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ORIGINAL RESEARCH ARTICLE

Prognostic value of resting cardiac power index depends on mean arterial pressure in dilated cardiomyopathy

Ryota Morimoto*, Takashi Mizutani, Takashi Araki, Hideo Oishi, Yuki Kimura, Shingo Kazama, Naoki Shibata, Tasuku Kuwayama, Hiroaki Hiraiwa, Toru Kondo, Kenji Furusawa, Takahiro Okumura and Toyoaki Murohara

Department of Cardiology, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya, Aichi 466-8550, Japan

Abstract

Aims In recent decades, haemodynamic parameters have been estimated for risk stratification and determining treatment strategies for patients with non-ischaemic dilated cardiomyopathy (DCM). In various invasive procedures, the cardiac pumping capability is defined as cardiac power output (CPO), which is calculated by multiplying cardiac output by the mean arterial pressure. Lower CPO values in advanced heart failure predict adverse outcomes. However, few studies discuss the prognostic value of CPO in mild-to-moderate phase patients. This study aimed to determine the value of the cardiac power index (CPI) obtained from the resting CPO for predicting the prognosis of patients with New York Heart Association Functional Class II or III DCM.

Methods and results From March 2000 to January 2020, a total of 623 cardiomyopathy patients were evaluated for haemodynamic parameters. Patients with secondary cardiomyopathy, ischaemic cardiomyopathy, valvular heart disease, and Class IV cardiomyopathy were excluded. A total of 176 DCM patients fulfilled the criteria for inclusion. Patients were 51.7 ± 12.5 years old (mean ± standard deviation) with a mean left ventricular ejection fraction of 32.1 ± 9.2%. The patients were divided into two groups by their median CPI (CPI $<$ 0.52, low-CPI; CPI $\geq$ 0.52, high-CPI). No significant differences were found in the left ventricular end-diastolic diameter, left ventricular ejection fraction, or pulmonary arterial wedge pressure between the groups. The probability of cardiac event-free survival was significantly lower for low-CPI than for high-CPI groups by Kaplan–Meier analysis (P = 0.012), even with no significant difference between the high and low cardiac index groups (P = 0.069). Furthermore, Cox proportional hazards regression analysis revealed that, in addition to the CPI, the systolic and mean arterial pressure involved in CPI calculation were independent predictors of cardiac events. Indeed, among these factors, mean arterial pressure had the strongest prognostic ability.

Conclusions Although CPI is effective for stratifying DCM and predicting cardiac events in patients with Class II/III DCM, this prognostic value depends on mean arterial pressure.

Keywords Cardiac power index; Mean arterial pressure; Dilated cardiomyopathy; Right ventricular catheterization; Prognosis

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**Correspondence to: Ryota Morimoto, Department of Cardiology, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya, Aichi 466-8550, Japan. Tel: +81-52-744-2147; Fax: +81-52-744-2210. Email: ryota.m0726@med.nagoya-u.ac.jp*
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Introduction

Right heart catheterization (RHC) is performed to estimate cardiac function, volume status, and intracardiac pressure. However, in one study,[1] cardiac output (CO) and pulmonary arterial wedge pressure (PAWP) did not demonstrate significant efficacy on the stratification of patients. Among haemodynamic parameters, CO is affected by the interactions of biventricular contractility, intravascular volume, and filling pressure. Vascular compliance and systemic compensating mechanisms help reduce clinical symptoms. In various haemodynamic parameters, cardiac power output (CPO)

simultaneously measures CO and mean arterial pressure (AP) and reflects both the cardiac power and the resistance of the conducting vessels. The CPO and cardiac power index (CPI) are calculated by dividing the resting CPO by the body surface area and are useful predictors in patients with cardiogenic shock and acute cardiac disease.[2,3] In 2017, Yildiz *et al*.[4] reported that resting CPO strongly predicted adverse clinical events. Although both the CPO and CPI are effective for the stratification of advanced patients, their long-term prognostic ability for patients with stable-status non-ischaemic cardiomyopathy is unknown. Given that CO is affected by the body surface area, we hypothesized that CPI could stratify dilated cardiomyopathy (DCM) patients, even those with mild-to-moderate disease defined as no symptoms at rest, and predict subsequent cardiac events.

