Anesthesia & Clinical Research

Navaie et al., J Anesth Clin Res 2014, 5:4
http://dx.doi.org/10.4172/2155-6148.1000403

Review Article Open Access

Use of Stellate Ganglion Block for Refractory Post-Traumatic Stress Disorder: A Review of Published Cases

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Abstract

Introduction: The lifetime prevalence of post-traumatic stress disorder (PTSD) is estimated to be 7.3% in the U.S. population, 10-20% among active duty service members and 35-40% among veterans. Overall success rates of evidence-based therapies for PTSD are low, leading clinicians to explore new therapeutic options. This study evaluated all published articles on the use of stellate ganglion block (SGB) as an adjunctive therapy for treatment-refractory PTSD.

Methods: EMBASE, PubMed, PsychINFO and Cochrane databases were searched using keyword combinations including stellate ganglion block, SGB, post-traumatic stress disorder, and PTSD. Articles were restricted to English language with no date delimiter. Twelve publications were identified, seven of which were eliminated due to lack of case data, duplicate patient sample, or descriptive reports with no standardized PTSD symptom assessment. Twenty-four cases from five articles were examined further by two independent evaluators who extracted data on sociodemographic and clinical characteristics including PTSD symptoms, comorbidities, and treatment history. Inter-rater reliability showed complete agreement (κ=1.0).

Results: Cases were predominantly male (n=21, 88%) and active duty military (n=14, 58%) or veterans (n=8, 33%) with combat-related PTSD. The average age was 40.5 years (±10.0 SD). All cases had received >1 year of psychotherapy and pharmacotherapy before SGB. Seventeen cases (71%) received one SGB, seven (29%) received multiple SGBs. Clinically meaningful improvements were observed in 75% (n=18) of cases after SGB, with significant differences in mean PTSD scores pre- (69.5 ± 26.6) and post-SGB (34.2 ± 32.5) across cases ($p<0.001$). The effect size was relatively large ($d=1.2$). On average, PTSD improved by 50.4% (± 30.9 SD; range: 6.3-98.4) for cases with one SGB and 69.0% (± 28.0 SD; range: 9.2-93.5) for cases with multiple SGBs.

Conclusions: Most patients with treatment-refractory PTSD experienced rapid improvement after SGB. Robust clinical trials are needed to determine SGB's treatment efficacy for PTSD.

Keywords: Stellate ganglion block; SGB; Regional anesthesia; Treatment-refractory post-traumatic stress disorder; Treatment-resistant PTSD; Military; Veterans

Introduction

Physical pain and psychological suffering are intimately connected. Therefore, it may not be surprising that as far back as 1947 a procedure that is normally used to treat pain, stellate ganglion block (SGB), was also reported to improve symptoms of psychological depression [1]. More recently, SGB has been shown to be helpful in the treatment of hallucinations in schizophrenia [2] and climacteric psychosis [3]. Another emerging use of SGB as a potentially beneficial treatment for psychiatric conditions has been in post-traumatic stress disorder (PTSD) [4].

An overview of ptsd: prevalence, diagnostic criteria, and treatment challenges

People often think of PTSD as a condition suffered by war veterans. Many earlier names for the condition, such as "shell shock" or "combat fatigue", emphasized war as a cause of the syndrome, and an estimated 10-20% of active duty service members and 35-40% of veterans do experience PTSD [5,6]. However, PTSD can also be caused by other traumas, such as sexual assault, life-threatening accidents, and natural disaster. In the United States, the lifetime prevalence of PTSD is estimated to be 7.3% in the U.S. population [7]. Epidemiologic studies have shown that women, African-Americans, Hispanics, and persons between the ages of 45 to 59 are at a higher risk for PTSD than other segments of the civilian population [5,8]. Familiar symptoms of PTSD include nightmares, flashbacks, exaggerated startle, emotional numbing, and an inability or unwillingness to engage in activities that remind the individual of the trauma [9].

