990s as a means to improve hemodynamic control of the neurosurgical patient during cranial fixation. The goal was to prevent acute increases in heart rate (HR) and blood pressure, which might lead to acute changes in intracranial pressure or possible rupture of cerebral aneurysms and other vascular lesions. Studies that have addressed the role of scalp blockade in promoting intraoperative hemodynamic stability are summarized in Table 1 [3,28,29,49-52].

Pinosky et al [3] first described scalp blockade as a means to improve hemodynamic control during cranial fixation. They found that neutrally directed scalp blockade with bupivacaine 5 minutes before cranial fixation, compared with a saline control, led to greater hemodynamic stability and a trend toward decreased isoflurane concentrations in the intervention group. Another study compared IV opioid analgesic controls, LA infiltration with 0.5% bupivacaine at each pin insertion site, and scalp blockade with 0.5% bupivacaine, and found that scalp blockade was superior in controlling hemodynamics during cranial fixation and for 3 minutes afterward. The scalp blockade group also showed lower levels of cortisol and adrenocorticotropic hormone 5 and 60 minutes after cranial fixation [50].

Table 2 Summary of studies of the impact of local anesthetic use by infiltration, scalp block, or superficial cervical plexus block on postoperative pain after intracranial * surgery

Study	Surgery type	Study type	Intervention (pts per group)	Timing	Findings
Acute pain					
Ayoub et al [43]	Supratent	RCT	Remifent-based GA. 0.1 mg/kg morphine sulfate or *scalp block* w/20 mL 50:50 0.5% bupiv & 2% lidocaine; saline controls used in each 25-pt group.	Postsurg	Pain scores, time to 1st request for analgesia, total SQ codeine use similar in both groups in 24-hr study pd.
Bala et al [4]	Supratent	RCT	*Scalp block*: 0.5% bupiv/1:400K epinephr vs saline controls (20 pts/group).	Postsurg	Decreased pain, less need for rescue med in intervention group in 1st 6 hrs of 12-hr observ pd.
Batoz et al [61]	Intracranl	RCT	*Infiltration*: 20 mL 0.75% ropiv (n = 25) vs no infiltration (n = 27).	Postsurg	Decreased pain, trend to less opioid use in intervention group 1st 24 hrs after surgery (see *Chronic pain* below).
Biswas & Bithal [62]	Supratent	RCT	*Infiltration*: 25 mL 0.25% bupiv + IV saline vs 25 mL saline + 2 μg/kg fentanyl 5 min preincis.	Preincis	No diffs in n/pts needing rescue in 1st 12 hrs. Bupiv delayed rescue a median 105 vs 60 min in fentanyl group.
Bloomfeld et al [29]	Supratent	RCT	*Infiltration*: 0.25% bupiv/1:200K epinephr vs saline/1:200K epinephr (18 pts/group). Pin sites injected before fixation.	Preincis & postsurg	Pain on PACU admission less in bupiv group. No diffs at 1 hr ($P = 0.06$).
Gazoni et al [53]	Supratent	RCT	Remifent-based GA. *Scalp block*: 0.5% ropiv (n = 14) vs no block (n = 16).	Preincis	Pain less in intervention group in 1st 4 postop hrs. Trend to less opioid use in 1st 24 hrs postop.
Girard et al [30]	Infratent & occipital	RCT	Remifent-based GA. Bilat *superficial cervical plexus block*: 20 mL 50:50 0.5% bupiv & 2% lidocaine + IV saline vs block with saline + IV 0.1 mg/kg morphine (15 pts/group).	Postsurg	No diffs in pain scores or time to 1st request for SQ codeine (median ≤ 25 min in both groups) in 24-hr study pd.
Gottschalk et al [42]	Intracranl	Observ	*Scalp block* (generally 0.5% bupiv, n = 30) vs standard practice (generally surg infiltration of incision with 0.25% bupiv, n = 157).	Preincis	Reduced rest pain over 1st 2 postop days & pain with movement on 1st, not 2nd, postop day.
Honnma et al [5] (in Japanese)	Supratent (aneurysm clipping)	RCT	NSAID given preop. *Scalp block* (supraorb & supratrochl nerves) w/0.25% bupiv preincision + incisional *infiltration* w/ lidocaine preincision + incisional *infiltration* w/bupiv postsurg vs surg site *infiltration* w/lidocaine preincision (10 pts/group).	Preincis & postsurg	Pain scores, NSAID use 6, 12, 24 hrs postsurg and 3, 5, 7, 14 days postsurgery less in intervention group (from *Abstract*; see *Chronic pain*)
Imaev et al [63] (in Russian)	Intracranl	RCT	5 groups: 1) postop NSAIDs (control), 2) postop NSAIDs (control), 3) preop & postop NSAIDs , 4) ropiv *scalp block* + *infiltration*, 5) preop & postop fentanyl.	Preincis	Scalp block group had less pain in the 54-hr observ pd vs 2 control groups (from *Abstract*).
Law-Koune et al [64]	Supratent	RCT	3 groups: *infiltration* of 20 mL of 1) saline (control, n = 40), 2) 0.375% bupiv/1:200K epinephr (n = 20), 3) 0.75% ropiv (n = 20).	Postsurg	Intervention groups showed opioid-sparing effect in 1st 2 hrs of 16-hr observation pd; no diffs in VAS.
Nguyen et al [6]	Supratent	RCT	*Scalp block*: 20 mL 0.75% ropiv vs saline controls (15/group).	Postsurg	VAS lower in scalp block group in 48 hrs; no diffs in 1st request or SQ codeine use postop.
Saringcarinkul & Boonsri [65]	Intracranl	RCT	*Infiltration*: 0.5% bupiv/1:400K epinephr vs saline/1:400K epinephr controls (25/group).	Postsurg	Lower median pain score tended to be in bupiv group in 1st 12 hrs; diff significant only in 1st hour.

