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Stellate Ganglion Blockade: an Intervention for the Management of Ventricular Arrhythmias

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Abstract

Purpose of Review—To highlight the indications, procedural considerations, and data supporting the use of stellate ganglion blockade (SGB) for management of refractory ventricular arrhythmias.

Recent Findings—In patients with refractory ventricular arrhythmias, unilateral or bilateral SGB can reduce arrhythmia burden and defibrillation events for 24–72 h, allowing time for use of other therapies like catheter ablation, surgical sympathectomy, or heart transplantation. The efficacy of SGB appears to be consistent despite the type (monomorphic vs polymorphic) or etiology (ischemic vs non-ischemic cardiomyopathy) of the ventricular arrhythmia. Ultrasound-guided SGB is safe with low risk for complications, even when performed on anticoagulation.

Summary—SGB is effective and safe and could be considered for patients with refractory ventricular arrhythmias.

Keywords

Stellate ganglion block; Ventricular arrhythmia; Ventricular tachycardia; Ventricular fibrillation

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Introduction

Ventricular tachycardia (VT) and fibrillation (VF) are life-threatening arrhythmias with an increasing prevalence [1]. Ventricular arrhythmias are especially common in cardiac and surgical intensive care units, and they are responsible for over 450,000 deaths every year in the USA [2]. Electrical storm is a particularly challenging clinical entity in which VT/VF events recur frequently (3 or more episodes of sustained or hemodynamically significant VT/VF in 24 h). This dangerous clinical entity is commonly driven by underlying structural heart disease, and approximately 10–40% of patients have a prior history of VT/VF [3]. Even in the presence of an implantable cardiac defibrillator (ICD), electrical storm confers an eightfold higher risk of arrhythmic death [4]. Despite the use of antiarrhythmic medications, acute mortality due to electrical storm remains over 20% [5].

The sympathetic nervous system is a key player in the genesis of ventricular arrhythmias and represents an important therapeutic target [6]. Established treatments for ventricular arrhythmias include β -blockers, other antiarrhythmic medications, mechanical circulatory support, and catheter ablation. However, these treatments are often inadequate and often do not successfully suppress VT/VF events. In some cases, the arrhythmias persist despite all therapies, while in other cases, patients may be too hemodynamically unstable to permit interventions like catheter ablation. Mounting evidence suggests that stellate ganglion blockade (SGB) can reduce myocardial sympathetic tone and thus serve as a useful adjunct in the management of ventricular arrhythmias and electrical storm. SGB has been described as a useful suppressive therapy for ventricular arrhythmias or as a bridge therapy to other interventions such as surgical sympathectomy, heart transplantation, or catheter ablation [7••].

In this manuscript, we provide a comprehensive review of the anatomy of the stellate ganglion, indications for SGB, procedural technique, safety considerations, and current evidence supporting SGB for treating refractory ventricular arrhythmias. This review concludes with a discussion of future directions.

Anatomy and Indications

The stellate ganglion is a collection of neuron cell bodies characterized by the fusion of the inferior cervical and first thoracic sympathetic ganglia (Fig. 1). The word “stellate” is used to describe the ganglion’s star-like shape. The ganglion is variably located in the lower part of the neck, typically anterior to the C7 or T1 vertebral body [8]. In the stellate ganglion, thoracic preganglionic sympathetic nerves synapse with post-ganglionic sympathetic nerves that provide efferent sympathetic output to the myocardium, upper extremity, neck, and face. In the context of normal physiologic function, SGB mediates multiple cardiovascular effects, including vasodilation of the upper extremity and face, anhidrosis (part of Horner’s syndrome), and reduced inotropy and chronotropy. This is why SGB has been successfully used to treat upper-extremity ischemia from Raynaud’s disease [9], hyperhidrosis [10], refractory angina [11], and ventricular arrhythmias (Table 1) [12••, 13••].

The stellate ganglia are bilateral structures. However, the right and left stellate ganglia do not have equivalent sympathetic effects on the myocardium. In canine models, left stellate ganglion resection results in higher thresholds for induction of VF compared with controls and those with right stellate ganglion resection [14]. Likewise, coronary artery occlusion amidst left SGB is less arrhythmogenic compared with controls and/or right SGB [15]. These results suggest that the left stellate ganglion contributes more sympathetic tone to the myocardium than the right. This explains why most human studies use unilateral left SGB for treatment of ventricular arrhythmias [7•, 13•]. However, it is also important to note that damage to the myocardium can result in remodeling of both the right and left stellate ganglia, which supports the use of bilateral blockade for treatment of ventricular arrhythmias [16•]. In a porcine model of myocardial infarction (MI), myocardial injury induced bilateral morphologic and neurochemical changes in the stellate ganglia about 5 weeks after the initial insult [16•]. MI led to an increase in neuronal size and synaptic density of both stellate ganglia, with a measurable increase in neuronal firing for up to 8 weeks. This illustrates that the stellate ganglion is not just a relay point for efferent nerve signals but also receives afferent input from the heart, resulting in the aforementioned changes. Further, remodeling with changes in gene expression and neuronal structure is not limited to stellate ganglia but is also found in the thoracic dorsal root ganglia (DRG) of the spinal cord [17, 18], suggestive of afferent crosstalk between the stellate ganglion and central nervous system as well.

It is important to understand that while SGB is a relatively recent addition to the electrophysiologic armamentarium, SGB is most commonly used for treatment of chronic pain conditions like complex regional pain syndrome (CRPS), orofacial pain, and post-mastectomy pain (Table 1) [19–22]. Given that pain relief can be achieved via sympathetic blockade, these pain syndromes are often characterized as “sympathetically mediated.” Although the underlying mechanisms remain to be fully elucidated, the coupling between sympathetic efferent fibers and afferent nociceptive fibers is believed to contribute to these pain states [23]. Central mechanisms have also been postulated [24]. There are also reported direct neural connections from the stellate ganglion to intracerebral areas like the amygdala, hypothalamus, and insula, which are known to be involved in pain perception [24]. These areas are also activated during hot flashes and post-traumatic stress disorder, which may explain why SGB has been used successfully for the treatment of these disorders as well (Table 1) [24–26].

Stellate Ganglion Block Procedure

Decades ago, SGB was a “blind” procedure performed without image guidance and based on the palpation of anatomical landmarks to guide the needle. The procedure is now most commonly performed using either fluoroscopic or ultrasound guidance. Ultrasound has increasingly become the imaging modality of choice, as it allows for direct visualization of critical structures that reside near the stellate ganglion, including the carotid and vertebral arteries (Fig. 2). Recent studies reporting on the use of SGB for the management of ventricular arrhythmias were performed predominantly with ultrasound guidance [12•, 13•]. The use of ultrasound is recommended as this can easily be utilized at the bedside. Additionally, many of these patients are anticoagulated when the procedure is performed,

and thus visualization of nearby vascular structures is helpful to actively avoid puncture and subsequent bleeding.

