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Efficacy of Stellate Ganglion Blockade in Managing Electrical Storm: A Systematic Review

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Abstract

BACKGROUND—The efficacy of percutaneous stellate ganglion block (SGB) for managing electrical storm (ES) is not well understood.

OBJECTIVE—To characterize the efficacy of SGB as a treatment for ES.

METHODS—We conducted literature searches using PubMed/Medline and Google Scholar, for mixed combinations of terms including “stellate ganglion block”, “*ganglion block (ade)”, “sympathetic block (ade)” and “arrhythmia”, “ventricular arrhythmia (VA)” or “tachycardia” (VT), “ventricular fibrillation” (VF), “electrical storm”. Inclusion criteria were presentation with guideline-defined ES and treatment with SGB. Exclusion criteria: presentation with any supraventricular arrhythmia, VA without ES, or surgical sympathectomy. Studies lacking basic demographic data, arrhythmia description, and outcomes were excluded.

RESULTS—Of 3,374 publications reviewed, 38 patients from 23 studies met study criteria (52 ± 19.1 years, 11 F, 17 with ischemic cardiomyopathy). Anti-arrhythmics were used in all patients. Mean Left ventricular ejection fraction was $31 \pm 10\%$. ES was triggered by acute myocardial infarction in 15 patients and QT prolongation in 7 patients. The most common local anesthetic used for SGB was bupivacaine (0.25–0.5%). SGB resulted in a significant decrease in VA burden (12.4 ± 8.8 vs. 1.04 ± 2.12 episodes/day, $p < 0.001$) and number of external and ICD shocks (10.0 ± 9.1 vs. 0.05 ± 0.22 shocks/day, $p < 0.01$). Following SGB, 80.6% of patients survived to discharge.

CONCLUSION—SGB is an effective acute treatment for ES. However, larger prospective randomized studies are needed to better understand the role of SGB in ES and other VAs.

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Keywords

Stellate ganglion block; electrical storm; neuromodulation; autonomic nervous system; sympathetic nerves

INTRODUCTION

Electrical storm (ES) is commonly defined as the occurrence of three or more episodes of sustained ventricular arrhythmia (VA) over 24 hours (1,2). Antiarrhythmic medications and catheter ablation remain the standard of care in patients with ES or other refractory VA (3). The role of the autonomic nervous system in ventricular arrhythmogenesis is well recognized (4), and neural modulation via a number of avenues is increasingly gaining traction (5). Cardiac sympathetic denervation (CSD), the surgical resection of the lower half of the stellate (cervicothoracic) ganglion and T2–4 sympathetic ganglia, has been shown to be effective in the setting of ES or other refractory VA (6–14). Other forms of neuromodulation include pharmacologic beta-adrenergic receptor blockade, thoracic epidural anesthesia (TEA) spinal cord stimulation (SCS), and stellate ganglion block (SGB) (5). Stellate ganglion block (SGB) is performed by injecting local anesthetic agents percutaneously to stellate ganglion, which is less invasive than CSD and can be performed at bedside in emergent setting in patients with hemodynamic instability. Currently, besides case reports and small series, limited evidence is available regarding the role of SGB in ES (15–37). To better understand the role of SGB in ES (particularly to address patient characteristics, techniques, and overall efficacy of SGB), we performed dedicated literature searches and performed this systemic review.

METHODS

Literature search, and criteria for inclusion or exclusion

Using PubMed/Medline and Google Scholar, we performed varying combinations of searches using the following terms, “left stellate ganglion block”, “ganglion block (ade)”, “sympathetic block (ade)”, “arrhythmia”, “ventricular arrhythmia (VA)” “ventricular tachycardia” (VT), “ventricular fibrillation” (VF), “electrical storm (ES)”. ES was defined as three or more episodes of sustained VT or VF within a 24-hour period (1). The search was limited to human subjects only. The search strategy was neither restricted by the language, nor the date of publication. Inclusion criteria were patients presenting with ES who underwent SGB. Bilateral SGB was also included. Exclusion criteria included patients presenting with any supraventricular tachycardia, ventricular arrhythmia without ES (for example, premature ventricular contractions), or patients treated only with surgical sympathectomy. The articles were carefully reviewed for inclusion and exclusion criteria. A lack of basic patient demographics, arrhythmia description, or outcomes, which are critical for this study, was also a reason for exclusion.

