Postoperative Headache

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in Anesthesia

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ
قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ
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List of Abbreviations

5-HT	5-hydroxytryptamine
Ach	Acetylcholine
ACTH	Adrenocorticotropic Hormone
ADP	Adenosine diphosphate
CGRP	Calcitonin gene-related peptide
CH	Cluster headache
CNS	Central nervous system
COX-2	Cyclo-oxygenase -2
CSD	Cortical spreading depression
CSF	Cerebral spinal fluid
CT	Computed tomography
EBP	Epidural blood patch
FMRI	Functional magnetic resonance imaging
GABA	Gamma-aminobutyric acid
HAH	High-Altitude Headache
HC	Hemicrania continua
ICH	Intracerebral hemorrhage
ICHD	International Classification of Headache Disorders
IIH	Idiopathic intracranial hypertension
LORA	Loss of resistance to air
LORS	Loss of resistance to saline
LP	Lumbar puncture
MMP-2	Matrix metalloproteinase-2
MMP-9	Matrix metalloproteinase-9
MMPs	Matrix metalloproteinases
MOH	Medication overuse headache
MRI	Magnetic resonance imaging
MS	Multiple sclerosis
NDPH	New daily persistent headache
NMDA	N-methyl-D-aspartate
NO	Nitric oxide
NOS	Nitric oxide synthase
OSA	Obstructive sleep apnea
PAG	Periaqueductal gray
PDPH	Postdural puncture headache
PET	Positron emission tomography

PLPH	Post–lumbar puncture headache
PRLES	Posterior Reversible Leukoencephalopathy syndrome
PSH	Primary stabbing headache
PTH	Posttraumatic headache
RVM	Rostral ventromedial medulla
SAH	Subarachnoid hemorrhage
SP	Substance P
TACs	Trigeminal autonomic cephalalgias
TIA	Transient ischemic attack
TIMP-1	Tissue inhibitors of metalloproteinase-1
TMJ	Tepromandibular joint
TN	Trigeminal neuralgia
TNC	Trigeminal nucleus caudalis
TTH	Tension-type headache
VIP	Vasoactive intestinal polypeptide

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Introduction

Since approximately 25 million people undergo surgery in the United States each year, a considerable number of these patients will have chronic headaches. When headaches occur perioperatively, it is important to determine whether they correspond to prior headache patterns or represent an entirely new symptom complex ***(Aminoff, 2008).***

Headache after general anesthesia has been reported to range from 10% up to 38% during the first 24 hours. When patients are monitored for the occurrence of headache during the first week after surgery, this number increases to 48%. However, the incidence of chronic daily headache in the general population ranges from 5% to 30%; thus a portion of patients, had they not had surgery, would have been prone for the development of a headache anyway ***(Martikainen et al., 2000).***

The perioperative period is stressful, and tension and stress related headaches may be exacerbated during this time. A significant percentage of the population consumes caffeine containing drinks on a regular basis, and the perioperative period often limits caffeine intake ***(Fennelly et al., 1991).***

In patients who consume caffeine on a daily basis, the incidence of postoperative headache is reduced when they are permitted a caffeinated drink on the morning of surgery. Similarly, patients at risk of caffeine withdrawal can reduce the likelihood of postoperative headache if they are administered intravenous caffeine 200 mg while still in the Phase I or II recovery areas. These patients should additionally be encouraged to drink a caffeinated drink after surgery when it is appropriate for oral intake. Taking caffeine tablets before and after surgery in a dose approximating the patient's

customary daily caffeine intake is equally effective in minimizing postoperative headache ***(Weber et al., 1997).***

The complicating feature is that spinal anesthesia (with dural puncture) may be associated with headaches in from 1 to 5 percent of patients, depending on gender, age, and type of needle utilized for the anesthetic. The headache related to spinal puncture (decreased intracranial pressure) is possibly related to traction on trigeminal, glossopharyngeal, or vagal fibers, though there is increasing evidence that cerebrovascular vasodilatation may also be involved ***(Brown, 2005).***

Pathophysiological Consideration in the Headache

Headache can occur as a result of activation of pain sensitive cranial structures, such as the dura mater, vasculature, and the cranial and cervical muscles and ligaments, which are innervated by primary afferent neurons originating from the trigeminal and dorsal root ganglia of the upper cervical spinal nerves. In relation to nociception in cases of headache, two types of nerve fiber are considered to be important: the smallcaliber, unmyelinated, slow-conducting fibers called C fibers, and the small-diameter, lightly myelinated, more rapid-conducting fibers called Ad fibers. Findings from nerve stimulation studies indicate that C fibers transmit aching, throbbing, or burning pain that builds up slowly, whereas the Ad fibers conduct sharper initial pain sensation ***(Basbaum and Jessell, 2000).***

Anatomy

The trigeminal nerve conveys sensory information from most extracranial and intracranial structures to the spinal trigeminal nucleus. To investigate the site of nociception in migraine, the structures of the head that are sensitive to pain must first be identified ***(Messlinger et al .,2006).***

Extracranial pain-sensitive structures:

Extracranial pain-sensitive structures include the skin, muscles, arteries, and periosteum. The skin of the scalp is sensitive to all usual forms of painful stimuli. The sensory afferent fibres in the adipose and connective tissue that surround the blood vessels in muscle in the scalp and suggested that impulses originating in these fibres give rise to the perception of muscle pain. Experimental studies in human

beings have shown that pericranial muscles, in particular the temporal and neck muscles, might be a source of pain and tenderness ***(Svensson and Ashina,2006).***

Although all the arteries of the scalp (superficial temporal, posterior, occipital, supraorbital, and frontal) are sensitive to pain from various stimuli, including distension, the veins of the scalp are much less or not at all sensitive to pain ***(Svensson and Ashina,2006).***