The technical details of ultrasound-guided SGB have been described previously [27, 28]. In brief, an echogenic needle is guided via continuous ultrasound visualization in an in-plane fashion until the tip of the needle is between the carotid artery and longus colli muscle at either the C6 or C7 level (Fig. 2). Caudal spread of local anesthetic leads to blockade of the stellate ganglion in the C7/T1 region. The risk of vertebral artery puncture is higher at the C7 level, but utilization of Doppler imaging to identify vessels minimizes this risk. As described by Elmoftly et al., discerning between the C6 and C7 levels can be difficult and using the location of the cervical nerve roots as a guide can be helpful [29]. Once the needle is in its final position, 8–10 mL of 0.2% ropivacaine is typically injected after negative aspiration for blood (to ensure the needle is not intravascular). Ropivacaine is a long-acting local anesthetic with duration of action ranging from 2 to 8 h depending on the site of injection [30]. Due to reduced potential for causing cardiotoxicity, it is preferred over bupivacaine, a more commonly utilized long-acting local anesthetic [30]. While small concentrations of epinephrine are often mixed with local anesthetic to prolong the block and also detect an inadvertent intravascular injection, this is avoided to prevent inadvertent worsening of underlying ventricular arrhythmia.

Measuring Technical Success of Stellate Ganglion Blockade

How does one know whether SGB has been performed successfully? Unfortunately, there are few reliable markers with high sensitivity and specificity. A rise of measured temperature in the ipsilateral hand has traditionally been viewed as a marker of successful block (Fig. 3). This is secondary to the vasodilation and reduced perspiration that SGB causes in the upper extremity. Recent studies, however, have shown that when SGB is used for treatment of pain, a rise in temperature does not necessarily correlate with pain reduction, thus questioning the utility of this measure [19, 31].

Some have used the presence of Horner's syndrome (ptosis, anhidrosis, miosis) as a marker for successful SGB [19, 32]. However, Horner's syndrome can also occur with blockade of the middle or superior cervical ganglion, and thus, this is not a finding that is specific to SGB. The presence of a Horner's syndrome after SGB has also not been predictive of pain reduction [19] and thus has poor sensitivity. Finally, Horner's syndrome can be difficult to observe in ICU patients who are sedated, as is the case for many patients who receive SGB for the management of refractory ventricular arrhythmias.

Another potential metric for the technical success of SGB is the perfusion index (PI) [32–34]. Described in detail by Ginosar et al., PI is an objective parameter that is measured from the pulse oximeter with higher values suggesting increased blood flow [35]. Sahin et al. performed SGB on 40 patients with vasospastic Raynaud's disease and observed higher PI in those patients with successful SGB [32]. Of note, they defined block success by the presence of Horner's syndrome which is controversial. Interestingly, PI was found to be superior to skin temperature readings in assessing for vasodilation after epidural anesthesia [35].

In summary, there is no consensus on the ideal way to confirm technical success of SGB. As SGB becomes more frequent in clinical practice, there will be a greater need to develop a confirmatory assessment of block. Future efforts may need to evaluate a measure that combines several potential indicators, including cutaneous temperature changes, perfusion index, and Horner's syndrome.

Safety of Stellate Ganglion Blockade

Goel et al. recently published a systematic review of complications associated with SGB when performed for mostly pain indications [36••]. The most common reported complications were hoarseness, hematoma, light-headedness, hypertension, and brachial plexus block. The probability of these complications occurring could not be ascertained, as this was just a review of published case reports. In general, SGB is regarded as a safe procedure and is routinely performed on outpatients for treatment of pain.

The most common reported complication from SGB is hoarseness [36••]. Hoarseness is transient and secondary to inadvertent recurrent laryngeal nerve (RLN) blockade [37]. In a study of 20 patients on whom SGB was performed without any image guidance, the incidence of RLN blockade was found to be 10% when 10 mL of local anesthetic was used and 80% when 20 mL was used [37]. Given the increasing use of ultrasound for this procedure, 10 mL of local anesthetic is typically injected, and thus, the probability of RLN blockade is likely lower than reported. While unilateral RLN blockade is considered benign, bilateral blockade could lead to acute closure of the vocal cords which may require emergent airway intervention, hence why many clinicians who use SGB for pain relief consider bilateral SGB contraindicated [38]. Similarly, surgeons often use intraoperative nerve monitoring during thyroidectomy surgery to detect inadvertent RLN injury so that if it occurs, total thyroidectomy is stopped to avoid potential injury to the contralateral RLN [39]. Given these concerns, bilateral SGB is safest in intubated patients. This was the protocol followed by Tian et al. for the 15 patients who received bilateral SGB for treatment of refractory ventricular arrhythmias [13••]. However, it is important to note that given the morbidity and mortality of ventricular arrhythmias, risk/benefit assessments for bilateral SGB are different from those for its use in pain relief. Interestingly, Fudim et al. performed 20 bilateral SGBs for treatment of refractory ventricular arrhythmias, and 7 of these patients were not intubated [12••]. One of these patients exhibited transient hoarseness, but no airway interventions were required [12••].

Neck hematoma is the second most common reported complication of SGB [36••]. Of the 41 reported cases of hematoma, most resolved without intervention, but 5 required urgent tracheostomy [36••]. As many patients with ventricular arrhythmias are on anticoagulation, the decision to continue or stop anticoagulation before SGB is a challenging one. When SGB is utilized for treatment of pain, anesthesiology and pain medicine societies recommend that anticoagulants like warfarin, clopidogrel, and direct oral anticoagulants (DOACs) be discontinued many days before the procedure to reduce the risk of neck hematoma [40]. However, hospitalized patients with VT/VF are often on anticoagulation for critical reasons (e.g., ventricular assist device, extracorporeal membrane oxygenation, intra-aortic balloon pump, coronary stents), and benefits of continuing anticoagulation during

SGB likely outweigh the small risk of hematoma. Additionally, performing SGB for treatment of ventricular arrhythmias is time sensitive, and it is not feasible to wait for a long-acting anticoagulant to metabolize before performing the procedure. Reassuringly, no bleeding complications were reported in recent studies when SGB was used for treatment of refractory VT/VF despite uninterrupted anticoagulation [12••, 13••].

A feared complication of SGB when used for management of ventricular arrhythmias is local anesthetic systemic toxicity (LAST). Patients with refractory ventricular arrhythmias may already be on lidocaine and/or procainamide infusions for arrhythmia suppression. In addition to functioning as antiarrhythmics, these medications are also local anesthetics. Given that local anesthetic toxicity is additive [41], use of local anesthetic (i.e., ropivacaine) during SGB could lead to LAST in the presence of ongoing lidocaine and/or procainamide infusions. This may be even more of a concern in patients also treated with amiodarone, as this has been reported to increase plasma lidocaine levels due to potential cytochrome inhibition by amiodarone [42]. To reduce the risk of LAST, some stop lidocaine and procainamide infusions prior to SGB. The context-sensitive half time of lidocaine after prolonged infusion is ~ 30 min, and thus, plasma levels are minimal ~ 2.5 h after discontinuing the infusion [43]. To our knowledge, the context-sensitive half time of procainamide after prolonged infusion has not been reported but is known to be longer than lidocaine [44]. Given that significant amounts of local anesthetic for SGB are not being injected and that performing the procedure is time sensitive, some do not wait for lidocaine and procainamide serum levels to reduce to negligible levels before performing SGB and proceed approximately 2 h after the infusions are stopped. In a small study of 11 patients who received non-image-guided unilateral SGB with 10 mL of 0.5% bupivacaine for treatment of pain, the median time to maximum plasma bupivacaine levels was 5 min, and levels significantly dissipated by 60 min [45]. Given that it takes many hours for lidocaine and procainamide to get back to steady-state levels after starting an infusion (without a bolus) [43, 44], restarting these infusions approximately 1 h after SGB seems reasonable. Should LAST occur after SGB, rapid treatment with lipid emulsion therapy is critical, as described in detail by Neal et al. [41].