Methods

Data from 623 consecutive patients with cardiomyopathy from March 2000 to January 2020 were retrospectively collected. All patients underwent laboratory measurements, echocardiography, and cardiac catheterization to facilitate estimations of their general conditions. A diagnosis of DCM was made based on echocardiographic measurements of left ventricular ejection fraction (LVEF) $\leq$45%, left ventricular end-diastolic dimension $\geq$55 mm, a lack of coronary artery stenosis requiring therapeutic intervention, prior evidence of primary valvular disease, and secondary cardiomyopathy. After excluding patients with New York Heart Association (NYHA) Class IV cardiomyopathy, 176 DCM patients were finally enrolled. The study protocol was approved by the Ethics Review Board of the Nagoya University School of Medicine, and written informed consent was provided by all study subjects.

Echocardiography

M-mode, two-dimensional echocardiography, Doppler blood flow, and tissue Doppler imaging using a Vivid 7 system (GE Healthcare, Milwaukee, WI) were performed in accordance with the American Society of Echocardiography guidelines.[5] LVEF was calculated by using two-dimensional apical images and the modified Simpson's method. The peak flow velocities at the mitral level during rapid filling (E) and atrial contraction (A) and E/A were calculated from pulsed Doppler data. The tissue Doppler imaging wave of the mitral annulus from the septal side of the apical four-chamber view was recorded and analysed to determine the early diastolic filling velocity (E′).

Cardiac catheterization

All patients underwent biventricular cardiac catheterization analysis. Primarily, RHC was performed using a 7 F triple-lumen Swan–Ganz thermodilution pulmonary artery catheter (Edwards Lifesciences, Irvine, CA) at rest. After collecting baseline haemodynamic data, a coronary angiography was performed using a 4 F catheter via the trans-radial or brachial approach to avoid ischaemic cardiomyopathy. Mean AP was estimated as follows: diastolic AP + 1/3 (systolic AP − diastolic AP). CO was measured by the thermodilution method and calculated as the average of three to five tests. CPO was calculated as the mean AP × CO/451, and CPI was obtained by dividing the CPO by the body surface area.

Composite cardiac events

Cardiac events were defined as sudden cardiac death, ventricular tachycardia, and heart failure (HF), with ventricular tachycardia being defined as ventricular arrhythmia sustained for $>$30 s and requiring therapeutic intervention. A cardiologist diagnosed HF, defined as hospitalization-required drug therapy such as inotropic agents, vasodilator, surgical treatment, and circulatory assist.

Statistical analysis

All statistical analyses were performed by using the Statistical Package for the Social Sciences 18.0 software (SPSS/IBM Inc., Chicago, IL) and R Version 3.1.2 (R Foundation for Statistical Computing, Vienna, Austria; ISBN 3-900051-07-0, http://www.R-project.org). The data are presented as means ± standard deviations, and non-normally distributed variables are presented as medians with inter-quartile ranges. Intergroup differences were compared using Student's *t*-test for parametric variables and the Mann–Whitney *U* test for non-parametric variables. Categorical variables were compared by using the Pearson χ^2 test or Fisher's exact test, as appropriate. Cumulative survival curves were constructed as time-to-first-event plots via the Kaplan–Meier method. Relative risks and 95% confidence intervals were calculated using univariate and multivariate Cox proportional hazards regression models. Multivariate analyses were performed using a forced-entry model, in which each significant variable included in the univariate analysis considering multicollinearity was entered into the model, as follows: Model 1: B-type natriuretic peptide (BNP), LVEF, PAWP, and CPI; Model 2: BNP, LVEF, PAWP, and systolic AP; and

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Model 3: BNP, LVEF, PAWP, and mean AP. Receiver operating characteristic (ROC) analysis was used to obtain the best prognostic predictor for cardiac index (CI), CPI, and mean AP, and comparisons of areas under the curve (AUC) were performed by statistical software R. A value of $P < 0.05$ was considered to be indicative of statistical significance.

