Wireless pulmonary artery haemodynamic monitoring in chronic heart failure: a randomised controlled trial

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Summary

Background Results of previous studies support the hypothesis that implantable haemodynamic monitoring systems might reduce rates of hospitalisation in patients with heart failure. We undertook a single-blind trial to assess this approach.

Methods Patients with New York Heart Association (NYHA) class III heart failure, irrespective of the left ventricular ejection fraction, and a previous hospital admission for heart failure were enrolled in 64 centres in the USA. They were randomly assigned by use of a centralised electronic system to management with a wireless implantable haemodynamic monitoring (W-IHM) system (treatment group) or to a control group for at least 6 months. Only patients were masked to their assignment group. In the treatment group, clinicians used daily measurement of pulmonary artery pressures in addition to standard of care versus standard of care alone in the control group. The primary efficacy endpoint was the rate of heart-failure-related hospitalisations at 6 months. The safety endpoints assessed at 6 months were freedom from device-related or system-related complications (DSRC) and freedom from pressure-sensor failures. All analyses were by intention to treat. This trial is registered with ClinicalTrials.gov, number NCT00531661.

Findings In 6 months, 84 heart-failure-related hospitalisations were reported in the treatment group (n=270) compared with 120 in the control group (n=280; rate 0·32 vs 0·44, hazard ratio [HR] 0·72, 95% CI 0·60–0·85, p=0·0002). During the entire follow-up (mean 15 months [SD 7]), the treatment group had a 37% reduction in heart-failure-related hospitalisation compared with the control group (158 vs 254, HR 0·63, 95% CI 0·52–0·77; p<0·0001). Eight patients had DSRC and overall freedom from DSRC was 98·6% (97·3–99·4%) compared with a prespecified performance criterion of 80% (p<0·0001); and overall freedom from pressure-sensor failures was 100% (99·3–100·0%).

Interpretation Our results are consistent with, and extend, previous findings by definitively showing a significant and large reduction in hospitalisation for patients with NYHA class III heart failure who were managed with a wireless implantable haemodynamic monitoring system. The addition of information about pulmonary artery pressure to clinical signs and symptoms allows for improved heart failure management.

Funding CardioMEMS.

Introduction

Despite current treatments, rates of hospital admissions for heart failure have improved little during the past three decades.¹ In the USA, between 1996 and 2006, hospital discharges for heart failure rose from 877 000 to 1 106 000.² Among beneficiaries of Medicare, 27% of discharged patients with heart failure were readmitted to hospital within 30 days.³ More than half the US$39·2 billion yearly direct cost of care for heart failure in the USA is attributable to the cost of treatment in hospital. The estimated average cost of the final 2 years of life for patients with heart failure is greater than $156 000, with more than 75% attributable to hospital admission for heart failure during the last 6 months of life.⁴ Improvements in outpatient management of patients with chronic heart failure are needed to address the increasing burden of worsening heart failure that requires admission to hospital.⁵ Patients are usually admitted to hospital for heart failure because of worsening signs and symptoms of congestion.⁶ Previous investigations have shown that increases in intracardiac and pulmonary artery pressures are the cause of this clinical congestion and are apparent several days to weeks before the onset of worsening signs, symptoms, and hospital admission,⁶,⁷ suggesting that early intervention targeting these pressures might reduce the risk of admission to hospital. In a clinical trial,⁸ increases in intracardiac pressures often arose independently of weight changes, such that monitoring of weight alone was inadequate to identify congestion in time to avert the events associated with heart failure. This finding might account for why telemonitoring systems that rely on patient-reported assessment of general health, symptoms of heart failure, and daily weight change have not reduced re-admission or mortality rates.⁷

Implantable systems for chronic monitoring of intracardiac and pulmonary artery pressures have been developed.⁹–¹⁵ Preliminary findings with the use of these systems suggest a reduction in hospital admissions for...
heart failure. The hypothesis in the present study, the CardioMEMS Heart Sensor Allows Monitoring of Pressure to Improve Outcomes in NYHA Class III Heart Failure Patients (CHAMPION) trial, was that management of heart failure by use of pulmonary artery pressures would greatly reduce the rate of heart-failure-related hospitalisation.