All clinical variables were extracted from the selected studies including number of patients, age, gender, type of VA, episodes of VA and shocks before and immediately after SGB, presence of underlying cardiomyopathy, left ventricular ejection fraction (LVEF), trigger of

ES, antiarrhythmic medications used, other procedures or treatment received prior to or after SGB, anesthetic administration techniques (i.e. laterality, bolus injection vs. continuous pump infusion, use of imaging guidance), type and volume of local anesthetic agent used, inpatient survival to discharge. Relative reduction of VA and defibrillator shocks are calculated as the difference in episodes per day pre-vs. post-SGB, divided by the number of episodes per day pre-SGB.

Statistical analysis

Continuous variables were summarized as mean \pm SD. Comparison of VA episodes and number of external or ICD shocks before and after SGB was performed using Wilcoxon signed-rank test, given the non-normal distribution of the data. Change in VA burden or defibrillator shocks was expressed as relative reduction (i.e. post-SGB/pre-SGB). The relationship between arrhythmia reduction and LV EF was examined by linear regression. Comparison of cardiomyopathy subtypes was performed using Analysis of Variance (ANOVA), and arrhythmia subtypes were compared using the Kruskal-Wallis test, as these data were non-normally distributed. A p-value < 0.05 was considered significant.

RESULTS

Patient characteristics

A total of 3374 publications were reviewed and 38 patients from 23 studies published between 1976 and 2016 were included based on inclusion and exclusion criteria. The mean age of the patient population was 52 ± 19.1 years. Twenty-seven (71%) patients were male. Cardiomyopathy (CMY) was present in 24 (63.2%) patients (ischemic CMY (ICMY) in 17 patients, and non-ischemic CMY (NICMY) in 7 patients, Table 1). The mean LVEF was 31 ± 10 %. Acute myocardial infarction (AMI) was the most common trigger of ES (15 patients), followed by prolonged QT (7 patients). Interestingly, intracranial hemorrhage was the etiology in 2 patients. In 37% patients, the trigger of ES was unspecified. Of the arrhythmia types, mixed VT/VF was the most common type encountered ($n=15$), followed by polymorphic VT ($n=12$). Four patients had monomorphic VT and 7 patients had primary VF without VT (Table 1). Almost all patients were treated with beta-blocker therapy (33/38). All patients except one received antiarrhythmic medications prior to SGB (Table 2) and the most common agents were amiodarone (82%) and lidocaine (68%). On average, 1.82 ± 0.82 anti-arrhythmic drugs were used, along with beta-blockers and/or calcium channel blockers prior to SGB. Regarding other interventions prior to SGB, 36.8% patients were intubated and deeply sedated, while 15.8% patients were treated with catheter-based ablation (Table 3).

Approach to SGB

SGB was achieved by administering local anesthetic percutaneously to the SG. We examined the delivery method, the type and amount of local anesthetic, as well as the utility of imaging guidance with SGB. 34 patients received only left SGB whereas in 4 patients, both left and right SGB were performed. As noted in Table 4, local anesthetic agents were administered as bolus injections in 28 patients (73.7%), whereas continuous infusion with pump system was used in nine (one patient had both bolus and continuous infusion). Among patients receiving bolus injections, bupivacaine was the most commonly used anesthetic in

patients with SGB (n=16), while ropivacaine was the next most commonly used. The mean volume of bupivacaine used was 9 ± 5.6 ml with concentration ranging from 0.25% to 0.5%. We also examined the utility of imaging when performing SGB. Ultrasound guidance was utilized in 21 patients, and fluoroscopy in 4 patients (Table 4). In the remaining 13 patients, SGB was performed with anatomic landmarks only without imaging guidance.

