Hemodynamic Improvement and Heart Rate Variability during Aortic Counterpulsation

MR Risk¹, AJ Ramírez², EIC Fischer²

¹Buenos Aires Regional Faculty, National Technological University, Argentina
²CONICET, Favaloro University, Buenos Aires, Argentina

Abstract

Introduction: aortic counterpulsation is a useful technique, frequently used in heart failure refractory to medical treatment. The objectives of this study were to evaluate the hemodynamic changes and the heart rate variability (HRV) produced by aortic counterpulsation. Methods: in seven sheep acute heart failure was pharmacologically induced. Pressure and flow signals were recorded in order to evaluate intraaortic counterpulsation hemodynamic improvement. Results: Intraaortic counterpulsation resulted in a significant improvement of hemodynamic parameters: increase of cardiac output and cardiac index, and decrease of systemic vascular resistance. The extent of aortic diastolic pressure augmentation in assisted heart failure, showed a significant increase compared with unassisted values. Heart rate variability in both, time and frequency domain showed a non significant increase. Conclusion: treatment of acute heart failure by aortic counterpulsation allows an effective hemodynamic improvement without significant changes in HRV.

1. Introduction

The aortic counterpulsation is a useful technique, frequently used in heart failure refractory to medical treatment [1]. Intraaortic counterpulsation can be performed without the need of special equipment and has been shown reproducible, simple and reliable.

The prognostic value of heart rate variability in cardiac failure has been previously reported [2]. Furthermore, the role of heart rate variability changes, were analyzed in human cardiac transplantation [3]. However, the increasing clinician experience in left ventricular assistance, like that that uses aortic counterpulsation, has not been accompanied by heart rate variability analysis.

The objectives of this study were to evaluate the hemodynamic changes and the heart rate variability produced by aortic counterpulsation, in an animal model of pharmacologically induced heart failure.

2. Methods

A total of seven adult Corriedale sheep ranging from 30 to 42 kg in weight and aged between 15-24 months, were chosen at the beginning of this study. All animals were vaccinated and treated for skin and intestinal parasites. During 20 days before surgery they were appropriately fed, watered, and assessed for adequate clinical status.

The seven sheep were studied after inducing acute heart failure using halothane (4%) in order to evaluate hemodynamic improvement with a counterpulsation balloon pump [3]. This was achieved using an intraaortic balloon catheter positioned in descending aorta through left femoral artery. Figure 1 shows the balloon catheter placement in the aorta.

Figure 1. Intraaortic balloon catheter (IABC) positioned in the descending aorta, through femoral artery puncture.
Measurements of aortic blood pressure, ECG, cardiac output, cardiac index were performed in all animals [5].

Heart rate variability analysis in both time and frequency domain was off line. The ECG was used to determine the RR intervals, and then a beat series was constructed. The Blackman-Tukey method is based on the Fourier’s transform [6], this means the RR intervals must be sampled at a constant rate, and we calculated from the beat series a time series at 4 Hz [6].

The time domain measurements were mean RR and SD RR, these indicate the baseline heart rate and the dispersion around the baseline, respectively, and these are the basic measurements of HRV, where the SD RR shows the total variability.

The frequency domain measurements of HRV were performed in two bands: a) low frequency (LF, from 0.04 to 0.15 Hz), and b) high frequency (HF, from 0.15 to 0.5 Hz); using the Blackman-Tukey method for spectral estimation, were calculated in segments of 6 minutes [6].

The LF band express both sympathetic and parasympathetic branches of the autonomic nervous system, on the other side the HF band express the parasympathetic branch alone [6].

The ratio between diastolic and systolic areas beneath the aortic pressure curve (DABAC/SABAC) is an index used to evaluate the effects of counterpulsation, calculated as we previously described [5]; figure 2 shows the areas for a couple of beats with and without counterpulsation.

3. Results

Figure 3 shows the diastolic signal pressure augmentation obtained with intraaortic counterpulsation; note the augmentation during the diastole due to the balloon inflation, in this case performed every other beat, marked with an asterisk.

