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The Treatment of Paroxysmal Supraventricular Tachycardia and Research Progress

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Abstract

Paroxysmal supraventricular tachycardia (PSVT) is a common rapid arrhythmia observed in clinical settings, characterized by abrupt and sustained episodes lasting minutes to hours. It presents significant risks, including heart failure, syncope, shock, and sudden death, profoundly affecting patients' quality of life. This review explores the epidemiology, pathophysiology, clinical manifestations, and diagnostic approaches of PSVT, emphasizing treatment modalities such as physical therapy, pharmacotherapy, interventional procedures. Additionally, it addresses current obstacles and future prospects in PSVT management to guide clinical practice and research initiatives.

Keywords

Paroxysmal Supraventricular Tachycardia, Pathophysiological Mechanism, Clinical Diagnosis, Drug Treatment, Interventional Treatment

1. Introduction

Paroxysmal supraventricular tachycardia (PSVT) refers to a rapid heart rate originating from the atrium or atrioventricular junction, primarily caused by reentry mechanisms (60% - 80%) or increased myocardial autonomy. PSVT manifests as abrupt onset tachycardia with heart rates typically ranging from 160 to 220 beats per minute [1]. While not inherently life-threatening, recurrent episodes can significantly impact patients' quality of life and potentially lead to tachycardia-induced cardiomyopathy. The prevalence of PSVT is rising with the aging population and improved diagnostic technologies. Recent advancements, notably in catheter ablation, offer a promising curative approach for most patients. However, managing certain PSVT presents challenges, and addressing the long-term efficacy and side effects of pharmacological interventions remains crucial. This re-

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view aims to delineate the current therapeutic landscape and research progress in PSVT to inform clinical decision-making.

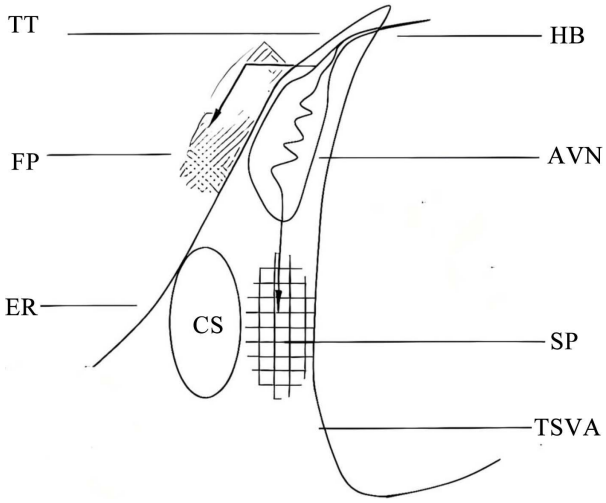
2. Epidemiological Characteristics of Paroxysmal Supraventricular Tachycardia

PSVT is a prevalent clinical arrhythmia characterized by abrupt onset, originating in or propagating through the atria or atrioventricular node. Its prevalence in the general population ranges from 168 to 332 cases per 100,000 individuals, with a higher occurrence observed with advancing age [2]. Epidemiological data indicates a slightly higher incidence rate among women compared to men, with a notable familial aggregation tendency. Notably, approximately half of PSVT patients fall within the 45 to 64 age bracket, with females constituting 67.5% of this subgroup [2] [3]. Recent and growing evidence suggests that AVNRT is associated with familial clustering and genetic factors. Key calcium signaling pathway components, including CASQ2, RYR2, TRDN, NOS1, ANK2, and ATP2C2, may be closely linked to the pathogenesis of AVNRT [4].

3. Pathophysiological Mechanism of Paroxysmal Supraventricular Tachycardia

Paroxysmal supraventricular tachycardia (PSVT) denotes rapid heart rate originating from the atrium or atrioventricular junction, characterized by sudden onset with heart rates typically ranging from 160 to 220 beats per minute. The underlying pathophysiology primarily involves reentry mechanisms, occasionally augmented automaticity, or triggering events. PSVT can be categorized into three subtypes based on the mechanism of initiation: atrioventricular nodal reciprocating tachycardia (AVNRT), atrioventricular reciprocating tachycardia (AVRT), and focal atrial tachycardia (AT). AVNRT constitutes approximately 56%, AVRT about 27%, and AT around 17% of cases [5].