Outcomes of Stellate Ganglion Blockade for Ventricular Arrhythmias

Prior to 2019, the successful use of SGB for treatment of ventricular arrhythmia was confined to case reports and series. A comprehensive meta-analysis of 22 case reports/series published between 1974 and 2017 identified only 35 patients treated with SGB for VT/VF [7••]. Prior to SGB, all patients were on 1 antiarrhythmic medication, 23% underwent prior catheter ablation, and 23% were on inotropic or mechanical circulatory support (ventricular assist device, extracorporeal membrane oxygenation, or intra-aortic balloon pump). Left SGB was performed in 30 of these cases and bilateral SGB in 5 cases. Either unilateral or bilateral SGB reduced the number of VT/VF episodes and internal and/or external defibrillation events for 24 h after blockade. The reduction in VT/VF after SGB was independent of the etiology of ventricular arrhythmia, with comparable reduction of VT/VF episodes and defibrillation events seen with ischemic and non-ischemic cardiomyopathy. There was also no difference seen between the type of ventricular arrhythmia, with polymorphic and monomorphic VT having comparable reduction of VT/VF episodes and

defibrillation events after SGB. Furthermore, patients who underwent either left or bilateral SGB were shown to have improved survival. This is likely due to SGB affording time for other therapies to be used and to exert an effect, as 23% of patients underwent surgical sympathectomy, 23% catheter ablation, and 9% heart transplantation. No complications were reported in any of the 35 patients who received SGB.

In 2019, Tian et al. reported the use of SGB for 30 patients with refractory ventricular arrhythmias, with half receiving left SGB and the other half receiving bilateral SGB [13••]. Over 90% of the patients were on mechanical circulatory support, and all were on 2 antiarrhythmic drugs prior to SGB. There was an overall reduction in VT/VF episodes (mean \pm standard deviation pre 26 ± 41 to post 2 ± 4 , $p < 0.001$) for 72 h after SGB. Fifty percent of patients had complete arrhythmia suppression for 72 h after SGB. No differences in outcomes were seen between unilateral (left) versus bilateral SGB. There were no procedure-related major complications.

In 2020, Fudim et al. reported the use of bilateral SGB in 20 patients with refractory ventricular arrhythmias [12••]. These patients failed management with β -blockade, 2 antiarrhythmic medications, sedation, and some had also failed catheter ablation and mechanical circulatory support. Given that these patients were critically ill with few remaining therapeutic options, bilateral SGB was performed initially rather than unilateral SGB. SGB was associated with a reduction in arrhythmia episodes from the 24 h before (median 5.5 [interquartile range (IQR) 2.0 to 15.8]) to 24 h after SGB (median 0 [IQR 0 to 3.8]) ($p < 0.001$). The sum of defibrillation events also decreased from 2.5 (IQR 0 to 10.3) to 0 (IQR 0 to 2.5) ($p = 0.002$). The findings extended to 48 h. Similar to prior results, reduction in VT/VF burden was independent of type (monomorphic vs polymorphic) and etiology (ischemic vs non-ischemic cardiomyopathy) of ventricular arrhythmia. Nine patients (45%) had no recurrence of VT/VF for 48 h after bilateral SGB, and 4 patients (20%) had no recurrence for the rest of their hospitalization. Repeat bilateral SGB was performed in 25% of the patients. Thirteen patients (65%) were ultimately discharged from the hospital after one or a combination of catheter ablation, ventricular assist device implantation, heart transplantation, or surgical sympathectomy. The remaining seven patients (35%) ultimately died. Of note, seven patients (35%) were not intubated at the time of bilateral SGB. We mentioned previously that bilateral SGB confers an increased risk for acute respiratory distress secondary to potential bilateral RLN blockade. However, only one patient exhibited transient hoarseness. No other complications were reported from bilateral SGB.

Plasma levels of local anesthetic dissipate within hours after SGB [45], yet ventricular arrhythmia suppression after SGB appears to last for days. While reasons for this discrepancy are not understood, this is likely due to neural remodeling that occurs at the level of the stellate ganglion and the central nervous system after blockade, leading to prolonged reduction of sympathetic outflow [16•, 46]. The phenomenon of the clinical effect of SGB outlasting the expected pharmacological duration is also seen when utilized for treatment of pain, further supporting a central mechanism [24].

Notably, to date there is a lack of prospective randomized clinical data that compared SGB to a standard of care control group. A large multicenter randomized trial remains an important unmet need in order to reliably demonstrate the efficacy of SGB across multiple centers.

Thoracic Epidural Anesthesia: an Alternative to Stellate Ganglion Blockade?

Reduction of myocardial sympathetic tone may also be accomplished by thoracic epidural anesthesia (TEA). TEA involves continuous infusion of local anesthetic via a catheter placed in the thoracic epidural space, effectively blocking nociceptive and sympathetic fibers traveling through the spinal cord in this area. TEA is most commonly performed by anesthesiologists to treat post-operative pain; however, it has been successfully employed in a small number of cases for treatment of refractory ventricular arrhythmias [47]. The main advantage of TEA over SGB is the ability to do continuous, bilateral blockade of thoracic sympathetic fibers with just one catheter. TEA can also be performed by most anesthesiologists without image guidance, whereas SGB is most often performed by anesthesiologists with subspecialty training in pain medicine. However, TEA does have some disadvantages compared with SGB. To minimize the risk of an epidural hematoma which could lead to irreversible neurological injury, TEA is not recommended in the presence of anticoagulants like warfarin, DOACs, or antiplatelet agents such as clopidogrel [48]. Since many patients with refractory ventricular arrhythmias are on these medications, it makes TEA less attractive compared with SGB. Additionally, TEA is ideally performed in an awake patient so that the anesthesiologist can be alerted to inadvertent spinal cord or nerve trauma occurring during the procedure. Unfortunately, many patients with refractory ventricular arrhythmias are intubated and sedated, making this safety measure not possible. Furthermore, the failure rate of epidural catheters without imaging guidance is between 10 and 30% and assessing effectiveness in a sedated/intubated patient is challenging [49]. Interestingly, many patients who responded to TEA had previous reduction of arrhythmia burden with deep sedation. Thus, reduction of sympathetic tone via deep sedation may predict success with TEA and possibly SGB. Overall, more data on the efficacy and safety of TEA for treatment of refractory ventricular arrhythmias are needed.

Surgical Sympathectomy

Treatment of refractory ventricular arrhythmias with left or bilateral surgical sympathectomy has been previously reported [50–55]. The surgery is typically performed thoracoscopically and involves resection of multiple upper thoracic sympathetic ganglia. Due to concern that these patients will not tolerate general anesthesia and the single lung ventilation required to facilitate the operation, this is often reserved for patients with no other options. However, surgical sympathectomy appears to be well tolerated with low complication rates [50–55]. It is argued that bilateral surgical sympathectomy is more beneficial than unilateral [55]. While it may seem logical that successful reduction of ventricular arrhythmia burden with SGB would predict success with surgical sympathectomy, this has not been the case, particularly in recent studies [7••, 12••, 13••]. Patients with successful SGB do not always respond to

surgical sympathectomy and vice versa [7•• 12••, 13••]. This may suggest that the sympathetic tone is an amplifier rather than a direct cause of ventricular arrhythmias, with severe cases still requiring addressing the underlying structural or other cardiac disorder.