Results

Complete data were available for 176 patients, and the median follow-up time was 4.58 years. The mean age of the study patients was 51.7 ± 12.5 years, and 22.7% were female. The baseline clinical characteristics and pulmonary artery catheterization haemodynamics are presented in *Table 1*.

Table 1 Baseline characteristics of the study patients ($n = 176$)

	Total ($n = 176$)	L-CPI (CPI < 0.52) ($n = 88$)	H-CPI (CPI ≥ 0.52) ($n = 88$)	*P* value
Age (years)	51.7 ± 12.5	53.1 ± 12.7	50.4 ± 12.4	0.144
Female, *n* (%)	40 (22.7)	21 (23.9)	19 (21.6)	0.719
BMI (kg/m^2)	24.5 ± 4.7	23.9 ± 4.2	25.1 ± 5.0	0.083
DM, *n* (%)	33 (18.8)	21 (23.9)	12 (13.6)	0.082
Af, *n* (%)	26 (14.8)	18 (20.5)	8 (9.1)	0.034
ICD at follow-up period, *n* (%)	9 (5.1)	8 (9.1)	1 (1.1)	0.017
CRT at follow-up period, *n* (%)	26 (14.8)	18 (20.5)	8 (9.1)	0.034
Laboratory measurements				
Creatinine (mg/dL)	0.93 ± 0.39	0.97 ± 0.30	0.90 ± 0.47	0.238
Estimated GFR (mL/min/1.73 m^2)	69.7 ± 20.1	65.1 ± 19.1	74.2 ± 20.2	0.003
Haemoglobin (g/dL)	14.2 ± 1.8	14.2 ± 1.7	14.3 ± 1.9	0.898
BNP (pg/mL)	101.0 (53.0–294.1)	192.3 (63.4–398.5)	78.8 (30.2–171.0)	<0.001
Adrenaline (ng/mL)	0.030 (0.020–0.049)	0.032 (0.020–0.051)	0.029 (0.020–0.046)	0.745
Noradrenaline (ng/mL)	0.481 (0.289–0.731)	0.538 (0.371–0.837)	0.404 (0.263–0.600)	0.006
Dopamine (ng/mL)	0.020 (0.013–0.028)	0.018 (0.013–0.026)	0.020 (0.014–0.034)	0.182
Echocardiography				
LVDd (mm)	65.1 ± 7.2	65.9 ± 7.6	64.3 ± 6.8	0.161
LVDs (mm)	55.1 ± 8.3	56.6 ± 8.6	53.6 ± 7.8	0.014
LVMI (g/m^2)	164.0 ± 49.6	161.7 ± 44.4	166.3 ± 54.9	0.550
LVEF (%)	32.1 ± 9.2	30.2 ± 9.5	34.0 ± 8.6	0.060
E/A ratio	1.2 ± 0.8	1.3 ± 0.9	1.1 ± 0.8	0.072
E/e′ ratio	16.2 ± 8.6	16.4 ± 8.8	16.0 ± 8.4	0.810
Dct (ms)	188.2 ± 69.6	190.6 ± 72.5	185.2 ± 66.9	0.654
Electrocardiogram				
QRS (ms)	116.9 ± 24.1	116.4 ± 23.1	117.4 ± 25.3	0.780
Cardiac catheterization				
HR (b.p.m.)	75.9 ± 14.7	74.1 ± 16.3	77.7 ± 13.0	0.109
Systolic AP (mmHg)	120.4 ± 23.7	107.5 ± 16.3	133.3 ± 23.2	<0.001
Diastolic AP (mmHg)	73.9 ± 13.1	67.8 ± 10.9	80.0 ± 12.3	<0.001
Mean AP (mmHg)	89.4 ± 15.1	81.0 ± 11.3	97.8 ± 14.0	<0.001
RAP (mmHg)	5.9 ± 3.5	5.7 ± 3.9	6.1 ± 3.2	0.499
Systolic PAP (mmHg)	30.3 ± 18.2	30.3 ± 13.4	30.2 ± 22.1	0.973
Diastolic PAP (mmHg)	12.8 ± 7.5	13.7 ± 8.9	12.0 ± 5.8	0.140
PAWP (mmHg)	13.2 ± 7.1	13.7 ± 7.8	12.6 ± 6.4	0.294
CI (L/min/m^2)	2.7 ± 0.7	2.3 ± 0.6	3.1 ± 0.5	<0.001
PVR (Wood units)	1.0 (0.6–1.5)	1.2 (0.7–1.9)	1.0 (0.5–1.2)	0.008
SVR (Wood units)	17.6 (15.1–21.3)	18.8 (15.9–22.5)	16.6 (13.3–20.2)	0.001
LVdP/dT_{max} (mmHg/s)	1030.8 ± 246.8	932.0 ± 201.7	1117.4 ± 253.1	<0.001
LVdP/dT_{min} (mmHg/s)	−1067.1 ± 625.1	−977.9 ± 387.9	−1151.4 ± 777.0	0.099
CPO (Wood units)	0.96 ± 0.33	0.71 ± 0.18	1.21 ± 0.25	<0.001
CPI (Wood units/m^2)	0.54 ± 0.17	0.40 ± 0.09	0.67 ± 0.11	<0.001
Medication at follow-up period				
RAS inhibitor, *n* (%)	140 (79.5)	68 (77.3)	72 (81.8)	0.364
Beta-blockers, *n* (%)	160 (90.9)	82 (93.2)	78 (88.6)	0.405
Carvedilol equivalents (mg/day)	10.0 (2.5–12.5)	10.0 (2.5–10.0)	10.0 (2.5–20.0)	0.269
Aldosterone antagonists, *n* (%)	92 (52.3)	54 (61.4)	38 (43.2)	0.019
Diuretics, *n* (%)	97 (55.1)	55 (62.5)	42 (47.7)	0.058
Amiodarone, *n* (%)	24 (13.6)	18 (20.5)	6 (6.8)	0.009

