INTRODUCTION

Migraine is a common, often disabling neurologic disorder characterized by its pulsatile nature, unilateral location, and prolonged duration. It can be associated with nausea, vomiting, photophobia, or phonophobia and potentially with sensory (most commonly visual), verbal, or motor aura. Common triggers include emotional stress, hormonal changes, inconsistent meals, weather, and sleep disturbances. Migraine affects one out of seven Americans annually, with the highest prevalence in women aged 25–55.\(^{[1]}\)

The pathophysiology of migraine is complex, involving activation of the trigeminovascular system that leads to inflammatory changes in the pain-sensitive meninges and alters the permeability of the blood–brain barrier.\(^{[2]}\) Given the complex neurobiology of this disorder, treatment of migraine is often difficult and refractory.

Current management of migraine includes abortive therapy for acute attacks and preventive therapy to reduce the frequency and severity of recurrent headaches. Abortive therapy includes nonsteroidal anti-inflammatory drugs (NSAIDs) and analgesics, with level A recommendations for acetylsalicylic acid, ibuprofen, naproxen, diclofenac, and acetaminophen.\(^{[1]}\) These therapies may also be combined with caffeine. Many migraine-specific therapies target the 5-HT serotonin receptors, as agonists at these receptors result in intracranial extra-cerebral vasoconstriction and inhibition of peripheral and central trigeminal nociceptive terminals. Triptans are 5-HT1B/1D agonists that can be used in moderate-to-severe migraines and are effective in 60% of NSAID non-responders; however,
sustained relief may not occur, with headache recurrence in
15–40% of patients.\textsuperscript{[1]} The most common side effects of triptan
use include paresthesia, flushing, tingling, and mild transient
chest pressure. Triptan use is contraindicated in patients with
myocardial infarction, stroke, peripheral vascular disease, or
untreated vascular risk factors.\textsuperscript{[3]}

A common complication of episodic acute migraine treatment
is the development of a medication overuse headache (MOH).
Development of MOH can be limited by reducing triptan
use to fewer than 10 days per month.\textsuperscript{[1]} Chronic migraine,
deﬁned as 15 or more headache days per month for more than
3 months, occurs in 1% of the United States and commonly
occurs in the setting of MOH.

In patients with a chronic migraine or frequent and/or disabling
episodic migraine, preventive treatment should be explored
to improve quality of life and decrease disability. Preventive
treatment for migraine has multiple aims, including decreasing
attack frequency by 50%, decreasing headache intensity
duration, improving acute therapy responsiveness,
improving overall function, and decreasing occurrence
of MOH. The choice of preventive treatment includes
consideration of patient comorbidities, drug interactions,
and side effects.\textsuperscript{[1]} In addition to non-pharmacologic methods such
as stress management and relaxation techniques, commonly
used medications for migraine prevention include beta-
blockers and antiepileptics.\textsuperscript{[4]} On average, 40–45% of patients
taking prophylactic medications experience a 50% reduction
in migraine frequency, possibly limited by adherence due to
drug side effects.\textsuperscript{[3]}

OnabotulinumtoxinA (BTA) is approved for the management
of chronic migraine. This treatment is delivered as
intramuscular injections targeting 31 different head and neck
sites every 12 weeks. BTA blocks acetylcholine release at the
synaptic cleft, resulting in the autonomic blockade. In a meta-
analysis by Jackson \textit{et al.}, BTA was associated with fewer
headaches per month and a greater likelihood of experiencing
a 50% reduction among patients with chronic migraine
headaches, compared to placebo.\textsuperscript{[5]} Further, Hepp \textit{et al.}
showed that when compared to oral migraine prophylaxis,
BTA was associated with a signiﬁcantly lower likelihood of
headache-related ED visits and hospitalizations at 6, 9, and
12 months after initiating treatment.\textsuperscript{[6]}

Sphenopalatine ganglion (SPG) blockade, the focus of this
instructional article, is another emerging therapy for patients
with chronic migraine. Greenﬁeld Sluder ﬁrst described
the SPG in association with facial pain syndromes in 1909,
and it is now targeted in the treatment of multiple disorders
including sphenopalatine neuralgia, atypical facial pain,
migraine, cluster headache, and herpes zoster.\textsuperscript{[7]}

The SPG is located in the pterygopalatine fossa and is one
of the four cranial parasympathetic ganglia. Activation of
parasympathetic synapses within the SPG releases vasoactive
peptides that contribute to neurogenic inﬂammation,
vasodilation, and the symptoms of migraine.\textsuperscript{[8]} Cranial
autonomic symptoms commonly associated with migraine,
such as lacrimation, conjunctival injection, eyelid edema,
nasal congestion, and facial swelling, may be mediated by this
parasympathetic outﬂow of the SPG.\textsuperscript{[9]} By applying topical
local anesthetic to the SPG, parasympathetic and sensory
outﬂow may be attenuated, thereby treating migraine and its
associated autonomic symptoms.

