Doctor of Medicine

RBBB Type Wide QRS Complex Tachycardia with a Reversed R/S complex in Lead V6: Electrocardiographic Differentiation of Supraventricular Tachycardia from Ventricular Tachycardia

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Abstract

Objective

Supraventricular tachycardia (SVT) may manifest as a wide QRS complex tachycardia (WCT). Differentiation of SVT with a right bundle branch block (RBBB) pattern from ventricular tachycardia (VT) is difficult particularly when R/S ratio in lead V6 is below 1.0. We sought to investigate the ECG criteria for distinguishing these two arrhythmias.

Methods

We investigated ECG parameters from a total of 111 consecutive patients with RBBB pattern WCT with a reversed R/S ratio in lead V6 (72 VTs and 39 SVTs). Arial flutter was diagnosed by visible flutter waves with variable atrioventricular conduction during treadmill test/adenosine infusion or reproduction of WCT by atrial overdrive pacing at the cycle length of clinical tachycardia. Diagnostic criteria from the previous algorithms were compared with our new criteria, RS/QRS ratio, which was defined as the ratio of QRS waveform durations, measured by the interval from the onset of the QRS to the nadir of S wave divided by the total QRS width in the precordial lead V6.

Results

The diagnostic accuracy of previous criteria (Brugada criteria, Vereckei criteria, R-wave peak time at lead II) was only modest (sensitivity; 80.6%, 90.3%, 48.6%, specificity; 30.8%, 61.5%, 92.3%, respectively). However, RS/QRS ratio in lead V6 was significantly lower in SVT than in VT (0.36±0.04 vs 0.50±0.08, P <0.001). On receiver operating characteristic curve analysis, cutoff value of the RS/QRS ratio >0.41 differentiated WCT with a high diagnostic accuracy (sensitivity; 97.2%, specificity; 89.7%).

Conclusions

RS/QRS ratio >0.41 in lead V6 is a simple and reliable index distinguishing VT from SVT in patients with a RBBB pattern WCT and a reversed R/S complex in lead V6.

Keywords: Electrocardiography; Supraventricular tachycardia; Ventricular tachycardia; Differential diagnosis

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Abbreviations

AAD = anti-arrhythmic drug

AFL = atrial flutter

AV = atrioventricular

BBB = bundle branch block

NPV = negative predictive value

PPV = positive predictive value

RBBB = right bundle branch block

ROC = receiver operating characteristics

RS = interval from the onset of R wave to nadir of S wave

SVT = supraventricular tachycardia

VT = ventricular tachycardia

WCT = wide QRS complex tachycardia

Introduction

Diagnosis of a wide QRS complex tachycardia (WCT) with a right bundle branch block (RBBB) pattern and reversed (<1.0) R/S ratio in the precordial lead V6 usually favors ventricular tachycardia (VT).[1] Some supraventricular tachycardias (SVT) mimic this ECG pattern of ventricular tachycardia (VT). Most notably, in the presence of class Ic anti-arrhythmic drugs (AADs), atrial flutter (AFL) with 1:1 atrioventricular (AV) conduction may present as a WCT with bizarre RBBB morphology and a right or northwest axis.[2] In this instance, differential diagnosis is challenging because the QRS morphology of tachycardia mimics VT, especially VT of fascicular origin. Previously proposed criteria for differential diagnosis of a WCT is of limited value.[3] The present study was motivated by a 67-year-old patient who underwent an ICD implantation as a result of failure in ECG differential diagnosis. The patient had undergone coronary artery bypass grafting and was prescribed flecainide 75mg twice daily for paroxysmal atrial fibrillation. He presented with a RBBB pattern WCT and reversed R/S complex in the precordial lead V6 (Figure 1A). Under the diagnosis of VT in the

presence of structural heart disease, he received an ICD implantation. Afterwards, had suffered from recurrent inappropriate shocks and was transferred to our hospital. In the electrophysiology study, a WCT with an identical QRS morphology was induced by burst atrial pacing under flecainide and isoproterenol infusion (Figure 1B). A cavo-tricuspid isthmus dependent AFL with the same QRS complex morphology was also induced, and was eliminated by catheter ablation.

Class Ic AADs can cause rate-dependent conduction slowing in the ventricular muscle, and markedly prolong the QRS duration of aberrantly conducted AFL.[2-4] The conduction delay due to class Ic AADs has been known to be more pronounced in the ventricular myocardium than in the His-Purkinje system, preserving the initial part of QRS complex less affected.[5] On the basis of this, we hypothesized that an SVT with aberrancy might have shorter interval from the onset of R wave to nadir of S wave (RS) compared to the markedly prolonged QRS duration, and lower ratio of RS/QRS width in lead V6 than those with VT. We measured ECG parameters of VT and SVT with aberrancy in patients who have WCT with RBBB pattern and R/S ratio in lead V6 <1.0 to differentiate these two types of tachycardias.

Methods

Study patients

We retrospectively reviewed the medical records of 111 patients who had WCT with RBBB pattern and R/S ratio in lead V6 <1.0 at the Asan Medical Center from June 2007 to December 2016. Among the 111 patients, 72 were diagnosed with VT after an electrophysiology study (fascicular VT in 39 and myocardial VT in 33), and 39 patients were diagnosed with SVT and aberrant ventricular conduction (AFL in 33 and AV reentrant tachycardia in 6). AV reentrant tachycardia was diagnosed by electrophysiology study. AFL with aberrancy was diagnosed when; 1) the morphology of the WCT introduced by atrial pacing was identical to that of clinical tachycardia (Figure 1A). This 'simulated tachycardia' was introduced by atrial overdrive pacing at the tachycardia cycle length after infusion of flecainide (2.0 mg/Kg over 10 minutes). If 2:1 AV block occurred, isoproterenol was infused to maintain 1:1 AV conduction; 2) the flutter wave was identified during the recovery phase of treadmill exercise test (Figure 1B); 3) intravenous infusion of adenosine (6-12mg) revealed flutter waves (Figure

1C).

The 12-lead ECGs of a WCT were analyzed and compared. This retrospective study was approved by the Institutional Review Board of the Asan Medical Center, which waived the requirement for individual informed consent because ECGs were acquired during routine clinical practice. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki, as reflected in a priori approval by the institution's human research committee.

Electrocardiography

The 12-lead ECG of WCT with RBBB pattern and R/S ratio in lead V6 <1.0 was used for analysis. ECGs were recorded at a gain of 10 mm/mV and paper speed of 25 mm/s. QTc interval was calculated by using the Bazett formula ($QTc = QT/\sqrt{RR}$). During WCT, QRS width in the precordial lead V6 was measured from the onset of R wave to the end of S wave, and RS interval was defined as the interval from the onset of QRS wave to the nadir of S wave in lead V6. RS/QRS ratio was defined as the value of RS interval divided by QRS width in lead V6. Two cardiologists (M. Kim and D.Y. Kang) independently measured these intervals.

An average of three measurements from stable and consecutive cardiac cycles was used for all analyses. The intra-observer and inter-observer variabilities of QRS width measurement demonstrated an intra-class correlation coefficient of 0.98 (95% confidence interval [CI] 0.94-0.99) and 0.98 (95% CI 0.95-0.99), respectively. The differences between the two observers were resolved by consensus.

