Bilateral Stellate Ganglion Blockade for Recalcitrant Oral Pain from Burning Mouth Syndrome: A Case Report

David R. Walega, MD
Associate Professor of Anesthesiology
Chief, Division of Pain Medicine
Northwestern University
Feinberg School of Medicine
Chicago, Illinois, USA

Clark Smith, MD, MPH
Instructor
Physical Medicine & Rehabilitation
Columbia University Medical Center
New York, New York

Joel B. Epstein, DMD, MSD, FRCD(C)
Collaborative Member
Samuel Oschin Comprehensive Cancer Institute
Cedars Sinai Medical Center
Los Angeles, California
Adjunct Professor and Director of Oral Medicine
City of Hope National Medical Center
Duarte, California, USA

Correspondence to:
Dr David R. Walega
Division of Pain Medicine
Northwestern University
Feinberg School of Medicine
251 East Huron Street, #5-704
Chicago, Illinois, USA
Email: d-walega@northwestern.edu

©2014 by Quintessence Publishing Co Inc.

Burning Mouth Syndrome (BMS) is a chronic painful disorder characterized by unremitting bilateral burning oral pain often associated with taste abnormalities and complaints of dry mouth. The diagnosis is made by history and symptom presentation in the absence of an identifiable cause or oral lesion. It is commonly seen in perimenopausal women but is also seen in men, and is considered a small-fiber neuropathy. Management can be challenging and few effective treatments are available. This article presents a case report of stellate ganglion blockade as a treatment for recalcitrant pain from BMS. J Oral Facial Pain Headache 2014;28:171–175. doi: 10.11607/ofph.1165

Key words: *burning mouth syndrome, neuropathic pain stellate ganglion injection, small-fiber neuropathy, trigeminal nerve*

Burning mouth syndrome (BMS) is a chronic debilitating oral pain disorder characterized by unremitting burning sensations in the oral cavity, which may be associated with alterations in taste and taste perception, in the absence of relevant clinical or laboratory findings to account for the pain.[1–4] The International Association for the Study of Pain recognizes BMS as a distinct clinical entity.[5] The prevalence of BMS is 0.7% in the general population and increases significantly with age.[6,7] BMS rarely presents before the age of 30 in either sex and has been identified in all races and socioeconomic classes.[3] It is seen in up to 15% of perimenopausal and postmenopausal women and is much less common in men. The relatively higher proportion of postmenopausal women with BMS has led to interest in the role of estrogen in BMS and other chronic orofacial pain states.[8] It appears to be a painful small-fiber neuropathy involving the trigeminal nerve,[9,10] although its mechanism is not fully understood.

Unlike the pain of trigeminal neuralgia, the pain associated with BMS is not triggered by tactile or thermal stimuli and is typically bilateral. Pain is constant, moderate to severe in intensity, and is usually localized to the tongue and/or lips. Altered taste sensation, dysguesia, and a sensation of oral dryness are frequently reported, although saliva production may be normal.[11,12] The management of pain symptoms can be challenging and few effective treatments are available.

The stellate ganglion, part of the cervical sympathetic trunk, is composed of preganglionic fibers for the head and face; it mediates vasoconstrictor, sudomotor, and glandular secretory functions. Stellate ganglion blockade has been used to manage multiple pain conditions of the head and face, including complex regional pain syndrome, herpes zoster, cancer pain, trigeminal neuralgia, sympathetically mediated pain, and vascular headache. In many of these conditions, dysesthesia, hyperalgesia, hyperpathia, or other abnormal sensations are diminished by stellate ganglion blockade. In this case report, the use of bilateral stellate ganglion blockade is described as a treatment for recalcitrant pain associated with BMS.

© 2014 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY.
NO PART MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITHOUT WRITTEN PERMISSION FROM THE PUBLISHER.

Table 1 Patient's Scores on Three Pain Rating Scales Before and After Stellate Ganglion Blockade

			Follow-up visit before repeat injection		
	Pre-injection	Post-injection	Week 2	Week 3	Week 4
VRS score					
Treatment 1	4	1.5			
Treatment 2	3	1			
Treatment 3	2	1			
PGIC score*					
Treatment 1		2			
Treatment 2		2			
Treatment 3		3			
SF-MPQ-2†	11		9	9	9

*0 = "Much Better; 10 = "Much Worse."
†Maximum score 20.
VRS, verbal rating scale; PGIC, patient's global impression of change; SF-MPQ-2, Short-Form McGill Pain Questionnaire-2.

