Baseline diastolic pressure gradient and pressure reduction in chronic heart failure patients implanted with the CardioMEMS™ HF sensor

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Abstract

Aims Remote haemodynamic monitoring (RHM) decreases hospitalization rates in patients with chronic heart failure (HF). Many patients with chronic HF develop pulmonary hypertension (PH) secondary to left heart disease with some acquiring combined pre-capillary and post-capillary PH (Cpc-PH). The efficacy of RHM in achieving pulmonary pressure reductions in patients with Cpc-PH vs. isolated post-capillary PH (Ipc-PH) is unknown. The purpose of this study is to evaluate whether a higher baseline diastolic pressure gradient (DPGbaseline) measured at the time of CardioMEMS™ HF sensor implantation is associated with lower reductions in pulmonary artery diastolic pressures (PADP).

Methods and results This was a retrospective analysis of 32 patients meeting clinical indications for CardioMEMS™ implantation. DPGbaseline categorized patients as Cpc-PH (DPG ≥ 7 mmHg) or Ipc-PH (DPG < 7 mmHg). Minimum achievable PADP (PADPmin) and ΔPADP (PADPbaseline − PADPmin) were determined. Pearson’s correlation analysis and comparison of mean pressure changes were assessed. Median age was 69 years, and median left ventricular ejection fraction (LVEF) was 25%. Eight patients (25%) had a LVEF ≥ 40%. Twenty-five patients (78%) met criteria for Ipc-PH and seven (22%) for Cpc-PH. Neither PADPmin (ρ = 0.27; P = 0.13) nor ΔPADP (ρ = 0.07; P = 0.72) was correlated with DPGbaseline. A trend towards higher ΔPADP was seen in Cpc-PH vs. Ipc-PH patients (15.2 vs. 9.88 mmHg; P = 0.12). There was a moderate positive correlation between baseline PADP and ΔPADP [ρ = 0.55 (0.26–0.76); P < 0.001].

Conclusions Decreased PADP reduction was not seen in Cpc-PH vs. Ipc-PH patients. Higher PADPbaseline was associated with greater ΔPADP. Larger studies are needed to elaborate our findings.

Keywords Combined pre-capillary and post-capillary pulmonary hypertension; Isolated post-capillary pulmonary hypertension; Implantable haemodynamic monitoring; Heart failure

Introduction

Heart failure (HF) patients with pulmonary hypertension (PH) secondary to left heart disease (PH-LHD) experience worse outcomes than those without coexistent PH.²–⁶ Isolated post-capillary PH (Ipc-PH) represents a unique subset of those with PH and can be defined by a diastolic pulmonary gradient (DPG) < 7 mmHg, while combined pre-capillary and post-capillary PH (Cpc-PH) patients have a DPG ≥ 7 mmHg.⁵ More importantly, Cpc-PH patients not only develop maladaptive pulmonary vascular remodelling but also have poorer right ventricular–pulmonary vascular coupling and worse clinical outcomes.¹,⁵,⁷–¹⁰

To date, there is no proven medical therapy to improve morbidity and mortality in patients with PH-LHD.¹¹ A recent retrospective analysis of the PH-LHD subset of patients from the CardioMEMS™ Heart Sensor Allows Monitoring of Pressure to Improve Outcomes in New York Heart Association Class III Heart Failure Patients (CHAMPION) Trial¹² found that pressure-guided therapy with remote haemodynamic monitoring (RHM) using the CardioMEMS™ HF sensor reduced the composite endpoint of death and HF

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hospitalization.13 The study did not evaluate serial haemodynamic changes in patients with Cpc-PH vs. Ipc-PH over time; the CardioMEMSTM HF sensor has been shown to have minimal pressure drift over time.14 Additionally, the clinical implications of a higher DPG at the time of sensor implantation were not explored. Given the unique phenotypic differences in patients with Cpc-PH and Ipc-PH, we felt it was important to characterize the clinical responses to determine if one group was more likely to achieve a higher or lower pressure reduction than the other. Should a substantial difference exist between groups, it would have clinical implications for patient selection and prioritization for device implantation.

