## Original Article

# *Concordance Between Exercise Stress Testing and Electrophysiologic Study for Supraventricular Tachyarrhythmia Induction*

**Fatememasume Manuchehr[1], MD; Sajjad Ghane Ezabadi[2], MD; AmirHossein RahimBakhsh[3], MS; Arash Zourouni[1], MD; Soraya Shahrzad[1*], MD**

## ABSTRACT

***Background:*** Supraventricular tachyarrhythmias (SVTs) are common arrhythmias with paroxysmal presentations that often hinder electrocardiographic documentation during symptomatic episodes. Exercise stress testing may provoke SVT through sympathetic stimulation and increased myocardial demand. This study evaluated the concordance between exercise testing and electrophysiologic study (EPS) in patients with EPS-inducible SVT.

***Methods:*** This cross-sectional study included 71 patients with confirmed or suspected SVT referred to a tertiary center (April 2020–March 2021). Exclusion criteria were pregnancy, age below 15 years, inability to exercise, refusal, and absence of inducible arrhythmia during EPS. Participants underwent maximal Bruce protocol exercise testing with continuous monitoring, followed by EPS. Nonparametric tests were used for analysis.

***Results:*** Participants were 17 to 67 years of age, and exercise duration ranged from 2 to 16 minutes. Exercise stress testing induced SVT in 6 patients (8.5%) among patients with EPS-inducible SVT. Exercise-induced SVT was not associated with demographic characteristics, medical history, symptom profile, or exercise capacity. Atrioventricular nodal reentrant tachycardia (AVNRT) was the most frequent SVT subtype overall and the most common subtype among exercise-induced cases. The distribution of SVT subtypes differed significantly according to exercise-induced SVT status. Metabolic equivalents were inversely correlated with age and body mass index.

***Conclusions:*** Concordance between exercise stress testing and EPS vis-à-vis SVT induction was low among patients with EPS-inducible SVT. Exercise-induced SVT was not associated with clinical or demographic parameters. Although AVNRT was the most common induced subtype, the limited concordance supports a restricted role for exercise testing in provoking SVT before EPS. ***(Iranian Heart Journal 2026; 27(3): 60-70)***

**KEYWORDS:** supraventricular tachycardia; exercise testing; arrhythmia inducibility; electrophysiologic study; atrioventricular nodal reentrant tachycardia

[1] Department of Cardiology, Shariati Hospital, Tehran University of Medical Sciences, Tehran, IR Iran.
[2] School of Medicine, Tehran University of Medical Sciences, Tehran, IR Iran.
[3] Department of Comparative Biosciences, Faculty of Veterinary Medicine, University of Tehran, Tehran, IR Iran.

***Corresponding Author:** Soraya Shahrzad, MD; Department of Cardiology, Shariati Hospital, Tehran University of Medical Sciences, Tehran, IR Iran.
**Email:** sshahrzad@sina.tums.ac.ir **Tel:** +982166348571

**Received:** February 19, 2026 **Accepted:** June 29, 2026

Supraventricular tachyarrhythmias (SVTs) comprise a heterogeneous group of arrhythmias originating at or above the His bundle and are characterized by rapid supraventricular rhythms. Common SVT mechanisms include inappropriate sinus tachycardia (IST), atrial tachycardia (AT), atrial flutter (AFL), atrioventricular nodal reentrant tachycardia (AVNRT), and atrioventricular reentrant tachycardia (AVRT).[1, 2] SVTs have an estimated prevalence of approximately 2.25 per 1000 individuals and an annual incidence of 35 per 100 000, with AVNRT showing a female predominance and AVRT occurring slightly more often in men.[3–5]

Although SVTs represent a common cause of outpatient evaluation, many episodes are managed in the ambulatory setting, underscoring the diagnostic challenges posed by their paroxysmal nature.[6, 7] SVTs may be asymptomatic; however, when episodes occur, they can manifest with palpitations, dyspnea, lightheadedness or dizziness, sweating, presyncope or syncope, and characteristic neck pulsations.[8, 9]

Standard evaluation includes medical history, physical examination, resting 12-lead electrocardiography (ECG), transthoracic echocardiography, and ideally ECG documentation during tachycardia. Additional diagnostic modalities include ambulatory ECG monitoring, wearable monitoring devices, ischemia assessment when clinically indicated, and electrophysiologic study (EPS) with catheter ablation when appropriate.[2, 10, 11]

EPS remains the reference standard for identifying the mechanism of SVT and provides the opportunity for definitive catheter ablation. Nevertheless, because of its invasive nature and resource requirements, interest persists in noninvasive approaches that may reproduce or provoke SVT before EPS. Exercise stress testing, through increased sympathetic activation and myocardial demand, may facilitate arrhythmia induction and, therefore, has been investigated as a potential means of reproducing clinically relevant tachyarrhythmias. Nonetheless, the ability of exercise testing to reproduce SVTs subsequently confirmed during EPS remains insufficiently characterized.

