Etiology of Emergence Hypertension Following Craniotomy

The avoidance of coughing, straining and hypertension is a reasonable goal for all anesthetics; however, when it comes to neuroanesthesia its importance cannot be overstated. Patients following craniotomy may have impaired cerebral autoregulation and thus arterial hypertension can contribute to vascular engorgement leading to increased ICP, intracranial bleeding and increased edema formation[1].

The incidence of emergence hypertension has been reported to be between 20-90%[2,3,4]. In an effort to delineate the etiology, researchers have looked at predictive factors for post craniotomy hypertension. Diddee et al. from Western University found only age to be a positive predictive factor for post craniotomy hypertension[2]. Whereas a prospective observational study conducted by Perez et al. discovered that, in their population, a history of hypertension was the only independent risk factor for post-craniotomy hypertension[3].

Several potential mechanisms of hypertension post-craniotomy have been studied[10]:

Pain-induced sympathetic stimulation

Pain-induced sympathetic stimulation may appear to be a plausible contributor to post-craniotomy hypertension since craniotomies cause significant trauma to highly innervated structures including the scalp, soft tissue, and dura with the potential for direct nerve disruption, traction, entrapment, and compression. The image in the references below demonstrates the complex sensory innervation to scalp structures[3].

However, Bloomfield et al. showed that although scalp infiltration with local anesthetic blunts intraoperative hemodynamic responses and reduces postoperative pain it has no effect on postoperative hemodynamics[11].

Epinephrine containing local anesthetics

Epinephrine is added to local anesthetics to decrease systemic absorption and increase duration of action of local anesthetics as well as reduce bleeding at the injection site. The hemodynamic effects of epinephrine are dose dependent through a1, b1, and b2-receptors.

To my surprise, a randomized prospective double blinded control study looking at hemodynamic changes due to scalp infiltration with epinephrine-containing local anesthetics in craniotomy patients conducted by Yang et al. discovered temporary but significant hemodynamic changes including *hypotension* likely via activation of b2-receptors[9].

Vasoactive modulators

Olsen et al. studied vasoactive modulators during and after craniotomy and discovered postoperative hypertension was associated with consistently high renin levels intraoperatively, followed by elevation of catecholamine levels post-operatively[4].

With Perez et al.'s observation that pre-existing hypertension is a risk factor for emergence hypertension in craniotomy patients, it fits that a dysfunctional renin-angiotensin system, as seen in the essential hypertension population, contributes to emergence hypertension and is likely only compounded by pre-operative discontinuation of anti-hypertensive medications.

Cerebrovascular reflexes and neurosurgical lesions

Cerebral pial blood vessels are innervated by sympathetic and parasympathetic fibers. These fibers contain vasoactive substances and manipulation of these fibers could cause catecholamines to enter systemic circulation leading to hypertension[6].

Certain lesions are associated with increased catecholamine release including astrocytoma, cerebellar tumours, pheochromocytomas, hemangioblastomas, and intraparenchymal brain tumours. In addition, procedure specific factors such as mechanical pressure in the posterior fossa may lead to increased blood pressure[6].

Hypothermia/hypoxia/hypercarbia and emergence excitement

While not unique to neurosurgery, universal extubation criteria necessities the avoidance of hypothermia, hypoxia, and hypercarbia. Therefore, these factors are only theoretical contributors to post-craniotomy hypertension.

Hypothermia is a known trigger for thermoregulatory vasoconstriction which increases arterial pressure by increasing circulating concentrations of epinephrine, norepinephrine, and cortisol[7]. Global brain hypoxia is associated with massive catecholamine release[6]. Hypercarbia and emergence excitement are also known contributors to emergence hypertension[8].

Conclusion

Ultimately, post-craniotomy hypertension is a well-recognized common phenomenon with potentially significant clinical implications. The mechanism is likely secondary to high intra-operative renin and post-operative catecholamine levels. The etiology of emergence hypertension is unsatisfyingly multifactorial. However, it appears pain induced sympathetic stimulation and epinephrine containing local anesthetics do not play a significant role.

