The Use of Stellate Ganglion Block in the Treatment of Panic/Anxiety Symptoms (Including Suicidal Ideation), with Combat-Related Posttraumatic Stress Disorder

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Tailgating Over the Valley, by MSG Martin J. Cervantez, courtesy of the Army Art Collection, US Army Center of Military History.

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Posttraumatic stress disorder (PTSD) is a chronic or pathological anxiety that is brought on by witnessing or experiencing severe trauma. In 1980, the American Psychiatric Association (APA) added PTSD to the third edition of its Diagnostic and Statistical Manual of Mental Disorders (DSM-III) [1]. However, multiple terms describing what is currently known as PTSD preceded its use. The first modern description of PTSD emerged during the US Civil War. Dr. Mendez DaCosta described war veterans as having irritable heart or "soldiers' heart" [2]. In his 1876 research paper, Dr. DaCosta described startle responses, hypervigilance, and heart arrhythmias.

The wisdom of this report is a biological description of the nervous system over activity. If PTSD can be recognized as biological trauma, it may respond to an approach that reverses or treats biological alteration of the nervous system. The division of the nervous system, which is likely to play a dominant role in the development and maintenance of PTSD, is the sympathetic system. If biological causes of PTSD are accepted, the utilization of a blockade of the sympathetic system supplying the brain is conceivable as opposed to a "FREAKY-PTSD-TREATMENT" as portrayed via an article in the Wired Magazine [3].

The focus of this chapter is to summarize clinical evidence available for the effectiveness of cervical sympathetic ganglion injection called stellate ganglion block (SGB), as well as demonstrate possible clinical applications of its use. The patient who is the subject of the case report, failed conservative treatments for over 40 years and presented to the author for an urgent SGB. What led the patient to present, including his follow-up, offers a glimpse into the understanding of SGB as a vital treatment option for PTSD treatment.

13.1 Methods

13.1.1 Right-Sided C6 Cervical Sympathetic Chain Blockade

Once written consent was administered, a right-sided SGB was performed. An intravenous line was started with a 22G IV in the right hand. The patient was positioned comfortably in the supine position, prepped and draped in the sterile fashion. After radiographic confirmation of the right C6 vertebral body, the skin was anesthetized with 1 cc of 2% lidocaine. Using an anterior paratracheal approach, a 22-gauge Quincke needle was passed under fluoroscopic guidance until it contacted the anterior lateral aspect of the C6 vertebral body, and then it was pulled back 1 mm. Appropriate needle position was then confirmed by the injection of 2 cc of iohexol (180 mg/mL) radiopaque dye, and fluoroscopy was used to monitor its spread. After negative aspiration, 7 cc of 0.5% bupivicaine was injected slowly in order to produce a sympathetic ganglion block. We observed the patient for facial anhidrosis (inability to sweat normally) and Horner's syndrome (i.e., enophthalmos-posterior displacement of the eye), ptosis (drooping of the upper eyelid), and miosis (constriction of the pupil) that was noted within 10 min. Horner's syndrome is considered demonstrative of a successful sympathetic block of the cervical sympathetic chain.

13.1.2 Psychometric Testing

The PTSD Checklist (PCL) is a 17-item psychometric test commonly used to screen for PTSD. It was developed based on PTSD criteria from the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The PCL's initial validation [4] found that it was an effective brief screen for identifying PTSD, although the sample population did not include patients who experienced combat-related PTSD.

The PCL has since been validated for screening troops returning from combat to identify those with PTSD [5] as well as assessing symptom improvement as a result of treatment [6]. Different cutoff scores have been recommended for identifying PTSD, with ranges spanning from 30 to 50 PCL scores. Forbes et al. [6] concluded that the optimal cutoff score for identifying combat-related PTSD is a score of 50, with maximal score being 85.

13.2 Case Report 1

The patient is a 67-year-old male veteran of the Vietnam War. His military service began at age 19, where he served as a Medic for 19 months during the Vietnam War. Although the patient reported not seeing direct war combat, he witnessed horrific injuries and incidents of fellow military personnel. He experienced a very difficult transition as he attempted to settle back into a civilian lifestyle. His family and friends informed him how he was no longer the same person he was before he left for war. He also knew that what he experienced had fundamentally changed him.

Following his return home, the patient reported struggling with depression, insomnia, nightmares, flashbacks, alcoholism, and suicidal ideation for years. He dealt with fatigue, frequent headaches, and problems with concentrating, thinking, and recalling. His VA doctors prescribed him various medications to help alleviate his depression and sleeping problems. However, he was fired from 13 different jobs within his first year back home.