Af, atrial fibrillation; AP, arterial pressure; BMI, body mass index; BNP, B-type natriuretic peptide; CI, cardiac index; CPI, cardiac power index; CPO, cardiac power output; CRT, cardiac resynchronization therapy; Dct, deceleration time; DM, diabetes mellitus; E/A ratio, ratio of early transmitral flow velocity to atrial flow velocity; E/e′ ratio, ratio of early transmitral flow velocity to early diastolic mitral annular velocity; GFR, glomerular filtration rate; HR, heart rate; ICD, implantable cardioverter defibrillator; LVDd, left ventricular end-diastolic diameter; LVDs, left ventricular end-systolic diameter; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; PAP, pulmonary arterial pressure; PAWP, pulmonary arterial wedge pressure; PVR, pulmonary vascular resistance; RAP, right arterial pressure; RAS, renin–angiotensin system; SVR, systemic vascular resistance.
Data are mean ± standard deviation or median (inter-quartile range).

The median CPI of all study patients was 0.525 (*Figure 1*). Study patients were divided into two subgroups according to the median CPI value: a high-CPI group (CPI $\geq$ 0.52; n = 88) and a low-CPI group (CPI $<$ 0.52; n = 88). No significant differences were found in haemoglobin, left ventricular end-diastolic dimension, LVEF, or PAWP between the high-CPI and low-CPI groups. By contrast, BNP, noradrenaline, left ventricular end-systolic diameter, pulmonary vascular resistance, pulmonary vascular resistance, and systemic vascular resistance were significantly higher, and estimated glomerular filtration rate, atrial blood pressure, CI, and $LVdP/dT_{max}$ were lower in the low-CPI group than in the high-CPI group (*Table 1*).

