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# HYPERHYDROSIS AND SPHENOPALATINE GANGLION BLOCK (A case report)

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## Introduction

Sweating is a physiological response of the body for thermal regulation influenced by the change in skin temperature (1) mediated by sympathetic nervous system. In about 1% of the population, the sympathetic nervous system is overactive, causing certain areas of the body to sweat at inappropriate times and beyond what is necessary to maintain thermal regulation. This disorder is known as primary hyperhidrosis (2). The definition of hyperhidrosis as excessive sweating is subjective. It is defined quantitatively as the production of more than 100 mg of sweat in 1 axilla over 5 minutes (3). Excessive sweating (hyperhidrosis) or its absence (anhidrosis) are distressing, often attributed to organic causes requiring investigation and treatment (4). Hyperhidrosis may be focal or generalized. Focal hyperhidrosis is most often essential, or idiopathic, and results from a neurogenic overactivity of the sweat glands in the affected area. Focal hyperhidrosis usually affects the axillae, palms, soles of the feet, face, and, rarely, other areas. Focal hyperhidrosis, a physiologic condition, which is psychologically distressing affects up to 0.5% of the population during the second or third decade of life (5). Facial sweating affects up to 10% of patients with idiopathic hyperhidrosis. Facial hyperhidrosis differs from gustatory sweating, which occurs on the cheek in response to salivation or anticipation of food. Craniofacial hyperhidrosis has been a clinical problem overshadowed by palmar, plantar, and axillary hyperhidrosis. There are very few reports in the literature dealing with craniofacial hyperhidrosis, despite being a psychologically and socially distressing problem. A case of idiopathic excessive sweating treated successfully with Sphenopalatine ganglion block is reported.

## Case History

25 year old female had a history of profuse sweating over forehead and scalp of 15 years duration. The sweating was profuse, constant, increasing during summer, and on exposure to heat source. The profuse sweating prevented her from cooking, socializing and going out in the sun. She tied a cotton cloth (as an absorber) covering her scalp, and forehead to prevent constant wiping or pouring down of sweat on the face. She had been treated earlier by physicians, neurologists, dermatologists, and ENT surgeons without benefit. The treatment she had received were benzodiazepines (diazepam), Betablocker (propranolol), Antidepressant (amitryptaline), and topical creams. She had been suggested to undergo stellate ganglion block or cervical sympathectomy which she had refused. On examination the sweat was non odorous, bilateral, extending from vortex in the scalp to the eyebrows in the front. She was depressed, with a withdrawn personality as many therapies had failed. She was not on any medications. Routine blood screening, including a thyroid profile, complete blood cell count, and complete metabolic panel, revealed normal results.

## Treatment

She received bilateral Sphenopalatine Ganglion block (SPG), on two separate occasions at weekly intervals using Prasanna and Murthy technique (6). This technique involves the identification of the sphenopalatine ganglion, located just behind and above the junction of posterior end of the middle and superior turbinates medial to the sphenopalatine foramen covered by the mucosa in the lateral wall of the nose. Using a nasal rigid endoscope in supine position with a 15° head up tilt, this site is recognizable at the vanishing point (7). The nose was sprayed initially with 4% xylocaine containing 1: 100,000 adrenaline (freshly prepared) for local anaesthetic and decongestant effect. A cotton tipped probe soaked in 4% xylocaine with 1: 100,000 adrenaline was introduced under vision and placed against the mucosa for 5 minutes. This is a surface anesthetic block and not an injection of the ganglion involved.

## Results

There was a 50% reduction in the sweating over the forehead and scalp with the first block and 90% improvement with the second block. There was slight discomfort for 24 hours in the nose after each block which did not merit intervention. The subjective improvement with the first block was, the patient reporting decreased sweating. The objective improvement with the second block was that she no longer tied the cloth around the scalp and forehead, could venture out in the hot sun or socialize, could go near a heat source and enjoy cooking without the previous discomfort. The sweating did not increase during summer. A follow up of 12 months has shown no recurrence of the complaint.

