In vivo Assessment of Central and Peripheral Hemodynamic Impact of the C-Pulse Device

OPTIONS HF CLINICAL STUDY

SUNSHINE HEART

[1]Francisco Javier Londoño Hoyos, PhD [2]Dimitrios Georgakopoulos, PhD; Oliver Fey; Dori Jones, MS; [3]Christian Schlensak, MD; David Schibilsky, MD; [4]Michael Weyand, MD; Daniel Bujnoch, MD; [5]Holger Hotz, MD; [6]Patrick Segers, PhD; [7]Les Miller, MD; [1]J. Eduardo Rame, MD, M. Phil.
[1]Hospital of the University of Pennsyvania, Philadelphia, PA; [2]Sunshine Heart Inc., Eden Prairie, MN; [3]University of Tübingen, Tübingen, Germany; [4]University of Erlangen, Erlangen, Germany; [5]Cardio Centrum Berlin, Berlin, Germany; [6]Ghent University, Ghent, Belgium[7]Tampa General Hospital, Tampa, FL

Introduction

The C-Pulse® Heart Assist System is a counterpulsation technology used to treat patients with moderate to severe heart failure (NYHA class III or ambulatory class IV) refractory to optimal medical and device therapy. The implantable device is placed outside the bloodstream and gives patients the ability to disconnect from the system.

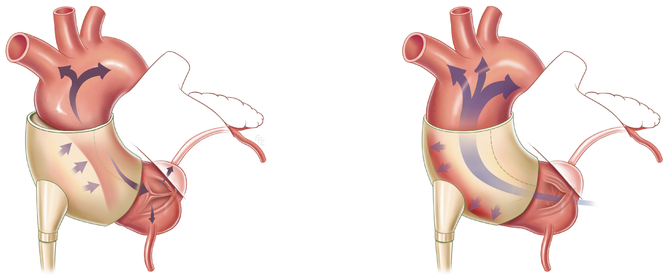


Figure 1. The cuff around the ascending aorta inflates during diastole and starts deflating shortly before ejection, thereby potentially increasing coronary blood flow and decreasing afterload.

Preliminary data, obtained from radial artery applanation tonometry and synthesized central pressure waveforms (using the Sphygmocor/Atcor system) indicated the presence of a pronounced secondary pressure peak in diastole markedly visible on radial artery pressure waveforms.

Synthesized central pressure waveforms suggested that the secondary peak persists (and originates) in the aorta, contributing to increased pressures in diastole (favoring coronary and organ perfusion). In addition, the data indicated an unloading effect of the device on the heart:
1) Lowering of the first systolic pressure peak, and
2) A reduction in the augmentation index.

The aim of this study was to extend and confirm these preliminary findings and to relate these effects to chronic remodeling of the heart. To this end, non-invasively measured pressure waves were combined with flow velocity waveforms for a more complete assessment of central hemodynamics in a cohort of patients implanted with the C-Pulse device.

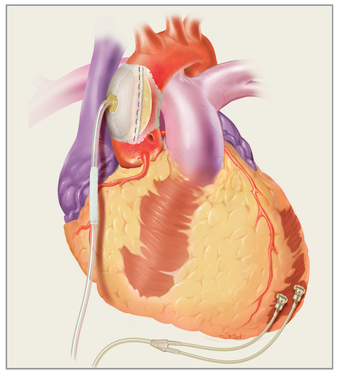


Figure 2: C-Pulse cuff on ascending aorta and epicardial leads used for timing of inflation and deflation of cuff.

Methodology

The study group consisted of 5 patients who had the C-Pulse System implanted, at three cardiac centers in Europe. Mean age was 63 ± 6 years, and 3 of 5 were male. The study protocol consisted of brachial cuff blood pressure measurements (brachial diastolic and systolic blood pressure), applanation tonometry at the right radial and right common carotid artery, and ultrasound Doppler velocity measurements on the right common carotid artery and the left ventricular outflow tract (LVOT) as a surrogate for ascending aorta flow velocity waveforms. Measurements were obtained with the C-Pulse switched off and on. Analysis included assessment of changes in pressure and flow velocity waveforms and derived indices, impedance analysis and separation of the pressure waveform in forward and backward propagating waves, and wave intensity analysis (WIA) to assess the timing and nature of wave reflections.

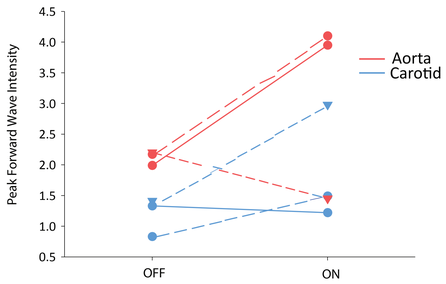
Author Disclosure Information: 1) M. Weyand: None; D. Bujnoch: None; 2) D. Georgakopoulos, O. Fey, D. Jones: Employee, salary; 3) H. Hotz: Consulting Fees/Honoraria; 4) C. Schlensak; D. Schibilsky: None; 5) P. Segers: Consulting Fees/Honoraria; 6) J. Rame: Consulting Fees; F. Hoyos: None; 7) L. Miller: Consulting Fees/Honoraria