We sought to evaluate patients with PH-LHD to determine if a higher baseline DPG measured at the time of CardioMEMSTM HF sensor implantation is associated with a lower serial pressure reduction of the pulmonary artery diastolic pressure (PADP).

Methods

Patient selection

This was a two-site study with an initial cohort of 55 patients meeting clinical indications for implantation of the CardioMEMSTM HF sensor. For the purposes of this analysis, we included only patients with PH-LHD, defined as mean pulmonary artery pressure (mPAP) ≥25 mmHg and pulmonary capillary wedge pressure (PCWP) >15 mmHg. Twenty-three patients who did not have PH-LHD by these criteria were excluded, yielding a final study cohort of 32. Patients were included regardless of ejection fraction, and all patients were required to have the CardioMEMSTM HF sensor implanted for at least 180 days. Written informed consent was not required for this retrospective analysis.

Remote haemodynamic monitoring and transmission

At the time of CardioMEMSTM HF sensor implantation, all patients were provided with device teaching and required to demonstrate their ability to obtain accurate pressure readings using their home unit prior to hospital discharge. Patients were instructed to transmit readings on a daily basis. A trained medical assistant monitored the CardioMEMSTM HF system website (Merlin.net) daily for patient compliance with pressure transmission. If patients did not have pressure transmissions for two consecutive days, they were contacted and encouraged to resume daily readings.

Haemodynamic measurements

Baseline haemodynamic indices were calculated based upon values obtained at the time of CardioMEMSTM HF sensor implantation, referred to as baseline pressures. DPG was calculated PADPbaseline minus PCWP. Transpulmonary pressure gradient (TPG) was calculated as mPAP – PCWP. Pulmonary vascular resistance (PVR) was calculated as TPG/cardiac output. Patients were categorized as having Cpc-PH if they met the criteria for PH-LHD and had a DPG ≥7 mmHg. Patients were categorized as having Ipc-PH if they met the criteria for PH-LHD and had a DPG <7 mmHg. The analysis was repeated using a TPG cut-off of >12 mmHg and a PVR cut-off of ≥3 Wood units to classify patients into the Cpc-PH group3 (Supporting Information). All available CardioMEMSTM pressure readings for each patient were evaluated by a single reviewer (A. M. W.) for a period of up to 180 days from the time of CardioMEMSTM HF sensor implantation. The minimum transmitted PADP (PADPmin) during the study period was recorded based upon review of the pressure transmissions. The maximum magnitude of pressure reduction over the study period was calculated as PADPbaseline – PADPmin and was defined as delta PADP (ΔPADP).

Statistical analysis

Baseline clinical characteristics are reported as either mean ± standard deviation or median and interquartile range. Baseline clinical characteristics were compared with t-tests or the Mann–Whitney U test for continuous variables or Pearson’s 𝜒² test or Fischer’s exact test for categorical variables. The Shapiro–Wilk test was used to evaluate for a normal distribution. Pearson’s correlation analysis was performed between baseline DPG, TPG, and PVR and either PADPmin or ΔPADP. Correlation between PADPmin and ΔPADP with PADPbaseline was assessed using Pearson’s correlation. Similar analyses were performed, categorizing patients into Cpc-PH and Ipc-PH groups based upon TPG and PVR cut-offs as specified above. Significance levels were two-sided with a P value of <0.05.

Results

Patient characteristics

Baseline patient characteristics are shown in Table 1. Median age for the entire cohort was 69 years, 22 (69%) were male, and 19 (59%) were White. Eight patients (25%) had a left ventricular ejection fraction ≥40%, and median ejection fraction was 25%. Based on a DPG ≥7 mmHg, 25 patients (78%) were categorized into the Ipc-PH group and 7 patients (22%) into the Cpc-PH group. There were no significant differences

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between the groups with respect to age, male gender, race, body mass index, ejection fraction, or co-morbid medical conditions. Baseline PADP (29.4 vs. 24.3 mmHg; \( P = 0.0526 \)) and mPAP (40.0 vs. 35.1 mmHg; \( P = 0.27 \)) were numerically greater in the Cpc-PH group than in the Ipc-PH group, while the PCWP (20.7 vs. 24.6 mmHg; \( P = 0.12 \)) was numerically smaller in the Cpc-PH than in the Ipc-PH group, suggesting the former group was closer to euvaemia than the latter. Not surprisingly, TPG was significantly higher in the Cpc-PH group compared to the Ipc-PH group (18.9 vs. 10.5 mmHg, \( P = 0.004 \), respectively). Additionally, cardiac output (4.78 vs. 3.6 L/min; \( P = 0.072 \)) and body mass index (32.7 vs. 29.1 kg/m\(^2\)) were both numerically higher in the Cpc-PH group than in the Ipc-PH group, respectively. Body surface area was not available in all patients; therefore, the calculation of cardiac index and subsequent comparisons were not performed.