Most reactions to trauma are not PTSD, and most individuals who experience stress-induced symptoms will recover without developing the full diagnostic criteria for PTSD. The specific syndrome of PTSD has been defined by the Diagnostic and Statistical Manual (DSM) as including symptoms of intrusively re-experiencing the trauma, avoiding trauma-related thoughts or actions, and a persistent state of hyper-

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Received April 09, 2014; **Accepted** April 28, 2014; **Published** May 20, 2014

Citation: Navaie M, Keefe MS, Hickey AH, McLay RN, Ritchie EC, et al. (2014) Use of Stellate Ganglion Block for Refractory Post-Traumatic Stress Disorder: A Review of Published Cases. J Anesth Clin Res 5: 403. doi:10.4172/2155-6148.1000403

arousal, all of which must be present for at least a month and cause significant impairment in social or occupational function. In 2013, the American Psychiatric Association through the fifth version of the DSM recognized a fourth cluster of symptoms, related to negative alterations in cognitions and mood [9]. The DSM-V definition also clarified the type of trauma that can cause PTSD and altered the exact pattern of symptoms that must be present, and specified sub-types within the diagnosis. This newer definition of PTSD has not yet been used widely in the scientific literature.

By any definition, PTSD has profound public health implications. From a clinical perspective, the condition is associated with many adverse outcomes including an increased risk of developing a wide range of health problems such as cardiovascular disease and dementia, premature mortality, alcohol and substance dependency, low health-related quality of life, and an inability to secure and maintain employment [10-17]. In addition, multiple studies have shown the association between PTSD and suicide ideation, attempts or completions [11,18]. Psychiatric comorbid conditions are present in >50% of patients with PTSD, the most common being depressive disorders, substance abuse, panic disorder, agoraphobia, generalized anxiety disorder, social phobia, bipolar disorder, and chronic pain [10]. Significantly, chronic pain and PTSD have been found to co-occur in 34-50% of veterans and in up to 24% of civilians, with the maladaptive chronification of fear avoidance emerging as a common factor for the progression, perpetuation and recalcitrance of both conditions [19-21]. From an economic perspective, the burden of PTSD is substantial with estimates ranging from $6,000 to $30,000 per patient [22,23]. According to the Congressional Budget Office, the costs of medical care associated with PTSD for veterans alone exceeded $2 billion in 2010 [24], a figure that is dwarfed in comparison to the $590 billion in cumulative disability costs, for service members and veterans following the Iraq-war, many of whom had PTSD [25].

Effective treatment options for PTSD remain challenging. Existing therapies for PTSD include pharmacotherapy, psychotherapy, or a combination of the two treatments [26]. Unlike conditions like Major Depressive Disorder, there is no universally agreed upon definition of what constitutes an adequate response for PTSD treatment and the DSM includes no definition of remission. Individual studies have required a particular decrease in score values on PTSD scales (i.e., a reduction of PTSD symptoms by a certain percentage), for example 30%, in order to consider a treatment response to be clinically significant [27]. Some studies consider anyone who falls outside the initial diagnostic pattern to be considered in remission, whereas others also require all symptom severity to have dropped below a particular threshold for a prolonged period of time [28]. These varying definitions may explain why there have been disagreements by reviewing bodies concerning what constitutes evidence-based treatment for PTSD. For example, the Institute of Medicine (IOM) took the position that there is insufficient evidence for any pharmacological therapy to be considered highly effective in the treatment of PTSD [26]. Even by looser standards, many treatments tried for PTSD have failed to produce detectable effect [28], and the overall success rate of PTSD treatment modalities is estimated to be <30% [26].

Substantial and consistent barriers contributing to the low impact of current therapeutic modalities for PTSD include the length of time required for treatment (typically weeks to months), stigma associated with the condition, transient mobility (e.g., deployments), high rates of co-morbidities that result in poor patient compliance, side effects, patient preferences, and perverse incentives [8,10,26]. Even when barriers to care are removed, approximately one-third of cases do not experience remission in PTSD symptoms despite many months to years of treatment with existing modalities [10]. These cases are often referred to as refractory PTSD, treatment-refractory PTSD or treatment-resistant PTSD, although such categorization is not precise due to definitional variability [9,10,29].

SGB: Non-psychiatric indications and hypothesized mechanism(s) of action in ptsd

SGB is a well-established pain management procedure in wide use since 1920 with a high safety profile [30] and an extremely low complication rate of 1.7 per 1,000 procedures [31]. The procedure is commonly used to treat pain associated with many conditions including chronic regional pain syndrome (CRPS) types I and II, vascular insufficiency/occlusive vascular disorders of the upper extremities, postherpetic neuralgia, phantom limb pain or amputation stump pain, Quinine poisoning, sudden hearing loss and tinnitus, hyperhidrosis of the upper extremity, cardiac arrhythmias, ischemic cardiac pain, Bell's palsy, trigeminal neuralgia, migraine headaches, neuropathic pain, Meniere's syndrome and hot flashes [32-40].