(continued on next page)

Table 2 (*continued*)

Study	Surgery type	Study type	Intervention (pts per group)	Timing	Findings
Chronic pain					
Batoz et al [61]	Intracranl	RCT	*Infiltration*: 20 mL 0.75% ropiv (n = 25) vs no infiltration (n = 27).	Postsurg	2 mos postsurgery, intervention group had less persistent pain, less likely to have neuropathic symptoms (see *Acute pain*, above).
Honnma et al [5] (in Japanese)	Supratent (aneurysm clipping)	RCT	See *Acute pain*, above	Preincis & postsurg	See *Acute pain*, above

RCT = randomized controlled trial, remifent = remifentanil, GA = general anesthesia, postsurg = postsurgical, 1st request = first request for pain medication, SQ = subcutaneous, bupiv = bupivacaine, hr = hour, pt = patient, pd = period, epinephr = epinephrine, med = medication(s), observ = observation, intracranl = intracranial, ropiv = ropivacaine, IV = intravenous, preincis = preincisional, n/pts = number of patients, no diffs = no differences, min = minute, PACU = Postanesthesia Care Unit, postop = postoperative, bilat = bilateral, surg = surgical, NSAID = nonsteroidal anti-inflammatory drugs, preop = preoperative, supraorb = supraorbital, supratrochl = supratrochlear, VAS = visual analog scale, mos = months.

* "Intracranial" includes supratrentorial (supratent) and infratentorial (infratent) procedures.

Investigators also have assessed the effect of scalp blockade on hemodynamics at the time of incision and until dural opening [49]. Sixteen patients were randomized to receive blockade with either 0.25% bupivacaine or saline as a supplement to a 50% nitrous oxide (N_2O) and isoflurane-based anesthetic. An increase in HR or mean arterial pressure (MAP) greater than 20% over baseline triggered a bolus of 2.5 mg/kg of thiopental sodium and 2 μg/kg of fentanyl. Only 25% of patients in the scalp blockade group required additional IV anesthetics, whereas 100% of the control group required additional treatment (Table 1). At dural opening, they did not observe a difference between groups with respect to MAP or HR. As scalp blockade does not anesthetize the dura, it is likely that the inhalational anesthetics blunted this hemodynamic response equivalently in each group. These investigators also did not find any correlation between hemodynamic variability and plasma catecholamine levels [49,50,53]. In another study in an adult population undergoing supratentorial intracranial surgery, 0.25% bupivacaine infiltration along the incisional line or at cranial fixation sites was compared to controls. Hemodynamic stability was assessed from the time of incision to dural opening. All patients received general anesthesia with 50% N_2O and isoflurane. During this period of observation, an improvement in hemodynamic stability, particularly MAP, was shown in the LA group [51].