For 40 years, the patient lived with these symptoms. All the while, he was receiving individual counseling and was treated with many different medications. The veteran grew frustrated with the lack of relief these treatment modalities were providing. When the patient presented himself for treatment, he was noted to be severely anxious and agoraphobic. Although he denied being suicidal during the first evaluation, he informed the author after the treatment, "If I could not have the injection done I would have killed myself that night."

When the patient received his first SGB treatment, that night was the first time he slept without having nightmares. The day after the procedure, his PCL score dropped from 74 to 54. A few days after his first injection, he had noticed nightmares returning, and another SGB was performed 16 days later. Two weeks after the second SGB, the patient reported sleep improvements and he had been able to decrease his daily dose of Trazadone from 600 mg down to 350 mg. He was feeling calmer during the day and was finding himself better able to socialize. His flashbacks decreased and he found himself much less tense.

Two weeks after SGB #2, he was able to sleep through the night, increasingly more social than he had been prior to the injections, significantly less tense, less prone to flashbacks, and he was no longer experiencing suicidal thoughts. He also informed our staff members that he had been able to discontinue all PTSD-related medications. The patient no longer felt the need to attend therapy secessions. He stated, "SGB literally saved my life. I had nightmares which pretty much led to destruction of my house every night since 1968. I can honestly say that after the first night of receiving SGB #1 that was the first time I slept all the way through the night without a nightmare. I felt like I was at the end of my rope. I was unfortunately ready to commit suicide. The procedure is so simple and the results are so great."

The patient still has memories of the war, but he states the memories no longer cause him anxiety. He no longer takes any medications for PTSD nor does he require any counseling. During a recent follow-up, nearly 4 years following the original SGB, the patient's PCL was 29.

13.3 Case Report 2

13.3.1 Chronologic PCL-C Report for the Patient

The focus of this chapter is strictly related to military-associated PTSD; however, PTSD can occur in the civilian population as well. A brief example of SGB application for nonmilitary PTSD is also presented below.

The patient is a 23-year-old Caucasian female, nonmilitary, and first seen 1-year posttrauma. A formal diagnosis of PTSD was made by her psychiatrist. The patient was a rape victim. She was subsequently placed on multiple selective serotonin reuptake inhibitor (SSRI), which she felt provided about 20% relief reduction in symptoms. The patient also attempted cognitive-behavioral therapy (CBT) but felt that it caused an increase in anxiety. The patient had a SGB at the C6 level and had a repeat block 4 months later. The procedure was unremarkable and no side effects were reported. The patient self-reported a reduction of symptoms by 90%, soon after SGB. She also self-reported an 80% reduction in anxiety 1 year after the last SGB and has returned to therapy; which is noted to be considerably more effective following SGB.

13.4 Discussion

13.4.1 PTSD Overview

The incidence of military-related PTSD is on the rise, partly due to increased awareness and better detection. The biggest driver, however, is the continued large-scale military mobilization in response to the sociopolitical violence of the past decade. The prevalence and profound impact on quality of life urgently demands effective PTSD treatments [7].

Although PTSD is the most commonly diagnosed service-related mental disorder among the US military personnel returning from Iraq and Afghanistan, an expert panel convened by the Institute of Medicine found little evidence for the efficacy of most currently employed PTSD treatment modalities [8]. As noted by Dr. Hoge in a JAMA 2011 editorial, current therapeutics has limited effect. He stated that veterans remain reluctant to seek care, with half of those in need not utilizing mental health services. Among veterans who begin PTSD treatment with psychotherapy or medication, there is a high percentage dropout which is commonly 20–40 % in randomized clinical trials (RCTs). "With only 50 % of veterans seeking care and a 40 % recovery rate, current strategies will effectively reach no more than 20 % of all veterans needing PTSD treatment" [9].

13.4.2 Conventional PTSD Treatment: Pharmaceuticals Plus Psychotherapy

13.4.2.1 Pharmaceuticals

Primarily used to address mood disorders, the SSRIs increase the amount of serotonin circulating in the synapse, but they have been shown to be helpful in mediating PTSD symptoms. The side effects, however, include sexual dysfunction [10], somnolence [11] and an increased risk for suicide [11]. Four SSRIs have undergone clinical trials for efficacy in treating PTSD; these include: citalopram (Celexa), fluoxetine (Prozac), paroxetine (Paxil), and sertraline (Zoloft). Of these SSRIs, only sertraline and paroxetine currently have a FDA indication for PTSD. The dual nighttime symptoms of PTSD—that is, nightmares and sleep disruption—are often unresponsive to medication [12].

While SSRIs are marginally effective for these symptoms among civilian populations [13], combat PTSD has been relatively impervious to pharmacological treatment [14]. Other drugs are beginning to take position on the front lines in the battle against PTSD, mainly atypical antipsychotics such as Seroquel.