SGB involves injecting a local anesthetic (e.g., ropivacaine or bupivacaine) into the sympathetic ganglion which is formed as a fusion of the inferior cervical and first thoracic sympathetic ganglion at the C6 or C7 vertebral level. A detailed description of SGB administered for PTSD has been described elsewhere [4].

Empirical data specific to the pathophysiologic role of the sympathetic nervous system that could explain SGB's potential mechanism of action in PTSD are lacking. However, several possibilities have been purported by investigators within the broader framework of the neural network connecting several cortical regions that regulate the formation of memory, cognition and behaviors. The framework highlights interactions that involve multiple neurochemicals such as corticotropin releasing hormone, cortisol, the locus coeruleus-norepinephrine system, neuropeptide Y, galanin, dopamine, serotonin, testosterone, estrogen and dehydroepiandrosterone (DHEA) [41,42]. A complex network of noradrenergic terminals project from the locus coeruleus and cell groups in the medulla and pons to innervate the entire neuraxis from the olfactory bulb to spinal cord, visceral organs and integument. This widespread organization permits the noradrenergic system, by means of both central connections and peripheral sympathetic nervous system to influence the nervous system under conditions of elevated levels of Norepinephrine (NE) [43].

Based on the complexity of neural networks, some investigators have proposed that SGB influences PTSD via connections that exist between the stellate ganglia and insular cortex and other intracerebral structures [44,45]. Others have suggested that SGB affects regional cerebral blood flow and its through this pathway that the procedure impacts PTSD symptom severity, a theory which is supported by numerous neuroimaging studies [46,47]. Another plausible hypothesis is that SGB may be altering melatonin rhythm and sleep [48]. Yet some researchers believe that SGB results in an overall decrease in sympathetic tone as a mode of improving PTSD symptoms [49]. Since SGB is known to result in decreased levels of circulating nor-adrenalin, researchers have hypothesized that decreased peripheral noradrenaline signals a reduction of central noradrenaline levels due to a shared nucleus controlling both systems. SGB can also reduce the expression of peptides, such as Nerve Growth Factor (NFG), that play a role in maintaining the perpetual hyperarousal state. This reduction of NGF by SGB has been thought to reduce the necessary peptide for maintenance

of PTSD, thereby reverting intracerebral sympathetic nerves to a pre-trauma state [50].

Evaluating the preliminary evidence-base for the use of SGB as an adjunctive treatment option for PTSD

Although SGB's precise mechanism of action in PTSD remains elusive, a growing number of case reports and case series have been published on its use for treatment-refractory cases of PTSD. However, to the best of our knowledge, there has been no collective review and synthesis of the literature on this topic. Thus, the purpose of this review was to examine all published cases on the use of SGB treatment for treatment-refractory PTSD in order to comprehensively evaluate its impact on PTSD symptom improvement. Furthermore, implications for research, practice and policy are presented in order to facilitate next steps in building the evidence-base for SGB as a potential adjunctive treatment option for PTSD.

Methods

Data sources and search strategy

EMBASE, PubMed, PsychINFO and Cochrane databases were reviewed using an iterative keyword search strategy by two independent researchers (MSK and JCS) who applied the following initial inclusion criteria: (a) SGB or stellate ganglion block, (b) PTSD, post-traumatic stress disorder or post-traumatic stress, and (c) English language. No date delimiter was applied. As depicted in Figure 1, the search yielded 12 articles, of which seven were subsequently eliminated due to the following exclusion criteria: (a) lack of case data, (b) duplicate publications on the same case, or (c) invalid PTSD outcome measures (i.e., no use of a standardized instrument to measure PTSD symptoms or severity). Twenty-four cases [51-55] from the five remaining articles were evaluated further. No published randomized or controlled trials of SGB for PTSD were identified in the literature.

Data extraction, synthesis and analysis

Two of the authors (MN and MSK) independently extracted the following information from eligible case reports: (1) patient's age, sex, military service and employment status; (2) clinical characteristics including PTSD diagnosis and severity, co-morbid conditions, past and concurrent PTSD treatment and duration; (3) SGB treatment history including number of injections, type and concentration of local anesthetic used, and follow-up time; and (4) PTSD symptoms and severity as measured by the Post-Traumatic Stress Disorder Checklist - Military Version (PCL-M) [56] or Clinician-Administered Post-Traumatic Stress Disorder Scale (CAPS) [57]. Assessments showed complete inter-rater reliability (κ=1.0).

