Circadian Variation of Paroxysmal Supraventricular Tachycardia

Shih-Huang Lee, MD; Pan-Chen Chang, MD; Huei-Fong Hung, MD; Peiliang Kuan, MD; Jun-Jack Cheng, MD; and Chi-Ren Hung, MD

Background: Various clinical manifestations of cardiovascular diseases have a pattern of circadian variation. In this study, we investigated whether the onset and duration of paroxysmal supraventricular tachycardia (PSVT) has a circadian variation.

Methods and results: In our analysis, we included 105 patients with 498 PSVT episodes. In this study, the onset of PSVT did not have a uniform distribution throughout the 24-h period. There were nearly equal peaks in the time periods from 8:00 to 9:00 AM, 12:00 to 1:00 PM, and 5:00 to 6:00 PM, with a trough at night. The duration of PSVT also did not show a uniform distribution throughout the 24-h period; it increased significantly during the daytime, with a peak between 1:00 and 2:00 PM, another peak between 6:00 and 7:00 PM, and a significant reduction at night.

Conclusions: The onset and duration of PSVT showed a circadian variation. However, the time-oriented antiarrhythmic therapy for preventing PSVT needs further study.

(CHEST 1999; 115:674–678)

Key words: circadian variation; supraventricular; tachycardia

Abbreviation: PSVT = paroxysmal supraventricular tachycardia

Circadian distribution has been noted in some cardiovascular phenomena, including heart rate, BP, transient myocardial ischemia, acute myocardial infarction, thrombotic stroke, and sudden cardiac death,1–10 showing a prominent clustering of events in the morning and another peak in the evening. This provided a clue to the recognition of rhythmic processes triggering these events, such as changes in catecholamine levels, and supported the importance of time-oriented therapy.11–13

Paroxysmal supraventricular tachycardia (PSVT) is a common arrhythmia, and it occurs sporadically in the affected patients. The distribution of the onset of PSVT has not been studied extensively,14–16 and it is not known whether the duration of PSVT has a circadian distribution. Therefore, the purpose of this study was to determine whether the onset and duration of PSVT showed a circadian distribution.

Materials and Methods

Study Population

Our study population was drawn from 2,689 consecutive patients who underwent 24-h Holter monitor recordings in our hospital from January 1996 to December 1997. Of this group, 105 patients had a total of 498 recorded PSVT episodes. None of the patients received antiarrhythmic drugs or any drugs with chronotropic effects, such as calcium channel blockers, digoxin, and β-blockers. The PSVT was defined by the following ECG criteria: (1) an episodic occurrence, (2) a ventricular rate > 120 beats/min, (3) a narrow QRS interval (< 0.08 s), functional right or left bundle branch block, and (4) a < 0.02-s variation in successive R-R intervals.14,17

Statistical Analysis

The distributions of the onset and duration of PSVT by 1- and 6-h intervals (1:00 to 7:00 AM, 7:00 AM to 1:00 PM, 1:00 to 7:00 PM, and 7:00 PM to 1:00 AM) were tested for uniformity by the χ² for goodness of fit. A p value of < 0.05 was considered statistically significant.

Results

Study Population

Of the 105 patients (mean [± SD] age, 53 ± 22 years), 47 were men. Of the 31 patients with associated cardiovascular diseases, 23 patients had hypertension, 5 had valvular heart disease, 7 had coronary artery disease, and 1 patient had congenital heart disease. Eleven patients were referred to receive
invasive electrophysiological study and radiofrequency catheter ablation because their PSVT was refractory to antiarrhythmic drugs.

**Onset of PSVT**

The onset of PSVT by 1-h intervals did not demonstrate a uniform distribution throughout the 24-h period (Fig 1, *Top, A*: p < 0.01). The frequency of onset showed nearly equal peaks in three hourly distributions (8:00 to 9:00 AM, 12:00 to 1:00 PM, and 5:00 to 6:00 PM) and a trough at night. Otherwise, the distribution of onset of PSVT was 56 episodes (11%) from 1:00 to 7:00 AM, 181 episodes (36%) from 7:00 AM to 1:00 PM, 178 episodes (36%) from

---

**Figure 1.** *Top, A*: the hourly frequency of the onset of PSVT. The peaks existed between 8:00 and 9:00 AM, 12:00 and 1:00 PM, and 5:00 and 6:00 PM. The onset of PSVT did not show a uniform distribution by 1-h intervals (p < 0.01). *Bottom, B*: the cumulative percentage of onset of PSVT by 6-h intervals. The onset of PSVT also did not show a uniform distribution by 6-h intervals (p < 0.01).
1:00 PM to 7:00 PM, and 83 episodes (17%) from 7:00 PM to 1:00 AM (Fig 1, Bottom, B; p < 0.01).