Following the blood-thinner Plavix, Seroquel (quetiapine fumarate) is the VA's second most largest prescription drug expenditure each year since 2007; where in fiscal year 2010, the agency spent $ 125.4 million on Seroquel, up from $ 14.4 million in 2001 [15]. Similarly, the Department of Defense's spending on Seroquel has increased nearly 700 % since 2001, to $ 8.6 million in 2004, according to purchase records [16].

While the FDA approved Seroquel only for schizophrenia, it is often used off-label for PTSD. However, the potential side effects include diabetes, weight gain, and uncontrollable muscle spasms. In the past few years, Seroquel has been the subject of more than 25,000 product liability lawsuits, including one brought by federal prosecutors. In April 2010, AstraZeneca paid $ 520 million to settle the federal government lawsuit, although thousands of other civil lawsuits are still pending [17]. Researchers at Vanderbilt University published a study in the New England Journal of Medicine suggesting a new risk: sudden heart failure. The investigators found three cardiac deaths per year for every 1000 patients taking antipsychotic drugs like Seroquel [18].

Similar risks were reported by Dr. Kuehn who observed that, "taking atypical antipsychotics doubles the risk of sudden cardiac death" [19]. Furthermore, clinical consideration for the use of atypical antipsychotics to treat patients presenting with PTSD may be problematic, since this class of medications can increase the risk of suicidal attempts, as demonstrated by Dr. Hering. His findings suggest that a noncompliant patient using atypical antipsychotics has a 3.6 times increased risk of suicide attempts as compared to compliant patient using atypical antipsychotic [20].

13.4.2.2 Cognitive-Behavioral Therapy (CBT) and Other Psychotherapy

CBT involves a therapist who helps the client change how he or she thinks about the traumatic event and the client's response to that event. Exposure therapy is one component of CBT and is likely the most effective [21]. The main objective is to help the patient identify feelings associated with PTSD and then develop methods to cope with these feelings. The CBT component of treatment helps the patient change how he or she thinks about the traumatic event and the response to that event. Using exposure therapy, the client is reintroduced to portions of the traumatic event in a controlled, safe environment. The typical CBT course is of 3 months with one to two visits per week. Alternatives to CBT, with potentially similar efficacy, include eye movement desensitization and reprocessing (EMDR).

13.4.2.3 Eye Movement Desensitization and Reprocessing (EMDR)

Francine Shapiro first developed the EMDR therapy upon noticing that certain eye movements reduced the intensity of disturbing thought. She proceeded to conduct a scientific study, sampling trauma victims in 1988 and the research was later published in the Journal of Traumatic Stress in 1989 [22]. In a 2007 review of 33 randomized controlled trials of various psychological treatments for PTSD, EMDR was rated as an effective method, not significantly different in effect from Trauma-Focused CBT or stress management (SM) treatments [23]. EMDR did significantly better than other therapies, according to patient self-reports [23]. Dr. Salkovskis reported that the eye movement is irrelevant, and the effectiveness of EMDR was solely due to having properties similar to CBT, such as desensitization and exposure [24]. Most recent meta-analyses conducted in 2013, including the Cochrane review, have indicated that CBT and EMDR therapies are well-supported by research and superior to all other psychotherapies [25].

13.4.3 New, Cutting Edge Treatments for PTSD, Complementary Alternative Medicine (CAM)

13.4.3.1 Methylenedioxymethamphetamine (MDMA)

One of the newest PTSD treatments is the use of the drug MDMA, a street drug called ecstasy. In the early 1900s, this compound was developed in Germany as a parent compound to be used to synthesize other pharmaceuticals. The drug gained a small following among psychiatrists in the late 1970s and early 1980s. During

which time, some psychiatrists even called it "penicillin for the soul" because MDMA was perceived to enhance communication in patient sessions and reportedly allowed users to achieve insights about their problems. Medical reviews have noted that MDMA has some limited therapeutic benefits in certain mental health disorders; however, it is unsafe due to the persistent adverse cognitive and neural effects associated with its use [26,27]. More research is needed in order to determine if the benefits of using MDMA in PTSD treatment outweighs the patients risk of persistent adverse neuropsychological harm [26,27].

13.4.3.2 Yoga

Patients experiencing PTSD which is exacerbated by stress and who have low heart rate variability (HRV) have been shown to improve in response to yoga-based interventions [28]. Dr. Streeter proposed a theory explaining the benefits of practicing yoga. It is hypothesized that stress induces (1) imbalance of the autonomic nervous system (ANS) with decreased parasympathetic nervous system (PNS) and increased sympathetic nervous system (SNS) activity, (2) increased allostatic load (allostatic load being defined as "the wear and tear on the body" which grows over time when the individual is exposed to repeated or chronic stress). It is further hypothesized that yoga-based practices (1) correct underactivity of the PNS in part through stimulation of the vagus nerves, the main peripheral pathway of the PNS, and (2) reduce allostatic load. According to the proposed theory, the decreased PNS underlying stress-related disorders can be corrected by yoga practices resulting in amelioration symptoms [27]. Interestingly, this theory is consistent with the SGB intervention because its effects are presumed to act via modulation of the SNS.