To assess the diagnostic accuracy of previous studies, we applied the Brugada criteria,[6] the Vereckei criteria,[7] and R-wave peak time index at lead II.[8] Briefly, the Brugada criteria to differentiate WCT were as follow: 1) absence of an RS complex in all precordial leads, 2) RS interval > 100 ms in any precordial lead, 3) AV dissociation, and 4) morphology of QRS in leads V1,2 and V6.[6] The criteria suggested by Vereckei et al. were; 1) presence of an initial R wave, 2) width of an initial r or q wave >40 ms, 3) notching on the initial downstroke of a predominantly negative QRS complex, and 4) estimation of the voltage change during the initial (Vi) and terminal 40 ms (Vt) of the QRS complex. A Vi/Vt $\leq$1.0 was suggestive of VT.[7] The R-wave peak time was defined as QRS duration from the initiation of depolarization until the first change of the polarity, independent of whether the QRS deflection was positive or

negative. R-wave peak time ≥50 ms at lead II was determined to suggest VT.[8]

Statistical analysis

All statistical analyses were performed by using SPSS 21.0 (SPSS Inc., Chicago, IL). All continuous variables are presented as mean ± standard deviation (SD). Categorical variables are presented as frequencies (percentage). Continuous variables were compared using Student's *t* test, Mann-Whitney U test, and ANOVA. And categorical variables were compared using a chi-square or Fisher's exact test. Receiver operating characteristics (ROC) analysis was used to determine the optimal cutoff values of the continuous variables for differentiating VT from SVT with aberrancy. A *P* value <0.05 was considered statistically significant.

Result

Baseline characteristics

The baseline clinical characteristics of the two groups are shown in Table 1. Patients with VT were significantly younger than those with SVT with aberrancy. The proportion of structural heart disease in the VT group was higher than that in the SVT group. Left ventricular ejection fraction of the VT group was lower than that of the SVT group.

ECG characteristics between VT and SVT with aberrancy

Table 2 shows the comparisons of ECG characteristics between the VT and SVT groups. Heart rate, PR interval, QRS width and QTc interval during normal sinus rhythm were comparable between the two groups. During WCT, heart rate was significantly faster in the SVT with aberrancy group than in the VT group (200.2 ± 33.7 beats/min [bpm] vs 172.7 ± 29.4 bpm, P <0.001). The QRS width during tachycardia was not significantly different between the two groups.

Differential Diagnosis using previously published data

When the diagnostic values of previous criteria (Brugada criteria, Vereckei criteria, and R-wave peak time at lead II) were tested in our study subjects, the sensitivity and specificity for diagnosis of VT were only modest (sensitivity; 80.6% and specificity; 30.8% for Brugada criteria/sensitivity; 90.3% and specificity; 61.5% for Vereckei criteria/sensitivity; 48.6% and specificity; 92.3% for R-wave peak time at lead II, table 3, Figure 2). Myocardial VT was correctly diagnosed using the previously published criteria. The Brugada and Vereckei criteria correctly diagnosed all of the 33 patients with myocardial VT, and the R-wave peak time criterion identified 32 (97.0%) of 33 myocardial VT patients. However, SVT and fascicular VT were not differentiated adequately. Details on stepwise application of the previous criteria for differentiation of posterior type fascicular VT and aberrantly conducted SVT are summarized in Figures 2. The first three steps in the Brugada criteria failed to distinguish SVT and fascicular VT (Figure 2A and C). Both tachycardias were discriminated by morphologic criteria of the fourth step. Fourteen (35.9%) of 39 fascicular VTs were classified as SVT due to the triphasic pattern in lead V1, and 27 (69.2%) of 39 SVTs showed monophasic R, qR, or

Rr' pattern in lead V1 and misdiagnosed as VT (Table 4). In the Vereckei criteria, 12 (30.8%) of 39 SVTs showed dominant R wave in lead aVR and were misdiagnosed as VT. In the second step, 2 SVTs had a qR wave with q wave more than 40 ms in lead aVR. None of SVT patients had negative dominant pattern in lead aVR. In the fourth step, 1 SVT was misdiagnosed as VT due to Vi/Vt ratio was less than 1 (Figure 2D). In the case of fascicular VT, 7 (17.9%) of 39 fascicular VTs were classified as SVT because the initial velocity was fast and Vi/Vt ratio was >1.0 (Figure 2F). Regarding the R-wave peak time criteria, 36 (92.3%) of 39 fascicular VTs were misdiagnosed as SVT, and 3 of 39 SVTs were misdiagnosed as VT. Overall, diagnostic accuracies for differential diagnosis of SVT and fascicular VT was 47.4% for the Brugada criteria, and 71.8% for the Vereckei criteria. Diagnosis of fascicular VT using the R wave criteria was poor. (Table 3)

New ECG index for distinguishing VT and SVT with aberrancy

RS interval in lead V6 was significantly shorter and RS/QRS ratio in lead V6 was significantly lower in SVT with aberrancy than in VT group (58.4 ± 10.5 vs 81.0 ± 24.6, P <0.001/0.36 ±

0.04. vs 0.50 ± 0.08, *P* <0.001, Table 2). Figure 3A shows a WCT due to AFL with 1 to 1 conduction. An enlargement of lead V6 shows RS/QRS ratio = 0.36. Figure 3B shows a typical 12-lead ECG of a WCT due to fascicular VT. An enlargement of lead V6 shows RS/QRS ratio = 0.48. Figure 4A and B show scatter plot graphs comparing RS interval and RS/QRS ratio between patients with aberrantly conducted SVT and those with VT. Figure 4C shows ROC curve analysis. For differential diagnosis of VT (fascicular and myocardial) from SVT, the best cutoff value of RS/QRS ratio in lead V6 was >0.41, with a sensitivity of 97.2%, specificity of 89.7%, positive predictive value (PPV) of 94.6% and negative predictive value (NPV) of 94.6% (area under curve [AUC] = 0.98, 95% CI 0.93-0.99, *P* <0.001) (Table 3, Figure 4C). For differential diagnosis of fascicular VT from SVT, diagnostic accuracy of the RS/QRS ratio criteria was 92.3% (Table 3).

The cutoff value for lead V6 RS interval was 59 ms, with a sensitivity of 85.2% and specificity of 59.4% (AUC = 0.78, 95% CI 0.68-0.86, *P* <0.001). RS/QRS ratio was superior to RS interval in distinguishing SVT from VT.

3D activation mapping of the left ventricle was performed in a patient with 'simulated

tachycardia'. Ventricular activation sequence and AH/HV intervals were compared between sinus rhythm and 'simulated tachycardia'. In sinus rhythm, the activation sequence was earliest in mid to apical septum of the left ventricular wall. Earliest activation changed to the inferior and posterolateral left ventricular wall when RBBB pattern and reversed RS ratio in lead V6 appeared during 'simulated tachycardia' (Figure 5).

Discussion

We identified a simple ECG discriminator differentiating a WCT with RBBB pattern and a reversed R/S ratio in the precordial lead V6. The ratio of QRS waveform durations as measured from the QRS onset to the nadir of S wave divided by total QRS width in lead V6 was able to differentiate SVT with aberrancy from VT. High (>0.41) RS/QRS ratio in lead V6 successfully discriminated VT from SVT with a high sensitivity (97.2%), specificity (89.7%), PPV (94.6%), and NPV (94.6%), and it was particularly useful in differentiating SVT from fascicular VT.

According to the classical criteria of Wellens, WCT with RBBB pattern and reversed R/S ratio in lead V6 favors tachycardia of ventricular origin.[1] AFL converted from atrial fibrillation during AAD therapy sometimes show the ECG features of RBBB pattern WCT with a reversed precordial R/S ratio. This aberrantly conducted SVT is extremely difficult to differentiate from VT, particularly from idiopathic fascicular VT of posterior type. Previous ECG criteria for differential diagnosis of SVT from VT are of limited clinical value because they included VTs mainly from patients with structural heart diseases.