Efficacy of SGB in immediate reduction of VA burden and shocks

To determine the efficacy of SGB, we quantified ES burden as the episodes of VA per day and number of external or ICD shocks per day. As shown in Figure 1, there was a significant reduction in both the episodes of VA, and number of shocks in patients with ES after SGB. SGB decreased VA burden from 12.4 ± 8.8 episodes/day to 1.04 ± 2.12 episodes/day ($p < 0.001$). The number of external or ICD shocks was decreased from 10.0 ± 9.1 /day to 0.05 ± 0.22 /day ($p < 0.01$).

In this cohort, 24 of the 38 patients demonstrated complete arrhythmia suppression following SGB during the immediate follow up period, while the remaining 14 showed partial success. The impact of SGB did not depend on the subtype of VA causing ES. Relative reduction in VA episodes for the four subtypes studied, monomorphic VT, polymorphic VT, mixed VT/VF, and primary VF were 0.94 ± 0.1 ; 0.76 ± 0.25 ; 1 ± 0 ; and 0.97 ± 0.05 , respectively ($p = 0.124$). The duration of clinical suppressive effect after bolus injection was 6–24 hours for ropivacaine, 8–18 hours for lidocaine, 6 hours to 1 week for bupivacaine, 11 hours to 4 weeks for mepivacaine.

We examined whether the anti-arrhythmic benefit of SGB was influenced by the presence and degree of LV dysfunction. As shown in figure 2A, there was no correlation between the LV EF, and arrhythmia reduction ($r^2 = 0.05$, $p = 0.384$). Patients with normal LV function, mild, moderate, or severe LV dysfunction, equally benefitted from SGB. Similarly, the presence and etiology of CMY did not influence the ability of SGB to exert anti-arrhythmic benefits (figure 2B). Relative reduction in VA burden was 0.95 ± 0.07 , 0.85 ± 0.19 , and 0.83 ± 0.29 respectively for no CMY, ICMY, and NICMY, respectively ($p = 0.78$). Using relative VA reduction $< 50\%$ as a cut off for poor response to SGB, 10% of patients were labeled as poor responders to SGB. The rest of the patients were considered to have good response. Characteristics of good and poor responders were summarized in Table 5. All poor responders were male and presented with PMVT. Mean LVEF in this group was $27 \pm 13\%$.

Following SGB, 80.6% of patients survived to discharge (hospital day 6–28), and terminal sympathectomy via surgical CSD was performed in 11 patients. One patient underwent orthotopic heart transplantation.

DISCUSSION

The major findings of the present study on the efficacy of SGB for ES are 1) SGB is effective in reducing the number episodes and therapies for VA, and 2) this efficacy was independent of the subtype of ventricular arrhythmia, the presence or absence of cardiomyopathy, and the degree of LV dysfunction in the patients studied. To our

knowledge, this is the first systemic review of the efficacy of the SGB in patients with ES, and strongly supports the use of SGB in patients with ES.

There is a strong link between cardiac sympathetic activity and ventricular arrhythmogenesis (38,39). In a rabbit myocardial infarction model, left stellate ganglion blockade (LSGB) prolonged the action potential duration (measured as MAPD₉₀) in all layers of the myocardial wall, reducing transmural repolarization heterogeneity, increased the effective refractory period (ERP), and reduced ventricular fibrillation threshold (40). SGB, by mitigating catecholamine release, likely reverses the findings in a canine post-infarct model of shortened ERP in abnormal ventricular myocardium, and increased inducibility of arrhythmias in the presence of catecholamines (41). SGB may also be particularly useful for attenuating burst discharge activity in left stellate ganglion, which has been shown to precede most VT and sudden cardiac death in a canine model of ischemic cardiomyopathy (42–43). Sympathoexcitation increases repolarization heterogeneity (44–46), increases risk of delayed after depolarizations (47,48), and increases arrhythmia inducibility (49). It also modulates peri-infarct tissues harboring circuits capable of facilitating MMVT (50). The efficacy of beta-adrenergic receptor antagonism in patients with cardiomyopathy (51), is related to minimizing the adverse effects of adrenergic activation signaling. However, despite use of beta-adrenergic receptor blockers, anti-arrhythmic medications (some of which further antagonize beta-adrenergic receptors), and catheter ablation, some patients continue to have arrhythmias. In this study, we examine the efficacy of SGB in this population.