Existing studies have largely evaluated unselected populations rather than patients with confirmed or strongly suspected SVT, and data regarding specific SVT subtypes, particularly AVNRT and AVRT, remain limited. Furthermore, the degree of concordance between exercise-induced arrhythmias and findings obtained during invasive electrophysiologic evaluation has not been well established.

Accordingly, this study aimed to evaluate the concordance between exercise stress testing and EPS in the induction of SVT among patients with suspected or previously documented SVT undergoing EPS. In addition, we examined the relationship between exercise-induced SVT and patient characteristics, symptom profiles, exercise capacity, and arrhythmia subtypes.

## METHODS

### Study Design and Participants

This cross-sectional study was conducted at Shariati Hospital, Tehran, Iran, between April 2020 and March 2021. Consecutive patients of both sexes who were referred for EPS because of confirmed or suspected SVT were screened for eligibility. Patients were eligible if they met at least 1 of the following criteria: (1) ECG documented SVT; (2) a definite or suspected accessory pathway associated with palpitations; or (3) a history of recurrent paroxysmal palpitations with abrupt onset and termination suggestive of SVT.

Exclusion criteria were pregnancy, age below 15 years, inability to perform exercise testing because of physical limitations, refusal to provide informed consent, and

absence of inducible SVT during EPS. Only patients with inducible SVT during EPS were included in the final analysis.

Because patients without inducible SVT during EPS were excluded, the study was designed to evaluate concordance in SVT inducibility between exercise stress testing and EPS rather than to estimate diagnostic accuracy indices such as specificity, predictive values, or overall diagnostic accuracy.

**Data Collection and Procedures**

Baseline demographic and clinical characteristics were recorded using a structured questionnaire prior to exercise testing. When clinically feasible, antiarrhythmic and rate-controlling medications were discontinued at least 48 hours before testing, corresponding to approximately 4 to 5 elimination half-lives.

Exercise stress testing was performed using the standard maximal Bruce protocol.[12] Continuous 12-lead ECG and hemodynamic monitoring was performed throughout exercise and recovery. Hemodynamic parameters were recorded at baseline, at 3-minute intervals during exercise, and throughout recovery. Before testing, patients were counseled regarding potential warning symptoms, including dyspnea, chest pain, dizziness, or presyncope. All tests were conducted in a fully equipped exercise laboratory containing a treadmill, monitoring system, resuscitation equipment, and emergency medications, under the direct supervision of a cardiology resident and a trained nurse, with immediate access to an attending cardiologist. All exercise tests were subsequently reviewed and interpreted by a cardiac electrophysiologist.

In cases of sustained tachyarrhythmia, exercise testing was immediately terminated, and a 12-lead ECG was obtained. Patients were placed in the supine position with continuous cardiac monitoring. Carotid sinus massage was performed when appropriate and in the absence of contraindications. If the arrhythmia persisted, pharmacologic termination was achieved using an appropriate antiarrhythmic agent, most commonly intravenous adenosine because of its short plasma half-life.

**EPS**

All participants subsequently underwent EPS according to standard institutional protocols. Intracardiac electrode catheters were positioned in standard locations under fluoroscopic guidance. Programmed electrical stimulation, including atrial pacing and delivery of extrastimuli, was performed to evaluate arrhythmia mechanisms and assess SVT inducibility. The type of SVT was determined based on established electrophysiologic criteria. When indicated, catheter ablation was performed during the same procedure.

**Statistical Analysis**

The final sample consisted of 71 patients with EPS-inducible SVT. Data were analyzed using IBM SPSS Statistics version 23 (IBM Corp, Armonk, NY). Normality of continuous variables was assessed using the Kolmogorov-Smirnov test. Because most variables were not normally distributed, nonparametric tests were applied.

Categorical variables were compared using the $\chi^2$ or Fisher exact test, as appropriate. Correlations between study variables were evaluated with the aid of the Spearman rank correlation coefficient. All statistical tests were 2-sided, and a *P* value of less than .05 was considered statistically significant.

Because patients without inducible SVT during EPS were excluded and the analysis included only individuals with EPS-inducible SVT, true negative cases were not available; therefore, specificity and Cohen κ for agreement between exercise testing and EPS could not be calculated, reflecting the presence of verification bias.