References:

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[8] Gal, T., Cooperman, L. Hypertension in the immediate postoperative period. *British Journal of Anaesthesia* **47,** 70–74 (1975).

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Appendix:

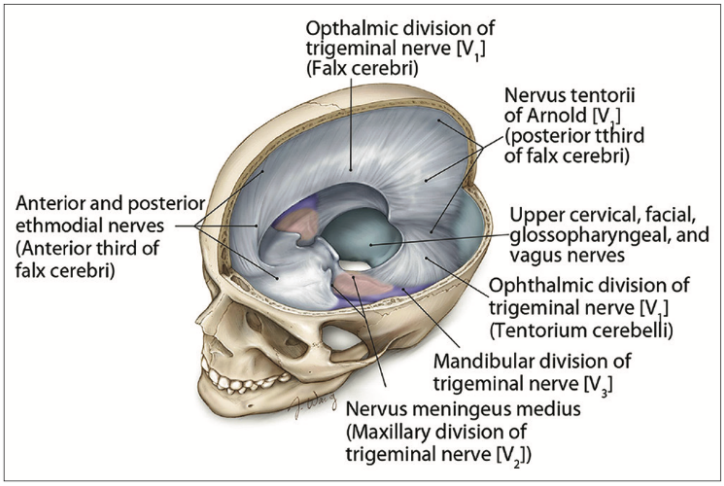

Image 1: Scalp sensory innervation

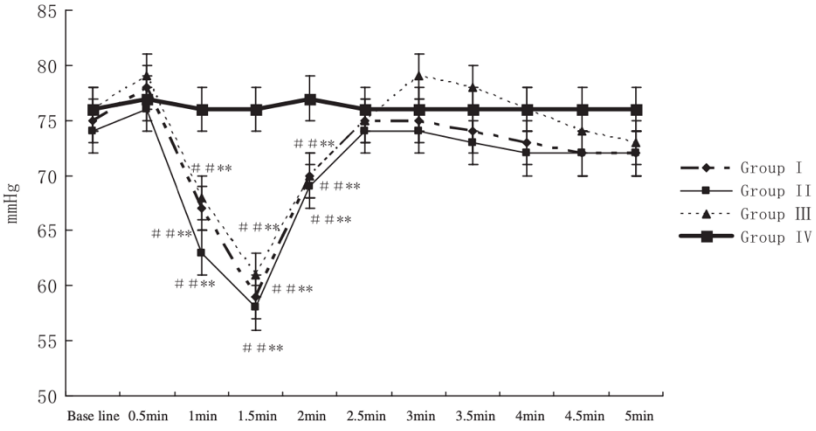

FIGURE 1. Changes in MAP after the beginning of local infiltration.

Figure 1: All patients received 1% lidocaine 16mL with different concentrations of epinephrine: Group 1 with 40mg (2.5 µg/mL); group 2 with 80 mg (5 µg/mL); group 3 with 160 mg (10 µg/mL); and group 4 (control group) without epinephrine.

	p	Odds ratio	95.0% C.I. for odds	
			Lower	Upper
Age	.0001	1.038	1.020	1.057
Sex	.255	1.302	.827	2.050
Meningioma	.293	.534	.166	1.719
Astrocytoma	.091	.275	.062	1.229
GBM	.186	.445	.134	1.476
Metastatic disease	.450	.630	.190	2.086
Oligodendroglioma	.079	.129	.013	1.272
Other	.598	.726	.221	2.383
ASA	.650	.914	.619	1.349
Chronic Hypertension	.660	.887	.522	1.509
Raised Intracranial Pressure	.162	1.471	.856	2.527
Operation Time	.818	1.000	1.000	1.000
Intraoperative Hypotension	.232	1.335	.831	2.145
Intraoperative Hypertension	.215	1.350	.840	2.168

Figure 2: Diddee et al.'s Predictive factors for postoperative hypertension in craniotomies for tumor. A logistic regression analysis for 506 patients.