A number of mechanisms may explain the therapeutic benefits imparted by SGB. The bulk of efferent sympathetic outflow to the heart travels through the SG. Preganglionic fibers mediating neurotransmission to the heart synapse on postganglionic neurons within the SG (and adjacent ganglia within the sympathetic chain) (Figure 3). In addition, postganglionic fibers from other ganglia may also travel through the SG to middle cervical ganglia and cardiac nerves (52). This represents an effective site to target cardiac adrenergic activation while limiting systemic effects. Further, post-ganglionic axons release a variety of neurotransmitters, of which noradrenaline, targeted by beta blockers, is only one. Additional neurotransmitters such as neuropeptide Y and galanin modulate adrenergic signaling at the myocardial level (53,54). However, in some cases, as evidenced by the patients examined in this study, beta-adrenergic blockade, antiarrhythmic drugs, and catheter ablation do not mitigate these other mechanisms. Interventions targeting the SG however, would attenuate not only noradrenaline signaling, but these additional signaling pathways.

Sensory neurotransmission is also mitigated by SGB. Sympatho-excitatory reflexes that occur at multiple levels are triggered by sensory afferent nerves that relay information intrinsic cardiac, mediastinal, SG, spinal cord and brain stem (38). At these centers, the sensory information is processed, and reflex sympathetic activity is generated. The bulk of spinal sensory afferents, which have been implicated in the pathogenesis of heart failure (55,56), and along the same lines arrhythmogenesis pass through the SG, enroute to the dorsal horn of the spinal cord. Infusion of anesthetic agents to block the SG also targets these fibers, and therefore attenuates both afferent and efferent neurotransmission at the SG

(Figure 3). Although SGB does not affect vagal afferents, the reduction of spinal afferents likely debulks overall cardiac afferent neurotransmission.

The burden of VAs and defibrillator shocks were significantly reduced after SGB in patients with ES. This occurred in patients independent of the etiology of the arrhythmia, triggering mechanisms, and LV function. VA suppression varied after bolus of injection of local anesthetics (LA), ranging from hours to weeks. The longer duration of efficacy relative to the anesthetic half-life is most likely related to short- and long-term adaptations in neurotransmission and neural processing. Due to the highly plastic nature of neurons, a short-term intervention can produce long-lasting effects, well beyond what is expected for the drug alone. That said, there is likely to be some variation related to pharmacodynamics of the agents used, proximity of the injection of the ganglion, and thickness of the ganglion sheath.

We present the most commonly used approaches, anesthetic agents and doses to achieve SGB. The patient characteristics and technical data generated by the present study may be helpful as a reference for the institution of SGB. These data may also be useful in the design and patient selection for randomized prospective studies to improve SGB techniques in clinical practice.

LIMITATIONS

This study has a number of limitations, which include the number of studies in the literature meeting inclusion/exclusion criteria. As these were predominantly case reports and case series, data reported here are retrospective. Further the small sample size limits potentially instructive subgroup analyses, as well as the reliability of the analyses made. To improve the accuracy of the present study, reports in the literature lacking basic patient demographics were excluded, reducing the overall number of patients in the study (59).

CONCLUSION

The findings of the present study support the routine use of SGB as an effective adjunct to contemporary therapies in managing ES. SGB is efficacious for a variety of VA subtypes and patient demographics. Prospective randomized studies are needed to better understand the role of the SGB in ES and other VA.