Methods
Patients
Patients (aged ≥18 years) were eligible for participation in the CHAMPION study if they had moderate (New York Heart Association [NYHA] functional class III) heart failure for at least 3 months, irrespective of left ventricular ejection fraction or cause, and a hospitalisation for heart failure within the past 12 months. Patients had to be given drug and device treatments for heart failure at optimum or best-tolerated stable doses, according to national guidelines. Exclusion criteria included a history of recurrent pulmonary embolism or deep venous thrombosis, cardiac resynchronisation with device implantation within the preceding 3 months, and stage IV or V chronic kidney disease (glomerular filtration rate <25 mL/min per 1·73 m²). The other inclusion and exclusion criteria have been described previously.
institutional review board of each participating centre approved the study protocol, and all patients provided written informed consent.

Study design
CHAMPION was a prospective, multicentre (n=64), single-blind, clinical trial undertaken in the USA. Eligible patients underwent implantation of a pulmonary artery sensor, an integral part of the wireless implantable haemodynamic monitoring (W-IHM) system (Champion, CardioMEMS, Atlanta, GA, USA). This system has a passive, wireless, radiofrequency sensor without batteries or leads, and has been described in detail elsewhere (figure 1). After sensor implantation, patients were admitted to hospital overnight for observation. Those receiving anticoagulant drugs were restarted on treatment. Participants who were not taking warfarin were placed on aspirin (81 mg/day or 325 mg/day, orally) and clopidogrel for 1 month (75 mg/day, orally) after insertion of the implant; after 1 month, only aspirin was continued. Before discharge from hospital, participants were randomly assigned to a treatment group, which allowed the clinician access to their pulmonary artery pressure readings that were obtained through the W-IHM system, or to a control group from which such access was blocked. Before discharge, patients were trained to operate the home electronic console. All patients, in treatment and control groups, took daily pressure readings. These measurements were transmitted through a modem to a secure patient database. The protocol-defined treatment goal for patients managed with clinician knowledge of W-IHM data was to lower pulmonary artery pressures when elevated, using neurohormonal, diuretic, or vasodilator drugs. All patients were on optimum drug and device therapies at the time of sensor implantation in accordance with the guidelines of the American College of Cardiology and American Heart Association. The data from the sensor was only available to the physician for patients in the treatment group, and drug changes were made based on information about the sensor haemodynamics and standard of care, which includes patients’ signs and symptoms. The control group continued to receive standard of care management, which included drug changes in response to patients’ clinical signs and symptoms. To assure a high background standard of care, only study sites with experience in management of heart failure were chosen to participate in the CHAMPION trial.

In the treatment group, review of pressure data was done at least once a week and more frequently, if changes occurred in treatment. All patients were scheduled to be seen by their clinician at 1 month, 3 months, and 6 months, and every 6 months thereafter. Visits included a physical examination, assessment of NYHA class and quality-of-life assessment by use of the 21-question Minnesota Living with Heart Failure questionnaire (MLHFQ), and review of drugs. Adverse events were recorded continuously.

Randomisation and masking
Randomisation was done by use of a computer-generated schedule stratified by study site, with block sizes of four, and maintained by use of a validated 21 Code of Federal Regulations, Part II, compliant system. Investigators enrolled patients who were randomly assigned in a 1:1 ratio by use of a centralised electronic system. To maintain patient masking, all patients were asked to
take pressure readings every day. Standardised clinician communication scripts were provided for telephone calls to patients about changes to drugs. Sites were required to balance the number of contacts between patients in the treatment and control groups. Patients were masked to their assignment group. The masking of patients was maintained until analysis of the 6-month data was complete for the entire patient population.

**Statistical analysis**

The primary efficacy endpoint was the rate of heart-failure-related hospitalisations during the 6 months after insertion of the implant in the treatment group versus the control group. The two primary safety endpoints were device-related or system-related complications (DSRC) defined as an adverse event that was definitely or possibly related to the wireless pressure sensor or external electronics, and was treated with invasive means other than intramuscular administration of drugs or a right-heart catheterisation; and pressure-sensor failure defined as an inability to obtain readings.

For the primary efficacy endpoint, a sample size of 248 patients per group, estimated through simulation, provided 90% power by use of a negative binomial regression procedure. To account for potential early discontinuations, 550 patients were enrolled. For the primary safety endpoints, assuming a final nominal significance continued to be shown. These were change hierarchically by use of α=0·05 at each stage, if superiority to rates of 0·80 and 0·90, respectively.

Each of the final primary safety and efficacy analyses required a significance level of 0·048, based on O’Brien-Fleming boundaries for one planned interim analysis. Freedom from DSRC and freedom from pressure-sensor failure defined as an inability to obtain readings.

