QRS Voltage Changes in SVT. Introduction: The aim of this study was to evaluate the changes in ventricular complex voltage associated with narrow QRS supraventricular tachycardia (SVT).

Methods and Results: One hundred forty-five patients undergoing catheter ablation for SVT, 85 with AV nodal reentrant tachycardia (AVNRT) and 60 with AV reentrant tachycardia (AVRT) due to a concealed accessory pathway, were studied. Four consecutive tachycardia beats and four consecutive sinus beats were analyzed, excluding the last tachycardia complex and the first sinus one. For each of the 12 leads, the QRS complex voltage was measured, and the results of four beats were averaged both in SVT and in sinus rhythm (SR). The sum (Σ) of the QRS voltages measured in the 12 leads during SVT (ΣSVT) and SR (ΣSR) were calculated, as well as the QRS axis during SVT and SR. QRS complex voltage was significantly increased during SVT, with respect to SR, in leads II, III, aVR, aVF, and V2 to V6. In addition, ΣSVT was significantly greater than ΣSR. Only lead V1 showed a significant voltage decrease during SVT. These voltage changes were almost identical in patients with AVNRT and patients with AVRT. No relationship was found between tachycardia rate and QRS voltage variation. The QRS axis showed a significant shift during SVT, from 55.8° to 64.5°.

Conclusion: QRS voltage increase occurs in reentrant SVT, independent of the underlying reentrant circuit. The phenomenon likely depends on tachycardia-related reduced ventricular filling. This could result in displacement of the heart in such a way that the left ventricle becomes closer to the precordial electrodes (proximity effect). Alternatively, decreased intracavitary blood mass could diminish the intracardiac short-circuiting of potentials, resulting in augmented transmission of cardiac vectors to the body surface. (J Cardiovasc Electrophysiol, Vol. 12, pp. 1358-1362, December 2001)

Brody effect, electrocardiogram, QRS complex, supraventricular tachycardia

Introduction

Paroxysmal reentrant tachycardia of supraventricular origin usually is associated with narrow QRS complexes that, in the absence of aberrant conduction, are identical to those in sinus rhythm (SR). It was reported recently that, during supraventricular tachycardia (SVT), QRS complex voltage is increased, with respect to SR, in precordial leads V2 to V6. The aim of the present study was to compare QRS voltages during SVT and SR in all 12 leads and to assess whether tachycardia-dependent QRS voltage changes are related to heart rate.

Methods

One hundred forty-five patients undergoing electrophysiologic study and catheter ablation for SVT were enrolled. There were 62 males (43%) and 83 females (57%) (age range 13 to 65 years, average 33.4 ± 12). In each patient, a sustained (>30 sec) reentrant SVT, either AV nodal reentrant tachycardia (AVNRT) or AV reentrant tachycardia (AVRT) due to a concealed accessory pathway, was initiated by programmed stimulation; the arrhythmia ceased spontaneously or after premature stimuli. The QRS complexes were narrow in any patient both in SR and during SVT. Cases showing bundle branch block, aberrant conduction, overt preexcitation, or QRS complex alternans during tachycardia were excluded. Attention was paid to ascertain that QRS duration was identical in SVT and in SR, in order to rule out even minimal degrees of tachycardia-dependent aberrant conduction.

In all patients, a 12-lead ECG at 25 mm/sec was available, including the final part of SVT and the beginning of SR. In all tracings, the tachycardia termination was selected. Four consecutive tachycardia beats and four consecutive sinus beats were analyzed, excluding the last tachycardia complex and the first sinus one. Whenever tachycardia was terminated with premature stimuli, paced beats were not considered for analysis. Escape beats or extrasystoles following tachycardia also were not taken into account. For each of the 12 leads, the QRS complex voltage (in millivolts) was measured manually as the sum of absolute voltages of any positive or negative deflection. Results of four consecutive beats were averaged both in SVT and in SR. For each tracing, we also calculated the QRS axis in SVT and in SR using the method proposed by Laiken et al. Axis values were obtained by averaging the results of three separate determinations, each based on two frontal plane orthogonal leads (I-aVF, II-aVL, III-aVR). QRS duration was measured in SVT and in SR using electronic calipers.
directly on the screen of the physiologic recorder device at a tracing speed of 100 mm/sec. The sum of the QRS voltages (Σ) measured in the 12 leads during SVT (ΣSVT) and SR (ΣSR) was calculated in all patients. QRS voltages, axes, and durations obtained during SVT were compared with those in SR. Statistical analysis was performed by t-test for paired data. P < 0.05 was considered statistically significant.