13.4.3.3 Mindfulness-Based Stress Reduction (MBSR)

Mindfulness-based stress reduction (MBSR) has shown promise as an intervention for PTSD [29]. Dr. Kearney reported significant improvements in PTSD symptoms; depression and behavioral activation, where 47.7% ofveterans had clinically significant improvements in PTSD symptoms [29]. Dr. Hölzel reported that reductions in perceived stress correlated positively with decreases in right basolateral amygdala gray matter density. Stressed but otherwise healthy individuals ($N = 26$) participated in an 8-week mindfulness-based stress reduction intervention. Following the intervention, participants reported significantly reduced perceived stress. Reductions in perceived stress correlated positively with decreases in right basolateral amygdala gray matter density, as measured by functional MRI. The more the participants' stress levels decreased, the greater the decrease of gray matter density in the right amygdala [30].

Dr. Hölzel went on to say that evidence suggests that mindfulness practice is associated with neuroplastic changes in the anterior cingulate cortex, insula, temporoparietal junction, fronto-limbic network, and default mode network structures. The authors suggested that the mechanisms described work synergistically by establishing a process of enhanced self-regulation [30]. As with Yoga, the above theory is consistent with the effect of SGB intervention, since the effects of MBSR and SGB are theorized to be due to the effects modulated by the amygdala [31].

This summary provides an abridged overview of current interventions for treatments of PTSD. By no means does this review offer an exhaustive treatise on the aforementioned approaches discussed. Each treatment modality has its advocates as well as detractors. The remaining part of this chapter focuses on a relatively new treatment to the field of psychiatry, SGB.

Essentially, SGB is an injection of a local anesthetic in the cervical spine that modulates the SNS and has marked impact on PTSD symptoms that are apparent in 30 min following the procedure and may be long lasting. In evaluating SGB, as well as other methods, as a viable treatment option of PTSD, it is important to recall Dr. Hoge statement, "Interventions that will have the greatest potential for improving care on a population level are those focused on enhancing the reach of treatment (e.g., engagement, adherence, and acceptability [9])."

13.5 Treatment of PTSD by Modulation of the Sympathetic Nervous System

13.5.1 Overview Sympathetic Nervous System as Related to PTSD

The focus of this section is on the manipulation of the SNS, which the author believes is one of the new frontiers for treating PTSD. The SNS is part of the ANS. Its main role is to mobilize the body's resources under stress and to induce the fight-or-flight response. It is also constantly active at a basal level in order to maintain homeostasis.

In large part, the activation of the SNS is accomplished by the increase of catecholamines, mainly epinephrine and norepinephrine (NE). The role of NE in the brain is that of a neurotransmitter leading to arousal, selective attention, and vigilance which has been demonstrated in preclinical studies [32]. Specifically, elevated urinary NE has been identified among patients with PTSD [33]. Similarly, NE concentrations in cerebrospinal fluid (CSF) are significantly higher in subjects with PTSD than among healthy controls, and have been correlated with the severity of PTSD symptoms [34]. Such notable increase in noradrenergic activity among subjects with PTSD suggest that reducing CNS noradrenergic activity could be effective, especially for arousal symptoms such as nightmares and startle reactions [35].

13.5.2 Orally Active Noradrenergic Blocking Agents in Treatment of PTSD

Orally active noradrenergic blocking agents have been used to moderate an overactive SNS with previously reported psychiatric effects on PTSD, which include clonidine and prazosin.

13.5.2.1 Prazosin

A sympatholytic drug typically used to treat hypertension, prazosin, is in the class of alpha-adrenergic blockers which lower the blood pressure by blocking the effects of NE. In doing so, this process relaxes the vessel walls. Interestingly, significant psychiatric effect was noted in double-blind placebo-controlled trials of prazosin, which demonstrated a dramatic 70–80 % reduction in combat-related PTSD nightmares [12]. Although the evidence was less compelling, prazosin also reduced PTSD-related anxiety during the day, comparable to that observed with the SSRIs [12].

13.5.2.2 Clonidine

Clonidine is an alpha-2 adrenergic receptor agonist that suppresses the SNS outflow throughout the brain. Because clonidine activates the post-synaptic alpha-2 receptors in the central nervous system (CNS), it inhibits sympathetic activity [36]. Contrary to the effects of prazosin, clonidine does not block the effects of NE directly, but reduces the sympathetic activation of CNS. Clonidine has been shown to reduce hyperarousal symptoms of PTSD [37].