Brugada's stepwise approach for the diagnosis of VT was only modestly effective (sensitivity; 80.6%, specificity; 30.8%, PPV; 68.2%, NPV; 46.2%). Diagnostic accuracy of the criteria was even worse (47.4%) for detection of fascicular VT from SVT (sensitivity; 64.1%, specificity; 30.8%, PPV; 48.1%, NPV; 46.2%) (Table 3). That is, the first step was passed because all tachycardias of our study had RS complex in precordial leads. In the second step, prolonged (>100 ms) RS interval criteria failed to distinguish between fascicular VT and SVT because all study patients of SVT and fascicular VT had RS interval ≤100 ms. In a previous study, RS interval was measured <80 ms in all precordial leads in all fascicular VT cases.[9] The third step of AV dissociation was rare in our patient cohort. There was no AV dissociation in patients with fascicular VT or SVT. AV dissociation was observed in only 10 of 33 myocardial VT patients. In morphologic criteria, typical triphasic RBBB pattern in lead V1 was recorded in 14 (35.9%) of 39 fascicular VTs, rendering about 1/3 of fascicular VT classified as SVT (Table 4). This is in contrast to the fact that all 33 patients of myocardial VT were correctly diagnosed as VT by the Brugada criteria. Sixteen of 33 myocardial VTs had RS interval of >100 ms. Five of the remaining 17 patients had AV dissociation. The final 12 myocardial VTs were diagnosed

as VT according to morphologic criteria. In patients with SVT, monophasic R, qR, or Rr' pattern in lead V1 were recorded in 27 (69.2%) of 39 SVTs rendering about 2/3 of SVTs classified as VT (Figure 2, Table 4). Figure 6 shows representative examples of misdiagnosis of both tachycardias.

Like the Brugada criteria, the Vereckei criteria also had a low differential test accuracy (sensitivity; 90.3%, specificity; 61.5%, PPV; 81.3%, NPV; 77.4%). The electrophysiologic basis of Vereckei finding was related to the different conduction velocities that occur in SVT (using the fast conducting His–Purkinje system) compared to VT (using the ventricular myocardium). Because, the mechanism of fascicular VT involves the His-Purkinje system, the initial ventricular activation in fascicular VT is rapid. The QRS duration of fascicular VT ranges 100 to 150 ms[9-12] and precordial RS intervals range 60 to 80 ms.[9] In 39 fascicular VT patients, 4 patients had initial R wave and 35 patients had qR pattern in lead aVR. Only one patient with qR pattern had q wave duration of >40 ms in lead aVR. Negative dominant pattern in lead aVR was not found. Only 5 of 39 fascicular VTs were correctly diagnosed as VT from the first three steps of the Vereckei criteria. In the final fourth step, 34 of 39 fascicular VT

patients were diagnosed according to Vi/Vt ratio. Fast initial QRS complex of fascicular VT led to misdiagnosis in 7 patients with fascicular VT (Figure 2F). In cases of SVT with aberrancy, 12 of 39 SVTs showed dominant R wave in lead aVR, and were wrongly classified as VT (Figure 2D). As already mentioned in their results,[7] the Vereckei criteria appeared to have only modest diagnostic value to discriminate this type of WCT; sensitivity (82.1%), specificity (61.5%), PPV (68.1%) and NPV (77.4%).

R wave peak time criterion also showed inadequate diagnostic accuracy for detection of VT (sensitivity; 48.6%, specificity; 92.3%, PPV; 92.1%, NPV; 49.3%). Similar to the other criteria, it was even poorer for differentiation of fascicular VT from SVT. (Table 3) In detail, almost all fascicular VT and SVT patients showed R-wave peak time <50 ms at lead II. Thirty-six (92.3%) of 39 SVT patients had R-wave peak time of <50 ms at lead II. Thirty-six (92.3%) of 39 fascicular VTs were misdiagnosed as SVT due to R-wave peak time of <50 ms at lead II. Fascicular VTs were incorrectly classified as SVT (sensitivity; 7.7%, specificity; 94.4%, PPV; 60.0%, NPV; 48.6%) (Table 3).

Class Ic AADs result in a slower flutter rate, at about 200 bpm rather than 250 bpm; this

facilitates 1:1 conduction through the AV node, resulting in a rapid ventricular response and potential cardiovascular compromise.[13,14] The explanation for the bizarrely shaped bundle branch block (BBB) during treatment with class Ic AAD, particularly flecainide, is yet uncertain.[15] In our patient in whom a 3D activation mapping was performed, the earliest ventricular activation shifted to the infero-postero-lateral left ventricular wall. This is in accordance with the dramatic ECG changes of RBBB in lead V1 and reversed RS ratio in lead V6, where the earliest ventricular intrinsicoid deflection is recorded. It is presumed that, in the presence of class Ic AADs, the normal septal activation initiated by the left anterior and posterior fascicle (and thus earliest in the left septum) may change to different sequence mediated by different Purkinje fibers other than those anterior/posterior fascicles. Flecainide-induced conduction delay has been shown to be more pronounced in the ventricular myocardium than in the His-Purkinje system.[16] Conduction is relatively less altered in areas activated by way of the His-Purkinje system, preserving the initial part of the QRS complex less affected. This may result in an exaggerated asynchrony of activation of the various parts of the heart, giving rise to the bizarre type of BBB.[15] As a result, combination of preserved

sharp initial part of QRS complex and widening of total QRS width probably make RS/QRS ratio lower in aberrantly conducted SVT than in fascicular or myocardial VT.

All SVT and VT patients of our study showed dominant R or qR pattern in lead aVR. Accordingly, in case with dominant R or qR in lead aVR along with reversed RS ratio in lead V6, we propose that applying the RS/QRS ratio in lead V6 can be critical to differentiate a WCT correctly.

In 8 of 33 AFL patients, tachycardias showed 1:1 AV conduction and bizarre QRS morphology even without taking AAD. Among them, 4 patients had structural heart diseases (non-ischemic cardiomyopathy, ischemic cardiomyopathy, cardiac sarcoidosis, and aortic valve replacement due to bicuspid aortic valve with significant stenosis). Of the 4 patients without structural heart disease, one patient had been on risperidone which has the sodium channel blocking property, one patient had RBBB in the baseline ECG, one patient suffered from acute respiratory disease syndrome due to sepsis, and the last one had no remarkable clinical finding.

Our present study showed that the tachycardia cycle length of SVT with aberrancy was

significantly faster than in VT (200.2 ± 33.7 bpm vs 172.7 ± 29.4 bpm, P <0.001). A mean tachycardia rate of VT is usually below 200 bpm. In the presence of class Ic AADs, decreased AFL rate to around 200 bpm can lead to 1:1 AV conduction in the absence of AV nodal blocker.[17,18] For this reason, it is recommended that AV nodal blocking drugs be used to prevent 1:1 AV conduction during class Ic AAD therapy. It is noteworthy that in 5 out of 33 AFL patients, 1:1 AV conduction occurred in the presence of concomitant AV nodal blocking agents.

Four of 39 SVT patients showed RS/QRS ratio >0.41 in lead V6. These 4 SVTs were AFL with aberrant conduction during class Ic AAD therapy. Among them, 2 patients had preexisting RBBB and one patient had wide QRS width of 138 ms during sinus rhythm. The reason for the prolonged RS/QRS ratio is not clear. Preexisting conduction disturbance of the His-Purkinje system might have caused, in part, decreased initial deflection in these 4 patients.