Gazoni et al [53] compared directed scalp blockade with 0.5% bupivacaine administered at least 15 minutes before cranial fixation with no blockade. They assessed the hemodynamic response to cranial fixation, intraoperative hemodynamic stability, postoperative pain and opioid use, and postoperative nausea and vomiting. With induction of anesthesia, a remifentanil infusion was initiated and titrated as per the anesthesiologist. Sevoflurane was used as the maintenance general anesthetic. Although the hemodynamic response to fixation was superior in the scalp blockade group, the remainder of the outcome variables were unaffected [53].

Overall, evidence indicates that scalp blockade can blunt the hemodynamic response to cranial fixation, and this benefit appears to extend to the moments before dural opening. However, any beneficial effect on hemodynamics for the remainder of the surgery is less established. Only two studies have evaluated the relationship between scalp blockade and hemodynamics in the period after dural opening, and they did not show clear benefits of the intervention [29,53].

7. Acute postoperative pain

Given that intracranial surgery can be executed with minimal IV supplementation after performing scalp blockade, it should not be surprising that this technique can provide some degree of postoperative analgesia. The procedure-specific extent and duration of this analgesic benefit remains to be fully established, as does the approach to scalp blockade that yields the greatest analgesic benefit. However, as noted above, scalp blockade is at best incomplete since the dura is not accessible to extracranial blockade. In addition, during infratentorial procedures the sensory innervation to some portion of the tissues dissected is not subserved by distinct, readily identifiable peripheral nerves. This region represents a source of intraoperative noxious stimulation and serves as a postoperative pain generator.

Traditionally it has been taught that pain following intracranial surgery is minimal. However, accumulated evidence indicates that the majority of patients experience some period of moderate to severe pain after intracranial surgery [42,54,55], and that such pain may be more severe for infratentorial procedures [42,56,57]. Nonetheless, even relatively recent surveys of analgesic practice following intracranial surgery have shown a reluctance to use analgesic regimens that are considered standard for other types of

surgery [58,59]. Concern about treating pain after intracranial surgery revolves around the side effects of sedation, miosis, and nausea and vomiting that could mask the signs of intracranial catastrophe, as well as the elevated arterial CO_2 concentrations and subsequent cerebral vasodilation that accompany opioid-induced respiratory depression [59,60]. Clearly, since pain after intracranial surgery is clinically relevant and realistic concerns exist about the use of opioids, it would appear that regional analgesia as provided by scalp blockade has a natural role after intracranial surgery.

A number of rather heterogeneous studies have examined the role of scalp blockade, LA infiltration of the surgical site and, in one instance, bilateral superficial cervical plexus blockade in reducing postoperative pain (Table 2) [4-6,29,30,42,43,53,61-65]. These studies differ with respect to type of intracranial surgery, general anesthetic technique, study design, nature and timing of the intervention, postoperative analgesia, and duration of the study period. The outcome variables generally are pain and analgesic consumption. The outcomes vary from meaningful long-term effects to those that are modest or absent with respect to the control group. Low concentrations of LA (eg, an actual or effective concentration of 0.25% or 0.375% bupivacaine or 0.5% ropivacaine) were not particularly effective. No pattern related to the timing of the intervention or the location of surgery was apparent in the studies described in Table 2, though there are relatively little data involving infratentorial surgery. The relatively short observation period in some studies would have prevented detection of the longer-term benefits observed in several studies. Therefore, the only conclusions to draw from Table 2 are: 1) study results related to the analgesic benefits of scalp blockade are heterogeneous, and 2) higher rather than lower concentrations of long-acting LA are associated with a greater likelihood of postoperative analgesic success. It may be that unidentified factors related to patient selection, surgery, or general anesthetic technique may be responsible for the rather disparate outcomes summarized in Table 2.

