The Link Between Abnormal Intra QRS Potentials and Premature Ventricular Beats

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Abstract

Cardiac late potentials have shown the value of high resolution ECGs for identifying a reentrant substrate, but they are usually obtained during sinus rhythm and have not been directly linked to an arrhythmia mechanism. Abnormal intra-QRS potentials (AIQP) are presumed to be a reflection of those late potentials which originate in early activated regions of the ventricles but which do not outlast the QRS complex. Using a high resolution, digital ambulatory ECG recorder (3 channels, 1000 Hz/ channel, 16 bit A/D resolution, and removable 340 or 510 Mbyte hard disc) 15 or 22 hours of XYZ lead data were recorded from 80 patients with frequent premature ventricular beats (PVBs). A data structure was centered on PVBs of highly similar morphology. Thirty-six patients met this criterion. Five sinus beats before and after the PVB were organized so that a signal averaged version of each indexed sinus beat was obtained. A total of 87 PVB sequences were analyzed. Using the previously published methods for obtaining AIQP resulted in 26 sequences with a significant change in the RMS of the AIQP signal in the sinus beat immediately preceding the PVB. It is hypothesized that the mechanism of the PVBs most likely associated with changes in the AIQP is reentry. For this data set one could conclude that 30% of the PVBs were caused by reentry.

1. Introduction

Cardiac late potentials, when detected from the body surface, have been used to identify patients at high risk for sudden cardiac death [1]. These signals originate from ventricular regions in and surrounding a myocardial infarct. It has been assumed that signals from these diseased regions may not always outlast the QRS complex. Gomes et al [2] developed a technique based on autoregressive modeling of the QRS complex to identify these abnormal intra-QRS potentials (AIQP). A clinical paper utilizing this method was published by Lander et al [3] and demonstrated a marked improvement in the specificity for identifying patients with life threatening ventricular arrhythmias.

Identifying the mechanisms of ventricular arrhythmias from the ECG has been problematic. Catheter based electrophysiology studies in both the clinical and experimental setting have been used to delineate these mechanisms. Reentry is one such mechanism and requires slow activation and unidirectional block in a circuit of tissue. In the case of ventricular arrhythmias cardiac late potentials are assumed to be a measurement of the signals which are partially activated in the reentrant substrate. They may be part of the actual reentrant circuit or are just a surrogate for the actual circuit.

Initial studies [4] used to link the late potentials with premature ventricular beats (PVBs) relied on a repeating PVB and the averaging of the sinus beat immediately prior to the PVB. No changes in the late potentials were observed in this set of selected sinus beats when compared to non-PVB related sinus beats.

This study uses the AIQP method of Gomes et al [2] and examines a data structure obtained from continuously recorded XYZ leads. Five sinus beats before and after the PVB were studied to determine if some of these PVB related sinus beats showed changes in the AIQP which could be linked to the PVB. Such a linkage could provide direct evidence for the reentry mechanism of some PVBs.

2. Methods

The Institutional Review Board approved our protocol and each patient signed an informed consent for the study. Ambulatory ECG recordings were collected from 80 patients who had frequent PVBs using the Altair Disc recorder (Diagnostic Medical Instruments, a Spacelabs Medical Inc. company, Karyn Beckley, CA). This device is a small, portable, battery powered, ambulatory ECG data acquisition and storage system. It allows the recording of three ECG signals with a dynamic range of 10 mV peak to peak and stores the data with 16 bits of amplitude resolution. The recorder has an input analog bandwidth of 0.05 to 500 Hz and sampling rates of 1000

Hz per channel. The recorder can store 15 or 22 hours of continuous uncompressed data on discs of 340 or 510 Megabytes, respectively.

The first-pass analysis included the detection and the classification of all the QRS complexes in the patient's record as well as beat feature extraction [5]. The outcome of the first-pass analysis was used for the selection of PVB and sinus beat classes to create beat sequences for the analysis. Beat sequences were defined as 5 sinus beats prior to a PVB and 5 sinus beats after the PVB. All the PVBs in the beat sequences had the same morphology. Following is a flow chart with complete set of processing steps used in this study.
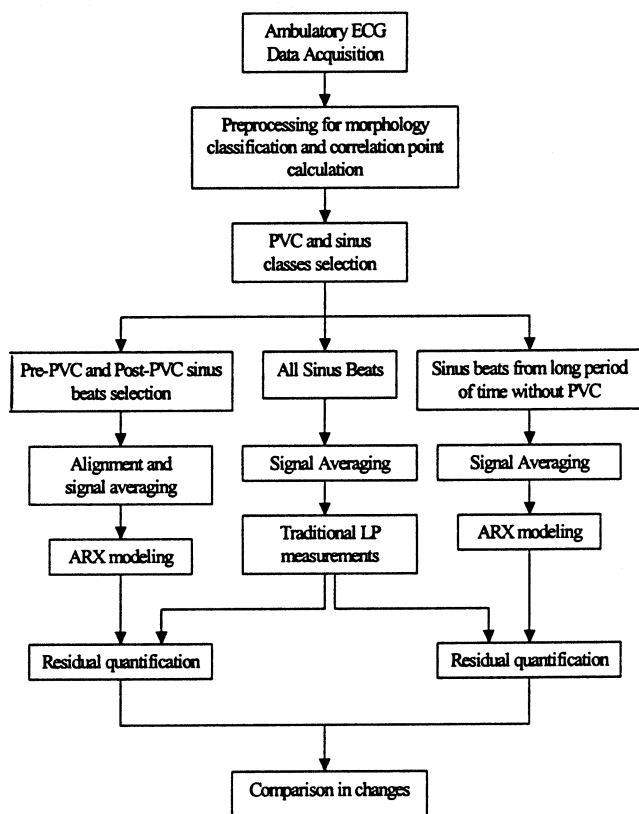