**Correlation analysis**

Correlation analysis was applied to assess the association of baseline DPG on the minimum achievable PADP (PADP\(_{\text{min}}\))

### Table 1 Baseline clinical characteristics of patients presented as the entire cohort and stratified according to isolated post-capillary pulmonary hypertension and combined pre-capillary and post-capillary pulmonary hypertension subgroups

| Variable                                      | Entire cohort (\( n = 32 \)) | Ipc-PH (\( n = 25 \)) | Cpc-PH (\( n = 7 \)) | \( P \) value |
|-----------------------------------------------|------------------------------|-----------------------|----------------------|--------------|
| Age (years)                                   | 69 (60–75)                   | 69 (60–74)            | 68 (58–74)           | 0.91         |
| Male                                          | 22 (69%)                     | 18 (72%)              | 4 (57%)              | 0.74         |
| Race (White)                                  | 19 (59%)                     | 14 (56%)              | 5 (71%)              | 0.72         |
| Body mass index (kg/m\(^2\))                 | 29.6 (24–35.1)               | 29.1 (24–35)          | 32.7 (30.7–35.3)     | 0.24         |
| Diabetes mellitus                             | 19 (59%)                     | 16 (64%)              | 3 (43%)              | 0.6          |
| Hypertension                                  | 22 (68%)                     | 19 (76%)              | 3 (43%)              | 0.44         |
| Atrial fibrillation                           | 20 (63%)                     | 17 (68%)              | 3 (43%)              | 0.54         |
| Ischaemic cardiomyopathy                      | 18 (56%)                     | 15 (60%)              | 3 (43%)              | 0.66         |
| Coronary artery disease                       | 19 (59%)                     | 16 (64%)              | 3 (43%)              | 0.6          |
| Heart failure with preserved ejection fraction| 8 (25%)                      | 5 (20%)               | 3 (43%)              | 0.36         |
| Left ventricular ejection fraction (%)         | 25 (20–39)                   | 25 (20–35)            | 38 (20–53)           | 0.29         |
| Creatinine (mg/dL)                            | 1.61 ± 0.47                  | 1.64 ± 0.51           | 1.48 ± 0.32          | 0.32         |
| Pulmonary artery diastolic pressure (mmHg)    | 25.4 ± 5.6                   | 24.3 ± 5.3            | 29.4 ± 5.5           | 0.0526       |
| Mean pulmonary artery pressure (mmHg)         | 36.1 ± 7.3                   | 35.1 ± 6.5            | 40.0 ± 9.3           | 0.27         |
| Pulmonary capillary wedge pressure (mmHg)     | 23.8 ± 5.9                   | 24.6 ± 5.9            | 20.7 ± 5.4           | 0.12         |
| Transpulmonary gradient (mmHg)                | 12.3 ± 5.8                   | 10.5 ± 4.5            | 18.9 ± 5.3           | 0.004        |
| Pulmonary vascular resistance (Wood units)    | 3.4 ± 1.6                    | 3.2 ± 1.6             | 4.1 ± 1.5            | 0.17         |
| Cardiac output (L/min)                        | 3.9 ± 1.4                    | 3.6 ± 1.4             | 4.78 ± 1.4           | 0.072        |

Data presented as mean ± standard deviation or median with interquartile range.