There are several limitations. This study involved a relatively small number of patients. Despite RS/QRS ratio in lead V6 has a relatively high diagnostic value to distinguish aberrantly conducted SVT from VT, our criterion failed to distinguish them in a small subset

of patients. Therefore, electrophysiology study is needed to determine the mechanism of these WCT. In borderline cases, overdrive atrial pacing under the infusion of flecainide and isoproterenol might reproduce QRS patterns of the tachycardia (simulated tachycardia), and provide useful information on differential diagnosis of the two tachycardias. Our study might be more useful when RS/QRS ratio in lead V6 is ≤0.41 and other clinical features including coronary artery status, arrhythmia, and medication history favor SVT with aberrancy, in facilitating performance of carotid sinus massage or adenosine injection for differential diagnosis of the two types of WCT.

Conclusion

In differential diagnosis of a WCT with RBBB pattern and reversed R/S ratio in the precordial lead V6, V6 RS/QRS ratio is a useful ECG marker to distinguish SVT with aberrancy from VT including posterior type fascicular VT.

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Figure legends

Figure 1. Atrial flutter with aberrancy was confirmed by the following methods. Figures in the left panel (A-1, B-1, and C-1) show 12-lead ECGs of clinical wide QRS complex tachycardia (WCT). (A-2) In the electrophysiology study, atrial overdrive pacing with intravenous flecainide (150 mg over 10 min) and isoproterenol (5 microgram/min) infusion reproduced WCT with identical QRS morphology. This 'simulated tachycardia' by atrial pacing was considered a strong evidence that the original tachycardia was supraventricular origin. The detailed protocol was described in the methods. (B-2) During the treadmill test, a WCT was induced and slowing of the ventricular rate during recovery was paralleled by narrowing of the QRS complex, suggesting that the diagnosis of WCT was atrial flutter. (C-2) After adenosine injection, flutter waves were clearly visible.

Figure 2. Stepwise application of the Brugada and Vereckei criteria for supraventricular tachycardia (SVT) with aberrancy (2A, 2D), all ventricular tachycardia (VT, 2B, 2E), and

fascicular VT (2C, 2F).

Figure 3. (A) A 12-lead ECG from a patient with atrial flutter with aberrancy and, magnified lead V6. RS/QRS ratio was 0.36. (B) A 12-lead ECG from a patient with fascicular ventricular tachycardia and, magnified lead V6. RS/QRS ratio was 0.48.

Figure 4. (A) Scatter plot comparing RS interval in lead V6 for supraventricular tachycardia (SVT) and ventricular tachycardia (VT). (B) Scatter plot comparing RS/QRS ratio in lead V6 for SVT and VT. Lines denote mean value with standard deviation ranges. (C) Receiver operating characteristic curve showing sensitivity and 1‑specificity of RS/QRS ratio >0.41. AUC, area under curve; CI, confidence interval.

Figure 5. Activation map of the left ventricular wall and the intracardiac electrograms during sinus rhythm and 'simulated tachycardia' (A) The earliest left ventricular activation was observed in the mid septum during sinus rhythm. (B) During atrial overdrive pacing under

isoproterenol and flecainide, QRS morphology changed to a wide QRS complex with RBBB pattern and reversed R/S ratio in lead V6. The earliest left ventricular activation was observed in the infero-postero-lateral wall. (C, D) The AH/HV intervals were 155/43 ms for sinus rhythm and 191/58 ms for 'simulated tachycardia', respectively. The HV intervals measured on the left side septum were only mildly prolonged while QRS duration was dramatically prolonged in the wide QRS complex. Yellow dot=His potential; Purple dot=Purkinje potential; RAO=right anterior oblique view; PA=posteroanterior view; HRA=high right atrium; CS=coronary sinus; p=proximal; d=distal.

Figure 6. Two representative examples of a misdiagnosis by previous criteria. (A) Atrial flutter (AFL) was misdiagnosed as ventricular tachycardia (VT) according to dominant R wave in lead aVR of the Vereckei criteria. When the Brugada algorithm was applied, first three steps could not discriminate the tachycardia origin. In the fourth step, AFL was misdiagnosed as VT according to the morphologic criteria. RS and QRS intervals were 64 and 163 ms, respectively, and the RS/QRS ratio of 0.39 indicated supraventricular tachycardia (SVT). (B) Fascicular VT

has qR pattern with q wave less than 40 ms in lead aVR. Fascicular VT was misdiagnosed as SVT according to Vi/Vt ratio of the Vereckei criteria. In the Brugada criteria, as in the case of AFL, the first three steps were not applicable. Fascicular VT was misdiagnosed as SVT according to the fourth step (morphologic criteria). RS and QRS intervals were 59 and 140 ms, respectively, and the RS/QRS ratio of 0.42 indicated VT.

Supplementary Figure. Scatter plot comparing RS/QRS ratio at lead V6 for supraventricular tachycardia (SVT), fascicular ventricular tachycardia (VT), and myocardial VT. When analyzed separately, fascicular VT showed ECG features that was intermediate between SVT and myocardial VT. Lines denote mean value with standard deviation ranges.

Table 1. Clinical characteristics of the study patients

Variables	SVT group (n = 39)	VT group (n = 72)	*p* value
Age, yrs	56.9±16.2	47.1±17.6	0.005
Male sex	29 (74.4%)	58 (80.6%)	0.45
Body mass index, kg/m^2	23.8±2.8	23.7±3.3	0.87
Diabetes	5 (12.8%)	9 (12.5%)	0.96
Hypertension	11 (28.2%)	16 (22.2%)	0.48
Chronic kidney disease, <60 ml/min	3 (7.7%)	10 (13.9%)	0.54
Coronary artery disease	3 (7.7%)	16 (22.2%)	0.052
Structural heart disease	8 (20.5%)	32 (44.4%)	0.014
Left ventricular ejection fraction, %	57.4±9.6	48.2±16.0	<0.001
Antiarrhythmic drug	25 (64.1%)	15 (20.8%)	<0.001
Flecainide	19 (48.7%)		
Propafenone	6 (15.4%)		
Amiodarone	0	12 (16.7%)	
Sotalol	0	3 (4.2%)	

Values are expressed as the mean ± SD or n (%).

SVT, supraventricular tachycardia; VT, ventricular tachycardia.

Table 2. Comparison of ECG parameters between patients with SVT and VT

Variables	SVT group (n = 39)	VT group (n = 72)	*p* value
Sinus rhythm			
Heart rate, bpm	69.5±19.3	69.9±12.8	0.90
PR interval, ms	169.8±29.2	177.2±33.4	0.27
QRS width, ms	107.1±20.2	106.5±22.3	0.89
QTc interval, ms	441.9±34.2	442.6±45.5	0.93
Tachycardia			
Heart rate, bpm	200.2±33.7	172.7±29.4	<0.001
QRS width, ms	161.7±22.1	159.5±27.8	0.68
RS interval, ms	58.4±10.5	81.0±24.6	<0.001
RS/QRS ratio	0.36±0.04	0.50±0.08	<0.001

Values are expressed as the mean ± SD or n (%).

SVT, supraventricular tachycardia; VT, ventricular tachycardia.