Figure 1. Flow Chart of processing steps used in this study.

In addition to these 10 PVB related beats a control set of 10 sinus beats were extracted from the recordings in the middle of 60 non-PVB related periods. In each case enough of these events were required in order to obtain low noise signal averages of each beat in the respective sequence. e.g., PVB-1, PVB-2, etc.

Not all of the patient recordings met these criteria but in some cases a single patient provided multiple PVB morphologies to allow multiple observations in each patient. Hence, data were analyzed from 36 patients comprising 87 total episodes for this study.

Also obtained from each patient was a signal averaged ECG using sinus beats irrespective of the PVBs. Late potentials were observed in 17 of the 36 patients using traditional criteria [1]. All of the analysis software was written in MATLAB.

The Gomes [2] method results in a residual signal which is most likely related to the AIQP. Figure 2 demonstrates the power of the method to detect a high frequency signal added to a QRS complex. Each panel in the figure has four traces. The top trace is the simulated QRS based on the autoregressive model. The second trace is the original QRS with the added high frequency signal; a single sawtooth signal shown in the bottom trace. The third trace is the residual signal obtained by subtracting the two QRS signals. The arrow indicates the presence of the sawtooth signal in the residual. Each panel shows a progressively later time where the sawtooth is added. In the lower right panel the sawtooth is also seen in the presence of a relatively larger residual signal.
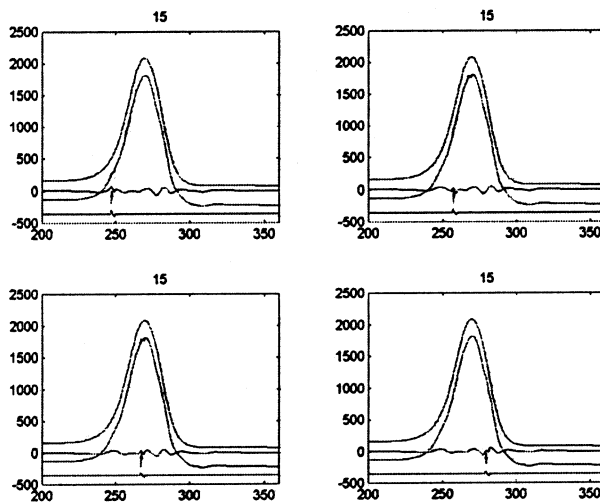

Figure 2. Example of the AIQP detection method using a simulated sawtooth added to the original QRS at 4 different times.

Quantifying the RMS voltage of the residual signal for each average allows for a comparison among the sinus beats in both the pre-PVB and post-PVB sequences as well as the non-PVB related sinus beats. This study focused on the sinus beat immediately prior to the PVB, or PVB-1. In the case of the pre-PVB beats the RMS of the PVB-1 was compared to the average of the other four pre-PVB sinus beats. Below are the two rules used to define if the PVB-1 sinus beat was significantly larger or smaller than the others in the sequence.

$$RMS_{PVC-1} < \frac{1}{4} \times \sum_{j=2}^{5} RMS_{PVC-j} - 3STD$$

or

$$RMS_{PVC-1} > \frac{1}{4} \times \sum_{j=2}^{5} RMS_{PVC-j} + 3STD$$

3. Results

Graphic examples of the RMS voltage for the pre- PVB and post-PVB sinus beats are shown in Figures 3 and 4. In addition the RMS values for the residuals obtained from the non-PVB related sinus beats are also plotted. The open circles (O) represent the PVB related beats and the plus signs (+) represent the 10 non-PVB related sinus beats.
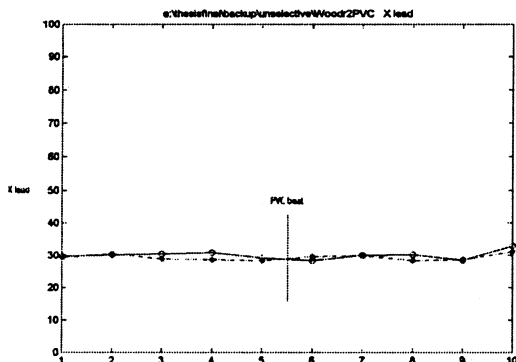

Figure 3. Plot of the RMS of the residual for the PVB related beats (O) and the non-PVB related beats (+).
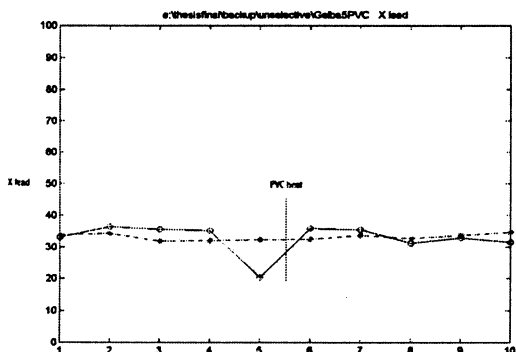

Figure 4. Plot of the RMS of the residual for the PVB related beats (O) and the non-PVB related beats (+).