## Discussion

Primary hyperhidrosis is diagnosed based on the patient's medical and family history (8), symptom presentation, and a physical examination with normal findings, since there are no diagnostic tests to confirm. The sweat glands and their innervation do not show any histologic abnormalities. A dysfunction of the central sympathetic nervous system, possibly of hypothalamic nuclei, or prefrontal areas or their connections is suspected (9,10). Sufferers display no other signs or symptoms of autonomic dysfunction. Secondary hyperhidrosis may be due to metabolic and infectious disorders, ruled out by performing laboratory tests such as a complete blood cell count, a thyroid-stimulating hormone level, follicle-stimulating hormone and luteinizing hormone levels, chemistry panel, fasting blood sugar, prolactin panels, and plasma-free testosterone. Innervation of the eccrine glands originates from the hypothalamic preoptic sweat center and travels down through the brainstem and medulla. The nerve fibers synapse in the intermediolateral cell columns of the spinal cord without significant crossing. The myelinated preganglionic fibers pass out in the anterior roots to the sympathetic chain and synapse. Unmyelinated postganglionic sympathetic C fibers arising from sympathetic ganglions join the peripheral nerves and end around the sweat gland. The supply to the skin of the upper limbs is usually from T2 to T8. The trunk is supplied by T4 to T12, and the lower limbs by T10 to L2. There is significant overlap of innervation in the sympathetic dermatome; a single preganglionic fiber can synapse with several postganglionic fibers. (11). Sweating over the face, forehead and vortex is mediated by the cholinergic parasympathetic fibers carried by the facial nerve via the greater superficial petrosal and vidian nerves to relay in the Sphenopalatine ganglion (12) and trigeminal nerve (13,14). It is not mediated by the cervical sympathetic outflow. Hence cervical chemical or surgical sympathectomy or stellate ganglion block will not be of benefit. Sympathetic outflow to the skin includes cholinergic neurons innervating sweat glands and adrenergic neurons innervating blood vessels and hair follicles (vasoconstrictor and pilomotor neurons). Acetylcholine is the major neurotransmitter, making eccrine gland sympathetic innervation unique; noradrenaline is generally the neurotransmitter in sympathetic nerves. Other mediators have been localized in the periglandular nerves, such as adenosine triphosphate, natriuretic peptide, calcitonin gene-related peptide, galanin, catecholamines, and vasoactive intestinal peptide. The significance of

these substances is not fully understood. The sweating over the face is a thermoregulated vascular response (15) during heat stress, influenced by the change in skin temperature (1). Neuropeptides play an important role in regulating this vascular response (16). This trigeminal parasympathetic vasodilator reflex increases the forehead circulation by the release of Vasoactive intestinal peptide (VIP) (13) during heat stress, mediated by the sympathetic and parasympathetic fibers passing through the sphenopalatine ganglion (17). The vascular tone and glandular secretion is modulated by the nitric oxide synthetase and Vasoactive intestinal peptide present in the sphenopalatine ganglion and trigeminal nerve (18). Sphenopalatine ganglion is the main contributor of nitric oxide synthetase containing nerve fiber (19). The preganglionic parasympathetic nerve in the SPG liberates nitric oxide synthetase to secrete acetylcholines (20). The cholinergic preganglionic parasympathetic fibres are mediated by acetylcholine, (4,13) vasoactive intestinal polypeptide (VIP) (18,19) and peptide histamine isoleucine (PHI) (21). Treatment of primary hyperhidrosis remains challenging. The treatment options depends on the presence or absence of an underlying cause. Topical agents used include aluminum chloride, potassium permanganate, glutaraldehyde, and formaldehyde, but their effects are only short-term (22). Oral anticholinergic agents have been used, but with many undesirable systemic side effects (23). Tranquilizers such as diazepam (2) as well as central-acting  $\alpha$ -adrenergic agonists such as clonidine (24,25), have been used but are limited by their neurocardiovascular side effects. The effectiveness of  $\beta$ -blockers in primary hyperhidrosis lacks support from the literature (26). The Surgical excision of affected areas has been useful in some cases but is generally limited to the axillae (27). Endoscopic thoracic sympathectomy (ETC) with a success rate of 92% to 99%, has been used in hyperhidrosis of palm although the complications are significant (2). Iontophoresis has been shown to successfully control palmar and plantar sweating via a mechanism thought to be due to portal plugging (28). Multiple injections of Botulinum toxin A have been popular with varied efficacy in palmar and axillary hyperhidrosis (29, 30). The modalities used for craniofacial hyperhidrosis include a combination of Sympathetic block with oral Clonidine and topical application of antiperspirant (25), combination of iontophoresis and topical application of anticholinergics (31), and local injections of botulinum toxin A (BTX-A) (32). The use of Alternative Therapies such as homeopathy, herbal remedies, biofeedback, acupuncture, and hypnosis therapies, have been universally disappointing. The various modalities have not proven to be completely effective. They are associated with side effects. Hence we contemplated the surface nerve block with local anaesthetic in this case, since the cause was an autonomic imbalance. The technique is a temporary chemical sympathectomy, with no side effects unlike the permanent surgical sympathectomy. Sphenopalatine ganglion (SPG) is a conglomeration of sympathetic, parasympathetic and sensory neurons situated in the lateral wall of the nose . It is the first relay station for the autonomic fibers after emerging from the pons (12). SPG block as a primary mode of pain therapy has been clinically used for a variety of disorders of the orofacial regions (33), eye (13) Sphenopalatine neuralgia (34), Otolgia (35,36), trigeminal neuralgia of V2 (37), Vasomotor rhinitis (38), cluster headache (39), and after endoscopic sinus surgery (40). In this case the local anaesthetic Xylocaine with adrenaline minimized the secretion of the acetylcholine possibly by blocking the preganglionic parasympathetic and the sympathetic fibers in the Sphenopalatine ganglion. Thus probably reducing the release of nitric oxide synthetase, and the vascular response resulting in minimizing the sweating. This technique is simple, safe and well accepted by the patient as an outpatient procedure.

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