Duration of PSVT

The duration of PSVT by 1-h intervals did not show a uniform distribution throughout the 24-h period (Fig 2, Top, A; p < 0.01). The duration of PSVT increased during the daytime, with a peaks between 1:00 and 2:00 PM and between 6:00 and 7:00 PM, and decreased at night. The distribution of duration of PSVT was 142 min (11%) from 1:00 to 7:00 AM, 389 min (29%) from 7:00 AM to 1:00 PM.

Figure 2. Top, A: the hourly duration of PSVT. The peaks existed between 1:00 and 2:00 PM, and 6:00 and 7:00 PM. The duration of PSVT did not show a uniform distribution by 1-h intervals (p < 0.01). Bottom, B: the cumulative percentage of duration of PSVT by 6-h intervals. The duration of PSVT also did not show a uniform distribution by 6-h intervals (p < 0.01).
680 min (51%) from 1:00 to 7:00 PM, and 125 min (9%) from 7:00 PM to 1:00 AM (Fig 2, Bottom, B; \( p < 0.01 \)).

**DISCUSSION**

**Major Findings**

This study demonstrated the circadian periodicity of the onset and duration of PSVT. The onset and duration of PSVT increased significantly during the daytime.

**Comparisons With Previous Studies**

Previous studies have shown that the onset of PSVT increased during the daytime and decreased at night.\(^{14,16}\) However, Kupari et al\(^ {15}\) showed no morning peak in the onset of PSVT. In these studies, researchers investigated the distribution of the onset of symptomatic PSVT episodes causing admission to the emergency department or telephone transmission of electrocardiography; they did not include asymptomatic PSVT episodes. In this study, we demonstrated that the onset and duration of PSVT, including symptomatic and asymptomatic episodes, showed a circadian variation.

**Onset of PSVT**

This circadian pattern of the onset of PSVT was similar to the pattern in some cardiovascular diseases,\(^ {3–10}\) suggesting that PSVT and some cardiovascular diseases share common mechanisms, such as an increase in physical or mental stress, and changes in autonomic nervous tone. A morning onset could result from the augmented sympathetic tone and the increased secretion of epinephrine that occurs as a consequence of awakening and of morning activities.\(^ {18}\) The increased catecholamines might increase the likelihood of starting the reentrant tachycardia.\(^ {19,20}\) The reasons for the evening peak in the onset of PSVT were less obvious. It was possible that late working shifts, heavy evening meals, or physical recreation activities resulted in the evening peak.\(^ {3,15}\) Our results suggest that it might be reasonable to keep a high drug level during the daytime and a low drug level at night. However, the time-oriented antiarrhythmic therapy for preventing PSVT needs further study.

**Duration of PSVT**

In this study, the total duration of PSVT was longer from 1:00 to 7:00 PM than from 7:00 AM to 1:00 PM, although the incidence of the onset of PSVT was similar in the two periods. When compared to the 1:00 to 7:00 PM time period, the PSVT was easier to terminate spontaneously between 7:00 AM and 1:00 PM; however, the exact mechanisms for this are unclear.

**Study Limitations**

First, in this study, the data were analyzed on the basis of Holter monitor recordings. Whether other episodes of PSVT that were not recorded followed a similar circadian pattern is unknown. However, prospective PSVT studies are difficult and unrealistic because of the relatively long periods between recurrences. Furthermore, there might be some bias to determine the time distribution of onset and duration according to the memory of the patients. Second, most of the patients had no symptoms after receiving antiarrhythmic drugs, and they did not receive electrophysiologic study and radiofrequency catheter ablation. The exact mechanisms of PSVT in these patients were unknown. However, our results clearly demonstrated the circadian distribution of the onset and duration of PSVT.

**CONCLUSION**

This study demonstrated that the onset and duration of PSVT showed a circadian variation. However, the time-oriented antiarrhythmic therapy for preventing PSVT needs further investigation.

**REFERENCES**