### Ethical Considerations

The study protocol was approved by the Ethics Committee of Tehran University of Medical Sciences (approval No. IR.TUMS.THC.REC.1399.031). All procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion.

## RESULTS

A total of 80 patients underwent exercise stress testing during the study period. Nine patients were excluded because of inadequate cooperation during testing or failure to demonstrate an inducible arrhythmia during the subsequent EPS. Consequently, 71 patients with EPS-inducible SVT were included in the final analysis, comprising 47 women (66.2%) and 24 men (33.8%), corresponding to a female-to-male ratio of 1.96:1 (Figure 1).

Exercise testing reproduced SVT in 6 patients (8.5%; 95% CI, 3.2% to 17.5%); however, the remaining 65 patients (91.5%) did not exhibit exercise-induced SVT despite documented inducibility during EPS. Exercise test duration ranged from 2–16 minutes, with a mean of 8.3 (SD, 2.2). Peak heart rate achieved during testing ranged from 111 to 226 beats/min, with a mean of 172 (SD, 22.1), corresponding to 68% to 135% of the age-predicted maximum heart rate (mean, 96%; SD, 11.2). Exercise capacity ranged from 5 to 16 metabolic equivalents (METs), with a mean of 10.1 (SD, 2.1) (Table 1).

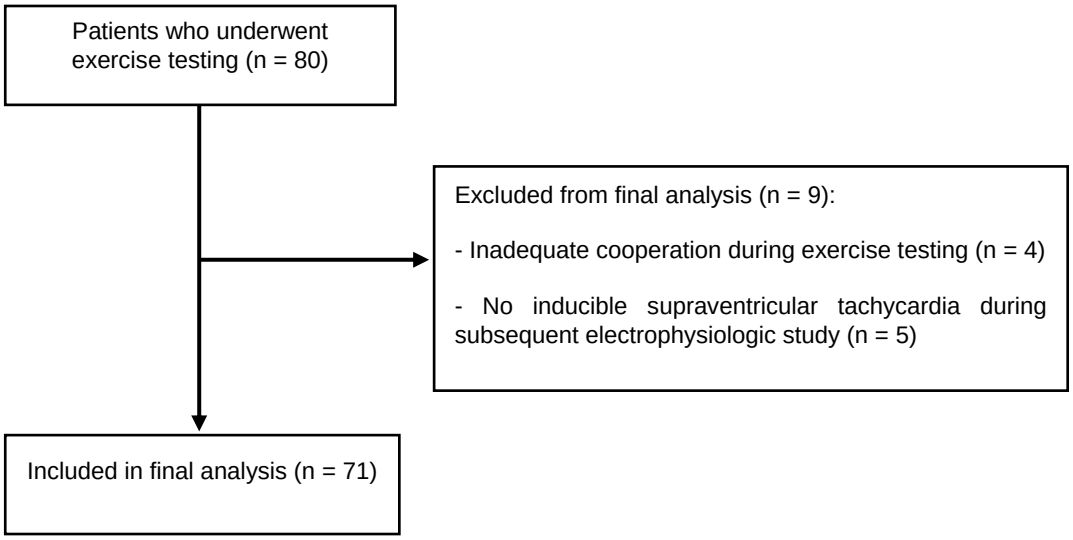


**Figure 1.** Flowchart of the study

**Table 1.** Exercise Test Characteristics (n = 71)

| Indicator | Min | Max | Mean (SD) |
|---|---|---|---|
| Duration, min | 2 | 16 | 8.3 (2.2) |
| METs | 5 | 16 | 10.1 (2.1) |
| Max HR, bpm | 111 | 226 | 172 (22.1) |
| Percent of age-predicted maximal HR (%) | 68 | 135 | 96 (11.2) |

METs: metabolic equivalents; HR: heart rate

**Table 2.** Baseline Demographic and Clinical Characteristics According to Exercise-Induced SVT Status

| Variable | | Total (n = 71) | Exercise-Induced SVT (n = 6) | No Exercise-Induced SVT (n = 65) | *P* |
|---|---|---|---|---|---|
| Age, years, mean (SD) | | 40.29 (12.57) | 38.5 (15.04) | 40.46 (12.44) | .378 |
| Sex, No. (%) | Male<br>Female | 24 (33.80)<br>47 (66.19) | 3 (50)<br>3 (50) | 21 (32.3)<br>44 (67.69) | .324 |
| BMI, kg/m² , mean (SD) | | 27.01 ( 524) | 24 ( 2.96) | 27.29 (5.33) | .485 |

SVT: supraventricular tachyarrhythmia; BMI, body mass index

Baseline demographic and clinical characteristics are summarized in Table 2. Participants ranged in age from 17 to 67 years, with a mean of 40.29 (SD, 12.57) years. The mean age was 38.5 (SD, 15.04) years among patients with exercise-induced SVT (group 1) and 40.46 (SD, 12.44) years among those without exercise-induced SVT (group 2); this difference was not statistically significant (*P* = .378). Sex distribution was also comparable between groups. In group 1, 50% of patients were male and 50% were female, whereas in group 2, 32.4% were male and 67.6% were female (*P* = .324).