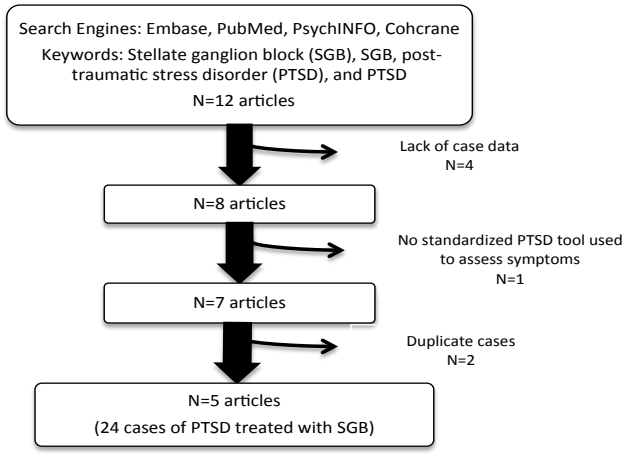

Figure 1: Flow Chart of Search Strategy and Article Selection Process.

Improvements in PTSD were calculated as the difference in either PCL-M or CAPS scores from pre-SGB 1 to post-SGB after each treatment episode. Unlike the CAPS where baseline PTSD scores are set as 0, baseline PTSD scores for PCL-M start at 17. Therefore, calculations of percent change in PTSD for outcomes measured by the PCL-M were adjusted by subtracting 17 from all pre- and post-SGB scores [56]. For cases with more than one SGB treatment, it was not always possible to compute changes in PTSD after every SGB treatment due to missing data. Under such a scenario, overall improvement in PTSD was computed for each case using pre-SGB 1 and post-SGB 2 or 3 PTSD scores. For the purposes of this review, a change of ≥30% in PTSD scores between pre- and post-SGB were considered clinically meaningful, irrespective of whether assessments were made using the CAPS or the PCL-M [57,58]. This approach was applied to permit a more uniform and conservative interpretation of results, despite a change of 20 points in PTSD scores being acceptable as clinically meaningful when using the PCL [58].

Patient follow-up time was computed from SGB 1 to the last point of contact with the patient when PCL-M or CAPS assessments were collected. SPSS was used to compute effect size by Cohen's *d* as recommended for a one-group pre-post study design [59] and to compare mean differences in PTSD scores pre- and post-SGB treatment. $P<0.05$ denoted statistical significance.

Results

Patient demographic and clinical characteristics across published case reports

Among the 24 cases, the majority were male (n=21, 88%) and active duty service members (n=14, 58%) or veterans (n=8, 33%) with combat-related PTSD [Table 1]. The patients ranged in age from 29 to 66 years old, averaging 40.5 years (±10.0 SD) across cases. Prior to SGB treatment, patients had received >1 year of psychotherapy, pharmacotherapy, or both without symptom relief. Comorbidity was reported in 87.5% of all published cases. Depression was the most commonly reported comorbid condition (50%), followed by chronic pain (33%), insomnia (29%), and alcohol abuse (21%).

Comparative summary of SGB treatments and overall improvements in PTSD across published case reports

Seventeen patients (71%) received one SGB, however seven (29%) received multiple SGBs. SGB administration was consistent across nearly all patients, with injections of 7cc of 0.5% ropivacaine or bupivacaine. Clinically meaningful improvements (i.e., ≥30% decline in PTSD symptoms) were observed in 75% (n=18) of patients after SGB, with statistically significant differences between mean PTSD scores pre- (69.5 ± 26.6) and post-SGB (34.2 ± 32.5) across all cases ($p<0.001$). The magnitude of the effect size was large (d=1.2). On average, PTSD improved by 50.4% (± 30.9 SD; range: 6.3-98.4) for patients treated with one SGB and 69.0% (± 28.0 SD; range: 9.2-93.5) for patients treated with multiple SGBs [Table 2 and Figure 2]. Patient follow-up time ranged from one to 264 days, with 54% observed for ≦7 days. The average time interval over which improvement was measured was 39.0 days (± 62.9) across all cases, 20.2 days (± 33.4) for cases with one SGB, and 84.57

Table 1: Characteristics ofPatientsTreated with Stellate Ganglion Block for Post-Traumatic Stress Disorder.

