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## CLINICAL REPORTS

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# Nerve Stimulator-Guided Repetitive Paravertebral Block for Thoracic Myofascial Pain Syndrome

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■ **Abstract:** Myofascial pain syndrome (MPS) may persist for many years and is often refractory to traditional therapeutic approaches including pharmacotherapy, focal tenderness infiltration by local anesthetic and corticosteroids, physical therapy and behavioral modification. This report describes three cases of MPS following coronary artery bypass graft, inadequate positioning during abdominal hysterectomy, and excessive physical effort refractory to conventional therapeutic approaches. Three patients were successfully treated with repeated nerve stimulator-guided paravertebral block using a mixture of bupivacaine and clonidine.

Physical examinations including a complete neurological assessments were unremarkable. Relevant diagnostic imaging (X-ray, magnetic resonance imaging, computed

tomography) and laboratory evaluations also failed to demonstrate any significant structural disorders or systemic diseases that might have been responsible for their pain. Nerve stimulator-guided paravertebral block was performed at the dermatomes corresponding to the thoracic myofascial pain region. Each point was injected with 4 mL of the local anesthetic solution. If the pain returned, a second paravertebral block was performed. The three patients were pain-free over a follow-up period up to 2 years. Our report suggests that nerve stimulator-guided paravertebral blockade could be a useful treatment for MPS refractory to traditional therapeutic approaches. ■

**Key Words:** myofascial pain syndrome, neuroplasticity, paravertebral block, trigger point, clonidine.

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## INTRODUCTION

Myofascial pain syndrome (MPS) is a chronic muscle pain disorder in one or more muscles associated with focal tenderness called trigger points.<sup>1,2</sup> The term Myofascial Trigger Point (MTrP) is defined as a focal tenderness in a taut band of skeletal muscle that is painful on compression and responsible for the pain in the zone of

reference.<sup>3</sup> Pain can be localized to the site of the taut band, distant from it, or referred to another part of the body.<sup>4</sup> MTrP varies from being spontaneously painful to being dormant and painful once it is stimulated.<sup>4</sup> Restricted range of motion and increase sensitivity may also cause tightness of the involved muscle.<sup>5-7</sup> MPS may persist for many years; it can be refractory to traditional therapeutic approaches including pharmacotherapy, MTrP infiltration, physical therapy, and behavioral modification.<sup>8-10</sup>

### CASE ONE

A 50-year-old man, ASA II, height 180 cm, weight 84 kg, underwent coronary artery bypass grafting followed by a 13-month history of moderate to severe bilateral parietal chest pain with a level of 6 to 8 on the Visual Analog Scale (VAS) (with 0 = no pain, and 10 = the worst pain imagined). A complete cardiovascular investigation ruled out pain of cardiac origin. His past medical history revealed stable chronic hypertension and a cerebral transient ischemic attack 3 years ago without neurological sequelae. Physical exam revealed bilateral T3-T5 deep intercostal muscular pain extending posteriorly to the back region with a trigger point at T4. No skin change, such as residual scarring, edema or inflammation, was noted. Pain was unrelieved by physical therapy, oral muscle relaxants, nonsteroidal inflammatory drug and tricyclic antidepressants. Bilateral Paravertebral Block (PVB) using 4 mL of the local anesthetic solution for each injection, guided by a nerve stimulator (Stimuplex, B.Braun, Melsungen, Germany), were performed at T3-T4 and T4-T5 levels respectively with a total anesthetic amount of 16 mL.<sup>11</sup> The local anesthetic mixture solution was prepared in 20 mL containing 19 mL of bupivacaine 0.5% and 1 mL of clonidine 150 µg/mL. The PVB blockades resulted in a total pain relief (VAS = 0/10) during a 2-year follow-up period.

### CASE TWO

A previously healthy 58-year-old woman patient, ASA II, height 170 cm, weight 53 kg, was referred with 8 months of left-sided parietal chest pain in the intercostal muscles. The pain, corresponding to the dermatomes T4-T6, began following abdominal hysterectomy under general anesthesia and was diagnosed as MPS. The pain was continuous, increasing progressively in intensity with movement and cough. The pain (VAS = 7-8/10) was associated with the presence of MTrP at T5. Previous treatment included acetami-

nophen, nonsteroidal anti-inflammatory drugs, tramadol hydrochloride, and tricyclic antidepressants. A few months later, gabapentin 300 mg three times per day was added and increased gradually to 3600 mg per day for a 6-month period. Moreover, MTrP infiltration with local anesthetic and corticosteroids at T5 was performed with little improvement. Upon presentation, the patient was still complaining of continuous myofascial pain with a VAS of 8/10 in the left thoracic region mainly at T4 extending to T6. Physical examination revealed hyperesthesia of the corresponding dermatomes. No skin change was noted. Chest X-ray, computed tomography (CT) scan of the thorax, and a magnetic resonance imaging (MRI) of the cervicothoracic region were unremarkable.