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Abbreviations

CMY	cardiomyopathy
ES	electrical storm
LA	local anesthetics

LQT	long QT syndrome)
LVEF	left ventricular ejection fraction
SGB	stellate ganglion block
VA	ventricular arrhythmias
VF	ventricular fibrillation
VT	ventricular tachycardia

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Perspectives

Competency in Medical Knowledge 1

The role of neuromodulation in managing electrical storm is gaining traction.

Competency in Medical Knowledge 2

Anesthetic blockade of the stellate ganglion achieved percutaneously represents an attractive approach that can be implemented in a variety of settings, including at the patient's bedside.

Competency in Patient Care

The types of anesthetic agents, doses, and methods of dosing are important in applying stellate ganglion block. Characteristics of patients, and the arrhythmias controlled by stellate ganglion block would guide patient care.

Competency in Interpersonal & Communication Skills

It is important to discuss the available options with patients and the medical team.

Translational Outlook 1

Stellate ganglion block is effective in controlling electrical storm, and is not limited by the type of ventricular arrhythmia, or the presence/type of structural heart disease.

Translational Outlook 2

Additional research is needed to understand the safety and efficacy of stellate ganglion block in prospective randomized trials to improve its use.

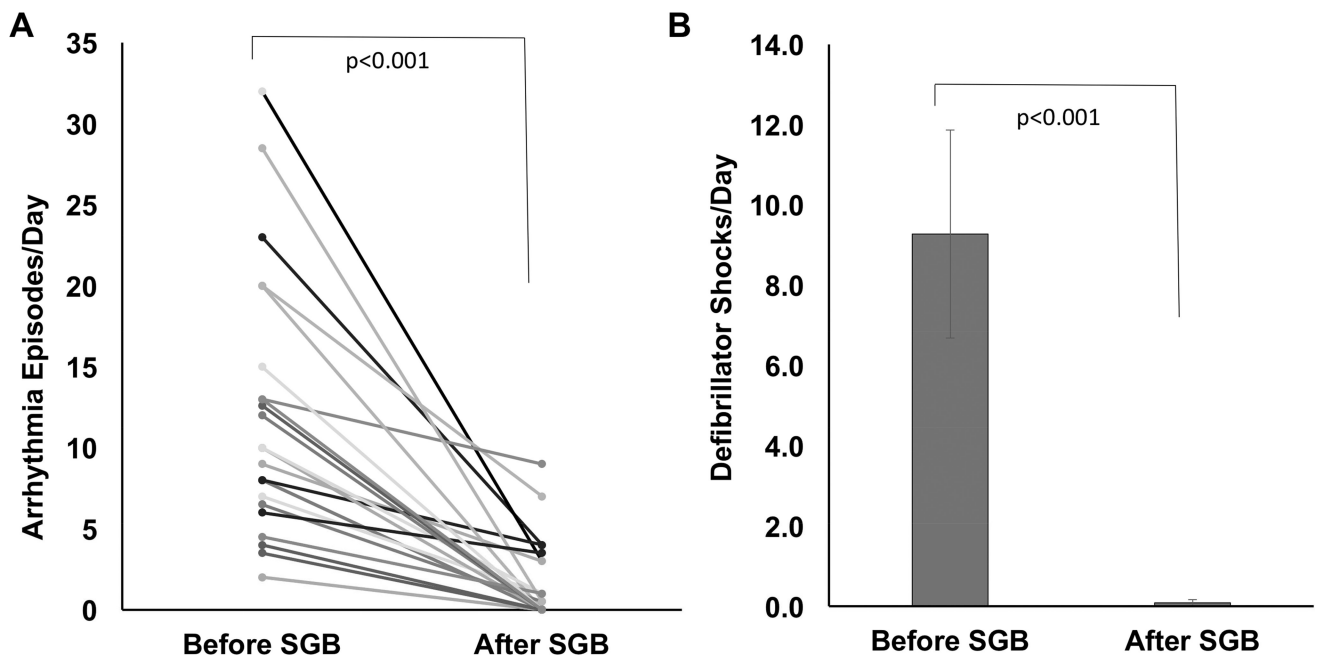


Figure 1. Impact of stellate ganglion block on ventricular arrhythmia episodes and defibrillator shocks