Article	N	Case Type	Sex	Age	PTSD[1] Treatment History	Co-Morbid Conditions
Mulvaney et al., 2010[51]	2	Veteran	Male	46	>1 year of psychotherapy[2] Anti-depressants Anti-psychotics Anxiolytics Hypnotics/Sedatives	Alcohol abuse Insomnia
		Active Duty	Male	36	>1 year psychotherapy[2] Anti-depressants Hypnotics/Sedatives	Insomnia
Hickey et al., 2012[52]	9	Active Duty	Male	46	>2 years of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants Anti-psychotic Hypnotics/Sedatives Anti-seizure Anti-hypertensive Anxiolytics	Chronic pain Depression
		Active Duty	Male	42	>1 year of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants Anxiolytics Hypnotics/Sedatives	Chronic pain Depression Obstructive sleep apnea
		Active Duty	Male	30	>1 year of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants	Chronic gastrointestinal pain Alcohol abuse Depression Chronic pain Traumatic brain injury
		Active Duty	Male	34	>1 year of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants Anxiolytics Anti-hypertensive Hypnotics/Sedatives	Depression Obsessive compulsive disorder Bulimia Panic disorder Chronic pain
		Active Duty	Female	46	>2 years of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants	Brain tumor Chronic pain
		Active Duty	Male	34	>2 years of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants	Depression Chronic pain
		Active Duty	Male	44	>2 years of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants	Depression Chronic pain
		Active Duty	Male	41	>1 year of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants	Chronic pain
		Active Duty	Female	44	>1 year of cognitive processing therapy and/or prolonged exposure therapy[3] Anti-depressants	Depression Chronic pain
Lipov et al., 2012[53]	8	Veteran	Male	55	Multiple years of psychotherapy[2] Meditation Anti-depressants Anti-hypertensive	Depression Insomnia
		Veteran	Male	31	Multiple years of psychotherapy[2] Medications unknown	
		Civilian	Male	29	Multiple years of psychotherapy[2]	
		Veteran	Male	33	EMDR[4] Medications unknown Multiple years of psychotherapy[2] Medications unknown	
		Veteran	Male	32	Multiple years of psychotherapy[2] Anti-depressants Anti-seizure Anxiolytics Hypnotics/Sedatives	Hypertension Arthritis Insomnia
		Veteran	Male	63	Multiple years of psychotherapy[2] Anti-depressants	Arthritis Depression Diabetes

days (± 93.7) for cases with multiple SGBs. No complications associated with SGB were reported in the cases compiled in this review.

Discussion

In this review of all published cases of SGB treatment for PTSD, although the results were not universally observed, 75% of cases showed clinically meaningful improvements in PTSD symptoms within a very short time after the procedure. Compared to an effect size of 0.41 to 1.63 for most evidence-based treatments of PTSD [60], the overall effect size for PTSD cases treated with SGB was 1.2, suggesting SGB had a relatively large impact on symptom reduction. However, it should be noted that it is not uncommon for newer treatments to report more dramatic improvements in PTSD. For example, a recent trial of the emotional freedom technique, a therapy often greeted with skepticism given its theoretical basis in mystic energy fields, reported 90% of patients who received the treatment experienced remission of their

Table 1: Characteristics of PatientsTreated with Stellate Ganglion Block for Post-Traumatic Stress Disorder(continued).

Article	N	Case Type	Sex	Age	PTSD[1] Treatment History	Co-Morbid Conditions
		Civilian	Male	66	Multiple years of psychotherapy[2] Anti-depressants Anxiolytics Hypnotics/Sedatives Anti-seizure	Depression Insomnia
		Veteran	Female	36	Multiple years of psychotherapy[2] Acupuncture Anti-depressants Anxiolytics Anti-seizure Anti-migraine	Depression
Lipov et al., 2013[54]	1	Veteran	Male	41	>1 year of psychotherapy[2] Anti-depressants	Alcohol abuse Depression Insomnia
Alino et al., 2013[55]	4	Active Duty	Male	34	Inpatient psychotherapy[2] Anti-depressants Anxiolytics	
		Active Duty	Male	35	>1 year of inpatient/outpatient psychotherapy[2] Anti-depressants Anti-psychotics	Alcohol abuse
		Active Duty	Male	46	Cognitive behavior therapy and prolonged exposure therapy Anti-depressants Anxiolytics Hypnotics/Sedatives	Insomnia
		Active Duty	Male	29	Inpatient/outpatient psychotherapy[2] Hypnotics/Sedatives	Alcohol abuse Insomnia

PTSD symptoms, often within three therapy sessions [61]. What differs in reports of SGB for PTSD, however, is that the cases were known to be refractory and improvements were often reported within just a few days of SGB treatment, both of which make the likelihood of a placebo response or coincidental improvement less probable.