Future Directions

As ventricular arrhythmia suppression via SGB appears to last for several days, many of the patients in the aforementioned studies required repeat SGB until other therapies could be applied. To mitigate the risks of repeated SGB and attain continuous arrhythmia suppression, placement of a catheter near the stellate ganglion for continuous local anesthetic infusion has been reported [56–58]. Peripheral nerve catheters are routinely placed by anesthesiologists to treat post-operative pain, and the infection risk is low when left in place for 7 days or less [59]. Studies are needed to evaluate the routine placement of stellate ganglion catheters in lieu of single-shot blocks. One drawback of continuous administration of local anesthetic via stellate ganglion catheter is that concomitant intravenous infusion of lidocaine and/or procainamide may lead to local anesthetic toxicity, and thus, these therapies may have to be discontinued.

Other ways of attaining longer duration SGB include thermal or chemical neurolysis and neuromodulation. Hulata et al. report a case of a patient with refractory VT who had more than 2 weeks of arrhythmia suppression after 100% ethanol was injected near the left stellate ganglion [57]. Luke et al. report a case of a patient with refractory ventricular arrhythmias who received bilateral stellate ganglion injection of 6% phenol, with resulting arrhythmia suppression lasting 13 days [60]. There may be a role for other injectable therapies such as liposomal bupivacaine or other novel agents aimed at longer-term blockade without the permanent changes of chemical denaturants.

Radiofrequency ablation of the stellate ganglion has been shown in several studies to afford long-term analgesia for various chronic pain states [61–63]. However, only one case report exists showing long-term (> 12 months) suppression of refractory ventricular arrhythmias after left stellate ganglion radiofrequency ablation [64].

Markman et al. report the successful treatment of refractory VT in five patients who had transcutaneous magnetic stimulator (TCMS) treatments [65]. A figure 8 TCMS coil was implanted lateral to the C7 spinous process, delivering magnetic stimulation to the area of the left stellate ganglion. The stimulation presumably caused neuromodulation of the left stellate ganglion, reducing sympathetic outflow [65]. Of note, none of the five patients had implantable cardiac devices which could limit applicability of this technology [65]. A sham-controlled study is ongoing ([NCT04043312](https://clinicaltrials.gov/ct2/show/study/NCT04043312)).

While these methods of attaining longer-term ventricular arrhythmia suppression are encouraging, more studies are needed to show the efficacy and safety of stellate ganglion neurolysis, radiofrequency ablation, and neuromodulation via devices.

Conclusions

Recent studies have shown that either unilateral or bilateral SGB is associated with a reduction in VT/VF and defibrillation events for days, allowing time for other therapies to be applied. SGB effectiveness appears to be independent of the type (monomorphic vs polymorphic) or etiology of ventricular arrhythmia (ischemic vs non-ischemic cardiomyopathy). Most importantly, SGB appears to be safe with low risk for complications, even when performed on anticoagulation. Given these observations, where should SGB stand in the treatment paradigm for ventricular arrhythmias? At our center, we start evaluating patients for SGB early after antiarrhythmic medications and sedation have failed to control ventricular arrhythmias (Fig. 4). These evaluations are done with a multidisciplinary team of cardiologists, electrophysiologists, anesthesiologists, and cardiothoracic surgeons. Often, these patients progress to invasive treatments like catheter ablation or mechanical circulatory support, and if these fail, SGB is then performed. If SGB suppresses VT/VF, it is performed serially until other therapies can be performed (heart transplantation, surgical sympathectomy, or catheter ablation). Randomized controlled studies are now needed to establish clinical efficacy compared with control treatments and to determine optimal timing for SGB.

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References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
 - Of major importance
1. Pokorney SD, Mi X, Hammill BG, Allen LaPointe NM, Curtis LH, Al-Khatib SM. Use of antiarrhythmic medications in Medicare part D patients with an implantable cardioverterdefibrillator and ventricular tachycardia. *Am J Cardiol.* 2017;119(9):1401–6. [PubMed: 28341360]
 2. Zheng ZJ, Croft JB, Giles WH, Mensah GA. Sudden cardiac death in the United States, 1989 to 1998. *Circulation.* 2001;104(18): 2158–63. [PubMed: 11684624]
 3. Conti S, Pala S, Biagioli V, Del Giorno G, Zucchetti M, Russo E, et al. Electrical storm: a clinical and electrophysiological overview. *World J Cardiol.* 2015;7(9):555–61. [PubMed: 26413232]
 4. Poole JE, Johnson GW, Hellkamp AS, Anderson J, Callans DJ, Raitt MH, et al. Prognostic importance of defibrillator shocks in patients with heart failure. *N Engl J Med.* 2008;359(10):1009–17. [PubMed: 18768944]
 5. Nademanee K, Taylor R, Bailey WE, Rieders DE, Kosar EM. Treating electrical storm : sympathetic blockade versus advanced cardiac life support-guided therapy. *Circulation.* 2000;102(7): 742–7. [PubMed: 10942741]
 6. Waldron NH, Fudim M, Mathew JP, Piccini JP. Neuromodulation for the treatment of heart rhythm disorders. *JACC Basic Transl Sci.* 2019;4(4):546–62. [PubMed: 31468010]
 - 7••. Fudim M, Boortz-Marx R, Ganesh A, Waldron NH, Qadri YJ, Patel CB, et al. Stellate ganglion blockade for the treatment of refractory ventricular arrhythmias: a systematic review and meta-analysis. *J Cardiovasc Electrophysiol.* 2017;28(12):1460–7. [PubMed: 28833780] Annotation: Systematic review and meta analysis of published case reports of stellate ganglion blockade for ventricular arrhythmia.

