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Sympathetic blockade for complex regional pain syndrome

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In the 1990’s, the dogma that sympathetic blockade was a first-line treatment for complex regional pain syndrome (CRPS) was shattered by accusations that benefits were due to placebo effects and that this treatment approach was “entrepreneurially inspired” [7]. Since then, systematic examination of the literature base has provided little support for the use of local anaesthetic sympathetic blockade for CRPS, and has highlighted the scarcity of high-quality double-blind placebo-controlled trials with an adequate sample size and follow-up period [1; 9]. However, as sympathetic blockade continues to be used therapeutically for CRPS in clinical practice [10], further examination of its efficacy is imperative.

In this issue of Pain, Dr Rocha and colleagues [8] report that thoracic sympathetic blockade, as an adjunct to pharmacological and physical therapy for patients with upper-limb CRPS, resulted in positive effects on mood and reductions in pain detectable 12 months later. During the blockade, 1 mL of contrast media, followed by 5 mL of local anaesthetic agent and another 5 mL of corticosteroid solution were injected under fluoroscopic guidance around the second thoracic ganglion of the sympathetic chain. In the control group, 10 mL of local anaesthetic agent and corticosteroid solution were injected subcutaneously at the T2 level to cover the possibility that placebo effects or systemic absorption of these agents might provide therapeutic benefits. Scores on the McGill Pain Questionnaire and the average evoked pain score on the Neuropathic Pain Symptom Inventory were significantly lower in the treated than control group at one and 12 months, and average pain and depression scores were significantly lower in the treated than control group at 12 months.

What might account for such persistent treatment gains after just one sympathetic block? It seems quite unlikely that a placebo response to sympathetic blockade would be detected 12 months later. Substantial increases in hand temperature indicated that sympathetic blockade was successful. However, patients apparently remained blind to the type of treatment they received, despite the increase in hand temperature and the occasional presence of Horner’s sign. Although a case might be made for temporary treatment gains in patients with a component of “sympathetically maintained” pain [3], it seems inconceivable that transient blockade of ganglia in the thoracic sympathetic chain would inhibit pain and lift mood 12 months later.

Might the infusion of corticosteroid solution around the sympathetic chain have mediated long-term benefits? Adding steroids to local anaesthetic agents for sympathetic nerve blocks is based on the premise that suppressing inflammation in the sympathetic chain inhibits pain. There is some support for this. For example, when administered repetitively over the course of a week to the sympathetic chain with local anaesthetic agent, steroids seemed to provide therapeutic benefits for patients with acute herpes zoster and reduced the rate of progression to postherpetic neuralgia [4].

However, one might question whether the sympathetic chain was the only target of the local anaesthetic agent and corticosteroid solution in Rocha’s study, as contrast media in 11 mL of fluid
(the volume injected by Rocha et al.) can disperse a considerable distance from the site of injection. For example, local anaesthetic agent in volumes as low as 2 mL spreads across approximately five spinal segments and produces physiological signs of sympathetic blockade [5]. In an animal model of low back pain, steroids applied locally to inflamed dorsal root ganglia inhibited mechanical sensitivity and reduced abnormal spontaneous activity in myelinated sensory neurons [11]. Furthermore, after spinal nerve ligation in rats, corticosteroids infused around the involved dorsal root ganglia inhibited mechanical sensitivity and sympathetic sprouting into the ganglia [6]. Potentially, then, absorption of the corticosteroid solution into nearby dorsal root ganglia of patients in Rocha’s study might have contributed to treatment gains. Perhaps this or some other effect of the steroids, local anaesthetic agent or blocking procedure augmented the efficacy of physical treatments or pharmacotherapy, resulting in progressive reductions in pain and positive effects on mood over the 12 months of follow-up.

Although this study casts little light on the role, if any, of local anaesthetic sympathetic blockade in the management of CRPS, therapeutic effects were promising and warrant further investigation. And we should not lose sight of the bigger picture. A clear distinction needs to be made between the use of sympathetic nerve blocks to identify “sympathetically maintained” pain (when compared against an active placebo procedure) and the therapeutic application of sympathetic blockade for CRPS. Indeed, we may be asking the wrong question by querying the therapeutic role of local anaesthetic sympathetic blockade for CRPS [1; 9] – it would seem more pertinent to determine whether and how to interrupt sympathetic activity in patients with “sympathetically maintained” CRPS rather than in CRPS patients overall. To avoid a tautology, new approaches may be required to identify sympathetically maintained pain (e.g., placebo-controlled electrical or pharmacological stimulation of the sympathetic chain or sympathetic nerve terminals), and to distinguish between central and peripheral forms of this disorder [2]. We might then be better placed to determine whether therapies that target the sympathetic nervous system provide benefits for an identifiable subgroup of patients with CRPS.

Conflict of interest statement

None declared.

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References


