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Conversion of sustained supraventricular tachycardia into sinus rhythm after stellate ganglion block: A case report and pathophysiological considerations

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Abstract

Considering the anatomy, physiology, and pathophysiology of the autonomic nervous system of the heart, therapy by means of stellate ganglion block may be a logical additional treatment option in certain cases of heart rhythm disorders. A woman in her late 60s presented with sustained paroxysmal supraventricular tachycardia and a heart rate of 170 beats per minute that had persisted for 2 h despite performing vagal maneuvers and orally administering 40 mg of verapamil. She had a history of several episodes of paroxysmal supraventricular tachycardia with spontaneous conversion. We performed a stellate ganglion block with 3 mL of 1% procaine. One minute later, the paroxysmal supraventricular tachycardia converted into a normal sinus rhythm. Since then (i.e. since 5 years), the patient has not experienced any further episodes of tachycardia. In addition to various other pathomechanisms of paroxysmal supraventricular tachycardia, neuroplastic and neurogenic inflammatory processes play a role, in which the sympathetic nervous system contributes to pathological positive feedback loops. Stellate ganglion block interrupts these feedback loops, allowing the autonomic nervous system to reorganize and achieve a better balance. This could exert a favorable effect on recurrences of heart rhythm disorders ("learning effect"). Stellate ganglion block via local anesthetics is a simple, safe, and virtually side effect-free procedure to terminate treatment-refractory paroxysmal supraventricular tachycardia. Further studies are needed to confirm these results.

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Introduction

Definition

Paroxysmal supraventricular tachycardia (PSVT) is a subtype of tachycardia with a narrow QRS complex, defined as intermittent supraventricular tachycardia (SVT) with an abrupt onset and termination as well as a regular ventricular response.[1]

Epidemiology

The available evidence on the epidemiology of PSVT is vague; however, it suggests that the prevalence of SVT is approximately 1–10/1000, with women being more frequently affected.[1]

Etiology and risk factors

PSVT can occur either idiopathically or in association with numerous underlying cardiac or extra-cardiac factors (e.g. rheumatic diseases or hyperthyroidism, stress, and various medications).[1]

Symptoms

The symptoms of PSVT usually include palpitations, thoracic pressure, shortness of breath, dizziness, and sweating.

Diagnosis

An electrocardiogram (ECG) recorded during an attack shows a regular rhythm with a fast heart rate (150–250 beats per minute (bpm)) and a narrow QRS complex, with the P wave normally hidden in the QRS complex.[1] Depending on the situation, laboratory and electrophysiological examinations may be necessary.

Treatment options

The initial approach for the acute treatment of SVT is usually vagal maneuvers and administration of oral or intravenous medications (e.g. verapamil); electrical cardioversion is considered if there is no response.[1]

Additional treatment option

Local anesthetics (LAs) are gaining attention for their role in managing cardiac arrhythmias. Regarding stellate ganglion block (SGB) performed under LA, the literature has shown positive effects of SGB on sinus tachycardia,[2] unusual types of atrial tachycardia,[3] and ventricular tachycardia.[4,5] A large, prospective, multicenter study[6] demonstrated the potential positive effect of SGB in cardiac arrhythmias, although the focus of the study was therapy-resistant electrical storm. We aimed to contribute a novel case report to the growing body of evidence on the therapeutic potential of LAs in cardiac arrhythmias, with a focus on SGB.

Case report

Medical history and findings

A woman in her late 60s presented to the emergency department with persistent tachycardia that had started suddenly 2 h ago without any apparent trigger. She reported a feeling of thoracic pressure, nausea, anxiety, and dizziness. She had

already undergone several vagal maneuvers and taken 40 mg of verapamil, both without any effect. Apart from arterial hypertension that was managed with medication (25 mg hydrochlorothiazide and 2.5 mg amiloride daily), the patient was in good health, and no other medication was ongoing. Till that time, there had been three short PSVT episodes with immediate conversion to vagal maneuvers and/or verapamil administration by mouth. Apart from five thyroid surgeries (the last one performed at the age of 46 years) and radioiodine therapy against the background of Graves' disease, the patient's medical history was unremarkable. At the time of presentation, the patient was euthyroid. Her blood pressure level was 150/100 mmHg, and her heart rate was 170 bpm. Physical examination revealed normal cardiopulmonary auscultation. ECG showed regular tachycardia with a narrow QRS complex, heart rate of 170 bpm, and absent P waves. The clinical picture and ECG results were consistent with the features of sustained PSVT. Electrophysiological examinations were not performed because the patient requested a wait-and-see approach.

Intervention and future course

Initially, vagal maneuvers were performed again; however, these could not terminate the PSVT. As a therapeutic LA (neural therapy), we performed a right-sided SGB with 3 mL of 1% procaine using the minimally invasive technique described previously.[7,8] The patient immediately exhibited Horner's syndrome, confirming the correct placement of the injection. One minute after performing SGB, the tachycardia converted into a sinus rhythm (verified via ECG), and the heart rate normalized to 90 bpm. The patient has not experienced any further episodes of tachycardia to date (the observation period was more than 5 years).