The incidences of cardiac events are summarized in *Table 2*. A total of 43 (24.4%) patients experienced cardiac events, including 4 (2.3%) sudden cardiac deaths, 5 (2.8%) patients with ventricular tachycardia, and 34 (19.3%) hospitalizations because of worsening HF. The cumulative probabilities of event-free survival curves are displayed in *Figures 2* and *3*. Although no significant differences were found between the high-CI and low-CI groups (P = 0.069) (*Figure 2*), Kaplan–Meier survival curves demonstrated a significantly higher probability of cardiac events in the low-CPI group than in the high-CPI group (P = 0.012) (*Figure 3*). Univariate and multivariate Cox regression analyses of factors possibly associated with cardiac events are shown in *Table 3*. In the univariate analysis, BNP, LVEF, systolic AP, mean AP, PAWP, and CPI were significantly associated with cardiac events. In the multivariate analysis under considering multicollinearity, BNP, CPI, systolic AP, and mean AP were independent predictors of cardiac events. ROC curve analysis identified cardiac event cut-off values for CI at 2.42 L/min/m^2 (AUC, 0.548; sensitivity, 46.5%; and specificity, 68.4%), CPI at 0.468 Wood units/m^2 (AUC, 0.627; sensitivity, 55.8%; and specificity, 69.9%), and mean AP at 78 mmHg (AUC, 0.72; sensitivity, 51.2%; and specificity, 84.2%). Mean AP showed a significantly larger AUC than those for CPI and CI (P = 0.019 and P = 0.0048) (*Figure 4*). Kaplan–Meier survival analysis for composite cardiac events using cut-off values is shown in Supporting Information, *Figure S1*.

Table 2 Composite cardiac events

	CPI $<$ 0.52 (n = 88)	CPI $\geq$ 0.52 (n = 88)
SCD, *n* (%)	1 (1.1)	3 (3.4)
VT, *n* (%)	5 (5.7)	0 (0)
HF, *n* (%)	21 (23.9)	13 (14.8)

CPI, cardiac power index; HF, admission due to worsening heart failure; SCD, sudden cardiac death; VT, ventricular tachycardia.

Discussion

This study's major finding was that low CPI was predictive of a high rate of cardiac events in patients with NYHA Functional Class II/III, even after adjustment for BNP, LVEF, and PAWP. Furthermore, this prognostic value was dependent on blood pressure and not CI estimated by cardiac catheterization. These results could support a revision of the therapeutic strategy for high-risk patients: even for those without HF symptoms at rest.

The stratification method of high risk in the mild-to-moderate phase of HF has received increased attention because the number of patients with HF has been rapidly increasing, reaching extreme levels in Asia.[6] Although RHC is a common technique that is very useful in the haemodynamic assessment of patients with HF, some studies[7–9] reported that RHC data were not associated with any benefit but were

Figure 1 A total of 176 consecutive patients with dilated cardiomyopathy who had undergone right ventricular catheterization from March 2000 to January 2020 were enrolled. The median cardiac power index (CPI) was 0.525. CPO, cardiac power output.

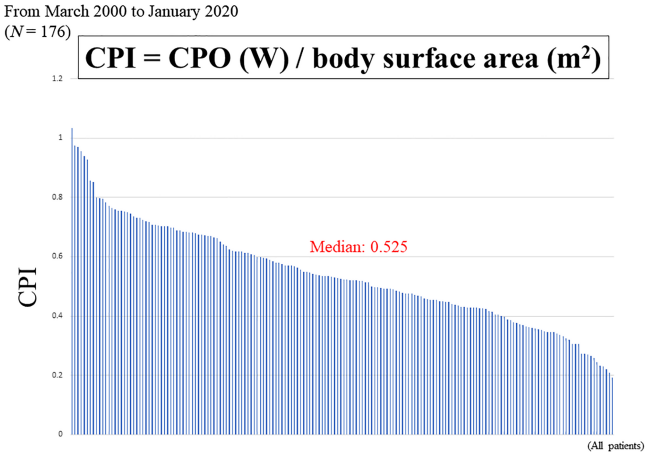


Figure 2 Kaplan–Meier analysis of the probabilities of cardiac events for 176 patients with dilated cardiomyopathy. The patients were divided into two groups according to their median cardiac index (CI). There were no significant differences in cardiac event rates between the low-CI (L-CI) and high-CI (H-CI) groups (P = 0.069; log-rank test).