Figure 3 demonstrates the case where there were no significant changes in the RMS of the PVB-1 sinus beat. Note also the similarity of the PVB related sinus beats with the non-PVB related beats. This is visually seen in the figure and was statistically true based on the above rules.

Figure 4 demonstrates the case where the RMS voltage of the PVB-1 sinus beat is both visually and statistically different than that of the surrounding beats as well as the non-PVB related sinus beats. The actual waveforms of the QRS residual for the X lead which formed part of the data in Figure 4 are shown in Figure 5. The five waveforms represent, from left to right, the PVB-5 through PVB-1 sinus beats. Note the consistency of the first four waveforms and the marked diminution of the fifth waveform.
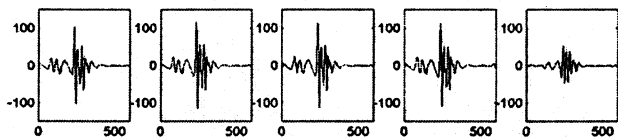

Figure 5. The QRS residual waveform for the five beats preceding the PVB in the X lead.

For the 87 episodes from the 36 patients, 26 of them met the criterion for a significant change as demonstrated in Figures 4 and 5. Seventeen of these cases were observed in only one lead, eight were observed in two leads, and in one patient the change was observed in all three leads. In addition, only seven of the seventeen cases were from patients with traditional late potentials.

Figures can fit across both columns if necessary. If possible include figures in the text document; otherwise paste them, taking care to keep the page flat. To make sure that all labels are clearly legible use at least a 9 point font. Use a line thickness of at least 0.5 mm.

4. Discussion and conclusion

The RMS of the residual QRS signal is one way to quantify the AIQP. There has been no formal study which has formally linked these two observations. However, the improved clinical results using the AIQP [3] certainly provides strong evidence linking these two observations. Using these methods and a rather straightforward statistical test has also provided some evidence that this measure of AIQP is related to the presence of a ventricular arrhythmia. The PVB is the simplest and most benign ventricular arrhythmia but has always been treated as singular event from the perspective of the ECG. In other words, except for the coupling interval, all PVBs were considered the same. There has been no attempt to understand the mechanism of the PVB from the ECG itself.

If one considers the variety of possible PVB mechanisms there should be some way to exploit or develop an ECG based method to identify the mechanism [6-7]. Understanding the mechanism may lead to a greater sense of morbidity for the PVB. A twenty year experiment in the use of anti-arrhythmic drugs to suppress PVBs without a knowledge of their mechanism may have permanently spoiled the opportunity to deal with these ventricular arrhythmias on a rational basis.

The data in this study suggest that a large percentage of PVBs may have a reentrant mechanism. This is based on the concept that AIQP have the same basis as late potentials except that they do not outlast the QRS complex.

Acknowledgements

References

[1] Steinberg JS and Berbari EJ. The signal-averaged electrocardiogram: Update on clinical applications. J Cardiovascular Electrophysiology, 7:972-988, 1996.

[2] Gomis P, Jones DL, Caminal P, Berbari EJ, Lander P. Analysis of abnormal signals within the QRS complex of the high resolution ECG. IEEE Trans. Biomed. Engr.,44:681-695, 1997.

[3] Lander P, Gomis P, Goyal R, Berbari EJ, Caminal P, Lazzara R, Steinberg JS, Analysis of Intra-QRS late potentials: Improved predictive value for arrhythmic events using the signal-averaged electrocardiogram. Circulation, 95:1386-1393, 1997.

[4] Narayanaswamy S, Berbari EJ, Lander P, Lazzara R. Selective beat signal averaging and spectral analysis of beat intervals to determine the mechanisms of premature ventricular contraction. Computers in Cardiology, 20:81-84, 1993.

[5] Lagerholm M, Peterson C, Braccini G, Edenbrandt L, Sörnmo L, "Clustering ECG complexes using Hermite functions and self-organizing maps", IEEE Trans. Biomed. Eng., 2000;47:838-848.

[6] Cházaro A, Sörnmo L, Sih HJ, Maass-Moreno R, Berbari EJ. Analysis of Ventricular Repolarization in Context of Premature Ventricular Beats. Computers in Cardiology, 27:339-342, 2000.

[7] O'Leary EA, Sörnmo L, Sih HJ, Berbari EJ. Detection of Low Level ST Segment Changes From the Ambulatory ECG And Their Correlation With Ventricular Premature Beats. Computers in Cardiology, 27:829-832, 2000.

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