

Amplitude Alterations Between Sinus Rhythm and Ventricular Tachycardia or Ventricular Fibrillation in Surface and Intracardiac Leads in Human Subjects

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Abstract

This study was performed to quantify the electrocardiographic amplitude alterations that occur in the transition from sinus rhythm (SR) to ventricular tachycardia (VT) or ventricular fibrillation (VF) in human subjects. Measurements were made of QRS complexes on surface ECGs and of intraventricular depolarizations. Patients were studied in three categories: Group A (no drugs), Group B (Amiodarone), and Group C (Procainamide). In ventricular tachycardia we found increases in every lead in all three groups. Lead I increases ranged from 12–52%; in Lead III increases were 49–148%; in Lead VI increases were 20–54%. Ventricular tachycardia measured from the right ventricular apex (RVA) showed increases ranging from 24–49%. In ventricular fibrillation, amplitudes exhibited both increases and decreases in Leads I and VI, and increases in Lead III. In the RVA, ventricular fibrillation amplitude decreased in all three groups, ranging from –2––55%. Results of VF for Amiodarone and Procainamide are inconclusive due to the small patient sample size. The data show that there is a significant increase in VT amplitude over SR, as well as a relationship in VT amplitudes between drug and no drug groups in every lead. Also, VF was characterized by decreased amplitudes or markedly smaller increases than VT.

1. Introduction

Heart rate is being used primarily as the decisive factor in algorithms for implantable cardioverter defibrillators (ICDs) to determine the occurrence of certain arrhythmias, such as VT and VF. [1, 2] With the introduction of new technologies, similar rate-based discriminators are utilized in other intelligent defibrillators, such as automatic external defibrillators (AEDs). These devices are now used on aircrafts and are proposed to become so extensive as to be available for use by untrained personnel in any public location or residence [3, 4]. However, while heart rate is a good differentiator in ICDs and AEDs, it would be desir-

able to have a supplemental means of classifying VT and VF. It is known that maximum ventricular amplitude generally increases from SR to VT [5] and generally decreases from SR to VF [6, 7], and since it is computationally simple to compare amplitudes, it would be logical to use it as a supplemental discriminant.

This is the premise behind our study. It is known that Amiodarone, which is a class III agent (Vaughan Williams classification), prolongs action potential duration, increases the Q-T interval [8], acts as a sodium channel inhibitor, and creates a more uniform action potential pattern throughout the heart [9]. Similarly, Procainamide (class IA agent) acts as a sodium channel blocker and also mildly increases the refractory period. Since a major field of research in the Medical Computing Laboratory at the University of Michigan for the last two decades has dealt with the digital analysis of features of the intraventricular electrogram [10, 11, 12, 13, 14], we wanted to investigate VT and VF amplitudes in surface leads and intracardiac leads with patients taking these antiarrhythmic drugs versus patients not taking these drugs, and examine the magnitude of changes that occur.

2. Methods

Patients were studied in three categories: Group A consisted of patients not taking antiarrhythmic drugs, Group B of patients on Amiodarone, and Group C of patients on Procainamide. Data were acquired from patients undergoing clinical electrophysiology studies in which ventricular arrhythmias were induced (Ann Arbor Electrogram Libraries, Ann Arbor, MI). Surface QRSs were acquired from two of three surface leads (I, III, or V1) at a standard ECG bandwidth of 0.05–100 Hz; ventricular electrograms were recorded (1–500 Hz) from a bipolar catheter (1 cm) located in the right ventricular apex (RVA). Filter and gain settings were held constant throughout the SR, VT, and VF passages.

Correct rhythm classifications of VT and VF were determined systematically based on rate. Ventricular tachycar-

dia was diagnosed if the ventricular rate in the RVA-Bipolar lead was between 120 and 250 cycles per minute (240–500 milliseconds). VF was diagnosed if cycle rate was predominantly above 330 cycles per minute (180 milliseconds). We excluded distinct episodes that exhibited regular sinusoidal morphology characteristic of ventricular flutter, although they may have met the VF rate criteria.