Table 3. Diagnostic accuracy of RS/QRS ratio in comparison with previously published criteria

	SVT group (n=39)	VT group (n=72)	Fascicular VT group (n=39)
Diagnosis of WCT according to Brugada criteria	Dx as SVT: 12 (30.8%) Dx as VT: 27 (69.2%)	Dx as SVT: 14 (19.4%) Dx as VT: 58 (80.6%) (SN 80.6%, SP 30.8%, PPV 68.2%, NPV 46.2%)	Dx as SVT: 14 (35.9%) Dx as VT: 25 (64.1%) (SN 64.1%, SP 30.8%, PPV 48.1%, NPV46.2%)
Diagnosis of WCT according to Vereckei criteria	Dx as SVT: 24 (61.5%) Dx as VT: 15 (38.5%)	Dx as SVT: 7 (9.7%) Dx as VT: 65 (90.3%) (SN 90.3%, SP 61.5%, PPV 81.3%, NPV 77.4%)	Dx as SVT: 7 (17.9%) Dx as VT: 32 (82.1%) (SN 82.1%, SP 61.5%, PPV 68.1%, NPV 77.4%)
Diagnosis of WCT according to R wave criteria	Dx as SVT: 36 (92.3%) Dx as VT: 3 (7.7%)	Dx as SVT: 37 (51.4%) Dx as VT: 35 (48.6%) (SN 48.6%, SP 92.3%, PPV 92.1%, NPV 49.3%)	Dx as SVT: 36 (92.3%) Dx as VT: 3 (7.7%) (SN 7.7%, SP 94.4%, PPV 60.0%, NPV 48.6%)
Diagnosis of WCT according to RS/QRS ratio criteria	Dx as SVT: 35 (89.7%) Dx as VT: 4 (10.3%)	Dx as SVT: 2 (2.8%) Dx as VT: 70 (97.2%) (SN 97.2%, SP 89.7%, PPV 94.6%, NPV 94.6%)	Dx as SVT: 2 (5.1%) Dx as VT: 37 (94.9%) (SN 94.9%, SP 89.7%, PPV 90.2%, NPV 94.6%)

SVT, supraventricular tachycardia; VT, ventricular tachycardia (both myocardial and fascicular VT); Dx, diagnosis; SN, sensitivity; SP, specificity; PPV, positive prediction value; NPV, negative prediction value.

Table 4. Morphologic characteristics of SVT with aberrancy and fascicular VT in lead V1 (Fourth step of the Brugada criteria)

Lead V1	SVT group (n = 39)	Fascicular VT group (n = 39)
Monophasic R	13 (33.3%)	5 (12.8%)
Rr’	2 (5.1%)	1 (2.6%)
qR	12 (30.8%)	19 (48.7%)
Rs	0	0
Triphasic (typical RBBB)	12 (30.8%)	14 (35.9%)

Values are expressed as n (%).

SVT, supraventricular tachycardia; VT, ventricular tachycardia; RBBB, right bundle branch block.

Supplementary Table. Summary of ECG analysis comparing between SVT, fascicular VT, and myocardial VT

Variables	SVT group (n = 39)	Fascicular VT group (n = 39)	Myocardial VT group (n = 33)	*P* value
Tachycardia				
Heart rate, bpm	200.2±33.7	177.4±29.1	167.0±29.2	0.001
QRS width, ms	161.7±22.1	141.3±13.7	181.1±24.7	<0.001
RS interval, ms	58.4±10.5	64.6±6.4	100.3±24.2	<0.001
RS/QRS ratio†	0.36±0.04	0.46±0.03	0.55±0.09	<0.001

Values are expressed as the mean ± SD or n (%).

SVT, supraventricular tachycardia; VT, ventricular tachycardia.

†Post hoc analysis showed that all settings were *p* value of less than 0.001 from each other.

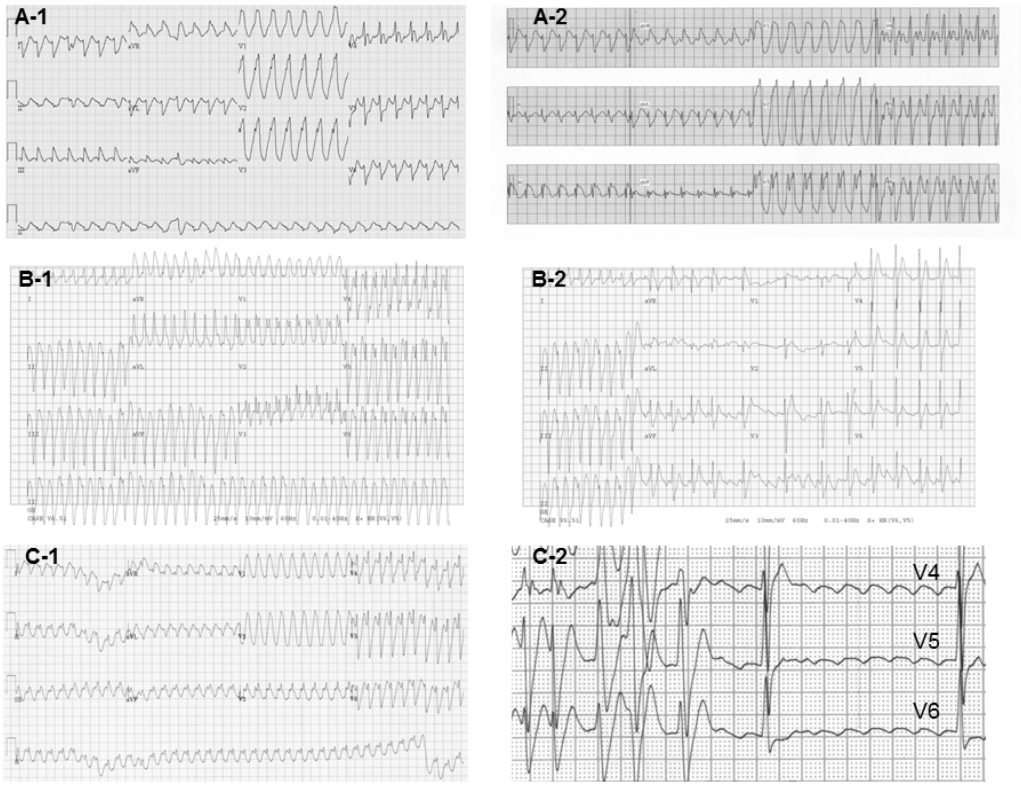


Fig 1. Atrial flutter with aberrancy was confirmed by the following methods. Figures in the left panel (A-1, B-1, and C-1) show 12-lead ECGs of clinical wide QRS complex tachycardia (WCT). (A-2) In the electrophysiology study, atrial overdrive pacing with intravenous flecainide (150 mg over 10 minutes) and isoproterenol (5 microgram/min) infusion reproduced WCT with identical QRS morphology. This "simulated tachycardia" by atrial pacing was considered a strong evidence that the original tachycardia was supraventricular origin. The detailed protocol was described in the methods. (B-2) During the treadmill test, a WCT was induced and slowing of the ventricular rate during recovery was paralleled by narrowing of the QRS complex, suggesting that the diagnosis of WCT was atrial flutter. (C-2) After adenosine injection, flutter waves were clearly visible.