Results

The mechanism of SVT was AV nodal reentry in 85 cases and AV reentry due to a concealed accessory pathway in 60 cases. The tachycardia rate ranged from 125 to 250 beats/min (average 181.4 ± 28.4). Mean rate was 180.5 ± 20.2 for AVNRT and 182.0 ± 27.7 for AVRT (P NS). Considering the whole group of patients, we observed that, during SVT, the QRS complex voltage in leads II, III, aVR, aVF, and V2 to V6 was significantly increased with respect to SR. In contrast, no voltage variation occurred in leads I and aVL, and only lead V1 showed a significant voltage decrease during SVT (Fig. 1). In addition, ΣSVT was 20.14 ± 4.7 mV and significantly greater than ΣSR, which measured 18.04 ± 4.4 mV (P < 0.001). Representative ECGs showing QRS voltage changes during SVT are shown in Figures 2 and 3.

Comparing the two groups of patients (AVNRT vs AVRT), the only difference was evident in V1, where QRS voltage reduction during tachycardia was slightly but significantly greater (P < 0.05) in AVRT than in AVNRT (Fig. 4).

The sum of QRS voltages was increased during SVT by >5% in 104 patients, unchanged or increased by <5% in 20 patients, and decreased in 21 patients. In the last subgroup, the average QRS voltage reduction during tachycardia was 6.9% ± 4.8%. The 21 patients showing QRS voltage decrease during tachycardia did not differ from the remaining ones: tachycardia mechanism was AVNRT in 12 subjects and AVRT in 9. Mean tachycardia rate was 169.14 ± 26.3, i.e., slightly less than the total average rate (181.4 ± 28.4) but not significantly different from it (P = NS).

A significant (P < 0.001) QRS axis shift was observed during SVT with respect to SR (Table 1). Considering the entire group, the axis changed from 64.5° ± 18.7° to 55.8° ± 21.9°, with a similar behavior for AVNRT and AVRT. There was no QRS duration change for both the whole group and for the AVNRT and AVRT groups (Table 1).

A search was made for possible correlation between QRS voltage change and tachycardia rate. For each of the 12 leads and for Σ, heart rate was plotted against absolute voltage change (millivolts) and against the percentage voltage change. No statistically significant relationship was found between tachycardia rate and QRS voltage variation; the highest r value was 0.163.

Intraobserver and Interobserver Variability of QRS Voltage Measurements

Two of the authors (F.L. and F.B.), each of whom evaluated about half of the tracings, performed manual measurements of QRS voltage. After completion of the study, we tested the intraobserver and interobserver vari-
ability of QRS amplitude measurements using a random sample of 15 tracings. The two observers analyzed these values independently, and measurements were repeated 1 week later. For each ECG, we calculated the difference between ∆SVT obtained by the two observers and between corresponding data measured by the same observer at different times. Differences are expressed as percentage ∆SVT. In no case was the maximal intraobserver or interobserver difference >5% of the ∆SVT value. The average intraobserver difference was 1.9%; the average interobserver difference was 2.7%.

Discussion

A definite increase in QRS voltage occurs during reentrant SVT, with respect to SR, in most subjects. This is evident in the majority of leads, but only lead V1 shows a slight but significant voltage decrease associated with tachycardia. These changes in voltage are not dependent upon the mechanism of SVT, because they occur in both AVNRT and AVRT. Theoretically, the simultaneous atrial and ventricular depolarization occurring in AVNRT could result in some apparent increase of QRS voltage, but this is not the case here, as the phenomenon also occurs in AVRT, where the P wave is well separated from the QRS complex.