Amplitude measurements were performed via a custom software package (AAELView, Ann Arbor, MI) on 12 bit data sampled at 1000 Hz. Shown in Figure 1 is the process used to measure individual cycle amplitudes. For each patient, ten to twelve cycles were measured per lead. These measurements were averaged and their magnitudes were used to compute the amplitude alterations per equation 1.

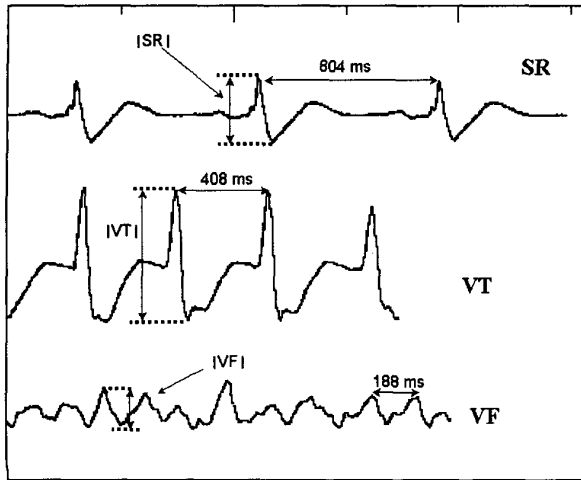


Figure 1. Passages of SR, VT and VF illustrating amplitude measurement for one patient (Lead III).

The percentage increase (+ percentage) or decrease (– percentage) of signal amplitude is given using the following equation, which relates the magnitudes of either VT or VF to SR.

$$X_{Amp}\% = \left(\frac{|\text{VT}| - |\text{SR}|}{|\text{SR}|} \right) \times 100\% \quad (1)$$

3. Results

The following average values were found for ventricular tachycardia in each category:

| Lead | Group A | Group B | Group C |
|------|-------------|-------------|--------------|
| I | +35% (n=10) | +12% (n=10) | +52% (n=12) |
| III | +69% (n=10) | +49% (n=11) | +148% (n=10) |
| V1 | +48% (n=11) | +20% (n=10) | +54% (n=10) |
| RVA | +49% (n=12) | +26% (n=12) | +24% (n=11) |

Note that not every patient has data in every surface lead, so enough patients were selected to yield a minimum of ten patients per lead measured. For a graphical depiction of these tabulated results see Figure 2.

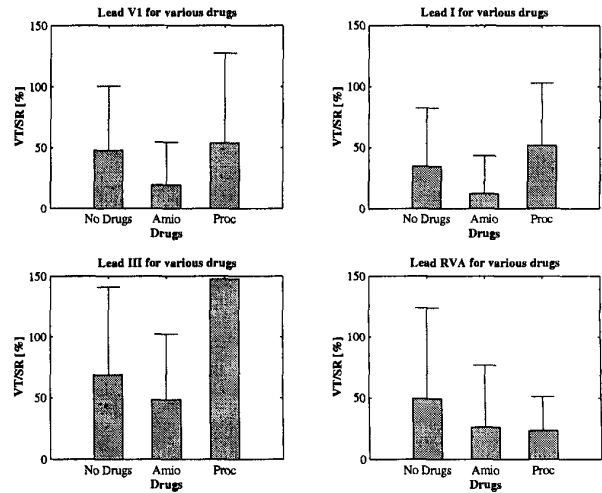


Figure 2. Ventricular tachycardia in Leads I, III, V1, and RVA-Bipolar.