8. Narouze S Ultrasound-guided stellate ganglion block: safety and efficacy. *Curr Pain Headache Rep.* 2014;18(6):424. [PubMed: 24760493]
9. Molnár I, Deák BZ, Hegyi G, Kovács Z, Kapócs G, Sz ke H. Effects of neural therapy on quality of life in patients with inoperable lower extremity artery disease. *Ideggyogyaszati szemle.* 2018;71(11–12):393–402. [PubMed: 30604938]
10. Heinig B, Koch A, Wollina U. Palmar hyperhidrosis treated by noninvasive ultrasound stellate ganglion block. *Wiener medizinische Wochenschrift (1946).* 2018;168(9–10):250–3. [PubMed: 27379849]
11. Lo JC, Nguyen D, Matthews TK. Usefulness of stellate ganglion block for refractory angina pectoris. *Proc (Baylor Univ Med Cent).* 2018;31(3):370–1.
- 12••. Fudim M, Qadri YJ, Waldron NH, Boortz-Marx RL, Ganesh A, Patel CB, et al. Stellate ganglion blockade for the treatment of refractory ventricular arrhythmias. *JACC Clin Electrophysiol.* 2020;6(5):562–71. [PubMed: 32439042] Largest single center case series of bilateral stellate ganglion blockade for ventricular arrhythmia.
- 13••. Tian Y, Wittwer ED, Kapa S, McLeod CJ, Xiao P, Noseworthy PA, et al. Effective use of percutaneous stellate ganglion blockade in patients with electrical storm. *Circ Arrhythm Electrophysiol.* 2019;12(9):e007118. [PubMed: 31514529] Largest single center case series of stellate ganglion blockade for ventricular arrhythmia.
14. Schwartz PJ, Snebold NG, Brown AM. Effects of unilateral cardiac sympathetic denervation on the ventricular fibrillation threshold. *Am J Cardiol.* 1976;37(7):1034–40. [PubMed: 1274864]
15. Schwartz PJ, Stone HL, Brown AM. Effects of unilateral stellate ganglion blockade on the arrhythmias associated with coronary occlusion. *Am Heart J.* 1976;92(5):589–99. [PubMed: 983934]
- 16•. Ajjjola OA, Yagishita D, Reddy NK, Yamakawa K, Vaseghi M, Downs AM, et al. Remodeling of stellate ganglion neurons after spatially targeted myocardial infarction: neuropeptide and morphologic changes. *Heart Rhythm.* 2015;12(5):1027–35. [PubMed: 25640636] Preclinical evidence to support the neuronal remodelling following cardiac insult.
17. Saddic LA, Howard-Quijano K, Kipke J, Kubo Y, Dale EA, Hoover DB, et al. Progression of myocardial ischemia leads to unique changes in immediate early gene expression in the spinal cord dorsal horn. *Am J Physiol Heart Circ Physiol.* 2018.
18. Gao C, Howard-Quijano K, Rau C, Takamiya T, Song Y, Shivkumar K, et al. Inflammatory and apoptotic remodeling in autonomic nervous system following myocardial infarction. *PLoS One.* 2017;12(5):e0177750. [PubMed: 28542617]
19. Aleanakian R, Chung BY, Feldmann RE Jr, Benrath J. Effectiveness, safety, and predictive potential in ultrasound-guided stellate ganglion blockades for the treatment of sympathetically maintained pain. *Pain practice : the official journal of World Institute of Pain.* 2020;20:626–38. [PubMed: 32255250]
20. Jeon Y Therapeutic potential of stellate ganglion block in orofacial pain: a mini review. *Journal of dental anesthesia and pain medicine.* 2016;16(3):159–63. [PubMed: 28884148]
21. Makharita MY, Amr YM, El-Bayoumy Y. Effect of early stellate ganglion blockade for facial pain from acute herpes zoster and incidence of postherpetic neuralgia. *Pain physician.* 2012;15(6): 467–74. [PubMed: 23159962]
22. Nabil Abbas D, Abd El Ghafar EM, Ibrahim WA, Omran AF. Fluoroscopic stellate ganglion block for postmastectomy pain: a comparison of the classic anterior approach and the oblique approach. *Clin J Pain.* 2011;27(3):207–13. [PubMed: 21178606]
23. Borchers AT, Gershwin ME. Complex regional pain syndrome: a comprehensive and critical review. *Autoimmun Rev.* 2014;13(3): 242–65. [PubMed: 24161450]
24. Lipov EG, Joshi JR, Sanders S, Slavin KV. A unifying theory linking the prolonged efficacy of the stellate ganglion block for the treatment of chronic regional pain syndrome (CRPS), hot flashes, and posttraumatic stress disorder (PTSD). *Med Hypotheses.* 2009;72(6):657–61. [PubMed: 19237252]
25. Lipov E, Lipov S, Stark JT. Stellate ganglion blockade provides relief from menopausal hot flashes: a case report series. *Journal of women's health (2002).* 2005;14(8):737–41.

26. Summers MR, Nevin RL. Stellate ganglion block in the treatment of post-traumatic stress disorder: a review of historical and recent literature. *Pain practice : the official journal of World Institute of Pain*. 2017;17(4):546–53. [PubMed: 27739175]
27. Gofeld M, Bhatia A, Abbas S, Ganapathy S, Johnson M. Development and validation of a new technique for ultrasound-guided stellate ganglion block. *Reg Anesth Pain Med*. 2009;34(5):475–9. [PubMed: 19920422]
28. Shibata Y, Fujiwara Y, Komatsu T. A new approach of ultrasound-guided stellate ganglion block. *Anesth Analg*. 2007;105(2):550–1. [PubMed: 17646541]
29. Elmofly DH, Eckmann M. Do not follow the bone, follow the nerve ultrasound-guided stellate ganglion block: a reconfirmation. *Br J Pain*. 2019;13(4):226–9. [PubMed: 31656628]
30. Hansen TG. Ropivacaine: a pharmacological review. *Expert Rev Neurother*. 2004;4(5):781–91. [PubMed: 15853505]
31. Cheng J, Salmasi V, You J, Grille M, Yang D, Mascha EJ, et al. Outcomes of sympathetic blocks in the management of complex regional pain syndrome: a retrospective cohort study. *Anesthesiology*. 2019;131(4):883–93. [PubMed: 31365367]
32. ahin ÖF, Tarıkçı Kılıç E, Aksoy Y, Kaydu A, Gökçek E. The importance of perfusion index monitoring in evaluating the efficacy of stellate ganglion blockage treatment in Raynaud’s disease. *The Libyan journal of medicine*. 2018;13(1):1422666. [PubMed: 29350104]
33. Yamazaki H, Nishiyama J, Suzuki T. Use of perfusion index from pulse oximetry to determine efficacy of stellate ganglion block. *Local and regional anesthesia*. 2012;5:9–14. [PubMed: 22915896]
34. Shiokawa Y, Morimoto Masahiro, Kamamoto Hiromichi, Kamamoto H, Uchida T, and Koga Y. Usefulness of perfusion index in evaluation of stellate ganglion block. *Acta Medica Kinki Univ* 2009;34(2):83–86.
35. Ginosar Y, Weiniger CF, Meroz Y, Kurz V, Bdoalah-Abram T, Babchenko A, et al. Pulse oximeter perfusion index as an early indicator of sympathectomy after epidural anesthesia. *Acta Anaesthesiol Scand*. 2009;53(8):1018–26. [PubMed: 19397502]
36. Goel V, Patwardhan AM, Ibrahim M, Howe CL, Schultz DM, Shankar H. Complications associated with stellate ganglion nerve block: a systematic review. *Reg Anesth Pain Med*. 2019;44:669–78. A systematic review of procedure related complications associated with stellate ganglion blockade.
37. Hardy PA, Wells JC. Extent of sympathetic blockade after stellate ganglion block with bupivacaine. *Pain*. 1989;36(2):193–6. [PubMed: 2919100]
38. Rathmell JP. *Atlas of image-guided intervention in regional anesthesia and pain medicine*: Lippincott Williams & Wilkins; 2011.
39. Al-Qurayshi Z, Kandil E, Randolph GW. Cost-effectiveness of intraoperative nerve monitoring in avoidance of bilateral recurrent laryngeal nerve injury in patients undergoing total thyroidectomy. *Br J Surg*. 2017;104(11):1523–31. [PubMed: 28707698]
40. Narouze S, Benzon HT, Provenzano D, Buvanendran A, De Andres J, Deer T, et al. Interventional spine and pain procedures in patients on antiplatelet and anticoagulant medications (second edition): guidelines from the American Society of Regional Anesthesia and Pain Medicine, the European Society of Regional Anaesthesia and Pain Therapy, the American Academy of Pain Medicine, the International Neuromodulation Society, the North American Neuromodulation Society, and the World Institute of Pain. *Regional anesthesia and pain medicine*. 2018;43(3):225–62.
41. Neal JM, Barrington MJ, Fettiplace MR, Gitman M, Memtsoudis SG, Mörwald EE, et al. The third American Society of Regional Anesthesia and Pain Medicine practice advisory on local anesthetic systemic toxicity: executive summary 2017. *Reg Anesth Pain Med*. 2018;43(2):113–23. [PubMed: 29356773]
42. Ha HR, Candinas R, Stieger B, Meyer UA, Follath F. Interaction between amiodarone and lidocaine. *J Cardiovasc Pharmacol*. 1996;28(4):533–9. [PubMed: 8891878]
43. Eipe N, Gupta S, Penning J. Intravenous lidocaine for acute pain: an evidence-based clinical update. *Bja Education*. 2016;16(9): 292–8.