Case Report

A 53-year-old healthy male attorney presented to a university-based hospital pain clinic complaining of 6.5 months of bilateral burning pain in the anterior tongue and buccal mucosa of the lips and noting no inciting factors or incident. His verbal rating scale (VRS) score for pain varied from 4 to 7 (10-point scale). Pain was present upon awakening and worsened throughout the day. Eating dry or spicy food also caused the pain to worsen. Pain significantly improved with gum chewing and was unchanged by food temperature, mood, sleep, or any specific activity. His ability to discriminate taste was significantly diminished, but the patient denied the presence of dysguesia. His past medical history was significant for hypothyroidism, diagnosed 5 years prior to the onset of pain symptoms, and he was euthyroid on levothyroxine. He had hypercholesterolemia treated with simvastatin. He had surgery to repair an inguinal hernia 23 years prior to presentation. He was married, had no children, did not use tobacco, and drank 1 to 2 glasses of wine 3 to 4 times per week. Despite the severity of his pain symptoms, he continued to work full time. Of note, he had no psychiatric history and no other chronic pain syndromes preceding the onset of BMS. He had been evaluated by multiple specialists and carried the diagnosis of BMS.

The patient had previously failed multiple medication trials to modulate his pain symptoms. Gabapentin caused sedation, confusion, and clumsiness when walking. He received a 6-week trial of pregabalin to a maximum dose of 150 mg twice daily, but it also caused intolerable sedation, cognitive slowing, and clumsiness when walking. Duloxetine 60 mg had no effect on the pain after 6 weeks. Alpha-lipoic acid and L-carnitine were used for over 2 months without any effect. Short trials of opiates caused sedation and were discontinued. He continued to use multivitamins and vitamin D 2,000 IU per day without any improvements in pain symptoms. A trial of vitamin B_{12} and zinc supplements for several weeks also produced no results. Using clonazepam 0.5 mg three times daily reduced the burning pain by 50%, but sedation was intolerable and prevented further dose titration.

Physical examination revealed a healthy-appearing male who was well developed and well groomed and in no distress. He had an appropriate mien and did not exhibit any pain behaviors or drug-seeking behaviors during his history, physical examination, or subsequent follow-up evaluations. He was 6 feet tall and weighed 85 kg. Vital signs were within the normal range. The oral cavity, tongue, and dentition were normal in appearance. There was no cervical or submandibular adenopathy. Cranial nerves were intact. Digital examination of the oral cavity showed no palpable masses. There was dysesthesia and hyperalgesia on the anterior tongue and hyperesthesia of the labial mucosa when these regions were stroked with a cotton-tipped swab and scratched with a coarse-tipped tongue depressor. There was no allodynia. There was normal sensation of the gingiva and frenulum with similar stimuli. Laboratory studies confirmed no hematologic abnormalities, and levels of electrolytes, testosterone, cortisol, thyroid hormone, glucose, transaminases, vitamin B_{12}, vitamin D, and zinc were all within the normal range.

Given the severity and neuropathic quality of the patient's pain symptoms and a history of multiple treatment failures, a trial of image-guided bilateral stellate ganglion injections with local anesthetic were recommended as a novel technique to alleviate pain. After informed consent was obtained, image-guided right stellate ganglion blockade was performed in the standard manner by using a 22-gauge, 1.5-inch needle placed at the right C6 vertebral body tubercle; a contrast study with radiopaque dye confirmed appropriate spread in the prevertebral fascial plane prior to the injection of 5 mL of 0.5% bupivacaine. The same technique was repeated on the contralateral side with the same volume of local anesthetic. Horner syndrome (miosis, ptosis, anhydrosis) was noted immediately and bilaterally. Within minutes following the procedure, the patient reported greater than 50% decrease in his VRS score for pain. Following a 1-hour recovery and observation period, the patient's global impression of change (PGIC), a validated measure assessing efficacy of treatment as an alternative to sole dependence on the VRS and commonly used in clinical pain studies, was assessed (Table 1). No hoarseness, difficulty swallowing, airway obstruction, cardiac arrhythmia, or hemodynamic changes

© 2014 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY.
NO PART MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITHOUT WRITTEN PERMISSION FROM THE PUBLISHER.

were observed. Following recovery and discharge, he maintained a pain diary and noted 75% to 80% reduction in pain for 20 hours.