Results

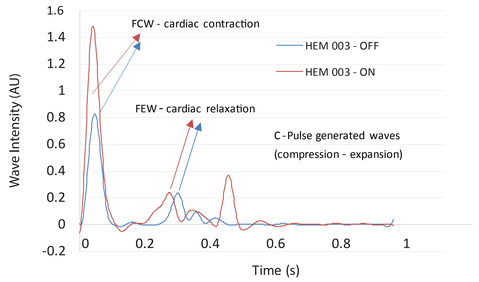
Carotid tonometry was successful in 3 subjects, and confirmed the marked pressure augmentation in diastole. Carotid flow waveforms, available for 4 patients, were clearly modified in diastole, with the appearance of a more pronounced secondary flow peak in early diastole. In 3 patients with complete carotid pressure and flow recordings, wave intensity analysis could be performed. The C-Pulse generated an additional forward compression wave followed by a strong forward expansion wave, generated by the inflation and deflation of the aortic cuff, respectively (Figure 3 A,B). There was a 71% and 53% increase in the forward compression wave generated by the heart with C-pulse ON at the carotid and aortic root, respectively (Figure 3 C).

The impact of C-Pulse on arterial system properties and central wave reflections was also assessed in 3 of the subjects based on carotid pressure and aortic flow analysis. There was no substantial impact on the input impedance of the aorta but a 34% reduction in impedance was observed in the periphery at the carotid artery (Figure 4). In addition, C-Pulse modified the timing and morphology of the backward reflected wave (Pb) at the aortic root. Long term follow-up of patients in the OPTIONS HF post market study has shown an improvement in ejection fraction EF from 21.9 ± 7.4% at baseline (N=13) to 42.5 ± 10% at 12 months (p<0.01; N=(6)) with a concomitant reduction in neurohormone levels as assessed by Nt-ProBNP (4641 ± 5751 vs 2122 ± 1018 pg/mL).

3. Carotid and Aorta Wave Intensity (C)



Carotid Artery (A)



Ascending Aorta (B)

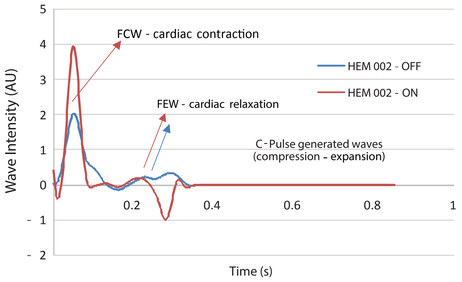


Figure 3. Pressure and flow waveforms were combined to derive wave intensity in the carotid artery (A) and ascending aorta (B). Forward compression wave generated by cardiac contraction and expansion wave generated by C-Pulse. Note the magnitude of the expansion wave from C-Pulse compared to forward wave generated by heart with C-Pulse off (C). Individual patient response showing significant increase in forward energy generated by the heart with C-Pulse.

4. Carotid Artery Impedance

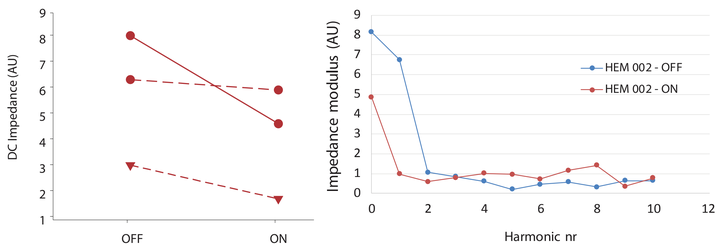
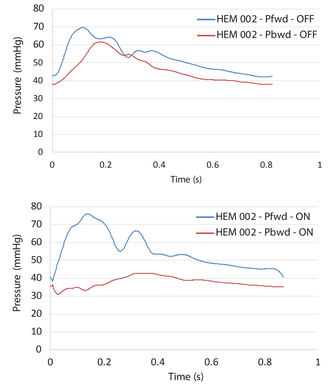


Figure 4. Individual patients response showing reduction in carotid impedance (left panel). Impedance was defined as the 0 Hz component derived from Fourier analysis (right panel).

5. Carotid Artery



Ascending Aorta

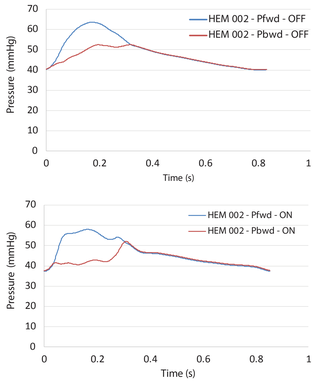


Figure 5. Separation of forward and backward pressure waves in the carotid artery (left panel) and ascending aorta (right panel). Note the marked reduction in amplitude and time of the reflected wave (Pb) in both carotid and aorta.

Conclusion

We conclude that C-Pulse increases central pressure in diastole. The waves generated by the device travel peripherally, and were clearly detectable in the carotid pressure and flow velocity waveforms, as well as in the radial pulse. In this cohort, C-Pulse led to an increase in carotid flow velocity and increased energy output of the heart related to peripheral unloading effects. Future studies will clarify the impact on potential myocardial recovery using chronic counterpulsation and identify the most likely mechanisms between enhanced pulsatility and chronic cardiac remodeling.

CAUTION: C-Pulse is an Investigational device. The device is limited by Federal (United States) Law to investigational use only.

Poster presented at the Utah Cardiac Recovery Symposium (UCARS) 3rd Annual Meeting, January 14–17, Salt Lake City, UT USA.