8. Chronic postoperative pain

Pain that accompanies surgery can persist and become chronic for a very large variety of surgical procedures [66], and pain following intracranial surgery is no exception [67]. This phenomenon is best established for posterior fossa procedures, for which postoperative pain is rather common, it may persist for many months, and it may be severe [68,69]. These issues are less appreciated for supratentorial procedures. However, according to one study [61], 56% of patients who underwent supratentorial procedures reported pain two months after surgery, and approximately 50% of those with pain reported symptoms with neuropathic qualities. It is important to note that infiltration of the incision site with 0.75% bupivacaine at the time of closure reduced the number reporting pain two months after surgery to only 8%, and only one of those two patients reported neuropathic symptoms. Another study suggested a long-term benefit of scalp blockade by showing a reduction in pain for 14 days after surgery [5]. Even if patients from the other studies in Table 2 did experience longer-term benefits, these would have gone unobserved as 48 hours was the next longest observation period. However, since the two studies just described also reported reductions in pain acutely, it seems unlikely that studies that failed to show a positive short-term benefit of the intervention would have produced an unobserved long-term benefit.

9. Conclusions

The history of scalp blockade, particularly for intracranial procedures, testifies to the efficacy, simplicity, and safety of this form of neural blockade. Over time, the anatomic basis for scalp blockade has become better defined and described, and its indications are expanding. These indications include extracranial surgery, blunting of the hemodynamic response to intracranial surgery, and reducing acute and chronic pain following intracranial surgery. Its evolving role in decreasing acute and chronic pain parallels that of other types of neural blockade, for which efforts to find efficacious combinations of procedure, patient, and technique are ongoing. However, the fact that extracranial blockade of the dura is not possible may represent a fundamental limit to the quality of postoperative analgesia provided by scalp blockade. Thus, for procedures that carry a likelihood of dural inflammation, the efficacy of scalp blockade with respect to postoperative analgesia may also depend on other analgesic adjuncts.

Acknowledgments

The authors gratefully acknowledge the the translation from the Japanese of Honnma et al [5] by Yoneyama Harumi, manuscript preparation by Claire Levine, and manuscript review by Jean-Pierre Ouanes, MD.

References

[1] Penfield W. The radical treatment of traumatic epilepsy and its rationale. Can Med Assoc J 1930;23:189-97.
[2] Penfield W. Some observations on the cerebral cortex of man. Proc R Soc Lond B Biol Sci 1947;134:329-47.
[3] Pinosky ML, Fishman RL, Reeves ST, et al. The effect of bupivacaine skull block on the hemodynamic response to craniotomy. Anesth Analg 1996;83:1256-61.
[4] Bala I, Gupta B, Bhardwaj N, Ghai B, Khosla VK. Effect of scalp block on postoperative pain relief in craniotomy patients. Anaesth Intensive Care 2006;34:224-7.
[5] Honnma T, Imaizumi T, Chiba M, Niwa J. Preemptive analgesia for postoperative pain after frontotemporal craniotomy. No Shinkei Geka 2002;30:171-4.
[6] Nguyen A, Girard F, Boudreault D, et al. Scalp nerve blocks decrease the severity of pain after craniotomy. Anesth Analg 2001;93:1272-6.