Blockade of the SPG has been achieved through transnasal,
transoral, and infrrazygomatic arch application of local
anesthetics. Today, there are three FDA approved devices
exist for delivery of blocking agents, namely SphenoCath\textsuperscript{®}
(Dolor Technologies, Scottsdale, Arizona, USA), Allevio\textsuperscript{™}
(Jet Medical, Schwenksville, PA, USA), and Tx360\textsuperscript{®} (Tian
Medical Inc., Lombard, IL, USA), all which approach the
ganglion transnasally.\textsuperscript{[10]}

The effectiveness of this treatment in acute and long-term
relief of migraine has been shown. Cady \textit{et al.} described
SPG blockade in patients with a chronic migraine using the
Tx360\textsuperscript{®} device. In this setup, the device is advanced below
the middle turbinate to the pterygopalatine fossa, where 0.3
cc of 0.5% bupivacaine is injected. In 26 patients treated
with bupivacaine compared to 12 patients treated with saline,
significant headache relief was noted at 15 and 30 min with
sustained relief at 24 h.\textsuperscript{[11]} In follow-up of the same patient
Group, decreased number of headache days at 1 month post-
treatment, decreased acute medication usage at 6 months, and
improved quality of life at 6 months were all reported.\textsuperscript{[12]}

Similar effectiveness of SPG blockade was reported in a study
by Mandato \textit{et al.} They studied the response of 112 patients
with a chronic headache who were treated with 4% of
xylocaine using the Allevio\textsuperscript{™} device. At 30 days, 88% of
patients required less medication for ongoing migraine relief,
with a 36% point reduction in the visual analog scale score
used to quantify the degree of debilitation.\textsuperscript{[13]}

Preliminary evidence shows SPG blockade is an effective and
safe option for treatment and prevention of migraine disorders.
We will describe how the authors use the SphenoCath\textsuperscript{®} device.

\textbf{Clinical evaluation of the patient}

All patients are seen in consult. A level III E and M visit is
documented with migraine disability assessment (MIDAS)
scores recorded. The nares and posterior oropharynx are
visually inspected with an otoscope. Informed consent is
obtained. We discuss transient hypotension, dysphagia
with possible aspiration, and arranging for a driver for the
treatment. Relative contraindications include pre-existing
cardiac rhythm abnormalities and severe hypotension. Patient
evaluation should include pre-existing medical conditions, current medications, and allergies. Baseline blood pressure and heart rate pre-procedure should also be obtained.

**Indications/contraindications for the procedure**

This procedure is indicated in patients with frequent, refractory episodic, or chronic migraine who have not achieved adequate migraine control with medical prophylaxis or who do not want or are not candidates for onabotulinumtoxinA injections. Contraindications include allergy to lidocaine, nasal canal atresia or stenosis, inability to thread the catheter, and unstable cardiac arrhythmia.

**Equipment needed**

a. SphenoCath® Device
b. Lidocaine (5 mL of 4%, lidocaine jelly)
c. Fluoroscopy

**Procedural steps**

a. Anesthetize nasal passageway before device insertion with a small quantity of lidocaine jelly on a Q-tip.
b. Place patient in supine position with cervical spine extension. The C arm is rotated into a cross-table lateral. The neck is extended, and the head is adjusted, so the mandibular angles are superimposed.
c. Insert SphenoCath® along the anterior nasal passage and place superior to the middle nasal turbinate (Figures 1 and 2).
d. Use fluoroscopy to confirm the location of the tip of the sheath.
e. Advance inner catheter. Slowly inject 1–2 mL of contrast under fluoroscopy to visualize a fluid/fluid level (or transient delay of flowing contrast) in the pterygopalatine fossa.
f. Next, administer 2 mL of 4% lidocaine to saturate the PPF.
g. Remove device and repeat on the opposite side.
h. The patient is maintained in a supine position for 10 min.\textsuperscript{[10]}

**Adverse effects**

a. Blood pressure and heart rate should be checked pre-procedure and post-procedure, as transient low blood pressure is a possible effect of treatment.
b. Patients may feel mild discomfort or burning during the procedure, and the medication may lead to an unpleasant taste.
c. There may be numbness in the back of the throat after the procedure. Patients should avoid eating or drinking until the numbness subsides to avoid choking.
d. Nausea and epistaxis may also occur.\textsuperscript{[10]}

**Clinical follow-up**

Patients are instructed to contact the provider in case of adverse events.

Unless the patient experiences complete relief for 1 week, treatments are repeated weekly for a total of three treatments, to achieve stepwise decrease in symptoms. If the patient is satisfied with the level of relief after the three treatments, maintenance treatments can be performed every 6–8 weeks. The authors have patients who have experienced lasting relief up to 6 months with just a single treatment. This, however, is the exception rather than the rule.

Our practice defines clinical success as a minimum of 50% reduction in severity and/or frequency of symptoms, lasting for at least 30 days. Other indirect signs of success are improved
MIDAS score, decreased use of medications, and decreased disability. Patients can choose to terminate the treatments at any time if they are not satisfied with the amount of symptom relief. Our success rate using the above criteria is 61%, well in line with other treatment modalities. SPG blockade can also be used as an adjunct with other treatment modalities.

**CONCLUSION**

Sphenopalatine ganglion blockade is a treatment option for those who suffer an episodic or chronic migraine, as it shown to be effective as acute migraine treatment and prevention of recurrent migraine. The SphenoCath® method described above is a safe and reproducible method to achieve SPG blockade. Research is currently being conducted to further characterize the effectiveness of this device in both acute and long-term migraine treatment.

**REFERENCES**


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