Figure 1 (A) Minimum pulmonary artery diastolic pressure (PADP\(_{\text{min}}\)) is plotted vs. baseline diastolic pressure gradient (DPG) for both isolated post-capillary pulmonary hypertension (Ipc-PH, closed circles) and combined pre-capillary and post-capillary pulmonary hypertension (Cpc-PH, open circles) groups. (B) Change in pulmonary artery diastolic pressure (\( \Delta \text{PADP} \)) is plotted vs. baseline DPG for both Ipc-PH (closed circles) and Cpc-PH (open circles) groups. The correlation coefficient (with 95% confidence interval and \( P \) value) and mean \( \text{PADP}_{\text{min}} \) (A) and mean \( \Delta \text{PADP} \) (B) for Ipc-PH vs. Cpc-PH (with \( P \) values comparing means) are shown superimposed on each figure. Each symbol represents one patient.
and the maximum achievable pressure reduction ($\Delta$PADP) (Figure 1). There was no significant association with baseline haemodynamic indices assessed across a range of DPG from $-6$ to $12$ mmHg. Scatterplots of the raw data are shown in Figure 1. There were no significant differences in the mean PADP$_{\text{min}}$ (14.4 vs. 14.0 mmHg, $P = 0.91$); however, there was a numerical, but not statistically significant, difference in the mean $\Delta$PADP (9.88 vs. 15.4 mmHg, $P = 0.13$) between patients in the Ipc-PH and Cpc-PH subgroups. See Figure S1 for similar analysis using TPG-specific and PVR-specific cut-offs. There was no significant correlation between PADP$_{\text{min}}$ and baseline PADP ($\rho = 0.23$ (-0.12 to 0.54), $P = 0.20$). However, baseline PADP had a moderate and significant positive correlation with $\Delta$PADP ($\rho = 0.56$ (0.26 to 0.76); $P < 0.001$).

**Discussion**

In this cohort of patients with the CardioMEMS™ HF sensor implanted for clinical indications, we found that the baseline haemodynamic index of Cpc-PH studied, DPG, was not correlated with either PADP$_{\text{min}}$ or $\Delta$PADP during a follow-up period of 180 days. However, there was a numerical difference in the mean $\Delta$PADP (15.4 vs. 9.88 mmHg, $P = 0.13$) between the Cpc-PH and Ipc-PH groups, respectively; albeit with only seven Cpc-PH patients (vs. 25 Ipc-PH patients). Nevertheless, this finding warrants investigation in a larger cohort to see if this difference becomes significant—upon completion of the CardioMEMS™ Post Approval Study (NCT02279888), a larger and more definitive analysis may soon be possible. Our finding that $\Delta$PADP increased with higher baseline PAPD may simply represent (1) regression to the mean and/or (2) that pressure reduction is unlikely in a patient with goal or near-goal baseline PAPD. Whether acquired pulmonary vascular disease in Cpc-PH patients poses a barrier to pressure reduction ($\Delta$PADP) remains a key question to answer. If so, it would require clinicians to re-calibrate pressure reduction goals with baseline haemodynamic status in mind. While our findings suggest that the efficacy of RHM may depend on baseline haemodynamic parameters, more investigation is needed.

The clinical implications of baseline haemodynamic status are especially important because current Food and Drug Administration criteria for CardioMEMS™ implantation only requires New York Heart Association functional class III symptoms and an HF hospitalization in the prior year. No consideration is given to concomitant Ipc-PH or Cpc-PH, and given worse outcomes in the Cpc-PH subgroup, we felt it important to explore the magnitude of pressure response. Furthermore, *a priori* knowledge of anticipated pressure reduction based on phenotype may assist in daily management and how best to titrate medical therapy.

To the best of our knowledge, this is the first study to report on the serial haemodynamic changes in a heterogeneous group of patients with PH-LHD undergoing RHM for clinical indications. While these findings are from a small, two-site cohort of patients implanted for clinical indications outside of clinical trials, they suggest that continued serial RHM for patients along a spectrum of PH-LHD is essential because many of these patients will proceed to end-stage HF and ultimately require more advanced therapies. A better understanding of the progression of the haemodynamic profile of patients with worsening HF will likely become a crucial element for