Injections were repeated weekly for a total of three injection treatments. The stellate ganglion injections were performed in the morning, using the same procedural technique, drug, and dose. For the second and third injections, pain relief lasted 6 and 18 hours, respectively. Although pain scores and GPIC ratings improved immediately following each stellate ganglion block, a Short-Form McGill Pain Questionnaire-2 (SF-MPQ-2) performed at follow-up visits prior to repeat injections showed minimal sustained longitudinal improvement over the course of treatment (Table 1). The outcomes indicate that there was very significant pain relief immediately following the stellate ganglion blockade, lasting for at least the half-life of the local anesthetic. Based on the reproducible degree and duration of pain relief from each of the local anesthetic blocks, neurolysis of the stellate ganglion was given consideration as a future treatment.

Discussion

Multiple studies have identified unique peripheral and central nervous system changes in patients with BMS, which may provide insight into the mechanism of this pain disorder. Lauria et al identified decreased epithelial and subpapillary nerve fiber density and diffuse axonal derangement in tongue biopsies of 12 BMS patients compared to normal controls.[9] Yilmaz et al found an increase in transient receptor potential vanilloid (TRPV1) receptors and very significant increases in nerve growth factor (NGF) in tongue tissue biopsies of BMS patients compared to controls.[10] Quantitative sensory testing of the tongue has consistently shown peripheral nerve and sensory dysfunction in BMS patients.[13–15] Koszewicz et al identified abnormal autonomic activity in BMS patients; increased heart rate variability, increased sympathetic skin response, and higher autonomic dysfunction scores were seen in BMS patients compared to controls.[16]

Central pain processing appears uniquely different in BMS. Hagelberg et al used positron emission tomography scanning in BMS patients and compared findings to normal controls; they identified decreased endogenous dopamine levels in the putamen of BMS patients.[17] Albuquerque et al used functional magnetic resonance imaging and showed brain-activation patterns in BMS patients subjected to thermal stimulation of the tongue that were distinctly hypoactive when compared to controls who were stimulated in the same manner.[18] There was increased signaling in the anterior cingulate cortex and precuneus regions, and decreased signaling in the thalamus, frontal gyrus, and cerebellum, in a manner similar to other painful neuropathic pain conditions. This study inferred that BMS patients process orofacial nociceptive information differently than those without chronic pain.

As a chronic pain disorder with few treatment options, BMS has a significant negative impact on quality of life.[19] Symptoms occur continuously for months or years without periods of cessation or remission. In a study by Sardella et al, spontaneous remission was seen in only 3% of the BMS patients within 5 years of onset of symptoms.[20]

Pain management of BMS is challenging due to the limited effective treatment options. A 2012 Cochrane review and a meta-analysis of randomized controlled trials affirmed the lack of effective treatments for BMS.[21] Controlled trials of clonazepam[22,23] and amisulpride[24] showed decreases in pain scores in treatment groups as compared to placebo. Lafutidine, an H2-receptor antagonist that also modulates capsaicin-sensitive afferent nerve fibers, significantly improved pain symptoms in BMS patients in a placebo-controlled trial in Japan,[25] but the drug is not available in the US. A prospective trial of duloxetine showed efficacy in reducing pain scores in BMS patients, independent of depression,[26] but the study lacked a placebo group. Low-level light therapy was introduced as a potential therapy to reduce pain and has shown mixed results.[27,28] Case reports of improvement in pain in BMS with gabapentin,[29] pregabalin,[30] topiramate,[31] and pramipexol[32] have been published, though each infers a potentially unique mechanism for improving pain. Cognitive behavioral therapy may also be effective in reducing pain in BMS patients.[33]

To date, stellate ganglion injections have not been used to treat BMS, but are commonly used as an effective treatment in sympathetically dependent pain syndromes, trigeminal neuralgia, atypical facial pain, hyperhidrosis, and vascular insufficiency of the head and neck (and upper extremity).[34,35] Although stellate ganglion blockade results in regional vasodilation and increased blood flow to several areas within the brain, the mechanism by which stellate ganglion blockade induces neural inhibition and decreases neuropathic pain is a complex and incompletely understood phenomenon. Furthermore, the role of placebo response as contributing to analgesia with injection treatments should be appreciated.[36]