A notable trend across cases treated with SGB for PTSD was that of more remarkable improvements among patients evaluated using the PCL-M as compared to the CAPS. The reasons for this observation are unclear. Some studies have demonstrated lower outcomes correlations and an effect size advantage in symptom change on the CAPS relative to the PCL [62]. By contrast, other studies have found an effect size advantage on the PCL compared to the CAPS [63]. More recent research has revealed that PTSD changes on the CAPS are typically 66% to 75% of the changes on the PCL [64]. Interpretively, researchers have attributed differences between outcomes assessed by the CAPS versus the PCL to discrepancies in ratings of symptom clusters associated with hyperarousal [65] and dysphoria [66,67]. An important methodological note in this regard is that many of the cases evaluated in this review reported improvements in PTSD within days of receiving SGB. Since the PCL-M asks about symptoms over the last month, post-SGB treatment assessments would theoretically have captured the state of the patient both before and after the procedure. Thus, the expectation would be to observe minimal symptom improvement after SGB. However, the opposite was observed in both the magnitude and rapidity of reported improvements after SGB. Future studies of SGB for PTSD could shed further light on this matter by measuring PTSD outcomes using both the PCL-M and the CAPS.

Another trend of interest was that cases who received multiple SGBs experienced greater PTSD symptom relief than cases who received a single SGB. Unfortunately, the small sample size and the lack of clarity regarding SGB's mechanism of action make it difficult to accurately interpret this observation. For example, it is unclear whether there is a dose response effect at play or a synergistic response of some sort with repeat SGBs. Additional research studies are clearly necessary.

Several methodological limitations merit mention. First, no published randomized or controlled trials of SGB treatment for PTSD were identified in the literature. Second, for the majority of the cases in this review, the follow-up periods were too short to ascertain the overall sustainability potential of SGB's effects. Third, a potential placebo effect could be at play, most particularly because SGB is an injection therapy [68]. In the field of analgesia and pain medicine, placebo-induced responses pose prominent clinical and methodological concerns [69,71]. Patient and investigator expectations are known to be basic mechanisms of the placebo effect, with investigator expectations reflecting larger effect size in observer ratings and patient expectations reflecting larger self-report effects [72]. Another methodological limitation is the potential for confounding related to an acupuncture effect [73-75]. Previous studies have shown the presence of an acupuncture effect when therapeutic needles are applied to acupuncture points [75,76] including the C6 location where SGB treatment was administered for the PTSD cases [77]. Future studies should consider designing placebo-controlled trials of SGB for PTSD that utilize sham injections of local anesthetic superficial to the stellate ganglion when administering SGB such that the plausible confounding effects of acupuncture can be blocked [77].

Implications for research, practice and policy

The state of the preliminary evidence related to SGB treatment for PTSD, as summarized in this review, points to unanswered research questions that are needed to guide clinical practice. Beyond the fundamental question of SGB's true efficacy for PTSD, it is unclear how and for whom SGB should be tested as a treatment modality for PTSD. Whether SGB treatment can, or should be, combined with other PTSD therapies also remains elusive. It is unknown what patient characteristics lend themselves to being well suited for SGB. For example, may SGB be more useful in patients with significant hyperarousal symptoms? Are patients with common co-morbid conditions to PTSD, such as chronic pain, more likely to experience greater PTSD improvement than others with SGB? Information about the frequency and optimal dosing