Four secondary efficacy endpoints were analysed hierarchically by use of α=0·05 at each stage, if significance continued to be shown. These were change in pulmonary artery pressures to 6 months, measured as area under the curve of pulmonary artery pressure relative to baseline (analysis of covariance with baseline pressure as the covariate); proportion of patients admitted to hospital for heart failure during the first 6 months (Fisher’s exact test); days alive outside hospital for heart failure during the first 6 months (Wilcoxon rank sum test); and the rate of heart-failure-related hospitalisations by baseline systolic function at 6 months (negative binomial regression procedure). All analyses were by intention to treat.

Cost-effectiveness was evaluated by use of a decision analytical model based on the Markov cohort simulation, developed to capture the clinical events and costs for a hypothetical cohort of patients from the time of intervention through a maximum of 5 years. Patients were assessed in terms of two health states (alive or dead) with separately estimated transition probabilities based on Kaplan-Meier survival curves for the treatment and control groups. Survival probabilities beyond the trial period were estimated with an exponential survival method; log-rank test); duration of heart-failure-related hospitalisation (Wilcoxon rank sum test); number of changes to drugs for heart failure (Wilcoxon rank sum test); and the rate of heart-failure-related hospitalisations by baseline systolic function at 6 months (negative binomial regression procedure). All analyses were by intention to treat.
model. For patients who were alive, the period of survival was weighted by patients’ utility measured with the European Quality of Life-5 Dimensions. Costs evaluated in the model included costs of W-IHM sensor implantation and device, heart-failure-related hospitalisation, drugs for outpatients, and end-of-life support for those who died. Total costs and quality-adjusted life years (QALYs) were modelled according to the time (in monthly intervals) patients spent in each health state. The model was constructed with TreeAge Pro 2006 Software (version 1.0).

The data analysis centre was Stat-Tech Services, Chapel Hill, NC, USA. An independent data safety monitoring board reviewed all available safety data during 25% and 75% masked interim analyses. An unmasked interim analysis of safety and efficacy was undertaken after 50% of patients completed at least 6 months of follow-up. An independent, masked clinical events classification committee reviewed all available clinical safety data and assessed the occurrence of heart-failure-related hospitalisations. All adverse events, hospital admissions, and deaths were reviewed and adjudicated by this committee and used in the final data analyses.

This trial was prospectively registered with ClinicalTrials.gov, number NCT00531661.

Role of the funding source
The study was designed by WTA, PBA, and advisers, including the steering committee, and the sponsor. Data were monitored, collected, and managed by the sponsor. WTA and PBA had full access to study data and final responsibility for the decision to submit for publication.

Results
Between Sept 6, 2007, and Oct 7, 2009, 550 patients were randomly assigned to the treatment (n=270) and control groups (n=280). Figure 2 shows the trial profile. All patients remained in their assigned group until the last patient completed 6 months of follow-up. The mean follow-up was 15 months (SD 7, total duration 250 176 patient days). The group sizes were similar with respect to baseline characteristics (table 1). All analyses were undertaken on patients in their original assignment groups.

Both primary safety endpoints were met. Patients had 98.6% freedom from DSRC (95% CI 97.3–99.4; table 2) and control groups (100%, 95% CI 99.3–100.0; table 2). The rate of heart-failure-related hospitalisations at 6 months (primary efficacy endpoint) was reduced by 28% in the treatment group (table 2). The numbers of non-heart-failure-related hospitalisations were not different in the treatment and control groups (146 vs 143, p=0.66).

Over the entire randomised follow-up, the rate of heart-failure-related hospitalisations was reduced by 37% in the treatment group (figure 3A). Additionally, the treatment group had a lower risk of death or first heart-failure-related hospitalisation (figure 3B).