Reduction in the number of ventricular arrhythmia episodes (A.) and number of internal or external defibrillator shocks (B.) before and after stellate ganglion block (SGB) are shown. Panel A (n=24) and panel B (n=11).

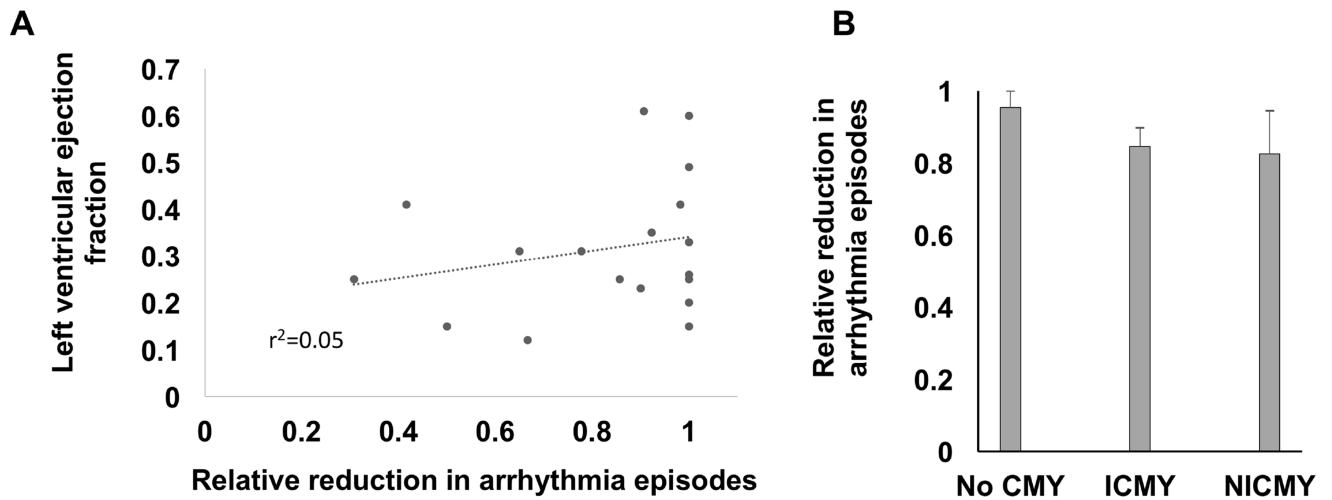


Figure 2. Relationship between arrhythmia burden reduction and left ventricular dysfunction
A. The relationship between left ventricular ejection fraction (LV EF) and the relative reduction in ventricular arrhythmia (VA) episodes (n=18), $r^2=0.05$, $p=0.384$. B. Reduction in arrhythmia episodes was independent of the presence and etiology of cardiomyopathy (CMY), n=24, $p=0.78$).