Wang et al hypothesized that stellate ganglion blockade reduces the release of substance P in response to noxious stimuli, leading to decreased nociceptive behavior and reduced hyperalgesia.[37] There is evidence that NGF is also a mediator of nociception in neuropathic pain. Herzberg et al identified up-regulation of NGF in injured nerves in an animal

© 2014 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY.
NO PART MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITHOUT WRITTEN PERMISSION FROM THE PUBLISHER.

model,[38] a finding confirmed with tongue tissue biopsies in BMS patients.[10] Birklein et al found that increases in NGF caused hyperesthesia.[39] Lipov et al hypothesized that stellate ganglion blockade may modulate NGF production,[40] which in turn decreases nociceptive transmission via central pathways, and stellate ganglion blockade could have an effect on peripheral trigeminal fibers, the trigeminal ganglion, or the spinal trigeminal nucleus, given the definitive increase in lingual NGF and hyperesthesia identified in BMS patients. It has also been demonstrated that the stellate ganglion has second-order and third-order synaptic connections to areas of the brain that modulate neuropathic pain, including the hypothalamus, amygdala, and infralimbic and insular cortices.[41] Stellate ganglion blockade and subsequent sympatholysis may mitigate the increased autonomic tone identified in BMS patients[16] and modulate regional blood flow, brain activation patterns, or central nociceptive processing. The potential interplay between these and other peripheral and central mechanisms points to the sympathetic nervous system in general and the stellate ganglion in particular as potential modulators in treating the pain of BMS, and requires further study.

The efficacy of injection treatments for BMS pain symptoms is not well described in the literature. Gremeau-Richard et al used lingual nerve injections in BMS patients and suggested subcategories of BMS based on peripheral or central response to these local anesthetic injections.[42] This classification was subsequently used in the study to predict a treatment response to benzodiazepines with a moderate degree of accuracy. No patient in this trial experienced pain relief beyond the half-life of the local anesthetic used.

The present case demonstrated meaningful analgesia immediately following stellate ganglion blockade with bupivacaine. This finding cannot be explained solely on plasma levels of local anesthetic. In two of the three injections, the analgesic effect of the stellate ganglion blockade persisted beyond the pharmacologic half-life of the local anesthetic, through an unclear mechanism. This case further supports the hypothesis that the stellate ganglion mediates nociception in BMS through a complex interplay of mechanisms including neural pathways between the stellate ganglion, trigeminal nerve, trigeminal ganglion, spinal trigeminal nucleus, or regions of the brain involved in central pain processing. The mechanism by which stellate ganglion blockade modulates pain in BMS requires further study.

Stellate ganglion injections are considered safe when performed in appropriate patients by experienced practitioners. The incidence of complications with stellate ganglion blockade, predating the use of image guidance as a standard practice for these injections, has been reported to be 1.7 per 1,000 procedures and has been related to intravascular injection of local anesthetic that resulted in temporary seizures.[43] It follows that image-guided stellate injections would have far fewer complications, as critical vascular or neural structures could be visualized in real time and avoided. The safety aspects of injection procedures in the era of image guidance bears further investigation.

Bilateral stellate ganglion blockade should be performed cautiously, as inadvertent bilateral recurrent laryngeal nerve blockade could result in the temporary loss of laryngeal reflexes and subsequent aspiration or airway obstruction. Phrenic nerve paralysis, if bilateral, could also cause ventilatory compromise for the duration of the local anesthetic half-life. Unilateral phrenic nerve paralysis, although temporary, is well tolerated in healthy patients but may be more problematic in those with underlying pulmonary disease and low pulmonary reserves. Bilateral stellate ganglion blockade could, in theory, result in decreased sympathetic tone of the cardiac accelerator fibers (T1-4) and result in bradycardia, posing a potential risk for BMS patients with underlying cardiac disease or arrhythmias.

Conclusions

Treatments that block the stellate ganglion may have a role in treating pain in BMS patients who fail conservative treatment. Further prospective controlled studies are needed to identify the clinical efficacy of stellate ganglion blockade in BMS and further elucidate the mechanism of pain modulation under such conditions.