44. Bigger JT Jr, and Giardina Elsa-Grace V. The pharmacology and clinical use of lidocaine and procainamide. *MCV/Q, Medical College of Virginia Quarterly* 1973;9(1):65–76.
45. Wulf H, Maier C, Schele HA, Wabbel W. Plasma concentration of bupivacaine after stellate ganglion blockade. *Anesth Analg.* 1991;72(4):546–8. [PubMed: 2006745]
46. Ajijola OA, Yagishita D, Patel KJ, Vaseghi M, Zhou W, Yamakawa K, et al. Focal myocardial infarction induces global remodeling of cardiac sympathetic innervation: neural remodeling in a spatial context. *Am J Physiol Heart Circ Physiol.* 2013;305(7):H1031–40. [PubMed: 23893167]
47. Do DH, Bradfield J, Ajijola OA, Vaseghi M, Le J, Rahman S, et al. Thoracic epidural anesthesia can be effective for the short-term management of ventricular tachycardia storm. *Journal of the American Heart Association.* 2017;6(11).
48. Horlocker TT, Vandermeulen E, Kopp SL, Gogarten W, Leffert LR, Benzon HT. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine evidence-based guidelines (fourth edition). *Reg Anesth Pain Med.* 2018;43(3):263–309. [PubMed: 29561531]
49. Hermanides J, Hollmann MW, Stevens MF, Lirk P. Failed epidural: causes and management. *Br J Anaesth.* 2012;109(2):144–54. [PubMed: 22735301]
50. Ajijola OA, Lellouche N, Bourke T, Tung R, Ahn S, Mahajan A, et al. Bilateral cardiac sympathetic denervation for the management of electrical storm. *J Am Coll Cardiol.* 2012;59(1):91–2. [PubMed: 22192676]
51. Coleman MA, Bos JM, Johnson JN, Owen HJ, Deschamps C, Moir C, et al. Videoscopic left cardiac sympathetic denervation for patients with recurrent ventricular fibrillation/malignant ventricular arrhythmia syndromes besides congenital long-QT syndrome. *Circ Arrhythm Electrophysiol.* 2012;5(4):782–8. [PubMed: 22787014]
52. Hofferberth SC, Cecchin F, Loberman D, Fynn-Thompson F. Left thoracoscopic sympathectomy for cardiac denervation in patients with life-threatening ventricular arrhythmias. *J Thorac Cardiovasc Surg.* 2014;147(1):404–9. [PubMed: 24268954]
53. Téllez LJ, Garzón JC, Vinck EE, Castellanos JD. Video-assisted thoracoscopic cardiac denervation of refractory ventricular arrhythmias and electrical storms: a single-center series. *J Cardiothorac Surg.* 2019;14(1):17. [PubMed: 30665431]
54. Vaseghi M, Barwad P, Malavassi Corrales FJ, Tandri H, Mathuria N, Shah R, et al. Cardiac sympathetic denervation for refractory ventricular arrhythmias. *J Am Coll Cardiol.* 2017;69(25):3070–80. [PubMed: 28641796]
55. Vaseghi M, Gima J, Kanaan C, Ajijola OA, Marmureanu A, Mahajan A, et al. Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm: intermediate and long-term follow-up. *Heart Rhythm.* 2014;11(3):360–6. [PubMed: 24291775]
56. Franklin AD, Llobet JR, Sobey CM, Daniels JM, Kannankeril PJ. Stellate ganglion catheter effective for treatment of ventricular tachycardia storm in a pediatric patient on extracorporeal membrane oxygenation: a case report. *A&A practice.* 2019;13(7):245–9. [PubMed: 31162228]
57. Hulata DF, Le-Wendling L, Boezaart AP, Hurley RW. Stellate ganglion local anesthetic blockade and neurolysis for the treatment of refractory ventricular fibrillation. *A & A case reports.* 2015;4(5):49–51. [PubMed: 25730409]
58. Scalercio L, Vitter J, Elliott CE. Placement of a continuous stellate ganglion block for treatment of refractory ventricular fibrillation in the setting of known Prinzmetal angina during pregnancy: a case report. *A&A practice.* 2019;12(4):106–8. [PubMed: 30102609]
59. Bomberg H, Bayer I, Wagenpfeil S, Kessler P, Wulf H, Standl T, et al. Prolonged catheter use and infection in regional anesthesia: a retrospective registry analysis. *Anesthesiology.* 2018;128(4):764–73. [PubMed: 29420315]
60. Luke WR, Daoud EG, Latif OS. Emergent phenol injection of bilateral stellate ganglion for management of refractory malignant ventricular arrhythmias. *Am J Case Rep.* 2020;21:e921465. [PubMed: 32188839]
61. Abbas DN, Reyad RM. Thermal versus super voltage pulsed radiofrequency of stellate ganglion in post-mastectomy neuropathic pain syndrome: a prospective randomized trial. *Pain physician.* 2018;21(4):351–62. [PubMed: 30045592]

62. Forouzanfar T, van Kleef M, Weber WE. Radiofrequency lesions of the stellate ganglion in chronic pain syndromes: retrospective analysis of clinical efficacy in 86 patients. *Clin J Pain*. 2000;16(2): 164–8. [PubMed: 10870729]
63. Kastler A, Aubry S, Saille N, Michalakis D, Siliman G, Gory G, et al. CT-guided stellate ganglion blockade vs. radiofrequency neurolysis in the management of refractory type I complex regional pain syndrome of the upper limb. *Eur Radiol*. 2013;23(5):1316–22. [PubMed: 23138389]
64. Hayase J, Vampola S, Ahadian F, Narayan SM, Krummen DE. Comparative efficacy of stellate ganglion block with bupivacaine vs pulsed radiofrequency in a patient with refractory ventricular arrhythmias. *J Clin Anesth*. 2016;31:162–5. [PubMed: 27185701]
65. Markman TM, Hamilton RH, Marchlinski FE, Nazarian S. Case series of transcutaneous magnetic stimulation for ventricular tachycardia storm. *Jama*. 2020;323(21):2200–2. [PubMed: 32372071]