Left PVB at T4-T5 and T5-T6 levels guided by a nerve stimulator were performed with 4 mL of the local anesthetic solution at each point. The pain disappeared for 30 days and then gradually reappeared. A second PVB was performed at the same levels and the patient became pain-free for a 2-year follow-up period.

### CASE THREE

A 73-year-old woman patient, ASA II, height 161 cm, weight 62 kg, previously healthy, was referred with an 11-month history of right-sided parietal chest pain in the intercostal muscles corresponding to the dermatomes T5-T7. This pain was initially mild (VAS = 3/10) appearing only after excessive physical effort, but progressively worsened over a few days becoming severe (VAS = 8/10) with a trigger point at T6. Upon presentation, the patient was complaining of persistent pain with a VAS of 8/10 in the right thoracic region mainly at T5 extending to T7. The pain intensity increased upon elevation of the right upper arm reaching a VAS of 10/10. CT scan of the thorax was unremarkable. The pain was treated initially by nonsteroidal anti-inflammatory drugs, tramadol hydrochloride, gabapentin up to 3600 mg per day and tricyclic antidepressants. However, no improvement was achieved with T6 infiltration of local anesthetic, corticosteroids, or with physical therapy.

Nerve stimulator-guided PVB was performed at the right T4-T5 and T5-T6 levels with 4 mL of the local anesthetic solution at each injection site. The pain disappeared for 1 week, and then gradually reappeared with a lesser intensity (VAS of 5/10). PVBs were then repeated on a weekly basis for 1 month with significant improvement. The patient remained pain-free over a 2-year follow-up period.

## DISCUSSION

Despite a variety of traditional therapeutic approaches for the treatment of MPS, it continues to be the most common cause of persistent regional pain.<sup>10</sup> A rationale mechanistic approach to the treatment of MPS is currently unavailable because of an incomplete understanding of the underlying pathophysiology.

Energy crisis theory, motor endplate hypothesis, and nerve entrapment are three main mechanisms proposed to be involved in MPS.<sup>7,12,13</sup> In the energy crisis theory, MPS results from a loss of oxygen and nutrient supply in the presence of an increased metabolic demand. This leads to the release of neuroactive biochemicals, which sensitize nearby nerves that in turn initiate the motor, sensory, and autonomic effects of MTrP via the central nervous system. The motor endplate hypothesis involves both the nerve terminal and the postjunctional muscle fiber, presenting as an abnormal pattern of endplate activity because of excessive acetylcholine, thus characterizing MPS as a neuromuscular disease. This abnormal endplate activity is modulated by sympathetic nerve activity. Finally, pain may be due to the entrapment of a nerve root or one of its branches, leading to the development of MTrP activity in the corresponding muscles. While these three mechanisms presumably may coexist in patients, one typically is thought to predominate.

Myofascial pain syndrome is characterized by complex changes in peripheral signal processing that may lead to altered central pain processing and the development of central sensitization.<sup>7</sup>

All three patients underwent physical examination with a complete neurological assessment; diagnostic imaging (X-ray, MRI, CT), laboratory evaluation, and cardiovascular evaluation in order to identify a structural disorder or systemic disease. However, all results were unremarkable.

The success of the PVB in relieving the chronic pain in our patients could be multifactorial. The nerve stimulator allowed precise localization of the paravertebral nerves resulting in a high quality sensory blockade capable of abolishing somatosensory evoked potentials from the blocked dermatomes.<sup>14</sup> This may have reduced central sensitization and eliminated continuous pain stimuli because of mechanical movements. Repeated PVB may have improved oxygenation and blood supply to the local area and might result in damaged tissue regeneration. It is also possible that providing a pain-free period might have interrupted the established reverberatory neural circuit between the nociceptive, the

central nervous system and the motor unit resulting in pain alleviation.<sup>15,16</sup> In addition, the administration of clonidine in close proximity of the nerve has been also found to be capable of reversing the neuroplastic changes in dorsal horn neurons.<sup>17</sup> Clonidine provides an interaction with the immune system resulting in reduced recruitment of macrophages and lymphocytes at the nerve injury site and shift from the pro- to the anti-inflammatory phenotype.<sup>17,18</sup> In conclusion, our report suggests that nerve stimulator-guided paravertebral blockade could be a useful treatment for MPS refractory to traditional therapeutic approaches.

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