[7] Finco G, Atzeni M, Musu M, Maxia S, Ribuffo D. Greater occipital nerve block for surgical resection of major infiltrating lesions of the posterior scalp. Plast Reconstr Surg 2010;125:52e-3e.
[8] Halldin CB, Paoli J, Sandberg C, Gonzalez H, Wennberg AM. Nerve blocks enable adequate pain relief during topical photodynamic therapy of field cancerization on the forehead and scalp. Br J Dermatol 2009;160:795-800.
[9] Young WB. Blocking the greater occipital nerve: utility in headache management. Curr Pain Headache Rep 2010;14:404-8.
[10] Frost EA. The contributions of the pioneers in neurosurgery to the development of neuroanaesthesia. In: Atkinson RS, Boulton TB, editors. The History of Anaesthesia. (International Congress and Symposium Series, No. 134). Royal Society of Medicine Services. London: Parthenon Publishing Group; 1989. p. 522-617.
[11] Klauder JV. Novocain dermatitis. Cent Cosmos 1922;64:305-9.
[12] Cocaine. Br Med J 1979;1(6169):971-2.
[13] Braun H. On some new local anesthetics (stovaine, alypin, novocaine). Dtsch Med Wochenschr 1905;31:1667-71.
[14] Girvin JP. Neurosurgical considerations and general methods for craniotomy under local anesthesia. Int Anesthesiol Clin 1986;24:89-114.
[15] Costello TG, Cormack JR. Anaesthesia for awake craniotomy: a modern approach. J Clin Neurosci 2004;11:16-9.
[16] Piccioni F, Fanzio M. Management of anesthesia in awake craniotomy. Minerva Anestesiol 2008;74:393-408.
[17] Feindel W, Penfield W, McNaughton F. The tentorial nerves and localization of intracranial pain in man. Neurology 1960;10:555-63.
[18] Ambrosini A, Vandenheede M, Rossi P, et al. Suboccipital injection with a mixture of rapid- and long-acting steroids in cluster headache: a double-blind placebo-controlled study. Pain 2005;118:92-6.
[19] Naja ZM, El-Rajab M, Al-Tannir MA, Ziade FM, Tawfik OM. Occipital nerve blockade for cervicogenic headache: a double-blind randomized controlled clinical trial. Pain Pract 2006;6:89-95.
[20] Saper JR, Dodick DW, Silberstein SD, McCarville S, Sun M, Goadsby PJ; ONSTIM Investigators. Occipital nerve stimulation for the treatment of intractable chronic migraine headache: ONSTIM feasibility study. Cephalalgia 2010;31:271-85.
[21] Lord SM, Barnsley L, Wallis BJ, Bogduk N. Third occipital nerve headache: a prevalence study. J Neurol Neurosurg Psychiatry 1994;57: 1187-90.
[22] Jeong SM, Park KJ, Kang SH, et al. Anatomical consideration of the anterior and lateral cutaneous nerves in the scalp. J Korean Med Sci 2010;25:517-22.
[23] Hwang K, Suh MS, Lee SI, Chung IH. Zygomaticotemporal nerve passage in the orbit and temporal area. J Craniofac Surg 2004;15:209-14.
[24] Kaminer MS, Arndt KA, Dover JS, editors. Atlas of Cosmetic Surgery. Philadelphia: Elsevier Health Science; 2009. p. 65.
[25] Andersen NB, Bovim G, Sjaastad O. The frontotemporal peripheral nerves. Topographic variations of the supraorbital, supratrochlear and auriculotemporal nerves and their possible clinical significance. Surg Radiol Anat 2001;23:97-104.
[26] Tubbs RS, Salter EG, Wellons JC, Blount JP, Oakes WJ. Landmarks for the identification of the cutaneous nerves of the occiput and nuchal regions. Clin Anat 2007;20:235-8.
[27] Eichenberger U, Greher M, Kapral S, et al. Sonographic visualization and ultrasound-guided block of the third occipital nerve: prospective for a new method to diagnose C2-C3 zygapophysial joint pain. Anesthesiology 2006;104:303-8.
[28] El Dawlatly AA, Abbas S, Turkistani A, et al. Use of tenoxicam for post craniotomy pain relief with or without bupivacaine scalp infiltration: a randomized trial. Internet J Anesthesiol 2008;15.
[29] Bloomfield EL, Schubert A, Secic M, Barnett G, Shutway F, Ebrahim ZY. The influence of scalp infiltration with bupivacaine on hemodynamics and postoperative pain in adult patients undergoing craniotomy. Anesth Analg 1998;87:579-82.
[30] Girard F, Quentin C, Charbonneau S, et al. Superficial cervical plexus block for transitional analgesia in infratentorial and occipital craniotomy: a randomized trial. Can J Anaesth 2010;57:1065-70.