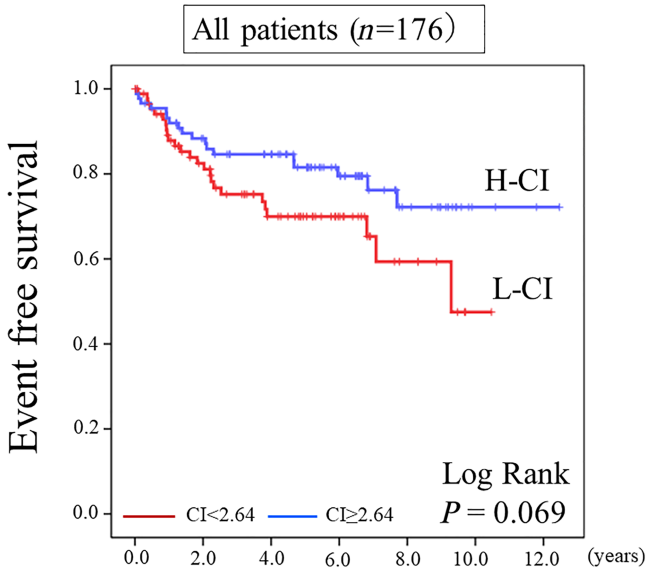
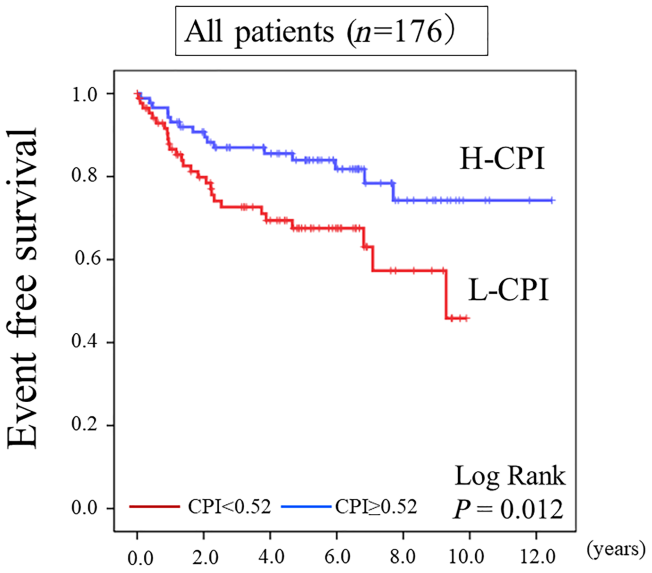


Figure 3 Kaplan–Meier analysis of the probabilities of cardiac events for 176 dilated cardiomyopathy patients. The patients were divided into two groups according to their median cardiac power index (CPI). The probability of cardiac events was significantly higher in the low-CPI (L-CPI) group than in the high-CPI (H-CPI) group (P = 0.012; log-rank test).



associated instead with increased complications. In current clinical practice, haemodynamic estimation and monitoring of parameters such as CO are performed for patients with known HF before major surgical operations and in patients with abnormal myocardial function. Therefore, these strategies are useful for risk stratification and guiding the treatment of these patients. Because CO is affected by contractility, vascular stiffness, systemic volume, and ventricular filling pressures, homeostasis might reduce its prognostic accuracy. Here, we found no significant differences in composite cardiac events between the high-CI and low-CI groups (*Figure 2*). However, our hypothesis that CI reflects

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both volume status and resistance was supported. The CPI is simultaneously calculated from the CO and intravascular pressure and can be used to stratify DCM patients, even when the CI is normal (average, 2.7 L/min/m^2) and systemic vascular resistance elevation is exceedingly small (median, 17.6 Wood units, 1408 dyn·s/cm^5). In 1986, a normal resting CPO was defined as 1 Wood unit; after dobutamine infusion, most patients with a maximum CPO of <1 Wood units died because of intractable cardiac failure.[10] Subsequently, from the SHOCK Trial Registry, Fincke *et al*.[11] reported that the CPO was a novel haemodynamic measurement in cardiogenic shock. A CPO of 0.53 Wood units in patients admitted to a coronary care unit was associated with the highest in-hospital mortality rate.[12] In a 2010 study,[13] CPO was used as a physiological marker of HF severity and as a guide for the management of patients with a left ventricular assist device. Furthermore, Grodin *et al*.[3] reported that the resting CPI was associated with common markers of worsening cardiac dysfunction in patients with advanced HF. Recently, Yildiz *et al*.[4] reported that a lower resting CPO (CPO ≤ 0.54 Wood units) predicted adverse outcomes. However, their study adopted the same cut-off value (i.e. 0.54 Wood units) used in the previous SHOCK Trial Registry, in which 56% of the patients had ischaemic cardiomyopathy, and it was expected that each ischaemic level dynamically affected the cardiac function. Therefore, to evaluate the efficacy of CPI for a homogeneous group in a relatively compensating phase, our study excluded patients with secondary cardiomyopathy, valvular disease, and NYHA Functional Class IV cardiomyopathy. In the enrolled patients without severe symptoms at rest, AP rather than CI was an independent haemodynamic predictor of composite cardiac events.