Aortic balloon pump counterpulsation in acute experimental heart failure resulted in a significant improvement of hemodynamic parameters: increase of cardiac output, from 0.86±0.04 L/min to 1.29±0.09 L/min, *P* < 0.05, and cardiac index, from 0.03±0.01 to 0.04±0.01 L/min/kg, *P* < 0.05, and decrease of systemic vascular resistance, from 89.76±6.69 to 66.56±6.02 mmHg/L/min, *P* < 0.05.

The extent of aortic diastolic pressure change evaluated through the DABAC/SABAC index in assisted cardiac failure induction, showed a significant increase compared with unassisted values, from 0.81±0.10 to 1.12±0.09, *P* < 0.05.

Heart rate variability in the time domain showed a non significant increase, mean RR interval from 616.35±30.53 ms for BHF to 641.51±32.26 ms for AHF; standard deviation of RR interval showed non significant changes.

Frequency domain measurements showed non significant increases for both LF and HF domain, from 6.12±3.21 ms² to 6.83±2.04 ms² and 5.5±1.94 ms² to 10.41±3.37 ms²; respectively. Table 1 summarizes the results.

4. Discussion and conclusions

We have previously shown that treatment of experimental acute heart failure by computerized
controlled aortic balloon pump counterpulsation improves hemodynamic parameters in a animal model of heart failure [4].

Our present results have shown that counterpulsation allows an effective hemodynamic improvement without significant changes in heart rate variability, in open chest sheep.

Heart rate variability analysis has been shown as a useful technique to evaluate prognosis and evolution of several diseases that involve dysfunction of autonomic cardiovascular control, such as diabetes, arterial hypertension, heart failure and acute myocardial infarction.

Table 1. Hemodynamics effects of counterpulsation and HRV in control and induced heart failure.

<table>
<thead>
<tr>
<th></th>
<th>BHF</th>
<th>AHF</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>104.6±25.7</td>
<td>107.7±23.9</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>0.86±0.04</td>
<td>1.29±0.09 †</td>
</tr>
<tr>
<td>CI (L/min/kg)</td>
<td>0.03±0.01</td>
<td>0.04±0.01 †</td>
</tr>
<tr>
<td>SVR (mmHg/L/min)</td>
<td>89.8±6.7</td>
<td>66.6±6 †</td>
</tr>
<tr>
<td>DABAC/SABAC</td>
<td>0.81±0.1</td>
<td>1.12±0.1 †</td>
</tr>
<tr>
<td>Mean RR (ms)</td>
<td>616.4±30.5</td>
<td>641.5±32.3</td>
</tr>
<tr>
<td>SD RR (ms)</td>
<td>3.6±1.3</td>
<td>5.5±4.6</td>
</tr>
<tr>
<td>LF RR (ms²)</td>
<td>6.12±3.21</td>
<td>6.83±2.04</td>
</tr>
<tr>
<td>HF RR (ms²)</td>
<td>5.5±1.94</td>
<td>10.41±3.37</td>
</tr>
</tbody>
</table>

HR: heart rate, CI: cardiac index, CO: cardiac output, SVR: systemic vascular resistance. Values are mean ± SD. † P < 0.05.

In mild cardiac heart failure patients, the baroreceptor inhibitory influence on heart rate is impaired [8]. Besides, it was demonstrated that HRV is an independent predictor of overall mortality and sudden death in chronic congestive heart failure [9]. Consequently, treatment of heart failure would involve changes in HRV.

In this study we evaluated the immediate effect of intraaortic counterpulsation on HRV, using short term periods, this was because the model is acute; future directions for this research could be the analysis of longer periods of time to evaluate chronic effects of counterpulsation on the regulatory mechanisms of heart rate in humans undergoing this treatment.

In this work, we demonstrated the feasibility of HRV analysis in an animal model of acute cardiac failure where, where pressure signals are remarkably modified by intraaortic counterpulsation. The lack of statistical significance could be attributed to biological sources.

References


Address for correspondence.

Marcelo R. Risk, Ph.D.
Center for Signal and Image Processing
Buenos Aires Regional Faculty
National Technological University
Medrano 951 CC1179AAQ
Buenos Aires - Argentina
E-mail: mrisk@cedi.frba.utn.edu.ar