Body mass index (BMI) ranged from 18–39 kg/m$^2$, with a mean of 27.01 (SD, 5.24) kg/m$^2$. More than half of the participants (57.7%; n = 41) were classified as overweight; 21.1% (n = 15) had normal weight, 19.7% (n = 14) were obese, and 1.4% (n = 1) were underweight. Mean BMI values did not differ significantly between patients with exercise-induced SVT (24.0 [SD, 2.96] kg/m²) and those without exercise-induced SVT (27.29 [SD, 5.33] kg/m$^2$) (*P* = .485).

Medical history characteristics are summarized in Table 3. Fewer than one-third of the study population reported a history of smoking, with 16 patients (22.5%) identified as smokers and 55 patients (77.5%) as nonsmokers. Although 33.3% of patients with exercise-induced SVT (group 1) and 21.5% of those without exercise-induced SVT (group 2) reported a history of smoking, no statistically significant difference was observed between the 2 groups (*P* = .409).

A history of alcohol consumption was reported by 7 participants (9.9%). Alcohol use was reported in 33.3% of patients with exercise-induced SVT and 7.7% of patients without exercise-induced SVT; however, no statistically significant between-group difference was identified (*P* = .104).

Most participants (n = 66 [93.0%]) had no history of diabetes mellitus or thyroid disease. None of the patients with exercise-induced SVT (group 1) had a history of either condition, whereas 7.7% of patients without exercise-induced SVT (group 2) had a history of diabetes mellitus and/or thyroid disease; still, the difference failed to constitute statistical significance (*P* = .634).

Most patients (n = 64 [90.1%]) had no history of hypertension. Hypertension was present in 16.7% of patients in group 1 and 9.2% of those in group 2; however, no statistically significant between-group difference was observed (*P* = .477).

Approximately two-thirds of the study population were receiving antiarrhythmic therapy, of whom 77% were treated with beta-blockers. In group 1, 66.7% of patients were receiving beta-blockers, 16.7% were taking other antiarrhythmic agents, and 16.7% were not receiving antiarrhythmic medication. Corresponding proportions in group 2 were 47.7%, 13.8%, and 38.5%, respectively; nonetheless, no statistically significant difference was observed between groups (*P* = .564).

A history of syncope was reported by 6 patients, all of whom were ultimately

diagnosed with AVRT during EPS. The between-group difference in the prevalence of syncope was 9.2% and did not reach statistical significance (*P* = .577).

Based on participant reports, symptoms were unrelated to physical activity in approximately two-thirds of participants (n = 45 [63.4%]). Activity-related symptoms were reported by 33.3% of patients in group 1 and 36.9% in group 2; nevertheless, no statistically significant between-group difference was observed (*P* = .617).

Frequent palpitations, defined as symptom occurrence more than once per month, were reported by 70% of participants. The proportion of patients with frequent vs infrequent palpitations did not differ significantly between group 1 and group 2 (*P* = .391).

Manifest accessory pathways were detected on resting ECG in 17 participants (23.9%). Although the between-group difference was not statistically significant (*P* = .180), no participant with exercise-induced SVT had evidence of an accessory pathway on baseline ECG, whereas 26.2% of those without exercise-induced SVT demonstrated ECG evidence of an accessory pathway.

Overall, no significant associations were identified between exercise-induced SVT and participants' medical history or symptom characteristics.

Most participants (85.9%) reported little or no history of regular sports activity (Table 4). Based on exercise test performance, participants were categorized into 5 predefined activity capacity groups: very low, low, moderate, high, and very high. Most demonstrated low or moderate exercise capacity.

In the exercise-induced SVT group (group 1), 33.3% and 50% of participants were categorized as having low and moderate activity capacity, respectively, compared with 12.3% and 53.8% in the no exercise-induced SVT group (group 2). High activity capacity was observed in 16.7% of participants in group 1 and 27.7% of those in group 2. No significant association was observed between activity capacity category and exercise-induced SVT (*P* = .501).