Intracranial pain-sensitive structures:

In 1940, Ray and Wolff found that electrical stimulation of dural and cerebral arteries and veins, but not the parenchyma of the brain, evoked nausea and the perception of headache-like pain in conscious human beings during brain surgery. They concluded that the dural and large cerebral (pial) arteries are pain sensitive, and mapped this sensitivity along the course of the middle meningeal artery and the margins of the dural sinuses. The middle meningeal artery is the principal artery of the dura; it branches off the external carotid artery and supplies all of the supratentorial dura except for the part that covers the floor of the anterior fossa, which is supplied by the anterior meningeal artery, a branch of the internal carotid artery ***(Denuelle , et al.2004).***

The meninges represent several layers of membranes situated between the skull and the brain to protect the brain from physical damage. The concept that the meninges may serve as one of the sources of headache originated from the results of intraoperative experiments in which electrical, mechanical, thermal, and chemical stimulation of the dural and large intracerebral arteries elicited a painful sensation ***(Denuelle , et al.2004).***

The meninges consist of three layers called, from the inside to the outside, the pia mater, arachnoid mater, and dura mater, the meninges separate three spaces called the epidural space, subarachnoid space, and the subdural space. Each of these spaces contains some important blood vessels, rupture of which can cause headache ***(Blumenfeld, 2002).***

Innervation of the dura mater:

The dura mater is innervated by afferent nerve fibers, most of which originate in the ipsilateral trigeminal ganglion**.** The afferent nerve fibers in the dura mater have been implicated in neurogenic inflammation, a phenomenon putatively linked to the development of migrainous pain.The dura mater in the middle cranial fossa and the middle meningeal vessels are supplied mainly by nerve fibers rising from the second and third divisions of the trigeminal ganglion ***(Shields et al.,2003).***

These meningeal afferent nerve fibers show positive immunoreactivity for substance P (SP), neurokinin A, and calcitonin gene-related peptide (CGRP) (***Messlinger et al., 1993***).

In addition to its supply from the afferent nervous system, the dura mater is also innervated by sympathetic fibers containing neuropeptide Y (NPY) arising from the superior cervical ganglion ***(Von During et al., 1990)***, and comparatively sparse parasympathetic fibers containing vasoactive intestinal polypeptide (VIP) and nitric oxide synthase (NOS) ***(Berger et al., 1994).***

Referred pain from the dura mater:

The main trunks of all the dural arteries and the smaller branches arising from the main divisions of the middle meningeal artery have been demonstrated to be sensitive to

pain**.**Furthermore, according to studies conducted by the aforementioned authors, the dura covering the convexities of the cerebrum and cerebellar hemispheres and the middle fossa floor are entirely insensitive to pain except for that in regions along the margins of the dural sinuses or along the meningeal artery.On the other hand, the entire dural covering of the floor of the anterior fossa and posterior fossa is believed to be uniformly sensitive to pain. Pain transmitted by the former is referr to the ipsilateral head and eye, and that transmitted by the latter is referred to the occiput near the midline **(Table 1).** The superior surface of the tentorium cerebelli is also reported to be sensitive to pain***(Shimizu and Suzuki, 2011).***

Table 1: The Regions of Referred Pain from Intracranial Structures ***(Shimizu and Suzuki, 2011).***

Region	Anatomy
The eyes and forehead	–Dura of the anterior fossa. –Anterior middle meningeal arteries. –Structures innervated by the tentorial nerve (superior surface of the tentorium, transverse and straight sinuses, and the posterior half of the superior sagittal sinus). –Sylvian vein. –Intracranial portion of the internal carotid artery. –Vessels of the circle of Willis and proximal portion of the larger cerebral branches. –First division of the fifth nerve.
The temporal and parietal regions	–Middle meningeal arteries. –Union of the inferior cerebral vein of the temporal lobe with the venous sinuses. –Anterior portion of the superior sagittal sinus.
The occipital and suboccipital regions	–In or behind the ear. –Inferior surface of the wall of the confluence of sinus, straight sinus, and the transverse sinus. –Wall of the sigmoid sinus. –Branches of the basilar artery.

The occiput near the midline	–Part of the dura of the posterior fossa. –Posterior meningeal arteries. –Proximal portion of the posterior inferior cerebellar arteries. –Vertebral artery –Basilar artery.

The superior sagittal sinus, transverse sinus, straight sinus, sigmoid sinus, occipital sinus, and superior petrosal sinus are sensitive to pain. In contrast, the inferior sagittal sinus, superior cerebral vein, and inferior cerebral vein are insensitive to pain (***Blumenfeld, 2002***).

Bleys et al. (2001) have reported that the cavernous ganglion has nerve fiber connections with the sympathetic and sensory nerves in the cavernous sinus, which may explain a variety of symptoms associated with injury or disease of the cavernous sinus (***Bleys et al., 2001).***

Stimulation of the cavernous sinus causes pain in the ipsilateral ophthalmic and maxillary nerve region. The maxillary nerve often runs through the lateral wall of the cavernous sinus for a short distance (***Blumenfeld, 2002***).

Cerebral Arteries:

It is well known that there are two cerebral arterial innervation systems: the extrinsic and intrinsic innervations systems. The extrinsic innervation system originates from the extracranial ganglia, and mainly innervates the vessels of the circle of Willis and their penetrating cerebral branches. When they enter the brain parenchyma, the cerebral arteries lose their peripheral nerve supply from the extrinsic innervation system. After the cerebral arteries leave the Virchow– Robin space, they receive neural input from neurons located within the brain itself, represented by the intrinsic innervation system ***(Shimizu and Suzuki, 2011).***