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**Figure 2** (A) Minimum pulmonary artery diastolic pressure (PADP$_{\text{min}}$) is plotted vs. baseline PADP and (B) $\Delta$PADP vs. baseline PADP. Patients with isolated post-capillary pulmonary hypertension (Ipc-PH) are represented by closed circles and those with combined pre-capillary and post-capillary pulmonary hypertension (Cpc-PH) by open circles. The respective correlation coefficient with 95% confidence interval and $P$ value is superimposed on each panel. Each symbol represents one patient.
identifying the optimal timing of advanced HF strategies such as durable mechanical circulatory support\textsuperscript{19} or heart transplantation, especially among the high-risk Cpc-PH population.\textsuperscript{17}

Limitations

There are several limitations to the current analysis. The haemodynamic data were obtained from a relatively small number of patients treated at two different medical centres. Inherent in this study were issues related to the retrospective analysis of the data. There was no pre-specified, standardized reporting method for haemodynamic assessment. The small sample size limits the statistical power and generalizability of our findings. Association with outcome data was not explored. Additionally, management of elevated pressures did not follow a standardized protocol and therefore was provider specific across both institutions. Pulmonary vascular vasoreactivity studies to evaluate reversibility of pulmonary pressures were not routinely performed and were not incorporated into our analysis.

Conclusions

During a 6 month follow-up period, there was no observed correlation with serial pressure reduction and baseline DPG in patients managed with the CardioMEMSTM HF system. The finding of a numerical, but not statistically significant, difference between mean $\Delta$PADP in the Cpc-PH vs. Ipc-PH groups warrants additional investigation. As such, larger studies of patients with PH-LHD along a wider spectrum of pulmonary vascular disease with associated outcome data are needed to better understand the implications of Cpc-PH in patients managed with RHM.

References

1. Gerges M, Gerges C, Pistritto A-M, Lang MB, Trip P, Jakowitsch J, Binder T, Lang IM. Pulmonary hypertension in heart failure. Epidemiology, right ventricular function, and survival. Am J Respir Crit Care Med 2015; \textbf{192}: 1234–1246.
2. Tampakakis E, Leary PJ, Selby VN, De Marco TA, Cappola TP, Felker MG, Russell SD, Kasper EK, Tedford RJ. The diastolic pulmonary gradient (DPG) does not predict survival in patients with pulmonary hypertension due to left heart disease (PH-LHD). \textit{JACC Heart Fail} 2015; \textbf{3}: 9–16.
3. Vachiéry J-L, Adir Y, Barberà JA, Champion H, Coghlan JG, Cottin V, De Marco T, Galié N, Ghio S, Gibbs JSR, Martinez F, Semigran M, Simonneau G, Wells A, Seeger W. Pulmonary hypertension due to left heart diseases. \textit{J Am Coll Cardiol} 2013; \textbf{62}: D100–D108.
4. Guazzi M, Naeije R. Pulmonary hypertension in heart failure: pathophysiology, pathobiology, and emerging clinical perspectives. \textit{J Am Coll Cardiol} 2017; \textbf{69}: 1718–1734.
5. Ghio S, Guazzi M, Scardovi AB, Klerys C, Clemenza F, Carluccio E, Temporelli PL, Rossi A, Faggiano P, Traversi E, Vriz O, Dini FL. On Behalf of All Investigators. Different correlates but similar prognostic implications for right ventricular dysfunction in heart failure patients with reduced or preserved ejection fraction. \textit{Eur J Heart Fail} 2017; \textbf{19}: 873–879.
6. Miller WL, Grill DE, Borlaug BA. Clinical features, hemodynamics, and outcomes of pulmonary hypertension due to chronic heart failure with reduced ejection fraction: pulmonary hypertension and heart failure. \textit{JACC Heart Fail} 2013; \textbf{1}: 290–299.
7. Guazzi M, Dixon D, Labate V, Beussink-Nelson L, Bander F, Curtica MJ, Shah SJ. RV contractile function and its coupling to pulmonary circulation in heart failure with preserved ejection fraction: stratification of clinical phenotypes and outcomes. \textit{JACC Cardiovasc Imaging}.

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Conflict of interest

D. M. S. has served as a paid consultant, is on the speaker’s bureau, and receives research support from Abbott Vascular. A. M. W has received speaking fees from Abbott Vascular. L. G. has received speaking fees from Abbott Vascular. L. S. has received research support from Abbott Vascular. R. J. is on the speaker’s bureau and receives research support from Abbott Vascular.