The distribution of arrhythmia subtypes identified during EPS is shown in Table 5. AVNRT was the most common arrhythmia overall. Among participants with exercise-induced SVT, AVNRT accounted for 50% of cases, whereas the remaining cases included AVRT, AT, and AFL, each observed in 1 participant. Among participants without exercise-induced SVT, AVNRT was present in 69.2%, AVRT in 27.7%, and AT in 3.1% of cases, with no cases of AFL. The distribution of arrhythmia subtypes differed significantly between groups, indicating a statistically significant association between arrhythmia type and inducibility during exercise testing (*P* = .003).

Finally, as shown in Table 6, METs demonstrated significant inverse correlations with both age and BMI.

**Table 3.** Medical History and Symptom Characteristics According to Exercise-Induced SVT Status

| Variable | | Total (n = 71) | Exercise-Induced SVT (n = 6) | No Exercise-Induced SVT (n = 65) | *P* |
|---|---|---|---|---|---|
| Smoking | Yes<br>No | 16 (22.5)<br>55 (77.5) | 2 (33.3)<br>4 (66.7) | 14 (21.5)<br>51 (78.5) | .409 |
| Alcohol use | Yes<br>No | 7 (9.9)<br>64 (90.1) | 2 (33.3)<br>4 (66.7) | 5 (7.7)<br>60 (92.3) | .104 |
| DM | Yes<br>No | 5 (7)<br>66 (93) | 0 (0)<br>6 (100) | 5 (7.7)<br>60 (92.3) | .634 |
| HTN | Yes<br>No | 7 (9.9)<br>64 (90.1) | 1 (16.7)<br>5 (83.3) | 6 (9.2)<br>59 (90.8) | .477 |
| Thyroid disease | Yes<br>No | 5 (7)<br>66 (93) | 0 (0)<br>6 (100) | 5 (7.7)<br>60 (92.3) | .634 |

| | | | | | |
|---|---|---|---|---|---|
| FH of arrhythmia | Yes<br>No | 3 (4.2)<br>68 (95.8) | 0 (0)<br>6 (100) | 3 (4.6)<br>62 (95.4) | .764 |
| HX of syncope | Yes<br>No | 6 (8.5)<br>65 (91.5) | 0 (0)<br>6 (100) | 6 (9.2)<br>59 (90.8) | .577 |
| Accessory pathway | Yes<br>No | 17 (23.9)<br>54 (76.1) | 0 (0)<br>6 (100) | 17 (26.2)<br>48 (73.8) | .180 |
| Palpitation frequency | Frequent<br>Not Frequent | 49 (69)<br>22 (31) | 5 (83.3)<br>1 (16.7) | 44 (67.7)<br>21 (32.3) | .391 |
| Activity-related palpitations | Yes<br>No | 26 (36.6)<br>45 (63.4) | 2 (33.3)<br>4 (66.7) | 24 (36.9)<br>41 (63.1) | .617 |
| Antiarrhythmic therapy | Beta-blockers<br>Other AAs<br>No Drugs | 35 (49.3)<br>10 (14)<br>26 (36.7) | 4 (66.7)<br>1 (16.7)<br>1 (16.7) | 31 (47.7)<br>9 (13.8)<br>25 (38.5) | .564 |

Data are presented as n (%). The Fisher exact test was used when expected cell counts were < 5.
SVT: supraventricular tachyarrhythmia; DM: diabetes mellitus; HTN: hypertension; Hx: history; FH: family history; AAs: antiarrhythmic agents

**Table 4.** Physical Activity–Related Characteristics According to Exercise-Induced SVT Status

| Variable | | Total (n=71) | Exercise-Induced SVT (n = 6) | No Exercise-Induced SVT (n = 65) | *P* |
|---|---|---|---|---|---|
| Pre-EPS sports activity, No. (%) | Regular | 10 (14.1) | 0 (0) | 10 (15.4) | .301 |
| | Irregular | 28 (39.4) | 4 (66.6) | 24 (36.9) | |
| | None | 33 (46.5) | 2 (33.3) | 31 (47.7) | |
| Pre-EPS exercise capacity, No. (%) | Very low | 4 (5.6) | 0 (0) | 4 (6.2) | .501 |
| | Low | 10 (14.1) | 2 (33.3) | 8 (12.3) | |
| | Moderate | 38 (53.5) | 3 (50) | 35 (53.8) | |
| | High | 19 (26.8) | 1 (16.7) | 18 (27.7) | |
| | Very high | 0 (0) | 0 (0) | 0 (0) | |

The Fisher exact test was used when expected cell counts were < 5.
SVT: supraventricular tachyarrhythmia; EPS: electrophysiologic study