The following values were found for ventricular fibrillation:

| Lead | Group A | Group B | Group C |
|------|-------------|------------|------------|
| I | –23% (n=10) | –18% (n=3) | +8% (n=1) |
| III | +21% (n=10) | +36% (n=4) | N.A. (n=0) |
| V1 | –26% (n=10) | +10% (n=3) | –2% (n=1) |
| RVA | –2% (n=12) | –17% (n=4) | –55% (n=1) |

See Figure 3 for a graphical depiction of these results. For ventricular fibrillation, there was a small patient population due the unavailability of study data; hence only group A has at least ten patients per lead.

For a breakdown on an individual patient basis see Table 1 for the VT category and Table 2 for the VF category.

4. Discussion

The data show that there is a significant increase in VT amplitude over SR in surface leads I, III, V1 and RVA-Bipolar. From Figure 2, one can see a clear relationship in the surface lead ratios between group A and groups B and C. Furthermore, a clear decrease from group A in groups B and C is seen for the intracardiac lead, RVA-B. Statistical significance between the groups in the surface and intracardiac leads could not be shown due to a small population size and large variance.

Also, increase in amplitude during VF is markedly less than during VT. In VF, the results were mixed. Some leads

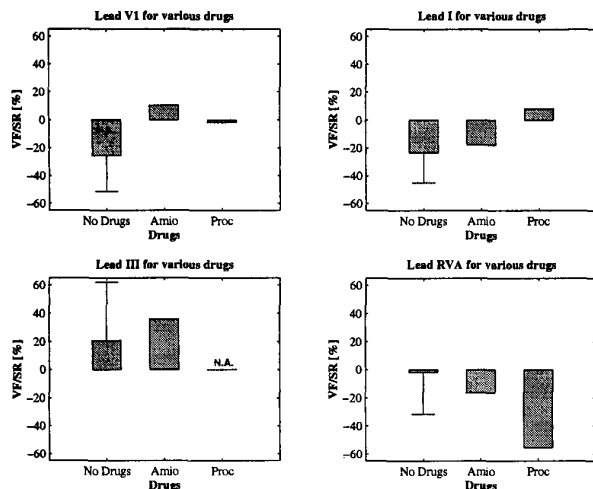


Figure 3. Ventricular fibrillation in Leads I, III, VI, and RVA-Bipolar.

showed increased amplitudes, others decreases. Group A (no drugs) saw increased amplitudes in Lead III, but a decrease in V1, I, and RVA-Bipolar. Group B (Amiodarone) decreased in Leads I and RVA-Bipolar, and increased in Leads III and V1. Group C (Procainamide) increased slightly in Lead I, decreased insignificantly in V1, and decreased dramatically in RVA-Bipolar. The sample set of VF data for groups B and C was too small to derive meaningful statistics.

For VF in group A, the RVA-Bipolar lead showed VF/SR ratio of 0.98. From other studies, such as Ellenbogen [6] (VF/SR = 0.77) and Leicht [7] (VF/SR = 0.55), one may recognize that our measured values are slightly higher. This can be attributed to multiple factors. Measurement values show some ambiguities due to our definition of VF based on rate. Some episodes met the rate criteria, but showed morphological characteristics of ventricular flutter. While our results are in disagreement with those of Leicht, this may be attributable to the fact that Leicht's data were heavily filtered (10–50 Hz) leading to distortion of the raw data prior to the measurement process. Effects of such filtering on waveform anatomy are described in Jenkins [15] and Morris [16].

Procainamide, classified as a class I antiarrhythmic agent, slows sodium channel conductance and hence inhibits the upstroke of the action potential [9]. Furthermore, Singh states that Amiodarone also exhibits a similar class I characteristic. From this, one could infer a decrease in overall signal amplitude for patients taking these drugs. This inference is based on the fact that on a cellular level the probability of myocardial cell firing is reduced, hence contributing to a decrease in the QRS amplitude, in comparison to patients that