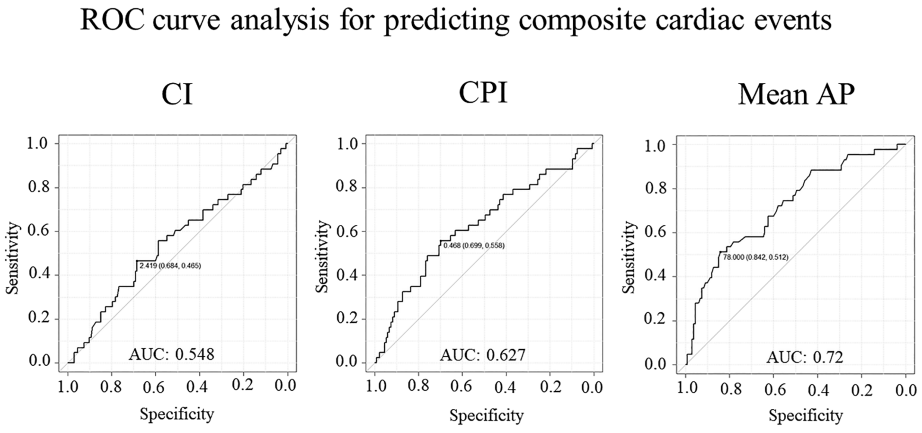
The prognostic importance of CPO assessed by stress echocardiography was reported previously in patients with chronic HF.[14] In 2017, Cortigiani *et al*.[15] reported that the power/mass at the peak of dobutamine echocardiography provided prognostically valuable information. Furthermore,

Table 3 Cox proportional hazard regression analysis for cardiac events

Variable	HR (95% CI)	*P* value
Univariate analysis		
Age (years)	0.980 (0.956–1.005)	0.110
BMI (kg/m^2)	0.960 (0.894–1.031)	0.265
Af	1.251 (0.557–2.813)	0.587
Estimated GFR (mL/min/1.73 m^2)	0.993 (0.977–1.009)	0.401
BNP (pg/mL)[b]	1.014 (1.007–1.021)	<0.001
Noradrenaline (ng/mL)	1.278 (0.602–2.712)	0.523
LVEF (%)	0.961 (0.931–0.992)	0.014
E/e′ ratio	1.010 (0.969–1.053)	0.637
Systolic AP (mmHg)	0.972 (0.957–0.988)	<0.001
Mean AP (mmHg)	0.949 (0.927–0.972)	<0.001
PAWP (mmHg)	1.068 (1.028–1.109)	0.001
CI (L/min/m^2)	0.729 (0.450–1.182)	0.200
PVR (Wood units)	1.095 (0.958–1.251)	0.182
SVR (Wood units)	0.944 (0.881–1.010)	0.095
LVdP/dT$_{max}$ (mmHg/s)	0.999 (0.998–1.001)	0.324
CPI (Wood units/m^2)[c]	0.752 (0.620–0.912)	0.004
Multivariate analysis[a]		
(1) BNP (pg/mL)[b]	1.009 (1.000–1.018)	0.041
LVEF (%)	1.001 (0.964–1.040)	0.945
PAWP (mmHg)	1.037 (0.992–1.084)	0.110
CPI (Wood units/m^2)[c]	0.809 (0.654–0.999)	0.049
(2) BNP (pg/mL)[b]	1.011 (1.002–1.019)	0.016
LVEF (%)	1.001 (0.964–1.040)	0.942
PAWP (mmHg)	1.033 (0.987–1.080)	0.163
Systolic AP (mmHg)	0.979 (0.963–0.996)	0.013
(3) BNP (pg/mL)[b]	1.010 (1.002–1.019)	0.018
LVEF (%)	1.003 (0.967–1.040)	0.889
PAWP (mmHg)	1.036 (0.991–1.083)	0.116
Mean AP (mmHg)	0.953 (0.930–0.977)	<0.001

