<Original Article>

Impact of Catheter Ablation on Jugular Venous Pulse Descent Pattern in Atrial Fibrillation: Insights into Heart Failure Prognosis

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ABSTRACT

Background: A dominant Y descent in the jugular venous pulse (JVP) is a marker of less right ventricular (RV) distensibility and poor prognosis in heart failure (HF), and is frequently observed in atrial fibrillation (AF). Whether catheter ablation (CA) can reverse this abnormal waveform remains unknown.
Methods: We conducted a single-center observational study of 166 patients who had undergone CA for AF. JVP waveforms were assessed at baseline, within 48 h after CA, and during follow-up.
Results: Among 65 patients in AF at baseline, 64 (98 %) exhibited a dominant Y descent in JVP. Following CA, 47 (73 %) normalized immediately, with additional patients improving during follow-up. Persistence of dominant Y descent in JVP was associated with longer AF duration and previous open-heart surgery. In 101 patients in sinus rhythm at baseline, no significant waveform changes were observed.
Conclusions: AF-related impairment of RV distensibility is largely reversible with CA. This reversal may represent an important physiological mechanism underlying CA-related improvement in HF outcomes. Further studies are warranted to determine the prognostic value of JVP normalization.

INTRODUCTION

Given its non-invasive accessibility, jugular venous pulse (JVP) waveform analysis has emerged as a fruitful field for clinical investigations into patients with heart failure (HF). The JVP waveform comprises several rises and descents within a single cardiac cycle, with the X nadir typically representing the lowest and most dominant point under normal conditions. An abnormal waveform pattern known as "dominant Y descent," characterized by a disproportionately deep Y descent, reportedly reflects a reduction in right ventricular (RV) distensibility and is also associated with the onset of

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HF and poor prognosis [1–4].

Dominant Y descent is frequently observed in patients with atrial fibrillation (AF), potentially due to the absence of atrial contraction and the presence of irregular ventricular filling. Moreover, since AF often develops in patients with underlying heart disease with compromised biventricular function, structural remodeling of the right ventricle may also contribute to the formation of this abnormal waveform [5–7]. A previous case report documented improvements in dominant Y descent following the restoration of sinus rhythm in patients with AF, suggesting that this waveform abnormality may be reversible in certain cases [8]. However, longitudinal investigations into JVP waveform dynamics remain limited and it has not yet been determined whether the dominant Y descent in AF derives from transient hemodynamic changes induced by arrhythmia or instead reflects a chronic impairment of RV compliance. The clinical determinants of waveform reversibility have also yet to be elucidated.

We hypothesized that while the dominant Y descent in patients with AF could be reversed by restoring sinus rhythm, its persistence would be associated with specific clinical factors. To test this hypothesis, we longitudinally evaluated changes in JVP waveforms among patients who had undergone catheter ablation (CA) for AF. This study aimed to: 1) determine the reversibility of the dominant Y descent after restoration of sinus rhythm; and 2) identify clinical characteristics associated with its persistence. Elucidating these points may provide new insights into how AF impacts HF prognosis from the perspective of RV distensibility.

MATERIALS AND METHODS

Study population

This single-center longitudinal observational study retrospectively identified consecutive inpatients who underwent CA treatment for AF at our hospital between July 2020 and January 2022. Those patients routinely received non-invasive JVP evaluation with simultaneous electrocardiogram recording both before CA and within 2 days after CA (following CA) by a highly trained cardiologist (T.O.). Patients with rhythms other than sinus rhythm or AF (such as atrial flutter, atrial tachycardia, or paced rhythm), those who did not revert to sinus rhythm following CA, and those with failed JVP tracing were excluded. We divided the remaining eligible patients into an AF group and a sinus rhythm (SR) group according to the rhythm identified at first JVP examination. Laboratory data and patient information, including demographics, vital signs, medical history and medication (e.g., heart failure drugs, antiarrhythmic agents, and anticoagulants), were collected prior to admission for CA. The duration of AF was estimated monthly using electronic medical records, and patients were classified into subtypes based on standard clinical definitions. Paroxysmal AF was defined as AF that terminated spontaneously or with intervention within 7 days of onset [9]. HF was determined using the universal definition [10].

All study procedures were performed in accordance with the ethical standards of the Institutional and National Research Committee and the Declaration of Helsinki and its later amendments or comparable ethical standards. The Ethics Review Board of Osaka Medical and Pharmaceutical University approved this retrospective study and waived the requirement for informed consent (approval no. 2024-258).