The atrioventricular node is situated deep within the endocardial surface of the Koch triangle (**Figure 1**), enveloped by a thin layer of atrial muscle on its right side. Within the atrioventricular node, there exist distinct fast and slow conduction pathways. Normally, atrial depolarization propagates swiftly down the fast pathway to the ventricles. However, in atrioventricular nodal reentrant tachycardia (AVNRT), during atrial systole when depolarization descends, the fast pathway is in a refractory state, impeding conduction, thereby necessitating propagation solely through the slow pathway. As the slow pathway conducts at a slower pace, by the time depolarization reaches the lower region of the atrioventricular node, the fast pathway has recovered from its refractory state, enabling retrograde conduction back to the atrium via the fast pathway, followed by anterograde conduction through the slow pathway. This reciprocal conduction pattern perpetuates to sustain tachycardia. AVNRT is further categorized into three subtypes based on the interplay of retrograde and anterograde conduction pathways: slow-fast type (most prevalent), slow-slow type, and fast-slow type [6] [7].



Abbreviations: TT: Tendon of Todaro; HB: His Bundle; AVN: Atrioventricular node; FP: Fast Pathway; SP: Slow Pathway; ER: Eustachian Ridge; CS: Coronary Sinus; TSVA: Tricuspid Septal Valve Annulus.

Figure 1. Triangle of Koch.

AVRT is caused by an atrioventricular bypass known as the Kent bundle connecting the atrium and the ventricle. Under normal circumstances, atrial impulses are conducted to the ventricles through the atrioventricular node, while the atrioventricular bypass tract itself is generally in the refractory period and does not participate in conduction. The atrioventricular bypass can be categorized based on conduction direction into dominant, concealed, and concealed with decremental conduction. In cases where pre-atrial contractions or other factors induce unidirectional block in the atrioventricular node or bypass tract, atrial impulses can bypass the block, reach the ventricle through an unblocked route, and then retrogradely activate the atrium, creating a reentry circuit. When conduction occurs through the bypass tract, some ventricular muscle fibers depolarize prematurely, resulting in delta waves on the electrocardiogram, a characteristic of pre-excitation syndrome, also known as Wolff-Parkinson-White syndrome (WPW syndrome) [6] [8]. These abnormal electrophysiological properties disrupt normal conduction, leading to reentrant circuits and rapid, regular ventricular activation.

AT originates from a focal point in the atria or structures connected to the atria, without involving the atrioventricular node. The episodes are characterized by paroxysmal or incessant onset, spreading centrifugally across the atria like ripples. The atrial rhythm is relatively regular with a rate ranging from 100 to 250 beats per minute. The underlying mechanisms include three types: micro-reentry, enhanced automaticity, and triggered activity [9].

4. Clinical Manifestations and Diagnosis of Paroxysmal Supraventricular Tachycardia

Paroxysmal supraventricular tachycardia (PSVT) typically presents with abrupt

palpitations, often accompanied by symptoms such as dyspnea, chest pain, dizziness, and fatigue. Severe cases may involve fainting or angina pectoris. Palpitations are the most prevalent symptom among PSVT patients (84%), followed by chest pain (47%), dyspnea (38%), syncope (26%), dizziness (19%), and excessive sweating (18%). Notably, 45% of patients experience abnormal and frequent polyuria (≥2 times) within 1 - 3 hours after the initial PSVT episode [10]. During an attack, heart rate usually ranges from 160-220 beats per minute, with a regular rhythm, potential blood pressure drop, and attack duration varying from minutes to hours. Termination may occur spontaneously or necessitate medical intervention.