The treatment group had a greater reduction in pulmonary artery mean pressure, fewer patients admitted to hospital for heart failure, more days alive outside hospital, and better quality of life than did the control group during 6 months of follow-up (table 2). For the quality-of-life assessment, analysis data were available at 6 months for 465 of 550 patients (or 9765 [85%] of 11 550 datapoints). 36 (<1%) of

<table>
<thead>
<tr>
<th>Primary efficacy endpoints*</th>
<th>Not enrolled (n=25)</th>
<th>Treatment group (n=270)</th>
<th>Control group (n=280)</th>
<th>All patients (n=575)</th>
<th>Risk (95% CI)</th>
<th>p value</th>
<th>NNT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart-failure-related hospitalisations up to 6 months (number; events per patient per 6 months)</td>
<td>NA</td>
<td>84 (32)</td>
<td>120 (44)</td>
<td>NA</td>
<td>0.721 (0.60–0.85)</td>
<td>0.0002</td>
<td>8</td>
</tr>
<tr>
<td>Primary safety endpoint†</td>
<td>Device-related or system-related complications</td>
<td>2 (8%)</td>
<td>3 (1%)</td>
<td>3 (1%)</td>
<td>8 (1%)</td>
<td>5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pressure-sensor failures</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>&lt;0.0001</td>
<td>NA</td>
</tr>
<tr>
<td>Prespecified supplementary efficacy endpoints‡</td>
<td>Heart-failure-related hospitalisations during entire randomised follow-up</td>
<td>NA</td>
<td>158</td>
<td>254</td>
<td>NA</td>
<td>0.631 (0.52–0.77)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Secondary efficacy endpoints</td>
<td>Change from baseline in pulmonary artery mean pressure at 6 months (mm Hg; days; mean area under the curve)</td>
<td>NA</td>
<td>−156</td>
<td>33</td>
<td>NA</td>
<td>NA</td>
<td>0.008</td>
</tr>
<tr>
<td>Patients admitted to hospital for heart failure at 6 months</td>
<td>NA</td>
<td>55 (20%)</td>
<td>80 (29%)</td>
<td>NA</td>
<td>0.721 (0.53–0.96)</td>
<td>0.03</td>
<td>NA</td>
</tr>
<tr>
<td>Days alive outside hospital at 6 months (mean, SD)</td>
<td>NA</td>
<td>174.4 (31.1)</td>
<td>172.1 (37.8)</td>
<td>NA</td>
<td>NA</td>
<td>0.02</td>
<td>NA</td>
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<tr>
<td>Minnesota Living with Heart Failure Questionnaire at 6 months (mean, SD)</td>
<td>NA</td>
<td>45 (26)</td>
<td>51 (25)</td>
<td>NA</td>
<td>NA</td>
<td>0.02</td>
<td>NA</td>
</tr>
</tbody>
</table>

Data are number (%) or number, unless otherwise indicated. NNT=number needed to treat; NA=not applicable. *p value from negative binomial regression for comparison of treatment group with control group. †Hazard ratio. ‡p value from exact test of binomial proportions of freedom from events compared with 80% (device-related or system-related complications) and 90% (pressure sensor failure) for all patients. §Risk difference not reported because analysis by randomisation group was not prespecified. ¶p value from the Anderson–Gill model for comparison of treatment group with control group. ||Relative risk.

Table 2: Effect of wireless implantable haemodynamic monitoring on safety and efficacy endpoints
of anticoagulation [n=2], prolonged hospitalisation secondary to resumption of therapeutic anticoagulation for mechanical heart valve [n=1]), two were exacerbations of pre-existing atrial dysrhythmias during right heart catheterisation, two febrile illnesses, one pulmonary in-situ thrombus during right-heart catheterisation that was treated with anticoagulation, one cardiogenic shock, one atypical chest pain, and one delivery-system failure that required a snare to remove the delivery system. No episodes of pulmonary infarction or embolism associated with the sensor (during or after the implant procedure) occurred during the trial. No events required removal of the sensor.

**Discussion**

W-IHM is safe and significantly reduces the risk of heart-failure-related hospitalisation in patients with NYHA functional class III heart failure. The 6-month risk of heart-failure-related hospital admission was 30% lower in the W-IHM group, managed with daily measurement of pulmonary artery pressures plus standard of care, than in the control group, managed according to standard-of-care monitoring of heart failure alone. This reduction in risk lasted the entire period of the randomised single-blind follow-up. The number needed to treat to prevent one heart-failure-related hospitalisation was highly favourable and similar to other standard treatments for heart failure. The treatment group also had significant reduction in pulmonary artery mean pressure, fewer patients admitted to hospital for heart failure, more days alive outside hospital, and better quality of life than did the control group. Compared with the other treatment approaches to heart failure, W-IHM produced reductions...
in heart-failure-related morbidity that were of similar but additive magnitude.20–25 By contrast with other treatments that have been proven to be effective only in patients with decreased systolic function, W-IHM similarly improved outcomes in patients with reduced and preserved systolic function.