Our results are similar to those reported by Wakimoto et al. and further demonstrate that QRS voltage change during SVT occurs not only in leads V2 to V5, but also in all of the precordial leads and in 4 of 6 limb leads. The mechanism underlying such a voltage variation is not immediately evident. It is easy to rule out an aberrant conduction and myocardial ischemia, the former because QRS duration was unchanged and the latter because most subjects were very young and no patient manifested clinical signs of coronary artery disease.

The factors leading to increased QRS amplitude in left ventricular hypertrophy were reviewed recently by Sura-wicz and include left ventricular mass, left ventricular surface area, intracardiac blood volume, and closer proximity of the enlarged ventricle to the chest wall. In the case of QRS voltage increase during SVT, there are two main possible explanations: the “proximity effect” and the changes in transmission of cardiac vectors to the body surface dependent on intracavitary blood mass. Feldman et al. demonstrated that the distance from the left ventricle to the precordial electrodes is a major determinant of R wave amplitude. They observed an R wave amplitude augmentation related to left ventricular end-diastolic diameter (LVEDD) increase during methoxamine infusion and, in contrast, an R wave amplitude reduction related to LVEDD decrease during Valsalva maneuver. All of these phenomena depended on the distance from the left ventricular posterior wall to the chest wall. Wakimoto et al. interpreted the QRS voltage changes observed during SVT in accordance with the proximity effect. They postulated that, as a result of tachycardia-induced diminished ventricular filling, the left ventricular chamber moves anteriorly and becomes

![Figure 3](image-url) Spontaneous interruption of AV reentrant tachycardia due to concealed accessory bypass tract. In several leads, particularly V2 and V3, QRS voltage is decreased at restoration of sinus rhythm. The first posttachycardia beat is an AV junctional escape complex and was not considered for QRS voltage analysis.

![Figure 4](image-url) Comparison between QRS voltages (in millivolts) measured during supraventricular tachycardia and during sinus rhythm (SR) in the 12 leads for AV nodal reentrant tachycardia (AVNRT) and AV reentrant tachycardia (AVRT). The only difference between the two types of SVT is in lead V1, where the voltage decrease during tachycardia, with respect to sinus rhythm, is significantly greater (P < 0.05) in AVRT than in AVNRT.
closer to the chest wall, so that the precordial electrodes of leads V2 to V5 record R waves taller than during SR. This interpretation is possible, but it does not account for QRS voltage variations in the limb leads. In the study by Feldman et al., no R wave amplitude change occurred in the limb leads during Valsalva maneuver or methoxamine infusion. On the other hand, the influence of cardiac blood mass on R wave amplitude cannot be ruled out. Ventricular load may affect the transmission of cardiac electrical forces to the body surface in two different ways: (1) the Brody effect and (2) the intracardiac shunting of potentials. According to Brody’s theory, the increase in ventricular blood mass may result in diminished transmission of radial cardiac forces, leading to increased ECG expression of vectors generated by the left ventricle, because the main direction of such vectors is radial. A reduced ventricular blood mass thus results in diminished transmission of left ventricular forces and vice versa. This phenomenon has been demonstrated in both the experimental setting and in clinical conditions. It also has been postulated that R wave increase during effort test reveals left ventricular dilation induced by ischemia. The Brody effect cannot be operative in determining the QRS voltage changes observed during SVT, because the reduced ventricular blood mass should lead, on such a basis, to QRS voltage reduction rather than increase. Several laboratory and clinical observations, however, have demonstrated that the effect of ventricular volume load on QRS voltage can be opposite to that expected based on Brody’s theory. Namely, a decrease in left ventricular volume, obtained with different factors (treatment of heart failure in patients with dilated left ventricle, hemodialysis, hyperosmolar contrast medium injection, furosemide in healthy volunteers) resulted in increased, rather than decrease, of QRS voltage on surface ECG. This was interpreted as due to low blood electrical resistivity. Because the resistivity of blood is lower than that of tissues surrounding the heart, some of cardiac forces undergo an intracavitary “shunting,” i.e., they are directed toward the cavity rather than toward the body surface. These inward vectors cancel each other and do not influence the ECG. An increase in intracardiac blood mass results in increased short-circuiting of potentials and hence diminished proportion of vectors that reach the body surface, and vice versa.