**Table 5.** Distribution of SVT Subtypes According to Exercise-Induced SVT Status and ECG Documentation

| Variable | Total (n = 71) | Exercise-Induced SVT (n = 6) | No Exercise-Induced SVT (n = 65) | ECG-Documented SVT (n = 50) | No ECG Documentation (n = 21) |
|---|---|---|---|---|---|
| AVNRT | 48 (67.6) | 3 (50) | 45 (69.2) | 40 (80) | 8 (38) |
| AVRT | 19 (26.8) | 1 (16.7) | 18 (27.7) | 6 (12) | 13 (62) |
| AT | 3 (4.2) | 1 (16.7) | 2 (3.1) | 3 (6) | 0 (0) |
| AFL | 1 (1.4) | 1 (16.7) | 0 (0) | 1 (2) | 0 (0) |

Data are presented as n (%). The Fisher exact test was used when expected cell counts were < 5. Because of small cell counts in some categories, *P* values may be unstable and should be interpreted with caution.
SVT: supraventricular tachyarrhythmia; AVNRT: atrioventricular nodal reentrant tachycardia; AVRT: atrioventricular reentrant tachycardia; AT: atrial tachycardia; AFL: atrial flutter

**Table 6.** Correlations Between METs and Clinical Variables

| Variable | Spearman ρ | *P* |
|---|---|---|
| Age | −.540 | <.001 |
| BMI | −.279 | .019 |

Data are presented as the Spearman correlation coefficient (ρ) with corresponding 2-tailed *P* values.
BMI: body mass index

## DISCUSSION

SVTs represent a common yet diagnostically challenging group of arrhythmias. Although careful history taking, physical examination, and resting ECG provide valuable initial information, definitive noninvasive confirmation typically requires ECG documentation during tachycardia. Be that as it may, the paroxysmal and unpredictable nature of SVT episodes often prevents timely ECG capture, limiting the diagnostic yield of ambulatory monitoring modalities such as Holter recording in many patients.

Exercise represents one of the most common forms of physiological stress and imposes substantial hemodynamic and autonomic demands on the cardiovascular system. Under these conditions, latent tachyarrhythmias may occasionally become apparent despite being absent on resting ECG.[13, 14] Nevertheless, previous studies have suggested that the occurrence of SVT during exercise testing is relatively uncommon.[15, 16] Given the widespread availability, noninvasive nature, and favorable cost profile of exercise testing, we aimed to evaluate the ability of exercise testing to reproduce SVT episodes in patients who were subsequently confirmed to have inducible arrhythmia during EPS.

Most previous studies addressing this topic have been conducted in unselected populations or without systematic confirmation of arrhythmia mechanisms using invasive electrophysiologic testing. In contrast, the present study focused on a clinically relevant cohort of patients with confirmed or highly suspected SVT who were referred for EPS, allowing definitive characterization of the arrhythmia mechanism. This design enabled evaluation of the concordance between SVT inducibility during exercise testing and arrhythmia induction during EPS.

In our cohort, SVT was reproduced during exercise testing in only 6 of 71 patients (8.5%), whereas inducible arrhythmia was confirmed in all patients during EPS. These findings indicate that exercise testing rarely reproduces SVT episodes even among patients with electrophysiologically confirmed arrhythmia.

Our results are consistent with previous reports. In a pediatric study using a similar methodological approach, Draper et al[15] observed SVT during exercise testing in only 3 of 53 patients (5.7%). Notably, all affected patients in that study reported exertion-related symptoms, and 2 had evidence of accessory pathways on resting ECG. Other studies have also demonstrated limited concordance between arrhythmia occurrence during physical activity and patients' reported symptoms or clinical histories.[17] Conversely, exercise testing may provoke arrhythmias in selected patients with known cardiac disease or established arrhythmogenic substrates.[18]

In the present study, no significant associations were observed between exercise-induced SVT and clinical manifestations such as syncope, palpitation frequency, or the relationship between symptoms and physical activity. Only 2 of the 6 patients with exercise-induced SVT reported exertion-related symptoms. All arrhythmic episodes occurred near peak exercise intensity, with no events observed during the recovery phase, findings that are consistent with previous

reports.[15, 19] One potential explanation for the low rate of SVT reproduction observed in our cohort may be the predominance of low to moderate functional capacity among participants. It is conceivable that higher levels of exercise intensity and greater peak heart rates could increase the likelihood of arrhythmia induction, although this hypothesis would require confirmation in larger cohorts with greater exercise capacity. In line with our findings, Jelinek et al[16] reported a limited overall role of exercise testing in provoking SVT, although in selected cases it facilitated identification of arrhythmogenic mechanisms that were otherwise difficult to establish.