| No Drugs | | | | |
|--------------|-------|-------|-------|-------|
| Pat. | RVA | V1 | I | III |
| No.1 | +28% | +182% | +210% | |
| No.2 | +56% | +76% | +80% | |
| No.3 | +76% | +218% | | -12% |
| No.4 | +31% | +12% | +22% | -20% |
| No.5 | +22% | -21% | +11% | +229% |
| No.6 | -20% | +26% | +44% | +6% |
| No.7 | +453% | +94% | | +100% |
| No.8 | +15% | -49% | -27% | +63% |
| No.9 | +11% | +6% | +85% | 0% |
| No.10 | -19% | +43% | +5% | +310% |
| No.11 | -14% | | -48% | +63% |
| No.12 | -48% | -62% | -35% | -54% |
| Amiodarone | | | | |
| No.13 | -51% | -19% | +62% | +243% |
| No.14 | -13% | -69% | -28% | -4% |
| No.15 | -31% | +1% | -34% | +50% |
| No.16 | +43% | +19% | | -23% |
| No.17 | +42% | +61% | +38% | +136% |
| No.18 | +41% | +140% | | +50% |
| No.19 | -19% | | +62% | -33% |
| No.20 | -10% | +10% | -30% | |
| No.21 | -24% | +53% | +50% | +81% |
| No.22 | +258% | | -20% | -37% |
| No.23 | +129% | +39% | | -21% |
| No.24 | -48% | -41% | -57% | +91% |
| No.25 | | | +77% | |
| Procainamide | | | | |
| No.26 | +39% | +76% | +62% | +31% |
| No.27 | +14% | -8% | +99% | +434% |
| No.28 | -46% | +28% | -6% | +269% |
| No.29 | +57% | +58% | +73% | +13% |
| No.30 | +16% | +286% | +12% | +3% |
| No.31 | +68% | | +3% | +17% |
| No.32 | -9% | -22% | +24% | +272% |
| No.33 | +26% | -76% | +125% | +136% |
| No.34 | +125% | -43% | -36% | |
| No.35 | -35% | | +54% | +285% |
| No.36 | +6% | +263% | +255% | |
| No.37 | | -24% | -44% | +16% |

Table 1. Amplitude for various leads and drugs in VT on a per patient basis.

were drug-free. This phenomenon manifests itself in the decreased average VT amplitude in the RVA-Bipolar lead.

5. Conclusion

This study was conducted to quantify the electrocardiographic changes that occur between normal sinus rhythm

| No Drugs | | | | |
|--------------|------|------|------|-------|
| Pat. | RVA | V1 | I | III |
| No.2 | -35% | -63% | -39% | |
| No.3 | +73% | +4% | | -66% |
| No.6 | +22% | -49% | -46% | +29% |
| No.7 | +94% | -9% | | -44% |
| No.12 | +14% | -44% | -28% | -16% |
| No.38 | -59% | -78% | -58% | |
| No.39 | -11% | -15% | -55% | -50% |
| No.40 | -3% | | +10% | +21% |
| No.41 | -76% | -25% | +38% | +76% |
| No.42 | +7% | | +23% | +120% |
| No.43 | -64% | -47% | -52% | +114% |
| No.44 | +16% | +69% | -28% | +22% |
| Amiodarone | | | | |
| No.17 | +9% | -46% | -37% | -13% |
| No.18 | -23% | | | -22% |
| No.19 | -20% | | -24% | +43% |
| No.21 | -32% | +40% | +8% | +135% |
| No.45 | | +37% | | |
| Procainamide | | | | |
| No.36 | -55% | -2% | +8% | |

Table 2. Amplitude for various leads and drugs in VF on a per patient basis.

and ventricular tachycardia and fibrillation. A comparison of amplitudes was made between drug-free patients and those treated with antiarrhythmics, Amiodarone and Procainamide.

The data show that there is a significant increase in VT amplitude over SR, as well as a relationship in VT amplitudes between drug and no drug groups in every lead. Also, VF was characterized by decreased amplitudes or markedly smaller increases than VT.

Acknowledgements

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