Acknowledgments

The authors report no conflicts of interest related to this study.

References

1. Danhauer SC, Miller CS, Rhodus NL, Carlson CR. Impact of criteria-based diagnosis of burning mouth syndrome on treatment outcome. J Orofac Pain 2002;16:305–311.
2. Woda A, Navez ML, Picard P, Gremeau C, Pichard-Leandri E. A possible therapeutic solution for stomatodynia (burning mouth syndrome). J Orofac Pain 1998;12:272–278.
3. Grushka M, Epstein JB, Gorsky M. Burning mouth syndrome. Am Fam Physician 2002;65:615–620.
4. Grushka M, Epstein JB, Gorsky M. Burning mouth syndrome and other oral sensory disorders: A unifying hypothesis. Pain Res Manag 2003;8:133–135.

© 2014 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY.
NO PART MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITHOUT WRITTEN PERMISSION FROM THE PUBLISHER.

5. Merskey H, Bogduk N. International Association for the Study of Pain. Task Force on Taxonomy. Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms. Seattle: IASP Press, 1994:xvi.
6. Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. J Am Dent Assoc 1993;124:115–121.
7. Bergdahl M, Bergdahl J. Burning mouth syndrome: Prevalence and associated factors. J Oral Pathol Med 1999;28:350–354.
8. Cairns BE. The influence of gender and sex steroids on craniofacial nociception. Headache 2007;47:319–324.
9. Lauria G, Majorana A, Borgna M, et al. Trigeminal small-fiber sensory neuropathy causes burning mouth syndrome. Pain 2005;115:332–337.
10. Yilmaz Z, Renton T, Yiangou Y, et al. Burning mouth syndrome as a trigeminal small fibre neuropathy: Increased heat and capsaicin receptor TRPV1 in nerve fibres correlates with pain score. J Clin Neurosci 2007;14:864–871.
11. Nagler RM, Hershkovich O. Sialochemical and gustatory analysis in patients with oral sensory complaints. J Pain 2004;5: 56–63.
12. Tammiala-Salonen T, Soderling E. Protein composition, adhesion, and agglutination properties of saliva in burning mouth syndrome. Scand J Dent Res 1993;101:215–218.
13. Grushka M, Sessle BJ, Miller R. Pain and personality profiles in burning mouth syndrome. Pain 1987;28:155–167.
14. Granot M, Nagler RM. Association between regional idiopathic neuropathy and salivary involvement as the possible mechanism for oral sensory complaints. J Pain 2005;6:581–587.
15. Forssell H, Jaaskelainen S, Tenovuo O, Hinkka S. Sensory dysfunction in burning mouth syndrome. Pain 2002;99:41–47.
16. Koszewicz M, Mendak M, Konopka T, Koziorowska-Gawron E, Budrewicz S. The characteristics of autonomic nervous system disorders in burning mouth syndrome and Parkinson disease. J Orofac Pain 2012;26:315–320.
17. Hagelberg N, Forssell H, Rinne JO, et al. Striatal dopamine D1 and D2 receptors in burning mouth syndrome. Pain 2003;101:149–154.
18. Albuquerque RJ, de Leeuw R, Carlson CR, Okeson JP, Miller CS, Andersen AH. Cerebral activation during thermal stimulation of patients who have burning mouth disorder: An fMRI study. Pain 2006;122:223–234.
19. Souza FT, Santos TP, Bernardes VF, et al. The impact of burning mouth syndrome on health-related quality of life. Health Qual Life Outcomes 2011;9:57.
20. Sardella A, Lodi G, Demarosi F, Bez C, Cassano S, Carrassi A. Burning mouth syndrome: A retrospective study investigating spontaneous remission and response to treatments. Oral Dis 2006;12:152–155.
21. de Moraes M, do Amaral Bezerra BA, da Rocha Neto PC, de Oliveira Soares AC, Pinto LP, de Lisboa Lopes Costa A. Randomized trials for the treatment of burning mouth syndrome: An evidence-based review of the literature. J Oral Pathol Med 2012;41:281–287.