No significant associations were observed between exercise-induced SVT and demographic variables, including age, sex, or BMI. Similarly, SVT reproduction during exercise testing was not related to prior medical history, including smoking status, alcohol consumption, diabetes mellitus, hypertension, or thyroid disease. Although minor differences were observed in some clinical variables, these did not reach statistical significance and may warrant further evaluation in larger studies.

None of the patients in the present cohort had documented atherosclerotic coronary artery disease. This finding is consistent with prior reports indicating that patients with paroxysmal SVT frequently have structurally normal hearts and unobstructed coronary arteries, with arrhythmia representing the principal clinical abnormality.[3, 20–22]

With respect to arrhythmia subtype, half of the patients with exercise-induced SVT were ultimately diagnosed with AVNRT, while the remaining cases included AVRT, AT, and AFL. A statistically significant association was observed between arrhythmia subtype and the likelihood of SVT reproduction during exercise testing. AVNRT was the most common arrhythmia overall in our cohort, accounting for more than two-thirds of cases. Importantly, none of the patients with manifest accessory pathways on resting ECG developed SVT during exercise testing.

No cases of atrial fibrillation were observed in the present study, likely reflecting the absence of significant structural heart disease among participants. Evaluation of exercise-related atrial fibrillation may therefore require more heterogeneous populations, including patients with underlying structural or ischemic heart disease.

Overall, the demographic and clinical profile of our cohort is consistent with previous reports from specialized electrophysiology centers, where patients referred for EPS and catheter ablation of paroxysmal SVT are typically younger and have a relatively low burden of concomitant cardiovascular disease.[3, 21, 22]

Previous epidemiologic studies have shown that AVNRT occurs more frequently in women, whereas AVRT predominates in men.[4, 5] Because AVNRT constituted the majority of arrhythmias in our cohort, the observed female-to-male ratio of 1.96:1 is consistent with the existing literature. Furthermore, our findings parallel reports from high-volume electrophysiology centers in which AVNRT represents the most frequently treated SVT subtype, followed by AVRT and AT.[23–25]

Because multiple statistical comparisons were performed across several clinical and demographic variables, the findings should be interpreted with caution. When a Bonferroni correction for multiple testing ($\alpha = .0025$) is applied, none of the observed associations remain statistically significant. Accordingly, these results should be regarded as exploratory and hypothesis-generating, and confirmation in larger prospective cohorts is warranted.

## Limitations and Future Directions

The present study has several limitations. First, because patients without inducible SVT during EPS were excluded from the

final analysis, the study was not designed to evaluate the diagnostic accuracy of exercise testing for SVT. Accordingly, diagnostic accuracy indices, including specificity, negative predictive value, and overall diagnostic accuracy, could not be estimated. Further, the absence of EPS-negative patients precluded determination of the false-positive rate of exercise testing. The findings should therefore be interpreted as reflecting the rate of SVT reproduction during exercise testing among patients with EPS-inducible arrhythmia, rather than the diagnostic performance of exercise testing in an unselected population.

Second, the number of patients in whom SVT was reproduced during exercise testing was small. This limited the statistical power to detect differences among arrhythmia subtypes and may restrict the generalizability of the findings. In addition, heterogeneity in exercise test duration, functional capacity, and participants' familiarity with treadmill-based stress testing may have influenced exercise performance and arrhythmia inducibility.

Future investigations should include larger, preferably prospective cohorts with both EPS-positive and EPS-negative participants to permit formal assessment of diagnostic accuracy. Studies with more homogeneous exercise protocols and better characterization of baseline functional capacity may help clarify which participants are most likely to have SVT reproduced during exercise testing. Long-term follow-up may further elucidate whether exercise-induced SVT has prognostic implications for arrhythmia recurrence or clinical outcomes.

Moreover, incorporating echocardiographic parameters, such as left atrial size and left atrial strain, may provide additional insight into the substrate associated with exercise-induced supraventricular arrhythmias. Finally, evaluation of exercise-induced atrial fibrillation and premature atrial contractions, as well as the potential role of exercise testing in identifying participants at increased risk for atrial fibrillation, remains an important area for future research.

## CONCLUSIONS

In this cohort of participants with electrophysiologically confirmed inducible SVT, exercise stress testing reproduced SVT in only a small proportion of cases. These findings indicate that exercise testing has a low yield for inducing SVT in participants with EPS-proven inducible arrhythmias. Accordingly, exercise testing should not be relied on as a routine screening tool for arrhythmia reproduction before electrophysiologic evaluation.