The primary diagnostic method for paroxysmal supraventricular tachycardia (PSVT) is electrocardiography, which typically shows narrow QRS complex tachycardia during episodes. The heart rate ranges from 160 to 220 beats/min, displaying a regular rhythm with possible fusion of P waves or QRS complexes [11]. For patients experiencing infrequent episodes, ambulatory ECG monitoring or wearable devices can aid in capturing episodes [12]. Electrophysiological study serves as the gold standard for confirming the type of PSVT, elucidating the tachycardia mechanism and critical sites through ventricular and atrial programmed stimulation, atrial incremental pacing or burst pacing. This examination offers precise guidance for catheter ablation therapy. Transesophageal electrophysiological study has emerged as a novel non-invasive diagnostic and therapeutic modality in recent years, featuring advantages such as non-invasiveness, simplicity, safety, and efficacy. It encompasses transesophageal atrial and ventricular pacing techniques [4] [13].

5. Treatment Methods for Paroxysmal Supraventricular Tachycardia

The treatment of PSVT can be divided into acute attack treatment and interventional treatment, as follows:

5.1. Acute Onset Treatment

The acute phase management of paroxysmal supraventricular tachycardia (PSVT) focuses on promptly terminating the arrhythmia. Clinical intervention is guided by factors such as hemodynamic stability, medical history, and the underlying mechanism of the arrhythmia. Treatment typically progresses in a systematic manner, starting with non-invasive vagus nerve stimulation and advancing to pharmacological interventions. In cases where patients present with hemodynamic instability, synchronized electrical cardioversion may be necessary [14].

5.1.1. Vaginal Nerve Stimulation Treatment

Vagus nerve stimulation is the initial treatment of choice for hemodynamically stable patients due to its simplicity, practicality, and ease of implementation. Successful vagus nerve stimulation techniques have shown efficacy rates ranging from 19% to 54% [15]. 1) Various approaches to vagus nerve stimulation include stand-

ard and enhanced Valsalva maneuvers, cold water immersion, carotid sinus massage, pharyngeal cold stimulation, and mechanical stimulation [16]. The Valsalva maneuver, named after the Italian anatomist Valsalva, involves taking a deep breath, closing the glottis, and forcefully exhaling [17]. In the treatment of paroxysmal supraventricular tachycardia (PSVT), patients are instructed to maintain a 45-degree semi-recumbent position and exhale forcefully using a 10mL syringe until the plunger moves for 10 - 15 seconds [18]. 2) Enhanced Valsalva maneuvers entail transitioning to a supine position immediately after exhalation, elevating the legs to 45 - 90 degrees, resting for 15 seconds, and then returning to the semi-recumbent position. Zhao Lu *et al.* conducted a study involving 2527 PSVT patients to compare the efficacy of modified Valsalva maneuvers with standard Valsalva maneuvers. Their findings revealed a significantly higher success rate with the modified Valsalva maneuver compared to the standard technique [19]. 3) The carotid sinus compression technique involves positioning the patient supine and extending their neck. A medical professional applies pressure to the right carotid sinus using the index and middle fingers for 5 - 10 seconds. If the desired response is not elicited, the opposite carotid sinus should be pressed after 1 - 2 minutes. It is crucial to avoid simultaneous compression of both carotid sinuses to prevent severe complications such as abrupt hypotension or cardiac arrest [1]. During medication use, if symptoms such as dyspnea, bradycardia, or chest pain occur, the drug should be immediately discontinued. It is contraindicated in patients with bronchial stenosis or spasm, second- or third-degree atrioventricular block, and sick sinus syndrome.

5.1.2. Drug Treatment

When the vagus nerve stimulation method is ineffective, drug treatment should be performed under electrocardiogram monitoring. Commonly used drugs are as follows: adenosine (first choice), verapamil, diltiazem, propafone, amiodarone, etc.