In addition to these oral agents, it is now possible to directly affect the SNS transmission to the brain. The neuroanatomy presented below provides a neuroanatomical explanation for brain sympathetic system manipulation outside the cerebrum. The stellate ganglion and upper thoracic ganglion (T-2) is the upper sympathetic ganglion that innervates the upper chest, the head, and the brain. Many of the efferent sympathetic fibers from the thoracic ganglia (T-2) pass through the stellate ganglion [38]. A connection from the stellate ganglion and the brain has been shown by the use of the pseudorabies virus injections [39]. Pseudorabies virus allows identification of neural pathway connections two to three synaptic connections from the injection site.

13.5.3 Minimally Invasive Modulation of SNS in Treatment of PTSD

13.5.3.1 Endoscopic Sympathetic Block (ESB) at the Second Thoracic Vertebra (T2)

Successful treatment of anxiety by the use of clipping the sympathetic ganglia via an endoscopic sympathetic block (ESB) at the second thoracic vertebra (T2) was first reported in 1998 [40]. In a follow-up publication to his 1998 paper, Dr. Telaranta noted the similarity in features between social phobias and PTSD—especially those caused by an overactive SNS, such as heart racing, hypervigilance, and avoidance of painful psychic situations [41].

13.5.3.2 Stellate Ganglion Block (SGB), a Cervical Sympathetic Injection

The SGB is an anesthetic injection in a group of nerves in the neck that are called the stellate ganglion. This procedure has been used to treat chronic pain since 1925, and recent studies have demonstrated great promise as a successful intervention for PTSD.

This author reported the first successful treatment of PTSD through the use of SGB in 2008 [42]. The subject of that report was a civilian robbery victim who presented for SGB treatment due to severe anxiety related to PTSD, 2 months post being robbed at gunpoint. The patient experienced excellent response to SGB and reported significant resolution of hyper vigilance and anxiety.

13.5.3.3 Potential Complications of SGB

SGB carry a very small risk of infection. Using prophylactic antibiotics can reduce the slight risk of infection. Although rare, severe complications following SGB do include bleeding, seizures, pneumothorax, and spinal cord trauma. A study of the incidence of severe complications was last undertaken in 1992 by German researchers Wulf and Maier; they reported 1.7 complications per 1000 blockades based on surveys completed by patients receiving a combined total of 45,000 blocks. No fatalities or persistent complications were reported [43]. This survey was conducted prior to the use of fluoroscopic guidance where the SGBs were performed at the C7 level rather than C6. The current improvements in guidance technology and changing the needle location to C6 are likely to reduce the chance of complications.

13.5.3.4 SGB and the Treatment of PTSD, Current Evidence

SGB has been used to treat PTSD since 2008. Dr. Navaie summarized available literature published between 2008 and 2013 on the use of SGB to treat PTSD [44]. She indicates that patients 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. All patients received more than 1 year of psychotherapy and pharmacotherapy before SGB. Seventeen patients (71%) received one SGB, seven (29%) received multiple SGBs. Clinically, meaningful improvements were observed in 75% ($n = 18$) of patients after SGB, with significant differences in mean PTSD scores, pre and post treatment.

In clinical case reports reviewed above, two have specific merit.

Dr. Alino reported on a patient with a 2 year history of suicidal ideation had become free of suicidal thought two days after SGB [45]. Dr. Mulvaney reported on two patients with severe PTSD who were able to completely stop taking psychiatric medications after SGB [46]. Recently, further validation of SGB efficacy has been published.

Dr. Mulvaney, in a follow-up to a 2010 publication [46] observed that 166 service members with symptoms of PTSD that received SGB had clinically significant reductions in PCL scores. Specifically, 70% of those treated with SGB reported significant reductions in PCL scores and the effects were sustained 3–6 months post procedure [47].

Further validation of SGB efficacy in 2014 came from Dr. Alkire. He presented an abstract titled: “Prolonged Relief of Chronic Extreme PTSD and Depression Symptoms in Veterans Following a Stellate Ganglion Block.” In this report, Dr. Alkire selected the most extreme PTSD cases in the veteran population and observed that SGB was greatly effective in helping 75% (9/12) of the subjects [48].

13.5.3.5 Proposed Mechanisms for the Clinical Effect of SGB

The hypothesis for potential mechanism of action for SGB (or cervical sympathetic chain blockade) has been described in multiple peer-reviewed publications [31, 46–49]. The hypothesis rests on previously demonstrated evidence and was originally proposed by the author.