[31] Gottschalk A, Yaster M. The perioperative management of pain from intracranial surgery. Neurocrit Care 2009;10:387-402.
[32] Scott DB, Lee A, Fagan D, Bowler GM, Bloomfield P, Lundh R. Acute toxicity of ropivacaine compared with that of bupivacaine. Anesth Analg 1989;69:563-9.
[33] Costello TG, Cormack JR, Hoy C, et al. Plasma ropivacaine levels following scalp block for awake craniotomy. J Neurosurg Anesthesiol 2004;16:147-50.
[34] Kopacz DJ, Emanuelsson BM, Thompson GE, Carpenter RL, Stephenson CA. Pharmacokinetics of ropivacaine and bupivacaine for bilateral intercostal blockade in healthy male volunteers. Anesthesiology 1994;81:1139-48.
[35] Salonen MH, Haasio J, Bachmann M, Xu M, Rosenberg PH. Evaluation of efficacy and plasma concentrations of ropivacaine in continuous axillary brachial plexus block: high dose for surgical anesthesia and low dose for postoperative analgesia. Reg Anesth Pain Med 2000;25:47-51.
[36] Scott DA, Emanuelsson BM, Mooney PH, Cook RJ, Junestrand C. Pharmacokinetics and efficacy of long-term epidural ropivacaine infusion for postoperative analgesia. Anesth Analg 1997;85:1322-30.
[37] Archer DP, McKenna JM, Morin L, Ravussin P. Conscious-sedation analgesia during craniotomy for intractable epilepsy: a review of 354 consecutive cases. Can J Anaesth 1988;35:338-44.
[38] Okuda Y, Matsumoto T, Shinohara M, Kitajima T, Kim P. Sudden unconsciousness during a lesser occipital nerve block in a patient with the occipital bone defect. Eur J Anaesthesiol 2001;18:829-32.
[39] Ohmura S, Kawada M, Ohta T, Yamamoto K, Kobayashi T. Systemic toxicity and resuscitation in bupivacaine-, levobupivacaine-, or ropivacaine-infused rats. Anesth Analg 2001;93:743-8.
[40] Graf BM, Abraham I, Eberbach N, Kunst G, Stowe DF, Martin E. Differences in cardiotoxicity of bupivacaine and ropivacaine are the result of physicochemical and stereoselective properties. Anesthesiology 2002;96:1427-34.
[41] Scott DB, Lee A, Fagan D, Bowler GM, Bloomfield P, Lundh R. Acute toxicity of ropivacaine compared with that of bupivacaine. Anesth Analg 1989;69:563-9.
[42] Gottschalk A, Berkow LC, Stevens RD, et al. Prospective evaluation of pain and analgesic use following major elective intracranial surgery. J Neurosurg 2007;106:210-6.
[43] Ayoub C, Girard F, Boudreault D, Chouinard P, Ruel M, Moumdjian R. A comparison between scalp nerve block and morphine for transitional analgesia after remifentanil-based anesthesia in neurosurgery. Anesth Analg 2006;103:1237-40.
[44] Costello TG, Cormack JR, Mather LE, LaFerlita B, Murphy MA, Harris K. Plasma levobupivacaine concentrations following scalp block in patients undergoing awake craniotomy. Br J Anaesth 2005;94:848-51.
[45] Hickey R, Blanchard J, Hoffman J, Sjovall J, Ramamurthy S. Plasma concentrations of ropivacaine given with or without epinephrine for brachial plexus block. Can J Anaesth 1990;37:878-82.
[46] Hickey R, Candido KD, Ramamurthy S, et al. Brachial plexus block with a new local anaesthetic: 0.5 per cent ropivacaine. Can J Anaesth 1990;37:732-8.
[47] Yang JJ, Liu J, Duan ML, Zhou ZQ, Li WY, Xu JG. Lighter general anesthesia causes less decrease in arterial pressure induced by epinephrine scalp infiltration during neurosurgery. J Neurosurg Anesthesiol 2007;19: 263-7.
[48] Osborn I, Sebeo J. "Scalp block" during craniotomy: a classic technique revisited. J Neurosurg Anesthesiol 2010;22:187-94.
[49] Lee EJ, Lee MY, Shyr MH, et al. Adjuvant bupivacaine scalp block facilitates stabilization of hemodynamics in patients undergoing craniotomy with general anesthesia: a preliminary report. J Clin Anesth 2006;18:490-4.
[50] Geze S, Yilmaz AA, Tuzuner F. The effect of scalp block and local infiltration on the haemodynamic and stress response to skull-pin placement for craniotomy. Eur J Anaesthesiol 2009;26:298-303.
[51] Mohammadi SS, Shahbazian E, Shoeibi G, Almassi F. Effect of scalp infiltration with bupivacaine on early hemodynamic responses during craniotomy under general anesthesia. Pak J Biol Sci 2009;12:603-6.