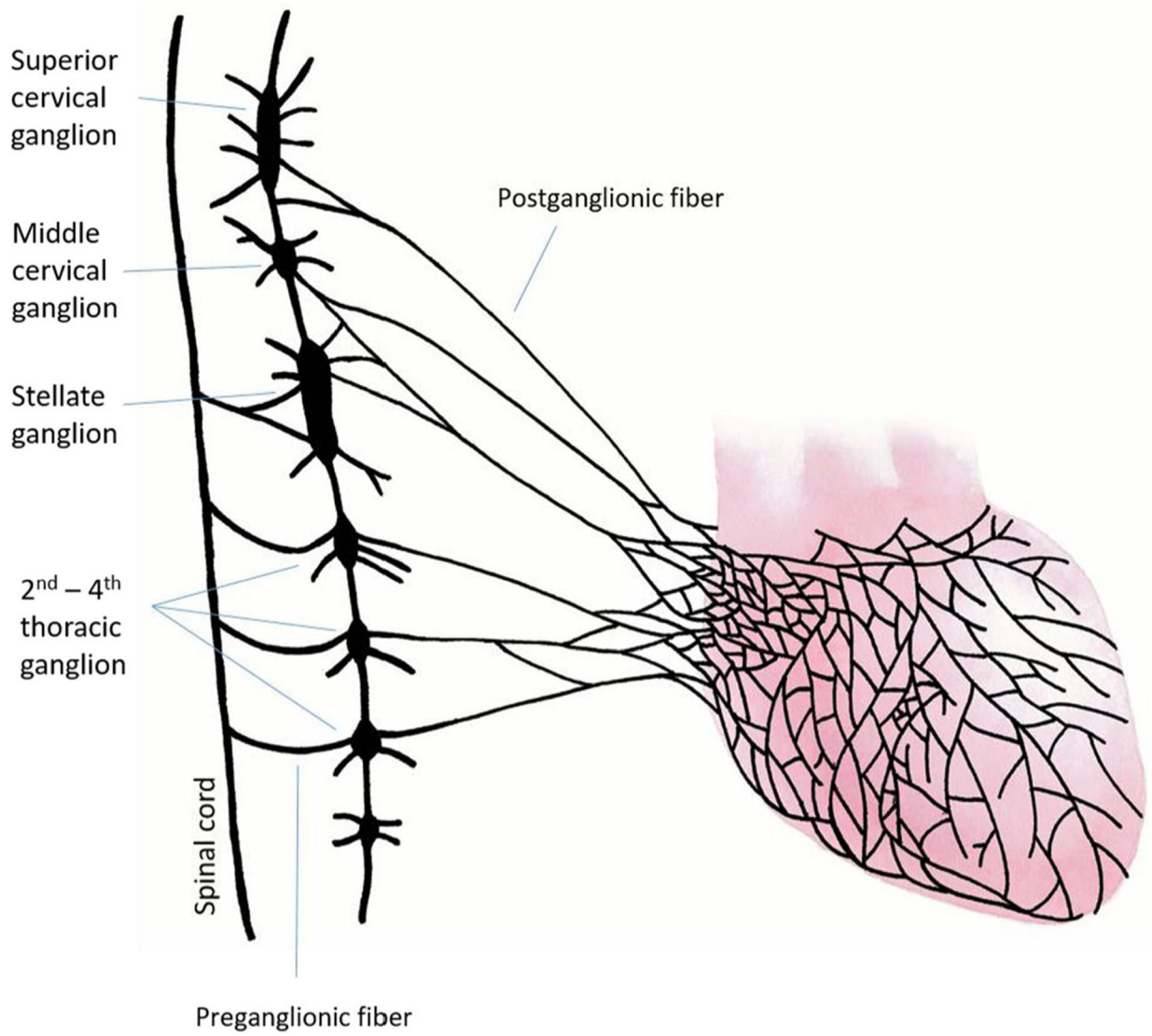


Fig. 1.
Sympathetic innervation to the heart

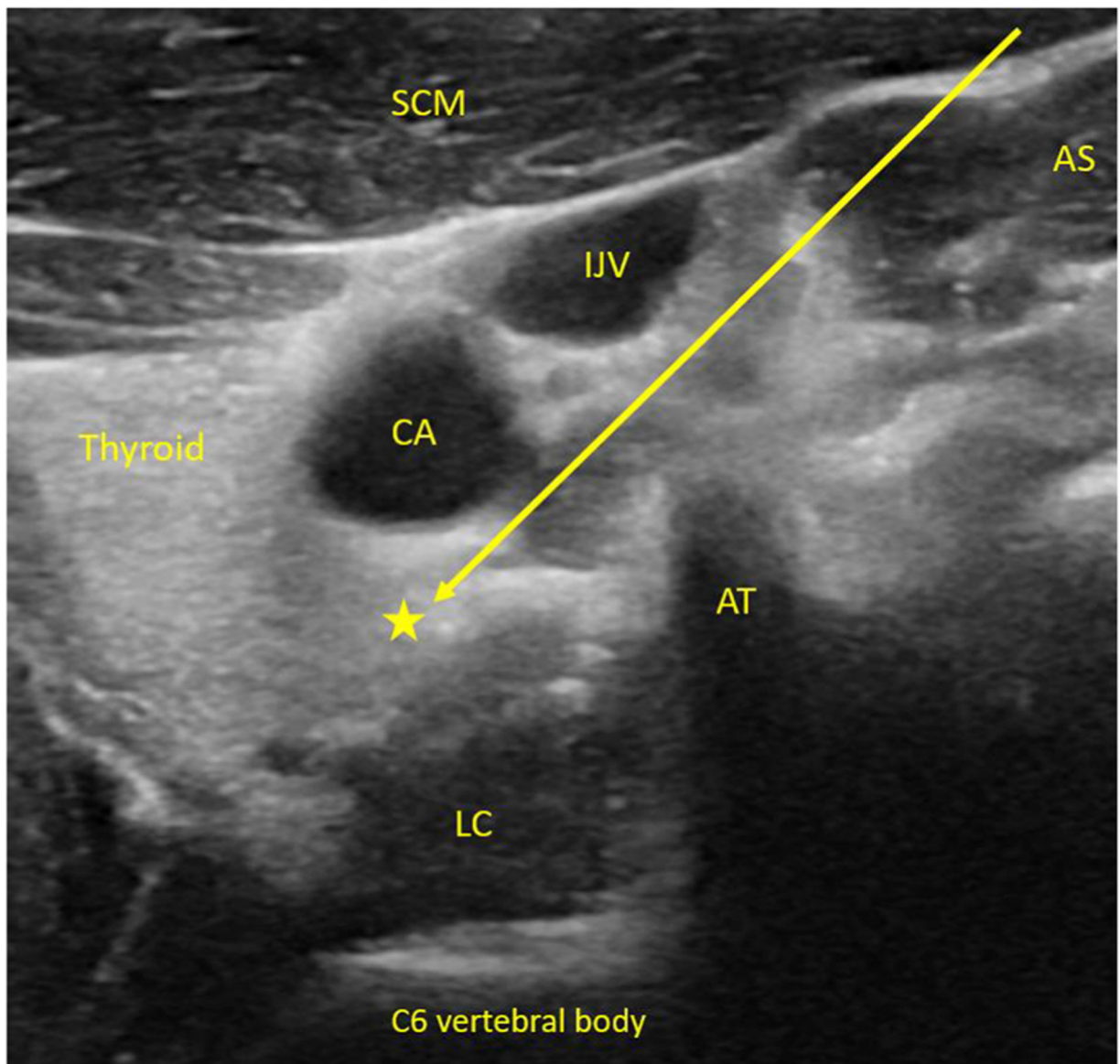


Fig. 2.

Ultrasound-guided left stellate ganglion block. The yellow line indicates the needle trajectory, and the star is the site of injection. The stellate ganglion resides between the carotid artery and the longus colli muscle at the C7/T1 level. The block is often performed at the C6 level to avoid the vertebral artery. The recurrent laryngeal nerve is not denoted here but is posterior to the thyroid gland, hence why this can be inadvertently blocked when targeting the stellate ganglion. SCM sternocleidomastoid muscle, CA Carotid artery, IJV Internal jugular vein, AS anterior scalene muscle, LC Longus colli muscle, AT Anterior tubercle of C6 vertebrae (Chassaignac's tubercle)