The first line of evidence in supporting the theory demonstrates a polysynaptic neurological connection from stellate ganglion to the part of the brain associated with PTSD, the amygdala [39] (Fig. 13.1). Specifically, Dr. Liberzon demonstrated increased activation of the amygdala in PTSD patients when compared to controls [50].

The second line of evidence relies on the nerve growth factor (NGF) increase observed as a physiological response to acute and chronic stress [51,52]. NGF increase is known to increase perivascular NE. This has been demonstrated by direct intracerebroventricular brain infusion of NGF into adult rats [53]. Stress-induced release of NE in amygdala and related structures has been shown to facilitate a number of anxiety-like behavioral responses that are mediated in these regions [54].

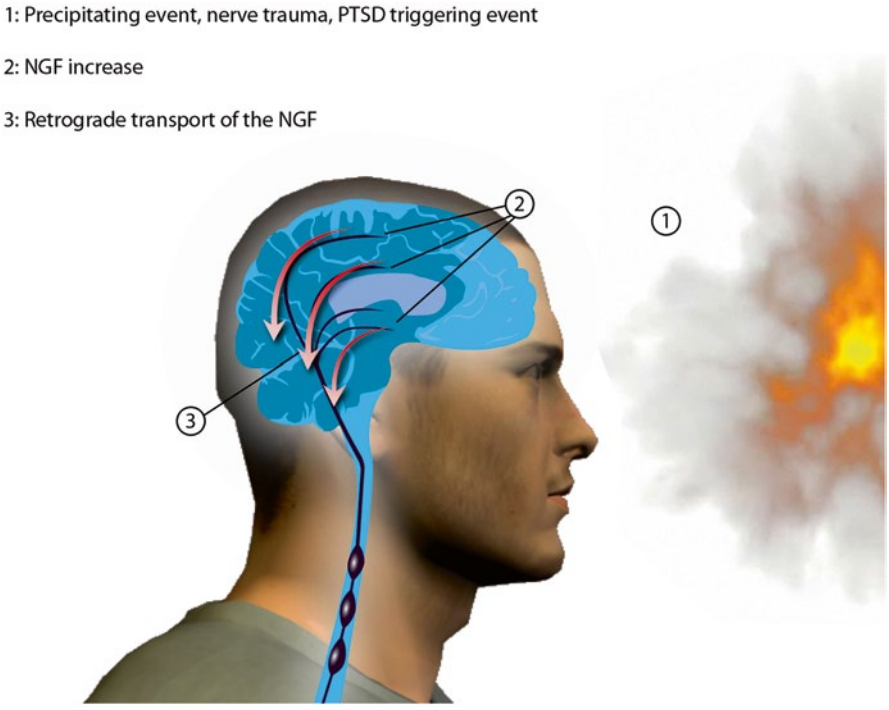

Fig. 13.1 The possible biologic mechanism exists for how sympathetic blockade may produce long-lasting systemic effects. There is a poly-synaptic neurological connection from stellate ganglion to the part of the brain associated with PTSD, the amygdala. (From Vlessides M, Anesthesia method suggests possible cure for post-trauma stress. Anesthesiology News 2012; 38:9, with permission)