Undesirable effects

Apart from a minimal feeling of dizziness for a few minutes, no undesirable effects were observed.

The reporting of this case conforms to the Case Report (CARE) guidelines.[9] Informed consent for treatment and publication was obtained from the patient. All patient details were de-identified.

Discussion

Anatomy and physiology of the cardiac autonomic nervous system (ANS)

The cardiac ANS comprises sympathetic and parasympathetic (vagal) nerve fibers, which form a dense network in the heart known as the cardiac plexus. The cardiac plexus helps modulate heart rate and myocardial inotropy and influences stimulus conduction in the atrioventricular node, blood pressure, and coronary blood flow. Information regarding the cardiac plexus is largely derived from the stellate ganglion (SG) and vagus nerve. The SG is a sympathetic ganglion that usually results from the fusion of the inferior cervical ganglion and the first thoracic ganglion. The SG innervates the entire equilateral upper body, including the heart, lungs, brain, skin, and musculoskeletal system (Figure 1).[7,8,10] The SGB influences not only pre- and postganglionic sympathetic fibers but also parasympathetic (vagal) and afferent nerve fibers, as described previously.[7,11]

Pathophysiological considerations and possible mechanisms of action of SGB

The decisive factor in the mechanisms of action of SGB is the restoration of a physiological balance between the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS), especially the vagus nerve. This allows the SNS or PNS to

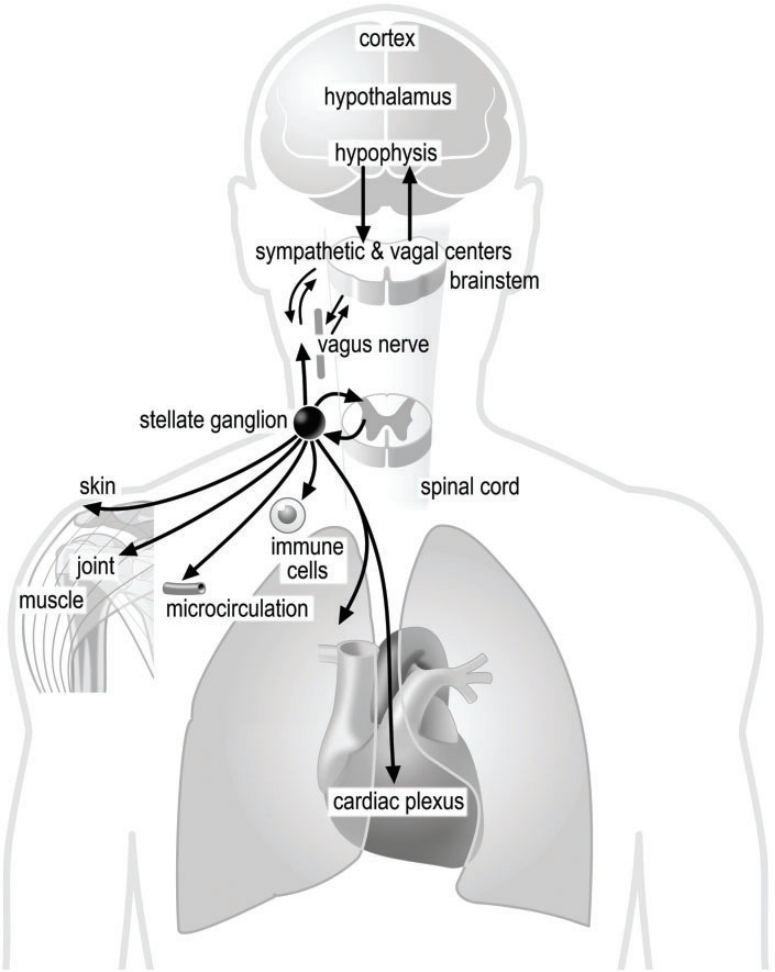

Figure 1. Simplified illustration of the anatomy and physiology of the stellate ganglion. Its efferent fibers supply the ipsilateral upper body quarter, including internal organs, skin, musculoskeletal system, blood vessels, and immune cells, via which the sympathetic nervous system can influence inflammatory cascades. Together with afferent fibers (in favor of a better overview not visualized in the figure), positive and negative feedback loops are formed. These can be interrupted by means of stellate ganglion block. Subsequently, the autonomic nervous system can reorganize itself to achieve a better balance.

temporarily dominate as required. A persistent imbalance of the ANS can lead to pain, inflammation, and dysfunction of internal organs, including rhythm disorders of the heart.[7,8,10,11] LAs, especially when used in SGB, play an increasingly important role in therapies that influence the ANS.[7,8,10,11] Various processes described below occur simultaneously after performing SGB. The extent to which these processes, as hypothesized by us, can play a role in the pathogenesis of PSVT and its resolution by SGB needs to be validated in larger studies.