Assessment of the pattern of JVP descent

A pulse-wave transducer (TY-501A; Fukuda Denshi, Tokyo, Japan) was placed over the internal jugular vein and secured with an adhesive tape cap (OA-256; Fukuda Denshi) in supine position. We recorded tracings at paper speed of 100 mm/s and evaluated the waveforms obtained while the patient held their exhalation to minimize respiration-related fluctuation. To avoid influences on waveform amplitude due to the Valsalva maneuver, the patient performed a gentle exhalation hold. For AF rhythm, we selected steady heartbeats and assessed the typical waveform during a cardiac cycle with a sufficiently long R-R' interval (> 0.8 s).

Two highly trained cardiologists (T.O. and K.S.) who were blinded to clinical data judged whether the JVP had a dominant Y descent where the nadir of Y descent was deeper than that of X descent. We defined ‘dominant Y’ as a dominant Y descent existence in the JVP waveform, and ‘dominant X’ as otherwise. Interobserver variation of the judgement was 2.9 %. As examples, **Figure 1A** depicts the dominant X state, while **Figure 1B** shows the dominant Y state.

CA procedure

CA was performed under intravenous dexmedetomidine sedation with non-invasive positive pressure ventilation. After coronary sinus catheter placement and a single transseptal puncture, circumferential pulmonary vein isolation was performed using a 3-dimensional mapping system (CARTO®; Biosense Webster, CA, USA) and an irrigated-tip ablation catheter. Additional CA (cavo-tricuspid isthmus, superior vena cava, box isolation, mitral isthmus, or atrial tachycardia) were performed at the discretion of the operator.

Echocardiography

All echocardiographic examinations were performed before CA by expert sonographers according to American Society of Echocardiography guidelines [11] and were interpreted by experienced cardiologists. Commercial ultrasound systems (Vivid E9; GE healthcare, Horten, Norway, EPIQ 7G; Philips Medical System, Andover, MA, USA; or Artida;

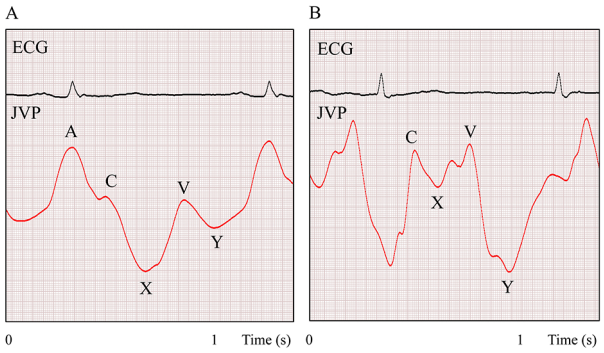

Figure 1 **Assessment of the descent pattern in JVP**
A) Control JVP tracing recorded from a healthy 60-year-old man. This waveform is classified as dominant X, as the lowest point corresponds to the nadir of the X descent.
B) Abnormal JVP tracing obtained from a 70-year-old woman with AF before CA. No A wave is observed due to atrial standstill in AF. The waveform is classified as dominant Y, with the lowest point corresponding to the Y nadir.
JVP, jugular venous pulse; AF, atrial fibrillation; CA, catheter ablation.

Canon, Tokyo, Japan) were used. Left atrial volume index (LAVI) was calculated by the biplane area–length method, and left ventricular ejection fraction (LVEF) by the modified Simpson's method. Doppler imaging was used to obtain E/A ratio, average e′ velocities (septal and lateral), and E/e′ ratio. The tricuspid regurgitation peak pressure gradient (TRPG) was derived using the modified Bernoulli equation, and tricuspid regurgitation severity was graded by a multiparametric approach. Right atrial pressure (RAP) was estimated from inferior vena cava diameter and collapsibility as per guideline algorithms.

Statistical analysis

We described the characteristics of patients in the AF and SR groups. In the AF group, patients were classified according to changes in JVP descent pattern via CA, and clinical characteristics were compared. Categorical variables are presented as numbers and percentages and were compared using the chi-square test or Fisher's exact test. Continuous variables are expressed as mean ± standard deviation or median with interquartile range (IQR). Normally distributed variables were compared between groups using Student's t-test, while non-normally distributed variables were compared using the Mann–Whitney U test. Statistical significance was defined at the level of $p < 0.05$. All statistical analyses were conducted using JMP Pro software (version 18.0; SAS Institute, Cary, NC, USA).