Article	Case No.	No. of SGBs	Anesthetic Type & Dosage	Instrument[1]	PTSD Severity Pre-SGB (1,2,3)[2]	PTSD Severity Post-SGB (1,2,3)[2]	Overall Improvement in PTSD (%)[2,4]	Follow-up Time (1,2,3)[2]
Mulvaney et al., [51]	1	2	Ropivacaine (7cc, 0.5% soln)	PCL-M	76, 67	26, 34	71.2*	1 day, 264 days
	2	1	Ropivacaine (7cc, 0.5% soln)	PCL-M	54	24	81.1*	50 days
Hickey et al., [52]	3	1	Ropivacaine (7cc, 0.5% soln)	CAPS	126	118	6.3	7 days
	4	1	Ropivacaine (7cc, 0.5% soln)	CAPS	87	60	31.0*	7 days
	5	1	Ropivacaine (7cc, 0.5% soln)	CAPS	83	56	32.5*	6 days
	6	2	Ropivacaine (7cc, 0.5% soln)	CAPS	106, 79	26, 18	83.0*	9 days, 103 days
	7	1	Ropivacaine (7cc, 0.5% soln)	CAPS	117	79	32.5*	7 days
	8	1	Ropivacaine (7cc, 0.5% soln)	CAPS	64	52	18.8	7 days
	9	1	Ropivacaine (7cc, 0.5% soln)	CAPS	104	43	58.7*	7 days
	10	2	Ropivacaine (7cc, 0.5% soln)	CAPS[3]	104, -	103, 96	7.7	7 days, 28 days
	11	1	Ropivacaine (7cc, 0.5% soln)	CAPS	101	79	21.8	7 days
Lipov et al., [53]	12	1	Bupivicaine (7cc, 0.5% soln)	PCL-M	58	51	17.1	16 days
	13	1	Bupivicaine (7cc, 0.5% soln)	PCL-M	68	35	64.7*	3 days
	14	1	Bupivicaine (7cc, 0.5% soln)	PCL-M	67	63	8.0	1 day
	15	1	Bupivicaine (7cc, 0.5% soln)	PCL-M	68	31	72.5*	48 days
	16	1	Bupivicaine (7cc, 0.5% soln)	PCL-M	55	29	68.4*	4 days
	17	1	Bupivicaine (7cc, 0.5% soln)	PCL-M	77	23	90.0*	6 days
	18	2	Bupivicaine (7cc, 0.5% soln)	PCL-M	70, 70	17, 29	77.4*	1 day, 2 days
	19	3	Bupivicaine (7cc, 0.5% soln)	PCL-M[3]	79, -, 30	54, -, 21	93.5*	1 day, 17 days, 59 days
Lipov *et al.*, [54]	20	2	Bupivicaine (6.5cc, 0.5% soln)	PCL-M	71, 66	40, 30	75.9*	43 days, 135 days
Alino *et al.*, [55]	21	2	Ropivacaine (7cc, 0.5% soln)	PCL-M	64, 35	22, 29	74.5*	3 days, 136 days
	22	1	Ropivacaine (7cc, 0.5% soln)	PCL-M	80	18	98.4*	2 days
	23	1	Ropivacaine (7cc, 0.5% soln)	PCL-M	69	34	67.3*	30 days
	24	1	Ropivacaine (7cc, 0.5% soln)	PCL-M	76	24	88.1*	1 day

*Clinically meaningful improvement in PTSD-related symptoms with ≥ 30% decline in PCL or CAPS scores. For cases with 1 SGB treatment, overall improvement was calculated using the pre-SGB 1 and post-SGB 1 scores. For cases with 2 SGB treatments, overall improvement was calculated using the pre-SGB 1 and post-SGB 2 or 3 scores.

[1]PCL=Post-Traumatic Stress Disorder Checklist - Military Version, CAPS=Clinician-Administered Post-Traumatic Stress Disorder Scale.

[2]Post-traumatic stress disorder symptom severity pre- or post-SGB treatments 1, 2 or 3 as applicable; Follow-up time was computed from SGB 1 to the last point of contact with patient when PCL or CAPS assessments were collected.

[3]Some data were missing. 4Overall Improvements calculated as the difference in either PCL-M or CAPS scores from pre-SGB 1 to post-SGB 2 or 3. PTSD improvement as measured by the PCL-M were adjusted by subtracting 17 from all pre- and post-SGB scores given the baseline value of PCL-M starts at 17, not 0 as is the case for CAPS.

Table 2: Comparative Summary of Stellate Ganglion Block Treatments and Overall Improvement in Post-Traumatic Stress Disorder

necessary to achieve short-term PTSD relief versus sustained long-term PTSD relief is also missing. For example, is there a role for multiple or bilateral injections? Additionally, the potential for synergism and prolongation of effects of local anesthetic agents combined with standard pain adjuncts, such as clonidine, should be explored [78]. Also, how long would we expect effects from SGB to last, based on its use for other disorders? Other relevant research questions include testing for a potential placebo effect that may be at play. Laboratory-based studies that could explain SGB's mechanism(s) of action in PTSD are also needed.