CI, confidence interval; HR, hazard ratio. Other abbreviations as in *Table 1*.
[a]The final model included all univariate predictors.
[b]Per 10 pg/mL increments.
[c]Per 0.1 Wood units/m^2 increments.

Figure 4 Receiver operating characteristic (ROC) curve analysis for composite cardiac events. ROC curve analysis identified cardiac index (CI), cardiac power index (CPI), and mean arterial pressure (AP) cut-off value for cardiac events of 2.42 L/min/m^2 [area under the curve (AUC), 0.548; sensitivity, 46.5%; and specificity, 68.4%], 0.468 Wood units/m^2 (AUC, 0.627; sensitivity, 55.8%; and specificity, 69.9%), and 78 mmHg (AUC, 0.72; sensitivity, 51.2%; and specificity, 84.2%). The AUC of mean AP was significantly larger than that of CI and CPI (*P* = 0.019 and *P* = 0.0048, respectively).



the exercise stress echocardiography-derived peak CPO to mass may contribute to the evaluation of functional status in patients with HF.[16] Although these estimations by echocardiography are safe, rapid, and non-invasive, the estimation of stroke volume depends on the accurate assessment of the LV outflow tract, so obtaining consistent values between institutions and observers is challenging. The cardiopulmonary exercise test also has been used to calculate peak CO and stratify patients with chronic HF.[17,18] This test also is an effective measurement of functional capacity and prognostic index, but it is an indirect estimation of CO and cannot be obtained in some patients for technical reasons. In the present study, the blood pressure determinate prognostic value of CPI was more useful than CI and PAWP, estimated by catheterization for prognosis prediction in DCM. Thus, patients with low mean AP should be aware of subsequent cardiovascular events. In ROC analysis, a mean AP of 78 mmHg was the best value for predicting cardiac events.

Our study had some limitations. First, this was a single-centre study with a small number of patients and cardiac events. Second, although the thermal dilution method potentially overestimates CO in low CO states,[19] we used the thermal dilution method instead of Fick's equation because it predicts mortality better in clinical practice.[20] Third, although mean AP was a more useful prognostic index than CI in patients with stable DCM, in retrospective studies, it was unclear how therapeutic interventions in blood pressure affect CI and prognosis.

In conclusion, the resting CPI designated by mean AP was a strong predictor of cardiac events and very useful for risk stratification of patients with DCM. Regardless of low CI or high PAWP, patients with low mean blood pressure are at increased risk of subsequent cardiovascular events and may require attention to prepare for therapies like resynchronization therapy, a ventricular assist device, or heart transplant.

Conflict of interest

T.O. received lecture fees from Ono Yakuhin, Medtronics, and Otsuka and received research grants from Ono Yakuhin, Bayer, Daiichi Sankyo, and Amgen Astellas (not in connection with the submitted work). Toyoaki Murohara received lecture fees and unrestricted research grants from Bayer, Daiichi Sankyo, Sumitomo Dainippon, Kowa, MSD, Mitsubishi Tanabe, Boehringer Ingelheim, Novartis, Pfizer, Sanofi-Aventis, Takeda, Astellas, Otsuka, and Teijin.

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Author contributions

T.M., T.A., H.O., Y.K., S.K., T.K., H.H., and K.F. contributed in the conception and design of the study, or acquisition of data, or analysis and interpretation of data; N.S., T.K., and T.O. in drafting the article or revising it critically for important intellectual content; and Toyoaki Murohara in the final approval of the version to be submitted.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. Kaplan–Meier analysis of the probabilities of cardiac events for 176 dilated cardiomyopathy (DCM) patients divided into two groups according to their cutoff value.

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