RESULTS

Baseline characteristics of the study population

Among 239 consecutive participants, we excluded 73 patients, including 30 with neither AF nor sinus rhythm at initial JVP examination, 6 who exhibited AF following CA, and 37 who displayed failure of JVP tracing (fatty neck, n = 4; tachycardia, n = 19; technical failure, n = 14). Ultimately, 166 patients were included in the final analysis, comprising 65 in the AF group and 101 in the SR group (**Figure 2**). **Table 1** summarizes the characteristics before CA of patients in the AF and SR groups. Median age of the entire cohort was 71 years, with men comprising 61 % (101 men, 65 women). Hypertension was present in 63 % (104/166), diabetes mellitus in 14 % (24/166), ischemic heart disease in 13 % (22/166), HF in 33 % (54/166), and prior open-heart surgery in 5 % (9/166), showing no significant differences between AF and SR groups. Similarly, 67 % (112/166) of patients received antiarrhythmic pharmacological therapy (Class I antiarrhythmic drugs, amiodarone or β-Blockers), 47 % (78/166) received renin–angiotensin system inhibitors (angiotensin-converting enzyme inhibitors,

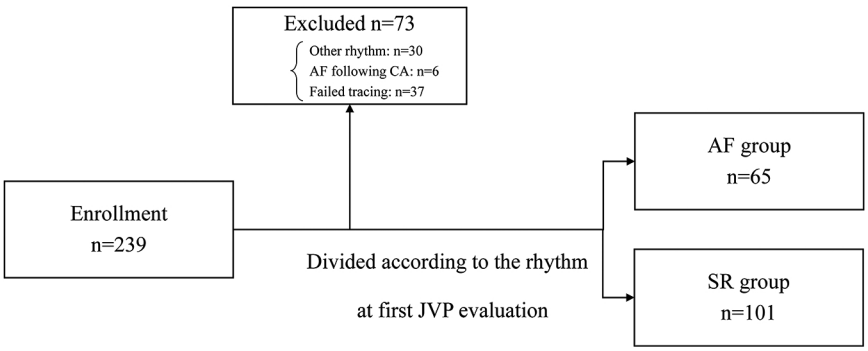

Figure 2 **Flowchart of patient enrollment in the present study**
This diagram outlines the inclusion and exclusion process and final group assignment based on cardiac rhythm at initial JVP assessment. Abbreviations are as in **Figure 1**.

Table 1 Patient characteristics in the AF and SR groups at baseline

Variables	AF group (n = 65)		SR group (n = 101)		*P*-value
Age, years	70	(63–77)	71	(63–77)	0.67
Male, %	39	(60)	62	(61)	0.86
BMI, kg/m^2	24	(21–26)	23	(21–26)	0.49
Systolic BP, mmHg	121	(± 18)	125	(± 20)	0.19
Heart rate, beats/min	81	(70–93)	70	(62–83)	0.0004
AF duration, months	2	(0–5)	0	(0–0)	< 0.0001
Paroxysmal AF, %	19	(29)	85	(84)	< 0.0001
Hypertension, %	45	(69)	59	(58)	0.16
Diabetes mellitus, %	13	(20)	11	(11)	0.10
Ischemic heart disease, %	8	(12)	14	(14)	0.77
Previous open-heart surgery, %	4	(6)	5	(5)	0.74
HF, %	24	(37)	30	(30)	0.33
Class I antiarrhythmic drugs, %	24	(37)	35	(35)	0.76
Amiodarone, %	7	(11)	10	(10)	0.86
β-Blockers, %	33	(51)	51	(51)	0.97
ACE-I/ARBs, %	26	(40)	39	(60)	0.86
MRAs, %	12	(18)	12	(12)	0.24
Loop diuretics, %	16	(25)	15	(15)	0.11
DOACs, %	51	(78)	79	(78)	0.97
Warfarin, %	5	(8)	5	(5)	0.47
eGFR, mL/min/1.73 m^2	59	(± 16)	64	(± 18)	0.06
Hemoglobin, g/dL	13.8	(± 1.9)	13.3	(± 1.8)	0.06
BNP, pg/mL	113	(59–262)	48	(22–126)	0.0002
LAVI, mL/m^2	49	(± 15)	36	(± 18)	0.001
LVEF, %	60	(51–64)	64	(57–70)	0.006
E wave of mitral inflow, cm/s	80	(67–97)	69	(56–83)	0.0002
Average mitral E/e' ratio	9	(7–13)	9	(7–12)	0.82
TRPG, mmHg	21	(17–22)	21	(16–25)	0.33
Estimated RAP, mmHg	3	(3–8)	3	(3–3)	< 0.001
Moderate or severe TR, %	4	(6)	1	(1)	0.06
Dominant Y in JVP, %	64	(98)	6	(6)	< 0.0001