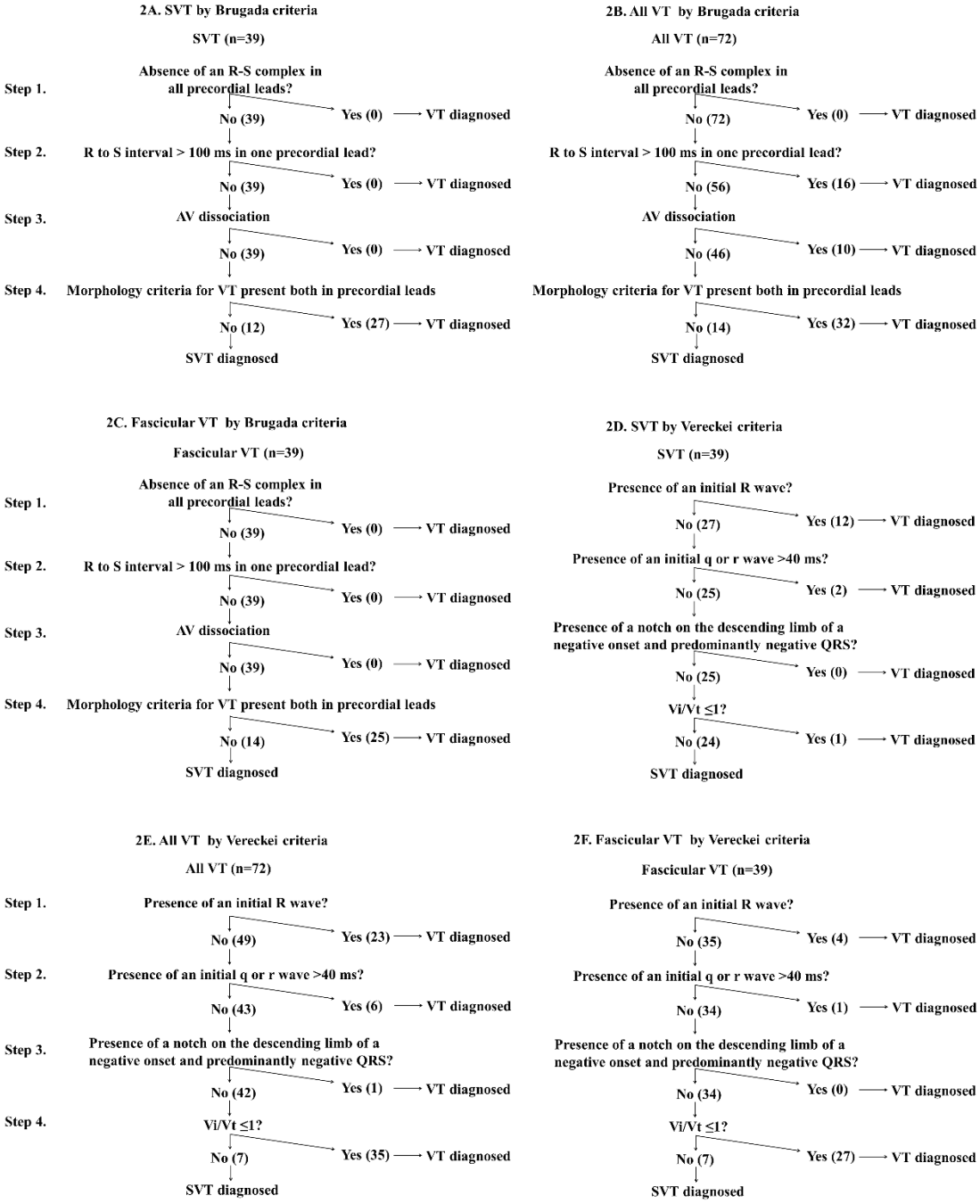


Fig 2. Stepwise application of the Brugada and Vereckei criteria for supraventricular tachycardia (SVT) with aberrancy (2A, 2D), all ventricular tachycardia (VT, 2B, 2E), and fascicular VT (2C, 2F).

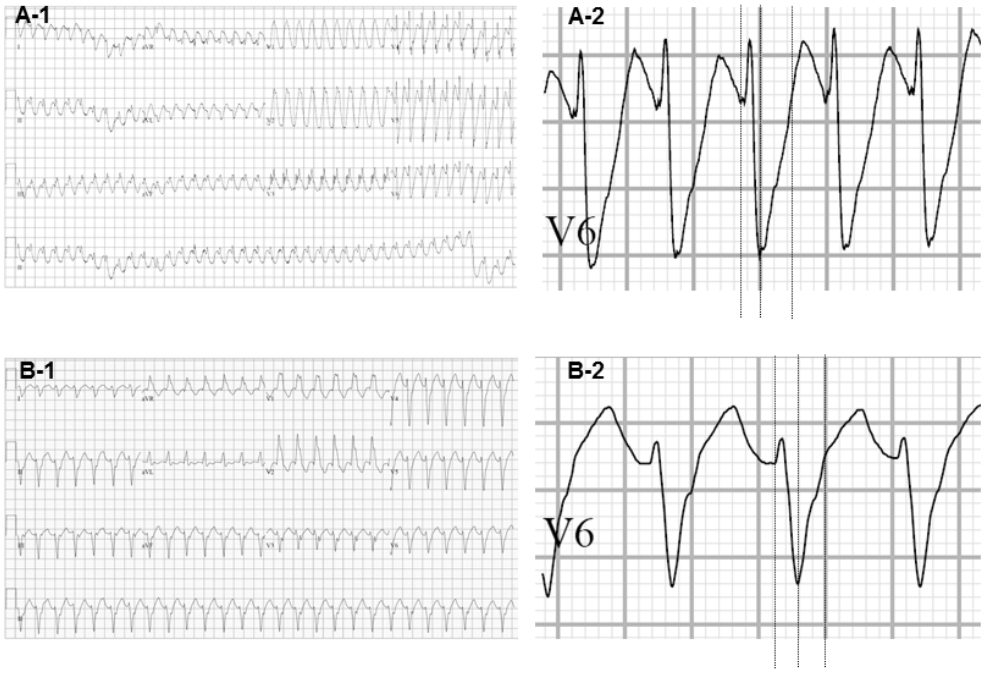


Fig 3. A-1: A 12-lead ECG from a patient with atrial flutter with aberrancy. A-2: Magnified view of the lead V6 from the ECG of panel A-1. RS/QRS ratio was 0.36. B-1: 12-lead ECG from a patient with fascicular ventricular tachycardia. B-2: Magnified view of the lead V6 from panel B-1. RS/QRS ratio was 0.48.

