Measurement of Atrial and Ventricular Heart rate variability Using Pacemaker-Mediated intracardiac Electrograms

Abbas F. Al-Hashimi MSc

Abstract

Background: Heart rate variability (HRV) measurements are usually performed from ventricular beat-to-beat intervals because of the difficulty to precisely locate the P-wave fiducial point in surface ECG recordings. Intracardiac electrogram can be recorded by pacemaker device. This provides useful signals to measure atrial and ventricular heart rate variability.

Objective: to describe a method which measure the atrial and ventricular heart rate variability using intracardiac electrogram recorded and stored by pacemaker devices.

Method: The study was conducted on 14 patients with dual chamber pacemakers. Those were suffering from intermittent sick sinus diseases or intermittent advanced A-V block attending the Cardiac Care Unit in Al-Kadhimia Teaching Hospital. The atrial and ventricular intracardiac electrograms were transmitted with the telemetry channel of the pacemaker to an external recorder for 20 minutes. The resultant intervals were used to calculate the standard deviation of all N-N intervals (SDNN), the squared root of the mean squared differences of successive N-N intervals (RMSSD), and the percentage of successive interval differences > 50 ms (pNN50). The differences between atrial and ventricular heart rate variability indexes (HRV-Indexes) were assessed for each patient with a cut-off point of 1%. Differences >1% were analyzed in detail.

Results: Fourteen patients with dual chamber pacemakers were included in this study. A total of 18788 heart cycles were analyzed. A manual correction due to false or not triggered atrial or ventricular events was necessary in 0.8%. The overall differences between atrial and ventricular pNN50 was -0.5%±2.1% and differences > 1% were observed in 4 patients. The N-N50 events occurred in the atrial and related ventricular interval in 84%. N-N50 events occurred only the atrium in 6% and only I the ventricle in 10%. The mean differences between atrial and ventricular SDNN and RMSSD were 0.4±2.1 ms and -0.1±3.5 ms with individual differences <1%.

Conclusion: This study describes the utilization of intracardiac electrograms to analyze differences between atrial and ventricular HRV. The differences for pNN50 indicate that ventricular HRV does not reflect the changes of sinus node activity in all patients.

Key words: Intracardiac electrogram, Heart rate variability HRV, Pacemaker.

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Introduction

Heart rate variability (HRV) measurements describe changes of the beat-to-beat intervals over time (1, 6,10). Ventricular beat-to-beat intervals are usually used for HRV determination because of difficulty to precisely locate the p wave fiducial point from surface ECG recordings (10).

Since the autonomic inputs to the sinus node and atrioventricular (AV) node are partially independent of each other, (3-5,7) the simultaneous determination of atrial and ventricular HRV can help to us better understand the underlying mechanism of changes in HRV and could increase their diagnostic impact (1,2, 8).

RR event marker intervals in combination with pacemaker implemented software have already been used for autonomic HRV analysis, which could be also used for the analysis of PP marker intervals in patients with dual chamber devices (5).
A limitation of this approach is the inability of exclude artifacts without the intracardiac electrograms reliably (1, 2).

Patients and Methods
The study was conducted on fourteen patients (56±1 years, male n=9) with the dual pacemaker (St, Jude Medical, Ireland). Those patients attending the Cardiac Care Unit in Al-Kadhimia Teaching Hospital, during the period between June 2006 to April 2007. Pacemaker indications were intermittent sick sinus diseases (SSS) in 7 patients or intermittent advanced A-V block (AVB) in the rest patients.

The pacemaker provided a high resolution telemetry, which enabled the continuous transmission of atrial and ventricular electrograms to an external recorder. Atrial and ventricular electrograms were continuously recorded for 20 minutes while the patient in a supine position and in sinus rhythm. The pacing mode was set during the recording time to DDI with a low basic rate of 40 pulse/ minute and a long AV delay of 250 ms to only record intrinsic activity. The recorded electrograms were stored on the programmer. Premature ventricular contractions (PVC) were identified by a mismatch between the atria and ventricular event. A premature atrial contraction (PAC) was classified as a beat-to-beat interval shortening >50%. In both cases these events and succeeding heart cycle were excluded from further analysis.

After computer analysis assisted triggering of atrial and ventricular events and manual correction of false and not triggered atrial or ventricular events. The resultant PP and RR intervals were used to determine the standard deviation of all NN intervals (SDNN), the squared root of the mean squared differences of successive NN intervals (RMSSD), and the percentage of successive interval differences >50 ms (pNN50). The means of atrial and ventricular HRV-Indexes were compared to each other. The differences for each patient were assessed with an arbitrary chosen cut off point of 1% in order to distinguish between patients with obvious and normal near zero differences. Differences > 1% were analyzed in detail.

Results
A total of 18788 heart cycles were analyzed with 1342±245 heart cycles in each patient. There was no atrial or ventricular pacing during the recording time. A manual correction due to false or not triggered atrial or ventricular events was necessary in 0.8%. The overall differences between atrial and ventricular pNN50 was -0.7±2.4%. Differences>1% between atrial and ventricular were observed in 5 (35%) patients with -2.6% in patient 10 with AVB, 5.2% in patient 13 with AVB, 4.8% in patient 4 with SSS, -3.2% in patient 1 with SSS, and -4.2% in patient 6 with SSS (figure 1,2,3).

There were 2818 NN50 events in the 14 patients. NN50 event occurred at the same time during atrial and related ventricular interval in 84% (Figure 4). NN50 events were observed only in the atrium in 6% (Figure 6), and only in the ventricle in 10% (Figure 5).

The mean differences between atrial and ventricular SDNN and RMSSD were 0.6±2.3 ms and -0.2±3.2 ms with no individual differences >1%.
Figure 1: pNN50 for each patient. Patient 1-7: intermittent sick sinus syndrome; patient 8-14: intermittent AV block.

Figure 2: RMSSD for each patient. Patient 1-7: intermittent sick sinus syndrome; patient 8-14: intermittent AV block.
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Figure 3: SDNN for each patient. Patient 1-7: intermittent sick sinus syndrome; patient 8-14: intermittent AV block.

Figure 4: shows the differences between atrial and ventricular NN50 events. Similar shortening and prolongation of atrial and ventricular events.
Figure 5: shows the differences between atrial and ventricular NN50 events. An exclusively shortening and subsequent prolongation of the ventricular, but not the atrial events.

Figure 6: shows the differences between atrial and ventricular NN50 events. An exclusive shortening of the atrial, but not the ventricular events.
**Discussion**

Little is known about interplay between PP and PR autonomic modulation. The present study demonstrated the feasibility and to measure atrial and ventricular HRV from pacemaker-mediated intracardiac electrograms. A limitation of common approach for HRV determination using digitalized surface ECG recordings is to locate the P wave fiducial precisely (10). The main benefit of the present approach is that intracardiac electrograms have a spike-like morphology not only for ventricular, but also for atrial events. Therefore the same method for triggering and data processing can be used for both the RR and PP interval determination.

A disadvantage of the described approach with present pacemakers is that the storage capacity for intracardiac electrogram is still limited. This problem was solved by continuous transmission of the signals through the high resolution telemetry channel of the pacemaker to storage floppy discs, then the data retrieved by the computer.

Another described approach is to use pacemaker events marker chains instead of intracardiac electrograms for automatic HRV analysis (9, 11). The benefit of this method is that less pacemaker memory is needed. However, the presence of artifacts and their rejection cannot be reliably verified without intracardiac electrograms.

**References**