Upon binding to the cardiac adenosine A1 receptor, adenosine can activate the acetylcholine-sensitive potassium channel, concurrently inhibiting adenylate cyclase and reducing cAMP levels. This dual mechanism significantly decelerates atrioventricular node conduction and prolongs the effective refractory period, thereby impeding atrioventricular node reentry. Adenosine is the preferred initial pharmacological intervention for paroxysmal supraventricular tachycardia due to its rapid onset, brief half-life, and high efficacy [20]. Typically administered intravenously at doses ranging from 6 mg to 12 mg, it was studied in a cohort led by Nichole Krug, involving 213 patients with PSVT. Among them, 117 individuals (54.9%) received the initial 6 mg dose, while 96 (45.1%) were administered the 12 mg dose. The reversion rates at initial doses of 6 mg and 12 mg were 56.4% and 79.1%, respectively ($p < 0.001$). Among the 46 patients who did not revert at the initial 6 mg dose, 33 (71.7%) responded after subsequent doses. The emergency department opted for a 12 mg initial adenosine dose over the guideline-recommended 6 mg dose to achieve a higher SVT reversion rate without raising the in-

cidence of adverse reactions [21]. During medication administration, symptoms such as dyspnea, bradycardia, or chest pain warrant immediate discontinuation. This medication is contraindicated in patients with bronchial stenosis or spasm, second- or third-degree atrioventricular block, or sick sinus syndrome.

Verapamil, a non-dihydropyridine calcium channel blocker, selectively inhibits calcium ion channels on cardiomyocyte membranes. This action reduces calcium ion influx, lowers cardiomyocyte excitability and conductivity, extends the effective refractory period of the Atrioventricular node, and ultimately decreases ventricular rate [22]. It is now considered a first-line treatment for paroxysmal supraventricular tachycardia, with an initial recommended intravenous dose of 5 - 10 mg administered slowly over more than 2 minutes. If ineffective after 10 - 15 minutes, a 5 mg dose may be repeated. Verapamil is contraindicated in patients with severe hypotension, second- and third-degree atrioventricular block, and sick sinus syndrome [23] [24].

Propafenone, classified as a Class Ic Na+ channel inhibitor, acts by impeding Na+ influx through the Na+ channels on cardiomyocyte membranes. This action results in the deceleration of depolarization, extension of the effective refractory period, elevation of the cardiomyocyte threshold potential, inhibition of Purkinje fibers' depolarization, reduction of myocardial excitability and automaticity, and slowing of both the response action potential and conduction velocity. Furthermore, Propafenone exhibits β-receptor blocking and calcium channel blocking properties [25] [26]. It is recommended as a first-line treatment for paroxysmal supraventricular tachycardia, with an initial dose of 70 mg administered slowly via intravenous injection over 5 - 10 minutes. A repeat dose may be administered after 10 - 20 minutes [27].

Amiodarone, a broad-spectrum antiarrhythmic drug classified as Class I - IV, exerts its main pharmacological effect by non-selectively blocking the potassium channels. This action leads to prolongation of the action potential duration and effective refractory period in the atria, Purkinje fibers, and ventricular myocytes, thereby impeding conduction in the atrioventricular nodes and bypass tracts, consequently slowing the heart rhythm [28]. The recommended initial dose is a slow intravenous injection of 150 mg over 10 minutes. If sinus rhythm is not restored within 20 minutes, a repeat dose may be administered. In comparison to propafenone, amiodarone has a slightly delayed onset of action but offers greater safety and a broader spectrum of clinical utility [29].

5.1.3. Emergency Electrical Cardioversion

For patients with refractory drug treatment, hemodynamic instability (systolic blood pressure <90 mmHg), or pre-excitation syndrome (Wolff-Parkinson-White syndrome) accompanied by atrial fibrillation or atrial flutter, the recommended initial energy for biphasic waveform cardioversion is 50 - 100 joules. If the initial attempt is unsuccessful, the energy level can be incrementally increased to 120 joules; for monophasic waveform cardioversion, the energy level is typically set at 100 joules [29]. In a study by Lorenzo Gamberini *et al.*, involving 1234 patients

with paroxysmal supraventricular tachycardia (PSVT), the success rate of restoring sinus rhythm using direct current synchronized cardioversion was reported to be 83.9% [30].