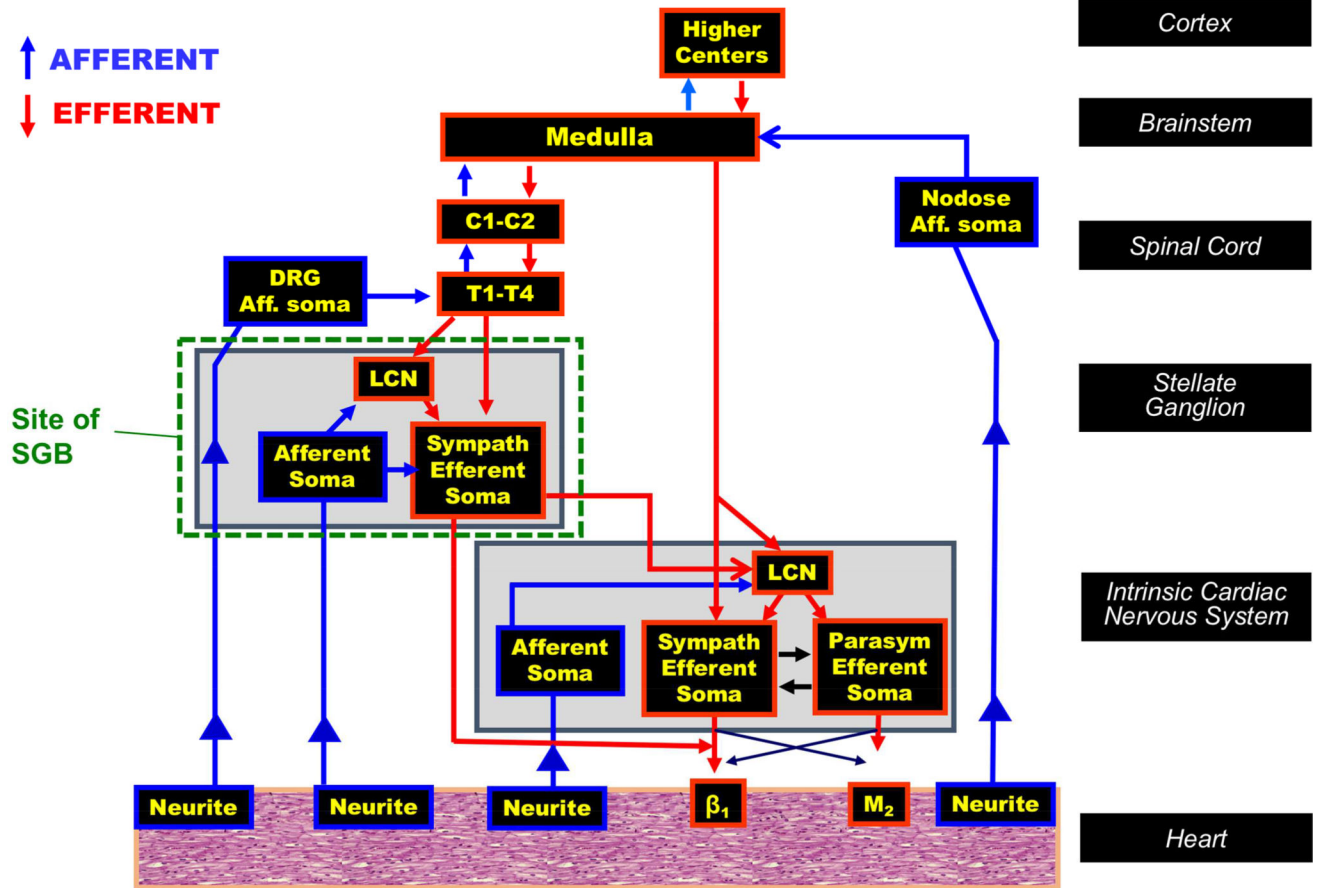


Figure 3. Schematic of cardiac innervation and site of stellate ganglion block

Stellate ganglion block directly impacts both efferent and afferent neurotransmission to the heart. The site of action and components are shown. Aff, afferent; β , β -adrenergic receptor; C, cervical; DRG, dorsal root ganglion; LCN, local circuit neuron; M, muscarinic receptor, Sympath, sympathetic, and T, thoracic.

Table 1

Patient Characteristics.