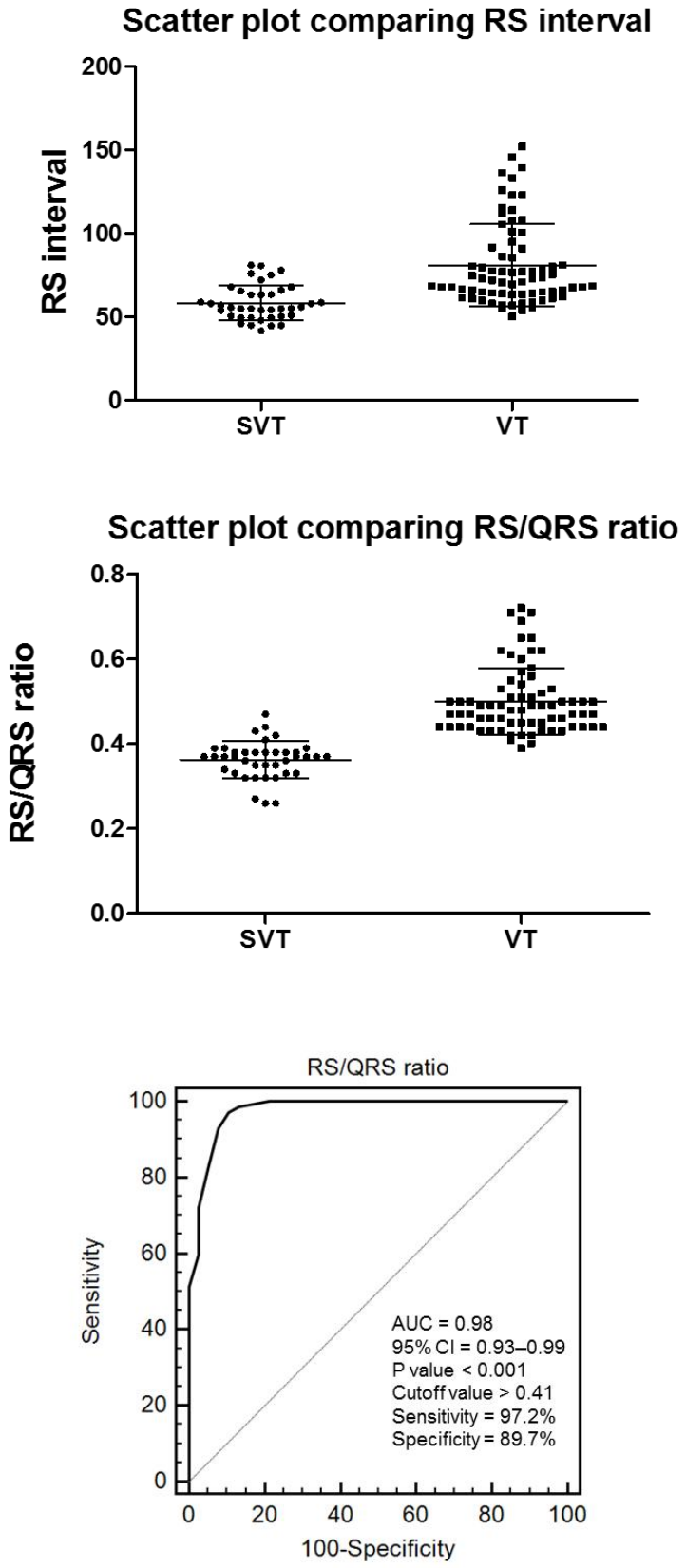


Fig 4. (A) Scatter plot comparing RS interval in the lead V6 for supraventricular tachycardia (SVT) and ventricular tachycardia (VT). (B) Scatter plot comparing RS/QRS ratio in the lead V6 for SVT and VT.

Lines denote mean value with standard deviation ranges. (C) Receiver operating characteristic curve showing sensitivity and 1‐specificity of RS/QRS ratio >0.41. AUC, area under curve; CI, confidence interval.

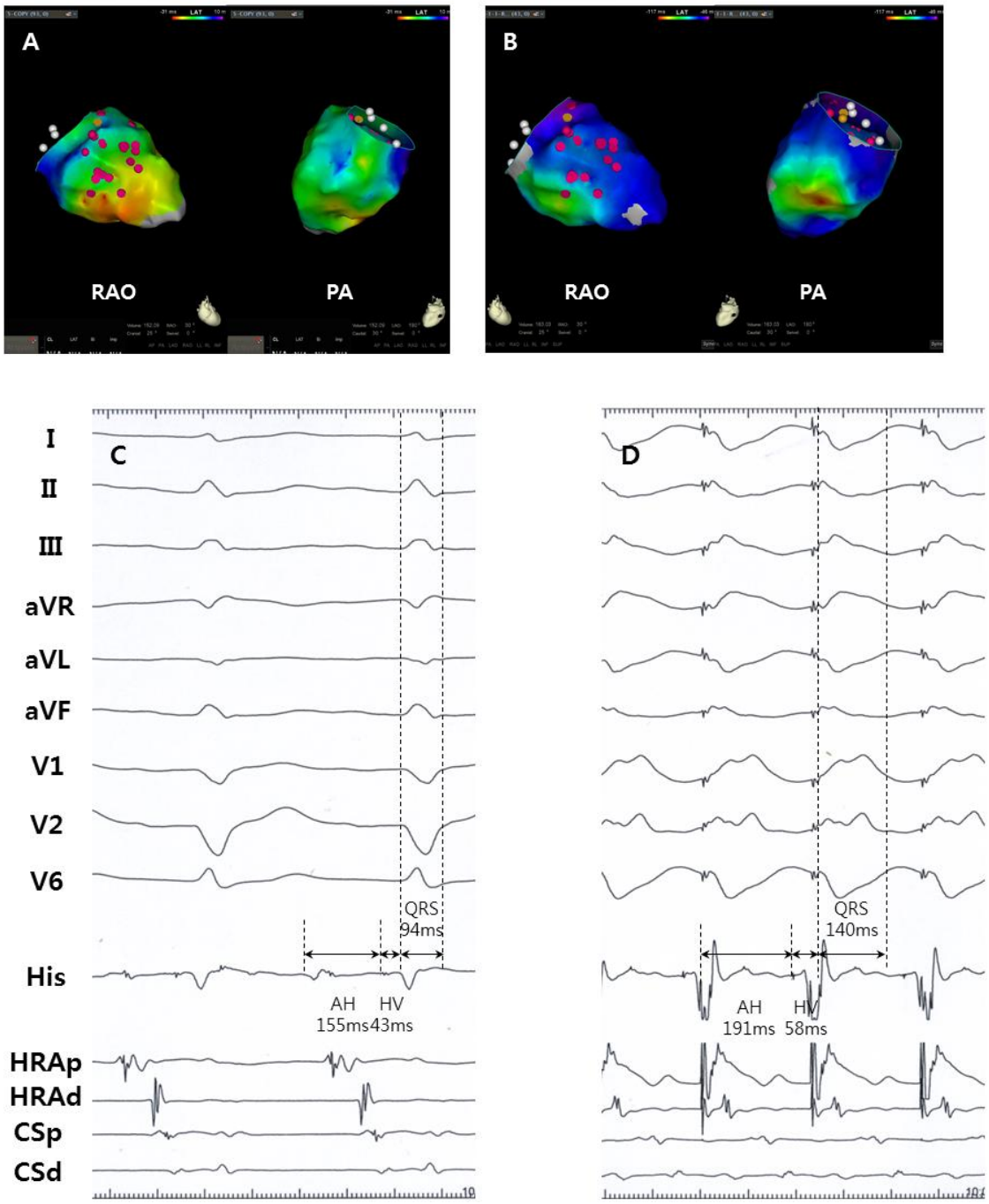
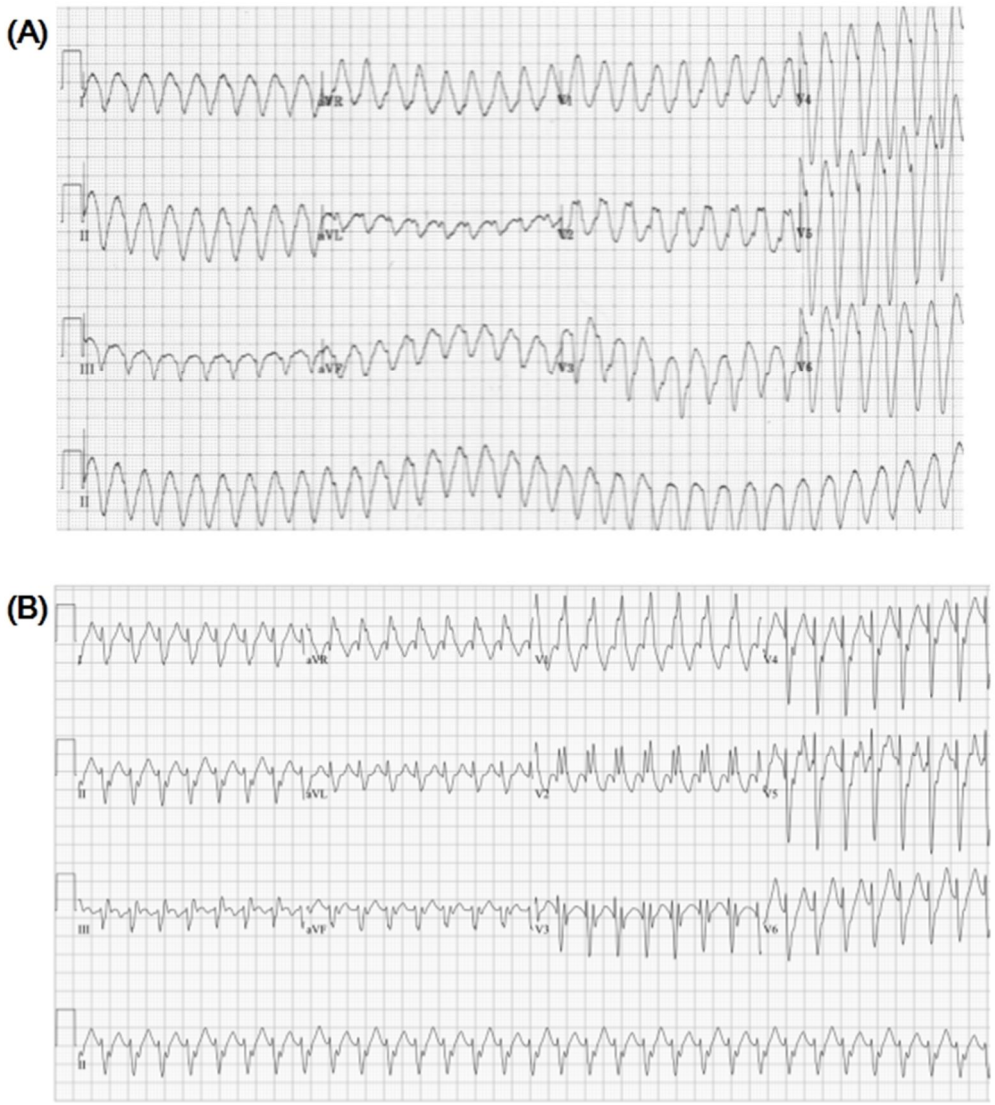