BMI, body mass index; BP, blood pressure; AF, atrial fibrillation; HF, heart failure; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; MRA, mineralocorticoid receptor antagonist; DOAC, direct oral anticoagulant; eGFR, estimated glomerular filtration rate; BNP, B-type natriuretic peptide; LAVI, left atrial volume index; LVEF, left ventricular ejection fraction; TRPG, tricuspid regurgitant pressure gradient; RAP, right atrial pressure; TR, tricuspid regurgitation; JVP, jugular venous pulse.
Class I antiarrhythmic drugs, class I antiarrhythmic drugs according to Vaughan Williams classification.

angiotensin II receptor blockers or mineralocorticoid receptor antagonists), 19 % (31/166) received loop diuretics, and 84 % (140/166) received anticoagulant therapy (direct oral anticoagulants or warfarin), with no significant difference between the two groups. The AF group showed significantly higher heart rate (81 [70–93] vs. 70 [62–83] beats/min, p = 0.0004) and B-type natriuretic peptide levels (113 [59–262] vs. 48 [22–126] pg/mL, p = 0.0002), and longer AF duration (2 [0–5] vs. 0 [0–0] months, $p < 0.0001$). In the AF group, 29 % of patients (19/65) had paroxysmal AF. In contrast, paroxysmal AF was present in 84 % of patients (85/101) in the SR group, while the remaining 16 % (16/101) had a history of AF lasting ≥ 7 days but had undergone successful cardioversion and were in sinus rhythm at the time of initial JVP examination. Echocardiographic findings revealed impaired left ventricular compliance and evidence of left atrial remodeling in the AF group, as indicated by significantly higher E wave velocities (80 [67–97] vs. 69 [56–83] cm/s, p = 0.0002) and larger LAVI (49 ± 15 vs. 36 ± 18 mm, p = 0.001), compared to the SR group. However, no clear evidence of elevated left atrial pressure was seen, given that both TRPG (21 [17–22] vs. 21 [16–25] mmHg, p = 0.33) and average E/e′ (9 [7–13] vs. 9 [7–12], p = 0.82) were comparable between groups. While left ventricular systolic function remained within the normal range in both groups, the AF group showed significantly lower LVEF (60 % [51–64 %] vs. 64 % [57–70 %], p = 0.006). With regard to right heart parameters, the AF group had a significantly higher estimated RAP (3 [3–8] vs. 3 [3–3] mmHg, $p < 0.001$), and tended toward a higher prevalence of moderate or severe TR (4/65 [6 %] vs. 1/101 [1 %], p = 0.06) relative to the SR group. A striking 98 % (64/65) of the AF group exhibited dominant Y in JVP before CA, compared with only 5 % (5/101) in the SR group. Among these five patients with dominant Y in the SR group, three had previously undergone open-heart surgery and one displayed severe tricuspid regurgitation.

Changes in JVP descent pattern after CA and additional follow-up

In the AF group, the only 1 patient with dominant X before CA showed no change in the descent pattern following CA. Of the 64 patients with dominant Y before CA, 73 % (47/64) transitioned to dominant X following CA, whereas 27 % (17/64) continued to show dominant Y. Conversely, patients in the SR group displayed no significant change in JVP descent pattern from before to after CA. In addition, we re-evaluated the JVP at follow-up in patients with residual dominant Y descent in the AF group after CA. These 17 patients were followed for a median of 7 months (IQR, 3–12 months). Three patients were lost to follow-up and one experienced recurrent AF. Among the remaining 13 patients who maintained sinus rhythm, nine showed a shift from dominant Y to dominant X (late modified group), whereas four continued to exhibit dominant Y (unmodified group). The 47 patients who demonstrated waveform improvement immediately after CA, as previously described, were designated as the ‘immediately modified group’, and the late and immediately modified groups were collectively termed the ‘modified group’. **Figure 3** illustrates patient classifications based on post-CA changes in the JVP waveform and corresponding alterations in the descent pattern.

Clinical characteristics associated with reversibility of the JVP descent pattern