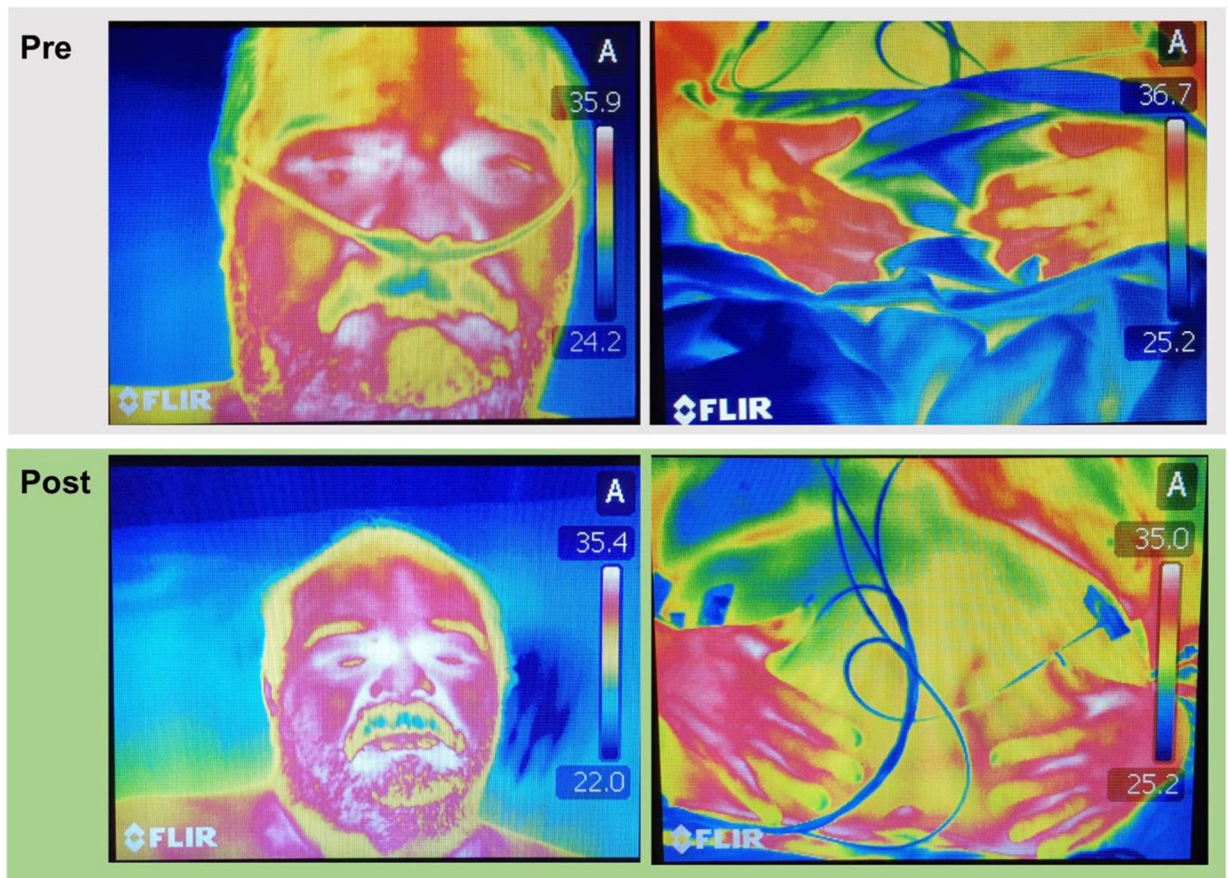


Fig. 3. Heat profile of patient before and 10 min after stellate ganglion blockade. From pre to post, there is an increase in the temperature on the forehead and hands, which is used as a surrogate for successful stellate ganglion blockade

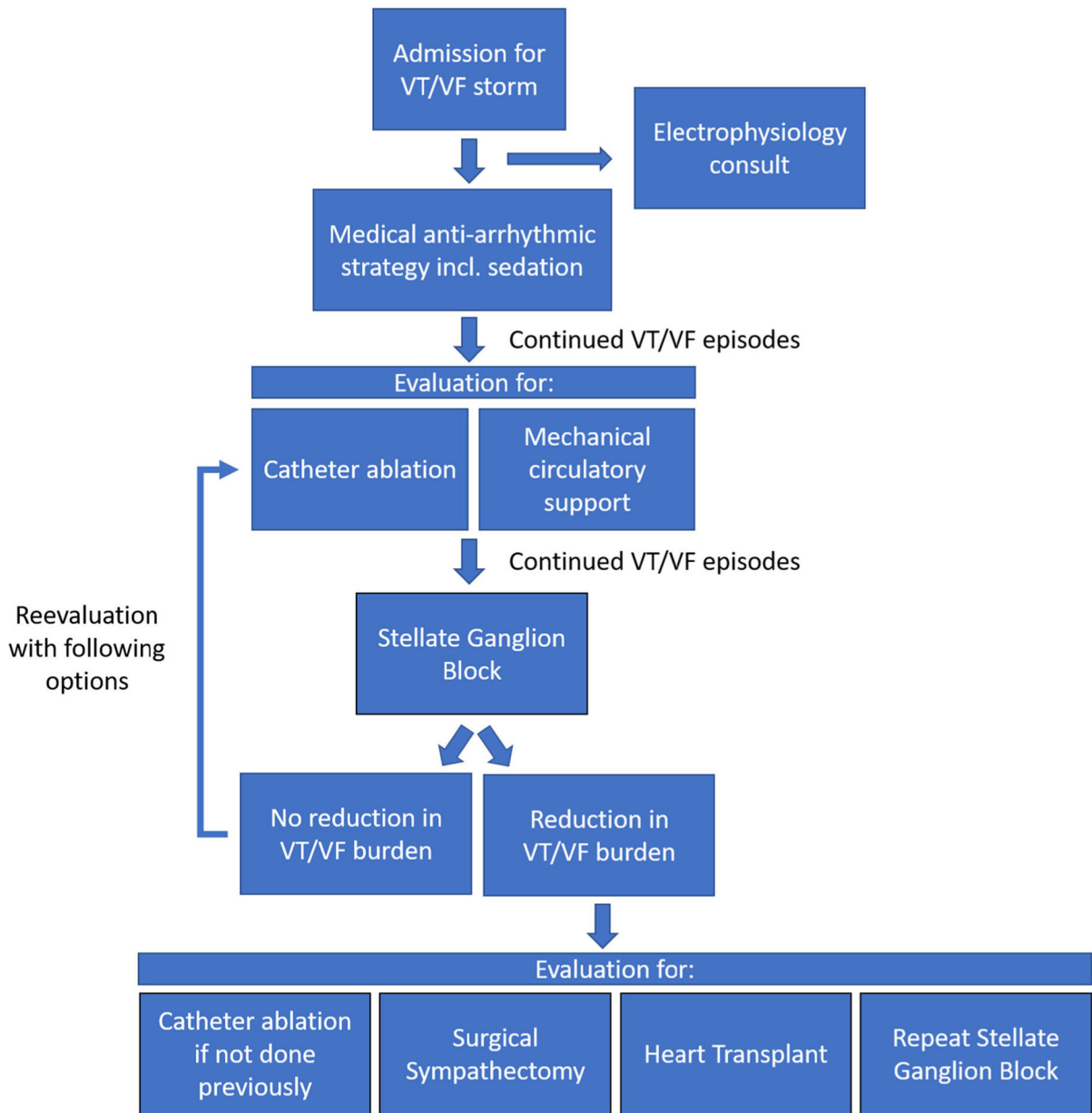


Fig. 4. Duke treatment algorithm for refractory ventricular arrhythmias. Figure modified from Fudim et al. (2020) [12••]. VT ventricular tachycardia, VF ventricular fibrillation

Table 1

Indications for stellate ganglion block. Complex regional pain syndrome (CRPS) is the most common indication. PTSD post-traumatic stress disorder

Pain	Cardiovascular	Other
CRPS	Ventricular arrhythmias	PTSD
Orofacial pain	Raynaud's disease	Hot flashes
Post-herpetic neuralgia	Refractory angina	
Post-mastectomy pain	Hyperhidrosis	

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