Elimination of the imbalance between SNS and PNS. Pathologically and chronically increased sympathetic activity can lead to various cardiac rhythm disorders.[7,12]

The "reset" induced by SGB can improve the imbalance between the SNS and PNS (vagus nerve).[11,13]

Desensitization may exert a beneficial influence on neuroplastic changes in the SNS. Under pathological sensitization, neuroplastic changes can occur, which include the formation of abnormal neurological short circuits (e.g. so called "sympathetic–afferent coupling"[14] and "sympathetic sprouting").[14,15] Another sensitization process leading to neuroplastic changes is the so called synaptic long-term potentiation.[16,17] Repeated pathological stimulation can potentiate the postsynaptic response in sympathetic ganglia.[16] Under these circumstances, even physiological stimuli can provoke an overshooting response.[11] In the SNS of the heart, we hypothesized that these neuroplastic changes can lead to hyperresponsiveness and thus arrhythmias.[7] These peripheral processes constitute positive feedback loops in a vicious circle.[7,11,18,19] These processes are perceived in the brain and amplified again.[11] SGB and LA in general[20] exert a positive effect on these sensitization and neuroplastic processes and can temporarily interrupt the feedback loops as a "reset," as described in a recent study.[11] Subsequently, the ANS can reorganize itself.[11,18,19,21–23] The SGB not only acts peripherally but also influences the central nervous system; therefore, we assume a positive long-term effect on arrhythmias as a learning process. This might explain why our patient has never experienced another episode of PSVT after SGB.

Influence of sympathetically maintained inflammation and neuroimmune communication on cardiac arrhythmias. The SNS and vagus nerve play important roles in neuroimmune communication and thus in the inflammatory process.[11,12,14,24] The basic pathomechanisms activate the same inflammatory cascades, regardless of whether it is a viral infection or autoimmune disease.[11] Thus, these inflammations can be regulated by means of SGB.[11,12]

Therefore, SGB could also be a treatment option for inflammatory heart conditions.[7,12] We recommend early treatment to prevent further tissue damage[11] with subsequent scarring changes, which can further cause arrhythmias. The SGB influences neuroimmune communication, both peripherally and centrally.[11] The central influence is observed in the sympathetic and vagal centers in the brain stem as well as in the cerebral cortex.[11]

Improvement of cardiac microvascular dysfunction. Overactivity of the SNS can cause microvascular dysfunction with myocardial ischemia, which can promote cardiac arrhythmias.[25,26] SGB also has a beneficial effect on microcirculation.[7,11]

Properties of procaine

In contrast to amide-structured LAs, procaine is not metabolized in the liver but is metabolized by the ubiquitous pseudocholinesterase,[8,23] indicating that there are practically no interactions with other medications. In addition, procaine (in contrast to amide-structured LAs) promotes microcirculation.[8] This can be beneficial for the SG itself because improved microcirculation may counteract its sensitization processes.[11]

Side-specific differences between right-side and left-side SGB

The side of injection does not play a significant role in the effectiveness of SGB in various heart diseases, as demonstrated in an anatomical–physiological study,[7] although some studies have concluded that the fibers of the right-sided SG are more involved in heart rhythm regulation, and those of the left-sided SG are more involved

in the action of coronary vessels and the myocardium. However, as the overlaps between the fibers of the right and left stellate ganglion within the plexus cardiacus are extremely large, the side of SG injection remains insignificant. Injections can also be administered bilaterally; however, we recommend performing SGB on the other side only after the Horner symptom complex of the first injection has subsided.[7,11]

Safety

With small amounts of short-acting procaine, SGB is considered a safe intervention, which was also apparent from the echocardiographic and hemodynamic evaluations.[7]

Limitations

There is an inherent limitation regarding the possibility of spontaneous resolution, particularly in relapsing–remitting diseases, as in our case report. However, the increasing number of studies with numerous patients showing the importance of SGB in various cardiac arrhythmias highlights the potential of this method.

Conclusion

The SGB, which enabled a "reset" of the ANS and subsequent self-organization of the system, may become a widely applied procedure in the future for various health conditions. We hypothesize that self-organization leads to an altered state of the ANS toward homeostasis, with effects far beyond the period of action of LAs. Thus, SGB has a regulatory effect. This also explains why there were negligible side effects. Further studies are needed on this topic to confirm these findings.

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Author contributions

C.C. performed the literature search and prepared the first draft of the manuscript. C.C. and L.F. interpreted the possible pathophysiology and mechanisms of action. L.F. treated the patient. Both authors have read and approved the final manuscript.

Data availability statement

The data are available from the corresponding author (L.F.).

Declaration of conflicting interests

Both authors declare that there are no conflicts of interest.

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