Mean age (years)	52±19.1
Male	27 (71%)
Presence of Cardiomyopathy	35 (92%)
Ischemic CMY	17
Non-ischemic CMY	7
Unspecified	11
Arrhythmia Type	
Mixed VT/VF	15
PMVT	12
MMVT	4
VF	7
Left Ventricular Ejection Fraction	31±10%
Acute trigger of ES	
Acute MI	15
Prolonged QT	7
Intracranial hemorrhage	2
Unspecified	14

CMY: cardiomyopathy, MI: myocardial infarction, MMVT: monomorphic ventricular tachycardia, PMVT: polymorphic ventricular tachycardia, VF: ventricular fibrillation, VT: ventricular tachycardia.

Table 2

Medications used before institution of stellate ganglion block.

Amiodarone	23 (82%)
Beta blocker	31 (82%)
Metoprolol	7 (25%)
Propranolol	6 (21%)
Esmolol	4 (14%)
Carvedilol	1 (4%)
Unspecified	14 (50%)
Lidocaine	19 (68%)
Sotalol	1 (4%)
Procainamide	8 (29%)
Mexiletine	3 (11%)
Quinidine	2 (7%)
Ajmaline	1 (4%)
Bretylum	1 (4%)
Phenytoin	8 (29%)
Digitalis	1 (4%)
Isoproterenol	1 (4%)
Verapamil	1 (4%)

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Table 3

Procedures/Interventions performed prior to stellate ganglion block.

Intubation and deep sedation	14 (36.8%)
Cardiac catheter ablation	6 (15.8%)
IABP	6 (15.8%)
ECMO	3 (7.9%)
TEA	2 (5.3%)
Tandem heart	1 (2.6%)

ECMO: Extracorporeal membrane oxygenation, IABP: Intra-aortic balloon pump, TEA: thoracic epidural anesthesia.

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Table 4

Approaches to anesthetic use for stellate ganglion block.

Anesthetic Agent	Number of patients	Dose (Concentration, %)	Volume (ml)
Bupivacaine	16	0.25–0.5	9 ± 5.6
Ropivacaine	11	0.2	6 ± 5.7
Lidocaine	9	1–4	8 ± 3.8
Mepivacaine	2	2	4 ± 0.0
Type of administration of anesthetics		Number of patients	
Bolus injections		28	
Continuous infusion		9	
Both bolus injections and continuous infusion		1	
Utility of imaging guidance		Number of patients	
Landmark only without imaging		13	
Ultrasound		21	
Fluoroscopy		4	

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Table 5

Comparison of patients with poor response and good response to LSGB.

	Poor response	Good response
Number of patients	3	35
Mean age (years)	61±9	51±20
Male	3 (100%)	24 (68.6%)
Presence of Cardiomyopathy	3 (100%)	24 (68.6%)
Ischemic CMY	2 (66.7%)	15 (42.9%)
Non-ischemic CMY	1 (33.3%)	6 (17.1%)
Unspecified CMY	0	3 (8.6%)
Arrhythmia Type		
Mixed VT/VF	0	12 (34.3%)
PMVT	3 (100%)	12 (34.3%)
MMVT	0	7 (20%)
VF	0	4 (11.4%)
Left Ventricular Ejection Fraction	27±13%	32±14%
Acute trigger of ES		
Acute MI	2 (66.7%)	14 (40%)
Prolonged QT	1 (33.3%)	7 (20%)
Intracranial events	0	2 (5.7%)
Unspecified	0	12 (34.3%)
Type of local anesthetics		
Bupivacaine	2 (66.7%)	14 (40%)
Ropivacaine	1 (33.3%)	10 (28.6%)
Mepivacaine	0	9 (25.7%)
Lidocaine	0	2 (5.7%)
Method of administration		
Bolus injection	2 (66.7%)	27 (77.1%)
Continuous infusion	1 (33.3%)	7 (20%)
Both bolus and continuous infusion	0	1 (2.9%)
Type of imaging guidance		
Ultrasound	3 (100%)	18 (51.4%)
Fluoroscopy	0	4 (11.4%)
Inpatient survival		
Discharged	1 (33.3%)	30 (85.7%)
Deceased	2 (66.7%)	5 (14.3%)