22. Gremeau-Richard C, Woda A, Navez ML, et al. Topical clonazepam in stomatodynia: A randomised placebo-controlled study. Pain 2004;108:51–57.
23. Heckmann SM, Kirchner E, Grushka M, Wichmann MG, Hummel T. A double-blind study on clonazepam in patients with burning mouth syndrome. Laryngoscope 2012;122:813–816.
24. Rodriguez-Cerdeira C, Sanchez-Blanco E. Treatment of burning mouth syndrome with amisulpride. J Clin Med Res 2012; 4:167–171.
25. Toida M, Kato K, Makita H, et al. Palliative effect of lafutidine on oral burning sensation. J Oral Pathol Med 2009;38:262–268.
26. Nagashima W, Kimura H, Ito M, et al. Effectiveness of duloxetine for the treatment of chronic nonorganic orofacial pain. Clin Neuropharmacol 2012;35:273–277.
27. Pezelj-Ribaric S, Kqiku L, Brumini G, et al. Proinflammatory cytokine levels in saliva in patients with burning mouth syndrome before and after treatment with low-level laser therapy. Lasers Med Sci 2013;28:297–301.
28. dos Santos Lde F, Carvalho Ade A, Leao JC, Cruz Perez DE, Castro JF. Effect of low-level laser therapy in the treatment of burning mouth syndrome: A case series. Photomed Laser Surg 2011;29:793–796.
29. White TL, Kent PF, Kurtz DB, Emko P. Effectiveness of gabapentin for treatment of burning mouth syndrome. Arch Otolaryngol Head Neck Surg 2004;130:786–788.
30. Lopez V, Alonso V, Marti N, Calduch L, Jorda E. Marked response of burning mouth syndrome to pregabalin treatment. Clin Exp Dermatol 2009;34:e449–e450.
31. Siniscalchi A, Gallelli L, Marigliano NM, Orlando P, De Sarro G. Use of topiramate for glossodynia. Pain Med 2007;8:531–534.
32. Stuginski-Barbosa J, Rodrigues GG, Bigal ME, Speciali JG. Burning mouth syndrome responsive to pramipexol. J Headache Pain 2008;9:43–45.
33. Bergdahl J, Anneroth G, Perris H. Cognitive therapy in the treatment of patients with resistant burning mouth syndrome: a controlled study. J Oral Pathol Med 1995;24:213–215.
34. Elias M. Cervical sympathetic and stellate ganglion blocks. Pain Physician 2000;3:294-304.
35. Salvaggio I, Adducci E, Dell'Aquila L, et al. Facial pain: A possible therapy with stellate ganglion block. Pain Med 2008;9: 958–962.
36. Benedetti F, Amanzio M. The neurobiology of placebo analgesia: From endogenous opioids to cholecystokinin. Prog Neurobiol 1997;52:109–125.
37. Wang QX, Wang XY, Fu NA, Liu JY, Yao SL. Stellate ganglion block inhibits formalin-induced nociceptive responses: Mechanism of action. Eur J Anaesthesiol 2005;22:913–918.
38. Herzberg U, Eliav E, Dorsey JM, Gracely RH, Kopin IJ. NGF involvement in pain induced by chronic constriction injury of the rat sciatic nerve. Neuroreport 1997;8:1613–1618.
39. Birklein F, Schmelz M. Neuropeptides, neurogenic inflammation and complex regional pain syndrome (CRPS). Neurosci Lett 2008;437:199–202.
40. Lipov EG, Joshi JR, Sanders S, Slavin KV. A unifying theory linking the prolonged efficacy of the stellate ganglion block for the treatment of chronic regional pain syndrome (CRPS), hot flashes, and posttraumatic stress disorder (PTSD). Med Hypotheses 2009;72:657–661.
41. Westerhaus MJ, Loewy AD. Central representation of the sympathetic nervous system in the cerebral cortex. Brain Res 2001;903:117–127.
42. Gremeau-Richard C, Dubray C, Aublet-Cuvelier B, Ughetto S, Woda A. Effect of lingual nerve block on burning mouth syndrome (stomatodynia): a randomized crossover trial. Pain 2010;149:27–32.
43. Wulf H, Maier C. Complications and side effects of stellate ganglion blockade. Results of a questionnaire survey [in German]. Anaesthesist 1992;41:146–151.

© 2014 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY.
NO PART MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITHOUT WRITTEN PERMISSION FROM THE PUBLISHER.