5.2. Interventional Treatment

Radiofrequency ablation is a sophisticated interventional therapy known for its precise localization and control of the treatment area, minimal invasiveness, rapid recovery, low pain levels, high success rate (exceeding 90% - 95%), minimal complications, and broad applicability. It is currently considered the most efficacious approach for managing paroxysmal supraventricular tachycardia (PSVT) [31] [32]. Initially, intracardiac electrophysiological assessment was conducted following puncture of the femoral vein or internal jugular vein under local anesthesia. Subsequently, a mapping electrode catheter was positioned sequentially in the high right atrium (HRA), His bundle (His), coronary sinus (CS), and right ventricle (RV). Tachycardia was provoked through standard procedural stimulation of the high right atrium and right ventricle to ascertain the subtype of paroxysmal supraventricular tachycardia (PSVT) [33]. For patients diagnosed with intraventricular reentrant tachycardia (AVNRT), the slow pathway is typically targeted for ablation through radiofrequency ablation (RFA) or pulsed electric field ablation. A standard 4 mm head catheter is utilized for this procedure. The initial ablation site is situated at the posterior aspect of the Koch triangle and is treated with 15 W power. In cases where there is no evidence of junctional rhythm during ablation, the ablation site will be revisited. Conversely, if a junctional heart rhythm is detected, the power output will be gradually escalated to 20 - 30 W for sustained ablation, with a cumulative duration exceeding 60 seconds. The ablation process is confined to the Koch triangular region. Post-ablation, atrial stimulation is conducted (with or without isoproterenol administration) within 30 minutes of the final ablation to assess the presence of a double pathway phenomenon. Subsequent ablations are performed until AVNRT is no longer inducible [34]. Isoproterenol can simulate a state of sympathetic excitation, which enhances the conduction capacity of the slow pathway and may make residual or damaged slow pathways "reusable." It also shortens the refractory period of the fast pathway. These two effects collectively create the most favorable conditions for inducing AVNRT. If AVNRT remains non-inducible after isoproterenol administration, with only discontinuous atrioventricular nodal echoes (or even no echoes) observed, the ablation is considered highly thorough, and the risk of postoperative recurrence is extremely low [13]. In patients with atrioventricular reciprocating tachycardia (AVRT), initial evaluation involves right ventricular (RV) stimulation to identify sites of earliest atrial retrograde conduction or ventricular pre-excitation. During ablation procedures, the left accessory pathway (adjacent to the mitral valve annulus) can be accessed retrogradely via the aorta towards the ventricular aspect for lateral ablation, or via the atrial septum pathway for standardization. The right accessory pathway (near the tricuspid valve annulus) can be cath-

eterized directly through the femoral venous route, utilizing support from the SR or RA sheath [35]. Radiofrequency (RF) energy parameters are set at 50˚C - 60˚C, with power outputs of 20 - 40 W for the right pathway and 15 - 30 W for the left pathway. Ablation duration may be prolonged to 60 - 120 seconds per point to ensure efficacy [36]. Ablation success is determined by the disappearance of delta waves, blockage of accessory pathway conduction, and the inability to induce AVRT [37].

5.3. Treatment for Special Populations

The management of paroxysmal supraventricular tachycardia (PSVT) in special populations (such as children and pregnant women) requires particular consideration, as standard treatment regimens may be limited by physiological status, variations in drug metabolism, and potential risks.

As first-line acute management for PSVT, vagal maneuvers constitute the primary intervention for both pediatric and pregnant populations. Should these prove ineffective, pharmacologic treatment (adenosine as the drug of choice) should be initiated. Immediate synchronized cardioversion is warranted upon emergence of hemodynamic instability. For long-term prophylaxis, beta-blockers (such as propranolol) represent the preferred pharmacologic strategy; notably, amiodarone is contraindicated during gestation due to its documented fetal thyrotoxic and neurotoxic potential. Catheter ablation is generally indicated for older pediatric patients (typically exceeding 4 years of age or 15 kg body weight) presenting with recurrent episodes or pharmacotherapy resistance, whereas its implementation should be deferred during pregnancy when feasible [38].