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Supporting information

Additional Supporting Information may be found online in the supporting information tab for this article.

Figure S1. Minimum pulmonary artery diastolic pressure ($\text{PADP}_{\text{min}}$) is plotted vs. baseline transpulmonary gradient (TPG) and baseline pulmonary vascular resistance (PVR) and shown in panels A and C, respectively. Change in pulmonary artery diastolic pressure ($\Delta$PADP) is plotted vs. baseline TPG and baseline PVR and shown in panels B and D, respectively. Patients with Ipc-PH are represented by closed circles and those with Cpc-PH by open circles. The respective correlation coefficient with 95% confidence interval and $P$ value is superimposed on each panel. Each symbol represents one patient.
8. Guazzi M, Labate V. Pulmonary hypertension in heart failure patients: pathophysiology and prognostic implications. *Curr Heart Fail Rep* 2016; 13: 281–294.

9. Guha A, Amione-Guerra J, Park MH. Epidemiology of pulmonary hypertension in left heart disease. *Prog Cardiovasc Dis* 2016; 59: 3–10.

10. Hoeper MM, Lam CSP, Vachiery J-L, Bauersachs J, Gerges C, Lang IM, Bondemier D, Olsson KM, Gibbs JSR, Dorfmuller P, Guazzi M, Galie N, Manes A, Handoko ML, Vonk-Noordegraaf A, Lankeit M, Konstantinides S, Wachter R, Opitz C, Rosenkranz S. Pulmonary hypertension in heart failure with preserved ejection fraction: a plea for proper phenotyping and further research. *Eur Heart J* 2016: ehw597.

11. Assad TR, Hemnes AR, Larkin EK, Glazer AM, Xu M, Wells QS, Farber-Eger EH, Sheng Q, Shyr Y, Harrell FE, Newman JH, Brittain EL. Clinical and biological insights into combined post-and pre-capillary pulmonary hypertension. *J Am Coll Cardiol* 2016; 68: 2525–2536.

12. Abraham WT, Adamson PB, Bourge RC, Aaron MF, Costanzo MR, Stevenson LW, Strickland W, Neelagari S, Raval N, Krueger S, Weiner S, Shavelle D, Jeffries B, Yadav JS. Wireless pulmonary artery haemodynamic monitoring in chronic heart failure: a randomised controlled trial. *The Lancet* 2011; 377: 658–666.

13. Benza RL, Raina A, Abraham WT, Adamson PB, Lindenfeld J, Miller AB, Bourge RC, Bauman J, Yadav J. Pulmonary hypertension related to left heart disease: insight from a wireless implantable hemodynamic monitor. *J Heart Lung Transplant* 2015; 34: 329–337.

14. Abraham WT, Adamson PB, Hasan A, Bourge RC, Pamboukian SV, Aaron MF, Raval NY. Safety and accuracy of a wireless pulmonary artery pressure monitoring system in patients with heart failure. *Am Heart J* 2011; 161: 558–566.

15. CardioMEMS HF System post approval study: full text view. ClinicalTrials.gov 2014. https://clinicaltrials.gov/ct2/show/NCT02279888 (6 Dec 2017).

16. Naeije R, Gerges M, Vachiery J-L, Caravita S, Gerges C, Lang IM. Hemodynamic phenotyping of pulmonary hypertension in left heart failure. *Circ Heart Fail* 2017; 10: e004082.

17. Davey R, Raina A. Hemodynamic monitoring in heart failure and pulmonary hypertension: from analog tracings to the digital age. *World J Transplant* 2016; 6: 542–547.

18. Feldman D, Naka Y, Cabuay B, Takayama H, Bauman J, Cowart P, Corcoran K, Levy W, Moazami N. A wireless hemodynamic pressure sensor before and after ventricular assist device placement: a sub-study of the CHAMPION trial. *J Heart Lung Transplant* 2011; 30: S86.

19. Al-Kindi SG, Farhoud M, Zacharias M, Gainwalla MB, ElAmm CA, Benatti RD, Oliveira GH. Left ventricular assist devices or inotropes for decreasing pulmonary vascular resistance in patients with pulmonary hypertension listed for heart transplantation. *J Card Fail* 2017; 23: 209–215.