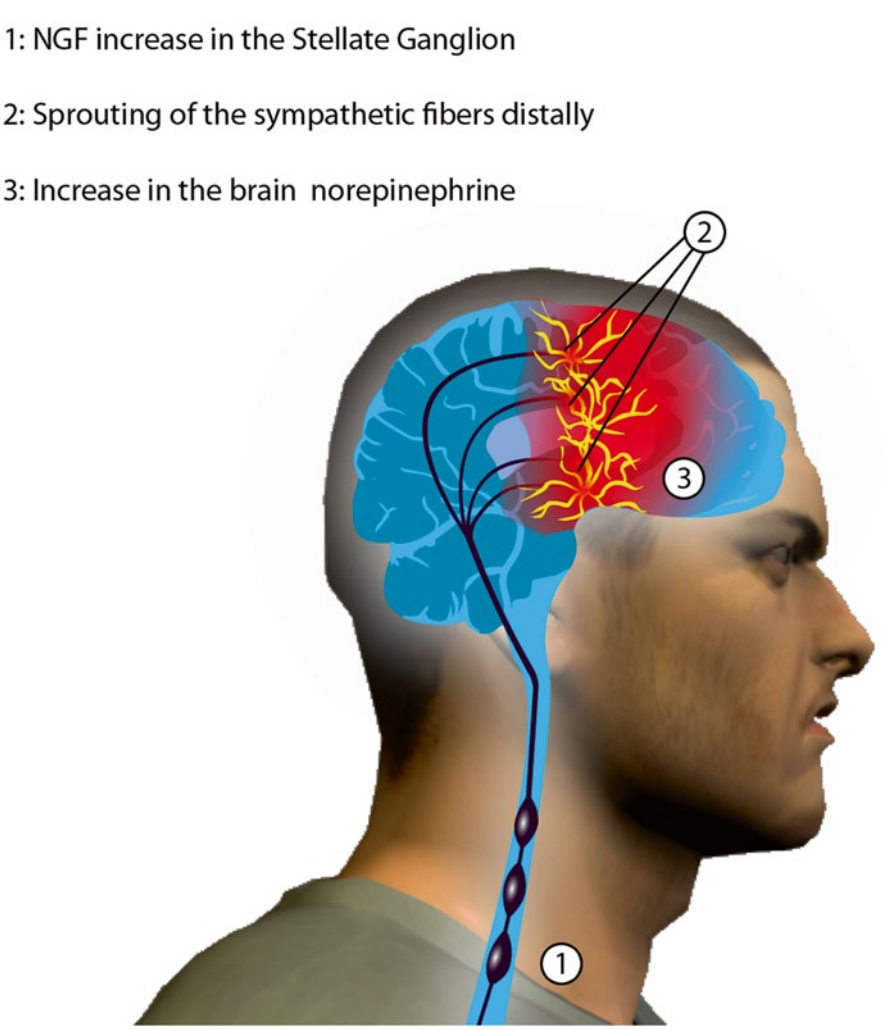

Fig. 13.2 The neurite outgrowth has been associated with NE increase. (From Vlessides M, Anesthesia method suggests possible cure for post-trauma stress. Anesthesiology News 2012; 38:9, with permission)

The NE increase has been shown to be associated with PTSD in urine [33] and cerebrospinal fluid [34] as discussed previously. The NE increase is likely due to NGF increase in the stellate ganglion which in turn is caused by retrograde NGF transport from the intracerebral site to the stellate ganglion [55]. NGF increase is also known to promote neurite outgrowth (sprouting) at the end terminals [56]. The neurite outgrowth has been associated with NE increase [57] (Fig. 13.2).

Finally, local anesthetic injections are known to suppress NGF [58], leading to dying of new nerve outgrowth since maintenance of sprouting is dependent on the

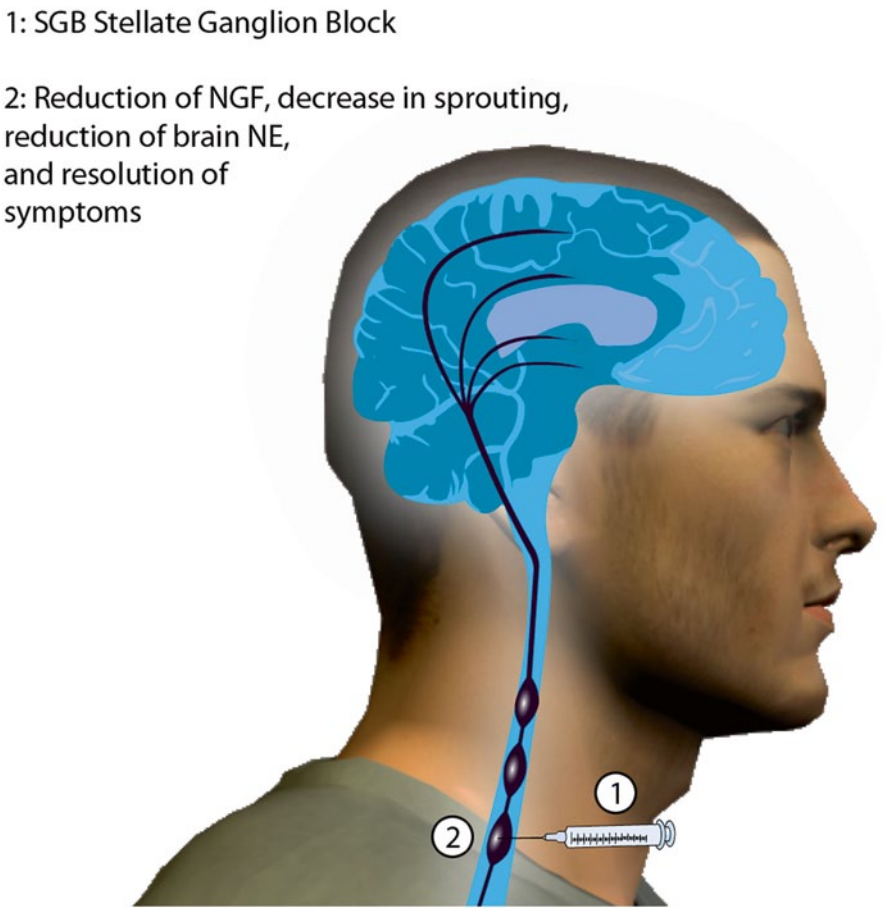

Fig. 13.3 It is hypothesized that the suppression of NGF would reduce NE levels and reverse the cascade of PTSD. (From Vlessides M, Anesthesia method suggests possible cure for post-trauma stress. Anesthesiology News 2012;38:9, with permission)

presence of NGF [59]. As a result, it is hypothesized that the suppression of NGF would reduce NE levels and reverse the cascade of PTSD [31] (Fig. 13.3).