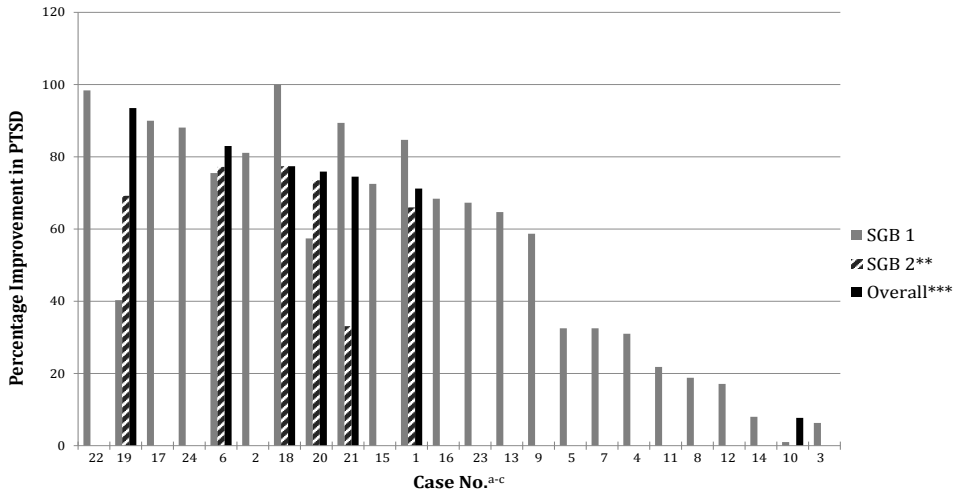

Figure 2: Percent Improvement in Post-Traumatic Stress Disorder (PTSD) After Treatment with Stellate Ganglion Block (SGB)

[a]Cases 1, 2, & 12-24 scores measured using the PTSD Checklist - Military Version (PCL-M), cases 3-11 scores measured using Clinician Administered PTSD Scale (CAPS). [b]For case 19, change measured using scores from pre-SGB 1 and post-SGB 3 due to limited data; [c]Overall=Improvements in PTSD calculated as the difference in either PCL-M or CAPS scores from pre-SGB 1 to post-SGB 2 or 3. PTSD improvement as measured by the PCL-M were adjusted by subtracting 17 from all pre- and post-SGB scores given the baseline value of PCL-M starts at 17, not 0 as is the case for CAPS.

While it is clear that before SGB can be considered for wide use in PTSD well-designed and more rigorous studies are needed, if shown to be an effective treatment option, the implications of SGB for enhancing psychiatric care are substantial. For example, SGB's rapid treatment cycle could address the high rates of attrition that often plague current evidence-based therapies for PTSD [79]. Another additional benefit of SGB could be that it offers a biologic approach to managing PTSD, thereby treating the condition on a more medical platform that could help lower the well recognized stigma of seeking treatment for the condition.

Initial policy-related implications of SGB for PTSD include considering incentives that would promote partnerships between interdisciplinary teams of specialists in anesthesiology, pain management and psychiatry, a necessary collaboration to optimize treatment delivery involving SGB. Another important consideration would be the need for additional cross-training of psychiatrists in pain management techniques, an issue which has recently been raised as an unmet need in psychiatry [80].

The treatment of PTSD continues to be a challenge for psychiatrists and other mental health practitioners. Pharmacotherapy and psychotherapy are only moderately helpful at best. More effective treatment options need to be sought. Given the substantial number of military and civilian populations that are adversely affected by PTSD, and the enormous economic burden of the condition, the growing evidence-base on SGB and PTSD as collectively presented in this review seem sufficiently compelling to motivate further action.

Acknowledgment

We would like to thank Natalie R. Hasbani, M.P.H., Jessica C. Smith, M.P.H., and Leighla H. Sharghi, B.S.N., M.P.A. from Advance Health Solutions, LLC for their administrative support, assistance with literature reviews, and preliminary presentations of the research at conference venues.

Disclaimer: The views expressed in this article are those of the authors and do not reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States government.

Previous Presentations: Earlier iterations of this manuscript, in part, were presented at the Annual Meetings of the American Public Health Association (APHA), November 2-6, 2013 in Boston, MA; the Association of Military Surgeons of the United States (AMSUS), November 3-8, 2013 in Seattle, WA; the American Society of Regional Anesthesia and Pain Medicine (ASRA), April 3-6, 2014 in Chicago, IL; the American Society of Interventional Pain Physicians (ASIPP), April 4-6, 2014 in New Orleans, LA; and the American Psychiatric Association (APA), May 3-7, 2014 in New York, NY.

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Citation: Navaie M, Keefe MS, Hickey AH, McLay RN, Ritchie EC, et al. (2014) Use of Stellate Ganglion Block for Refractory Post-Traumatic Stress Disorder: A Review of Published Cases. J Anesth Clin Res 5: 403. doi:10.4172/2155-6148.1000403