The latter mechanism is an alternative explanation to the “proximity effect” for QRS voltage changes observed during SVT. Reduced intracardiac blood mass due to shortening of diastole results in diminished intracavitary short-circuiting of potentials and increase of cardiac vectors directed to the body surface.

Theoretically, body mass could influence tachycardia-induced QRS voltage changes. However, we could not investigate the relationship between voltage changes and body mass, because this was a retrospective study and data on body mass index were lacking for several patients.

The significant QRS voltage reduction in lead V1 during SVT could express either reorientation of cardiac vectors in a direction more perpendicular to lead V1 or increased

![Figure 5. Two episodes of supraventricular tachycardia (SVT) due to AV nodal reentry are interrupted, in the same patient, by ventricular extrastimuli. The tachycardia rate is 140 in the left panel and 230 in the right panel. In both episodes, QRS complex voltages are higher during SVT than during sinus rhythm, but the difference is far more evident when the tachycardia rate is higher (right panel). At 140 beats/min, the sum of QRS voltages is 11 mV (+10% with respect to SR), whereas it is 13.4 mV (+31%) at a rate of 230. It is worth noting that the sum of QRS complex voltages during SR is identical in both tracings (10 and 10.2 mV in the left and right panels, respectively). The high-rate tachycardia is associated with some ST-T changes.](image-url)
distance of the heart from the recording electrode. It is surprising that QRS amplitude reduction during SVT in lead V1 is significantly greater (P < 0.05) in AVRT (1.56 ± 0.65 mV in SR vs 1.29 ± 0.64 mV in SVT) than in AVNRT (1.46 ± 0.54 mV in SR vs 1.36 ± 0.63 mV in SVT). We do not have any reasonable theory to explain such a small but significant difference, provided that, in the remaining 11 leads as well as in the sum (Σ) of QRS voltages, the behavior of the two types of SVT does not differ.

Independent of the mechanism responsible for the voltage changes (proximity effect or intracavitary shunting of potentials), it should be pointed out that the lack of correlation between heart rate and QRS amplitude variation suggests that the latter is neither entirely dependent on heart rate nor linearly related to it. It has been observed that heart rate increase as a result of rapid atrial pacing leads to reduction, rather than to augmentation, of QRS complex voltage. In addition, ventricular volume does not depend only on heart rate, but may be affected by several factors, including venous return and heart chamber compliance. Furthermore, any change in ventricular volume theoretically may exert two opposite effects on transmission of cardiac vectors. For example, in the case of diminished intracardiac blood mass, the Brody effect reduces, whereas the intracardiac shunting of potentials increases, the QRS complex voltage. The unpredictable interplay of these factors determine the net effect, which in most cases of SVT leads to increased QRS complex voltage.

Correct investigation of the relationship between QRS voltage change and tachycardia rate should compare tracings obtained in the same patient during separate episodes of SVT at different rates. We had a single patient with AVNRT in whom two episodes of tachycardia at different rates were available. At 140 beats/min ΣQRS was 11 mV (+10% with respect to SR), whereas at a rate of 230 beats/min it was 13.4 mV (+31% with respect to SR) (Fig. 5).

In conclusion, this study confirms that, in the majority of subjects, reentrant SVT is associated with an increase in QRS complex voltage. At first glance, this suggests an incorrect diagnosis of aberrant conduction or left ventricular hypertrophy. The presence of ST-T changes, which commonly occur in SVT, could speak in favor of such a misdiagnosis.

References