Fig 5. Activation map of the left ventricular wall and the intracardiac electrograms during sinus rhythm and 'simulated tachycardia' (A) The earliest left ventricular activation was observed in the mid septum during sinus rhythm. (B) During atrial overdrive pacing under isoproterenol and flecainide, QRS morphology changed to a wide QRS complex with RBBB pattern and reversed R/S ratio in lead V6. The earliest left ventricular activation was observed in the infero-postero-lateral wall. (C, D) The AH/HV intervals were 155/43 ms for sinus rhythm and 191/58 ms for 'simulated tachycardia',

respectively. The HV intervals measured on the left side septum were only mildly prolonged while QRS duration was dramatically prolonged in the wide QRS complex. Yellow dot=His potential; Purple dot=Purkinje potential; RAO=right anterior oblique view; PA=posteroanterior view; HRA=high right atrium; CS=coronary sinus; p=proximal; d=distal.



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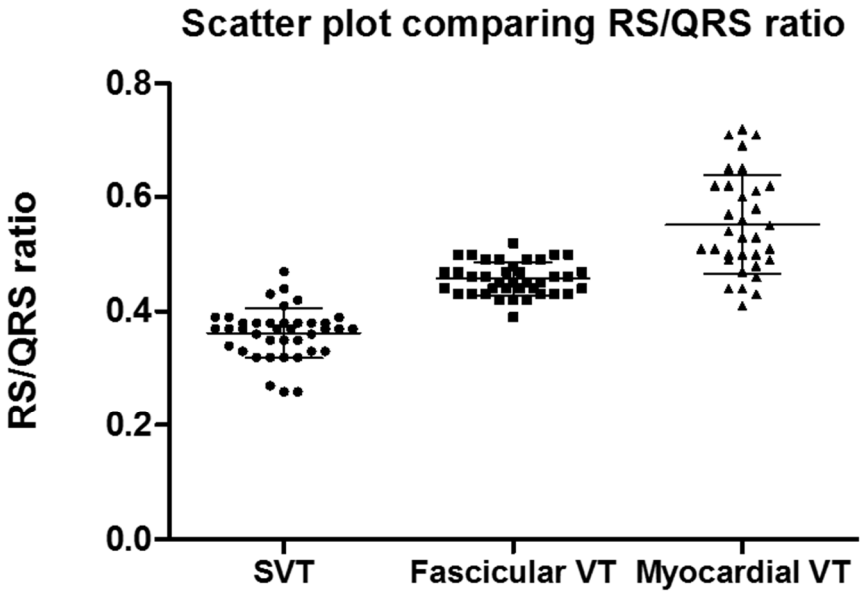

Supplementary Fig. Scatter plot comparing RS/QRS ratio at lead V6 for supraventricular tachycardia (SVT), fascicular ventricular tachycardia (VT), and myocardial VT. When analyzed separately, fascicular VT showed ECG features that was intermediate between SVT and myocardial VT. Lines denote mean value with standard deviation ranges.

국문요약

연구 목적

상심실성 빈맥은 넓은 QRS 복합성 빈맥으로 나타날 수 있다. 우각차단 형태의 상심실성 빈맥과 심실성 빈맥의 감별 진단은 흉부유도 V6에서 R/S 비가 1.0 미만일 때 특히 어렵다.

연구 방법

우리는 흉부유도 V6에서 R/S 비가 1.0 미만인 우각차단 형태의 넓은 QRS 복합성 빈맥, 총 111명(심실성 빈맥 72명, 상심실성 빈맥 39명)의 환자로부터 심전도 매개 변수를 조사하였다. 심방 조동의 진단은 아데노신 정맥주입 또는 운동부하검사 동안 다양한 방실 전도를 보이는 조동파가 확인 되는 경우와 임상적 빈맥의 주기로 심방을 고박동조율 하였을 때 넓은 QRS 복합성 빈맥이 유도되는 경우로 하였다.

이전 알고리즘 진단 기준들과 QRS 시작 시점에서 S 파의 최하점까지의 QRS 파형 지속 시간을 전체 QRS 폭으로 나눈 QRS 파형 지속시간의 비율로 정의 된 우리의 새로운 기준, RS/QRS 비를 비교하였다.

연구 결과

이전 기준들(Brugada 기준, Vereckei 기준, 사지유도 Ⅱ에서 R파 정점까지 시간)의 진단 정확도(민감도; 80.6%, 90.3%, 45.6%, 특이도; 30.8%, 61.5%, 94.4%)는 낮았다. 하지만, 흉부유도 V6에서 RS/QRS 비는 상심실성 빈맥에서 심실성 빈맥보다 유의하게 낮았으며(0.36 ± 0.04 대 0.50 ± 0.08, P <0.001), Receiver Operating Characteristic 곡선 분석에서 절단값, RS/QRS 비 >0.41은 높은 진단 정확도(민감도 97.2%, 특이도 89.7%)로 넓은 QRS 복합성 빈맥을 감별 진단하였다.

결론

흉부유도 V6에서 RS/QRS 비 >0.41은 흉부유도 V6에서 R/S 비가 1.0 미만인 우각차단 형태의 넓은 QRS 복합성 빈맥 환자에서 상심실성 빈맥과 심실성 빈맥을 감별할 수 있는 간단하고 신뢰할 수 있는 지수이다.