[52] Hartley EJ, Bissonnette B, St-Louis P, Rybczynski J, McLeod ME. Scalp infiltration with bupivacaine in pediatric brain surgery. Anesth Analg 1991;73:29-32.
[53] Gazoni FM, Pouratian N, Nemergut EC. Effect of ropivacaine skull block on perioperative outcomes in patients with supratentorial brain tumors and comparison with remifentanil: a pilot study. J Neurosurg 2008;109:44-9.
[54] De Benedittis G, Lorenzetti A, Migliore M, Spagnoli D, Tiberio F, Villani RM. Postoperative pain in neurosurgery: a pilot study in brain surgery. Neurosurgery 1996;38:466-70.
[55] Quiney N, Cooper R, Stoneham M, Walters F. Pain after craniotomy. A time for reappraisal? Br J Neurosurg 1996;10:295-9.
[56] Irefin SA, Schubert A, Bloomfield EL, DeBoer GE, Mascha EJ, Ebrahim ZY. The effect of craniotomy location on postoperative pain and nausea. J Anesth 2003;17:227-31.
[57] Thibault M, Girard F, Moumdjian R, Chouinard P, Boudreault D, Ruel M. Craniotomy site influences postoperative pain following neurosurgical procedures: a retrospective study. Can J Anaesth 2007;54:544-8.
[58] Roberts GC. Post-craniotomy analgesia: current practices in British neurosurgical centres–a survey of post-craniotomy analgesic practices. Eur J Anaesthesiol 2005;22:328-32.
[59] Stoneham MD, Walters FJ. Post-operative analgesia for craniotomy patients: current attitudes among neuroanaesthetists. Eur J Anaesthesiol 1995;12:571-5.
[60] Cold GE, Felding M. Even small doses of morphine might provoke "luxury perfusion" in the postoperative period after craniotomy. Neurosurgery 1993;32:327.
[61] Batoz H, Verdonck O, Pellerin C, Roux G, Maurette P. The analgesic properties of scalp infiltrations with ropivacaine after intracranial tumoral resection. Anesth Analg 2009;109:240-4.
[62] Biswas BK, Bithal PK. Preincision 0.25% bupivacaine scalp infiltration and postcraniotomy pain: a randomized double-blind, placebo-controlled study. J Neurosurg Anesthesiol 2003;15:234-9.
[63] Imaev AA, Dolmatova EV, Lubnin A. Comparative evaluation of preventive analgesia with xefocam, ropivacaine, and transdermal drug delivery system of durogesic in patients after craniotomy. Anesteziol Reanimatol 2010;4:15-9.
[64] Law-Koune JD, Szekely B, Fermanian C, Peuch C, Liu N, Fischler M. Scalp infiltration with bupivacaine plus epinephrine or plain ropivacaine reduces postoperative pain after supratentorial craniotomy. J Neurosurg Anesthesiol 2005;17:139-43.
[65] Saringcarinkul A, Boonsri S. Effect of scalp infiltration on postoperative pain relief in elective supratentorial craniotomy with 0.5% bupivacaine with adrenaline 1:400,000. J Med Assoc Thai 2008;91:1518-23.
[66] Perkins FM, Kehlet H. Chronic pain as an outcome of surgery. A review of predictive factors. Anesthesiology 2000;93:1123-33.
[67] Flexman AM, Ng JL, Gelb AW. Acute and chronic pain following craniotomy. Curr Opin Anaesthesiol 2010;23:551-7.
[68] Schankin CJ, Gall C, Straube A. Headache syndromes after acoustic neuroma surgery and their implications for quality of life. Cephalalgia 2009;29:760-71.
[69] Vijayan N. Postoperative headache in acoustic neuroma. Headache 1995;35:98-100.