## REFERENCES


1. Page, R.L., et al., 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Journal of the American College of Cardiology, 2016. 67(13): p. e27-e115.
2. Katritsis, D.G., et al., European heart rhythm association (EHRA) consensus document on the management of supraventricular arrhythmias, endorsed by heart rhythm Society (HRS), Asia-Pacific heart rhythm Society (APHRS), and Sociedad Latinoamericana de Estimulación cardiaca Y Electrofisiologia (SOLAECE). Europace, 2017. 19(3): p. 465-511.
3. Orejarena, L.A., et al., Paroxysmal supraventricular tachycardia in the general population. Journal of the American College of Cardiology, 1998. 31(1): p. 150-157.
4. Porter, M.J., et al., Influence of age and gender on the mechanism of supraventricular tachycardia. Heart Rhythm, 2004. 1(4): p. 393-396.
5. Liuba, I., et al., Gender-related differences in patients with atrioventricular nodal reentry

tachycardia. The American journal of cardiology, 2006. 97(3): p. 384-388.

6. Fuster, V., et al., ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation. Journal of the American College of Cardiology, 2001. 38(4): p. 1266-1266.

7. Campbell, R., Supraventricular tachycardia: occasional nuisance or frequent threat? European heart journal, 1996. 17(suppl_C): p. 21-25.

8. Skov, M.W., et al., Electrocardiographic preexcitation and risk of cardiovascular morbidity and mortality: results from the Copenhagen ECG Study. Circulation: Arrhythmia and Electrophysiology, 2017. 10(6): p. e004778.

9. Kotadia, I.D., S.E. Williams, and M. O'Neill, Supraventricular tachycardia: An overview of diagnosis and management. Clinical Medicine, 2020. 20(1): p. 43-47.

10. Wang, R., et al., Accuracy of wrist-worn heart rate monitors. Jama cardiology, 2017. 2(1): p. 104-106.

11. Katritsis, D.G., D.B. Mark, and B.J. Gersh, Revascularization in stable coronary disease: evidence and uncertainties. Nature Reviews Cardiology, 2018. 15(7): p. 408-419.

12. Bruce, R.A., Exercise testing of patients with coronary heart disease: principles and normal standards for evaluation. Ann Clin Res, 1971. 3: p. 323-332.

13. Faris, J.V., P.L. McHenry, and S.N. Morris, Concepts and applications of treadmill exercise testing and the exercise electrocardiogram. American Heart Journal, 1978. 95(1): p. 102-114.

14. Bruce, R.A., Methods of exercise testing: step test, bicycle, treadmill, isometrics. The American journal of cardiology, 1974. 33(6): p. 715-720.

15. Draper, D.E., et al., Diagnostic usefulness of graded exercise testing in pediatric supraventricular tachycardia. Canadian Journal of Cardiology, 2009. 25(7): p. 407-410.

16. Jelinek, M.V. and B. Lown, Exercise stress testing for exposure of cardiac arrhythmia. Progress in Cardiovascular Diseases, 1974. 16(5): p. 497-522.

17. McHenry, P.L., et al., Cardiac arrhythmias observed during maximal treadmill exercise testing in clinically normal men. The American journal of cardiology, 1972. 29(3): p. 331-336.

18. Gooch, A.S., Exercise testing for detecting changes in cardiac rhythm and conduction. The American Journal of Cardiology, 1972. 30(7): p. 741-746.

19. Maurer, M.S., E.A. Shefrin, and J.L. Fleg, Prevalence and prognostic significance of exercise-induced supraventricular tachycardia in apparently healthy volunteers. The American journal of cardiology, 1995. 75(12): p. 788-792.

20. Srinivasan, C. and S. Balaji, Neonatal supraventricular tachycardia. Indian Pacing and Electrophysiology Journal, 2019. 19(6): p. 222-231.

21. Calkins, H., et al., Diagnosis and cure of the Wolff–Parkinson–White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. New England Journal of Medicine, 1991. 324(23): p. 1612-1618.

22. Kay, G.N., et al., Role of radiofrequency ablation in the management of supraventricular arrhythmias: experience in 760 consecutive patients. Journal of cardiovascular electrophysiology, 1993. 4(4): p. 371-392.

23. García-Fernández, F.J., et al., Spanish catheter ablation registry. 17th official report of the Spanish society of cardiology working group on electrophysiology and arrhythmias (2017). Revista Española de Cardiología (English Edition), 2018. 71(11): p. 941-951.

24. Hosseini, S.M., et al., Catheter ablation for cardiac arrhythmias: utilization and in-hospital complications, 2000 to 2013. JACC: Clinical Electrophysiology, 2017. 3(11): p. 1240-1248.

25. Holmqvist, F., et al., A decade of catheter ablation of cardiac arrhythmias in Sweden: ablation practices and outcomes. European heart journal, 2019. 40(10): p. 820-830.