6. Selection of Treatment Methods for Paroxysmal Supraventricular Tachycardia

Schematic diagram of the current clinical treatment pathway selection for paroxysmal supraventricular tachycardia (PSVT) (**Figure 2**). In cases of initial onset with stable hemodynamics, vagus nerve stimulation is the preferred initial treatment option due to its simplicity, practicality, and ease of implementation. Should conservative measures prove ineffective, pharmacological interventions may be considered. Commonly utilized medications in clinical practice include adenosine (first-line), verapamil, diltiazem, propafenone, and amiodarone. Close monitoring of vital signs and electrocardiogram readings is imperative during drug therapy. In instances of critical condition, hemodynamic instability, or failure of conservative and pharmacological treatments, immediate electrical cardioversion is warranted. In cases of recurrent PSVT leading to significant impairment of quality of life and patient preference for definitive treatment, radiofrequency ablation emerges as a highly effective option. This method offers precise localization, minimal invasiveness, rapid recovery, high success rates (>90% - 95%), low complication rates, and broad applicability, rendering it the current gold standard for the curative management of PSVT.

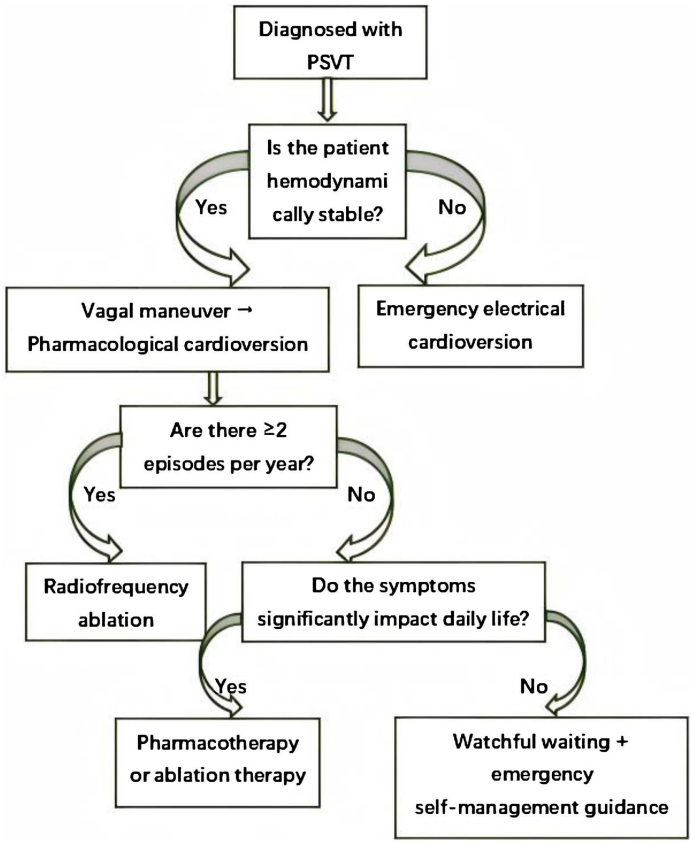


Figure 2. Schematic diagram of the treatment selection pathway for paroxysmal supraventricular tachycardia.

7. Conclusion

The management of paroxysmal supraventricular tachycardia (PSVT) has transitioned from conventional pharmacological interventions to a predominant reliance on catheter ablation as a radical therapeutic approach. Recent advancements in ablation techniques and antiarrhythmic medications have expanded the therapeutic armamentarium for PSVT patients. Nonetheless, addressing unique clinical presentations and ensuring long-term management remain formidable clinical hurdles. Future efforts should prioritize bolstering both fundamental and clinical investigations, fostering the evolution of tailored and precise therapeutic protocols, and refining the diagnostic and therapeutic continuum to enhance the long-term prognosis of individuals with PSVT. With the continual progress of technology, the management of PSVT is poised to become increasingly secure, efficacious, and convenient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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