The third and final line of evidence in support of the theory is based on EEG evaluation of rats following SGB. Dr. Jeong found that SGB with bupivacaine resulted in significantly decreased EEG activities in rats. These results suggest that SGB can induce a sedative effect in rats. The proposed mechanism of the effect described above was reduction in brain NE [60].

A possible new nomenclature for organization of PTSD etiology and treatment is based on the sympathetic system involvement. Complex regional pain syndrome (CRPS) and PTSD correlates.

13.5.4 Background

Complex regional pain syndrome (CRPS) is a chronic pain condition most often affecting one of the limbs, and is associated with mechanical hyperesthesia. Dr. Bogdak has recognized CRPS as a central phenomenon [61]. Repeated SGB utilizing local anesthetic are documented as being successful in treating patients suffering from upper limb CRPS, in prospective and retrospective trials [62]. CRPS and PTSD share common brain structure activation, mainly in the insular cortex. Activation of the insular cortex has been demonstrated on fMRI during mechanical hyperesthesia with CRPS [63], and in PTSD [64]. Because CRPS and PTSD seem to be mediated—at least in part—via insular cortex, one would anticipate that both conditions may occur simultaneously or one may lead to the other. One such report exists, where a Vietnam veteran had a recurrence of PTSD symptoms and simultaneous onset of CRPS in the leg, where the authors felt both the conditions have a common supraspinal mechanism [65].

13.5.4.1 A Possible New Nomenclature

CRPS is classified as being, sympathetically mediated pain (SMP) or sympathetically independent pain (SIP). By definition, SMP is responsive to SGB. Thus, CRPS with SMP diagnosis is made following a significant reduction in pain after SGB.

The SIP is defined as CRPS pain that is not responsive to SGB. The distinction of SMP and SIP is made to direct therapeutic intervention. Similarly, the author is proposing a similar diagnostic organization for PTSD, that is, the PTSD patients who respond to SGB can be diagnosed with sympathetically mediated PTSD (SMP), and those sympathetically independent PTSD who are irresponsive to SGB treatment (SIP). Sympathetically mediated PTSD (SMP) would be the equivalent of SMP, the way to diagnose and treat SMP; PTSD is by doing the SGB. The percentage of patients with SMP PTSD seems to be 70 % of PTSD patients based on current data. Sympathetically independent PTSD (SIP) would be equivalent of SIP. This diagnosis would be made following inadequate effect of SGB on PTSD symptoms, and no further SGB would be done.

The advantage of this nomenclature is a way to organize PTSD patients that have reversible sympathetic system activation and will respond to SGB.

The integration, of seemingly unrelated medical fields of study, has been predicted by Dr. Schore in 2002. He stated that neuropsychiatry and psychiatry would integrate for the treatment of PTSD [66]. He went on to report that data exists documenting episodes of trauma expressed in episodes of hyperarousal and dissociation; which are imprinted into the limbic and ANS of the right brain. These enduring structural changes lead to the inefficient stress-coping mechanisms that lie at the core of PTSDs [66]. SGB has been described as having therapeutic effect by affecting limbic and ANS, amygdala, and SNS, respectively [31].

13.6 Conclusion

Using the sympathetic system modulation as a new target for the treatment of psychiatric symptoms seems to have promise in the battle to resolve PTSD scourge. SGB has already changed a number of lives and has been used to help "save" four patients from suicide (unpublished data). It seems difficult to believe that an injection in the neck may lead to psychiatric effect at first glance; however, if one considers the persuasiveness of the sympathetic system and the well described SNS activation in PTSD, this effect becomes increasingly more plausible.

At the time of writing this chapter, over 2,000 military personal have been treated with SGB for PTSD in four military hospitals, with an over 70 % success rate. The advantages of the SGB as a PTSD therapy lie in the fact that it offers virtually immediate relief, increases compliance with the therapy, consistently maintains a high efficacy rate, reduces the use of psychiatric medications, and significantly improves the psychotherapy efficacy. As such, this innovation may not only improve the quality of life for millions of patients but may also reduce the overall socioeconomic burden of treating PTSD on the health-care system. If SGB indeed lives up to its early potential, and is validated by formal studies, it may have substantial short-term and long-term benefits by alleviating suffering and hardships for PTSD patients and their loved ones.

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