Traditional clinical methods and interventions known to reduce heart-failure-related morbidity were active in both groups of this trial. The high-quality management of heart failure provided by trial sites is shown by the very low rate of heart-failure-related hospitalisation in the control group. This rate was much lower than the rates noted in other trials.8,9 Thus, the benefits of W-IHM were in addition to the proven treatments and management strategies for heart failure. The management strategy tested in the CHAMPION trial allowed further optimisation of standard treatments for heart failure, and titration of diuretic treatment to significantly reduce increased pressures and improve clinical outcomes. By contrast, non-implantable telemonitoring systems for heart failure do not seem to improve outcomes compared with standard of care. In the largest randomised, controlled trial done so far, a comprehensive non-invasive telemonitoring system did not reduce morbidity or mortality in 1653 patients who were randomly assigned to the telemonitoring system versus usual care.9

The W-IHM system was safely implanted by a variety of cardiology subspecialists, including heart failure specialists, electrophysiologists, and interventional cardiologists. The safety profile and adverse event rates were similar to those reported for right heart catheterisation,26 and better than those reported for other permanent implants used in the management of heart failure (eg, pacemakers, defibrillators),27–29 mainly because of a lack of the complications associated with the placement of transvenous leads and subcutaneous impulse generators. Thus, the overall risk and benefit of management of heart failure by use of this W-IHM system compares favourably with current device-based approaches to treatment of heart failure.

Previous studies of implantable haemodynamic monitoring systems provided pilot data and suggestions of clinical benefit (panel).8,13,15 These reports were difficult to interpret because they had small numbers of patients, were statistically underpowered, or lacked a control group. Our study did not have these limitations. The CHAMPION protocol also differed from previous haemodynamic monitoring studies in that specific recommendations were made on how to use pressures in the management of heart failure. The potential limitations of the present trial included the challenges inherent in maintaining patient masking and in minimisation of the effect of investigator–patient and device–patient interactions on outcome. Great care was taken to successfully assure patient masking and a singular, objective primary endpoint was chosen to minimise the potential effect of unmasking. A strength of this trial was the prolonged single-blind follow-up after the assessment of the primary endpoint at 6 months. This prolonged follow-up allowed demonstration of the durability of the treatment effect. This trial was not powered to detect a mortality benefit, and larger trials of haemodynamic monitoring will be needed to address this important question.

The generalisability of these results to most patients with NYHA class III heart failure is likely to be good because the exclusion criteria for this study were few.

The major restriction to inclusion was stage IV or V chronic kidney disease because these patients might be difficult to treat (ie, diurese) even with knowledge of increased pulmonary artery pressures. The CHAMPION trial represents the first positive, randomised, adequately powered clinical trial of implantable haemodynamic monitoring in patients with moderately symptomatic heart failure. The addition of information about pulmonary artery pressure to clinical signs and symptoms allows for improved heart-failure management and leads to a reduction in heart-failure-related hospitalisations.

**Contributors**

The authorship group was made up of the CHAMPION co-principal investigators (WTA, PBA), steering or publication committee members (WTA, PBA, RCB, MFA, LWS, and JSY), and other key members of the trial execution team (MRC, WS, SN, NR, SK, SW, DS, and BJ). These individuals contributed to the conception, design, and execution of the study, analysis and interpretation of data, drafting the report (WTA, PB, BJ, and JSY) or revising it critically for important intellectual content (all others), and final approval of the submitted report.

**Conflicts of interest**

WTA, PBA, RCB, MFA, and LWS are consultants to CardioMEMS. RC, WS, SN, NR, SK, SW, and DS received research grants from CardioMEMS. BJ and JSY are employees of CardioMEMS. PBA is a consultant for Medtronic and St Jude Medical. MRC is a consultant for CHP Solutions.
Medtronic, and St Jude Medical. RCB has received grants from Medtronic. MRC has received grants from St Jude Medical and Gilead Sciences.

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Acknowledgments
CardioMEMS, Atlanta, GA, USA, sponsored this study. We wish to acknowledge the substantial contributions of key CHAMPION trial personnel—Susan Neville, Pam Cowwart, Greg Giron, John Henderson, Jordan Bauman, Melissa Webb, Kevin Corcoran, David Stern, Sandeep Yadav, Stephan Ogenstad, Rich Holcomb, and Spencer Kubo.

References
17. Adamson PB, Abraham WT, Bourge RC, et al. CHAMPION trial rationale and design: the long-term safety and clinical efficacy of a