Table 2 summarizes patient characteristics between the immediately modified group (n = 47) and the late modified group (n = 9) at baseline (prior to CA, corresponding to the same time point as **Table 1**). The two groups were comparable in terms of age, sex, body mass index, blood pressure, heart rate, AF duration, and proportion of paroxysmal AF. Likewise, no significant differences were seen between the two groups in the prevalence of comorbidities (including hypertension, diabetes mellitus, and ischemic heart disease), in the use of medications other than loop diuretics, or in the laboratory parameters (including estimated glomerular filtration rate, hemoglobin, and B-type natriuretic peptide levels). Of note, the late modified group had a significantly higher prevalence of HF (56 % vs. 27 %, p = 0.03), higher proportion of loop diuretic use (56 % vs. 18 %, p = 0.01), and elevated estimated RAP (3 [3–15] vs. 3 [3–3] mmHg, p = 0.03). Despite the higher E wave velocity observed in the late modified group, other parameters of elevated left ventricular filling pressure remained comparable between groups, with estimated RAP as the only parameter showing a significant difference. Further, to identify predictors associated with persistent dominant Y at follow-up, we compared baseline characteristics between the modified group (comprising patients with either immediately or late modified group, n = 56) and unmodified group (n = 4) as summarized in **Table 3**. The unmodified group was characterized by a significantly lower heart rate (65 ± 11 vs. 83 ± 17 beats/min, p = 0.03), indicating a relative bradycardic tendency, and a longer duration of AF (7 [5–8] vs. 2 [0–4] months, p = 0.02). Notably, all patients in the unmodified group (4/4, 100 %) had a history of open-heart surgery, compared to none in the modified group (0/56, 0 %; $p < 0.0001$).

DISCUSSION

This first longitudinal study reveals that impaired RV distensibility in AF, identified by a dominant Y descent on JVP, is often reversible with successful CA. Prior to CA, a dominant Y descent was nearly universal in the AF group, suggesting poor RV compliance. Following CA, this abnormal waveform resolved in most AF patients,

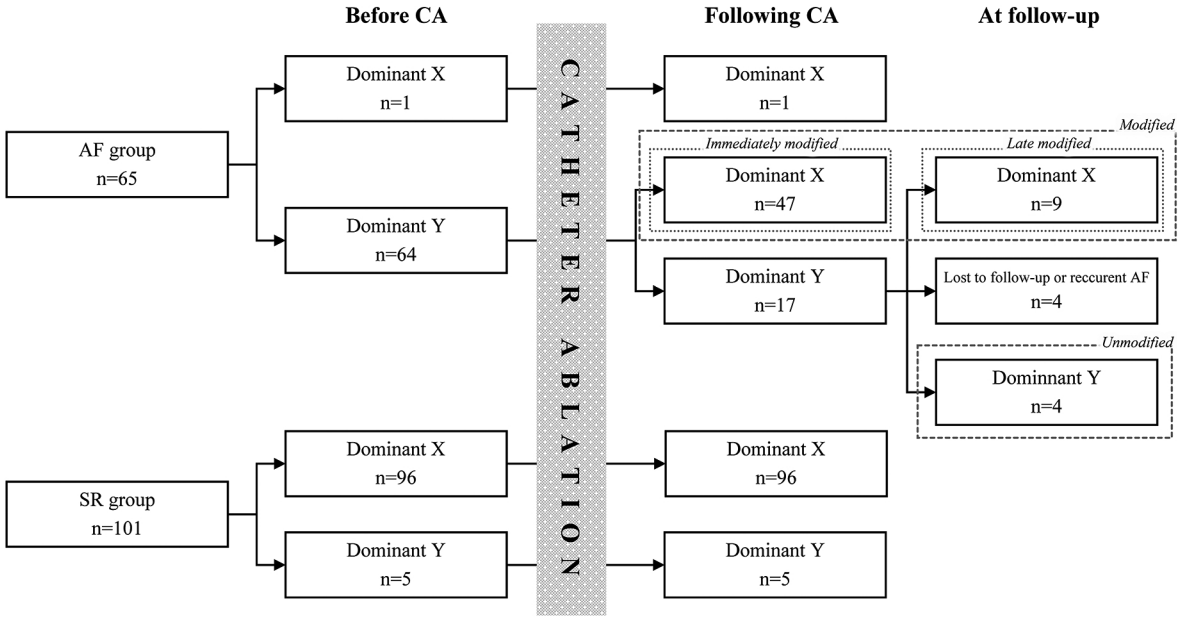

Figure 3 **Longitudinal changes in JVP descent pattern in the AF and SR groups**
This figure illustrates the main findings of the study. The upper panel shows changes in descent pattern among AF patients. Each subgroup (immediately modified, late modified, modified, and unmodified) is enclosed by dashed boxes. The lower panel demonstrates that SR group patients exhibited no significant change in JVP waveform throughout the study period. Abbreviations are as in **Figure 1**.

either immediately or during follow-up. In contrast to this dynamic change observed in the AF group, patients in the SR group exhibited no significant change in JVP descent pattern following CA (**Figure 3**). These findings suggest that AF itself drives this reversible RV dysfunction. Furthermore, baseline clinical characteristics differed among those who showed immediate, delayed, or no improvement. These distinct recovery patterns offer novel physiological insights into the mechanisms by which CA improves HF outcomes.

The right ventricle is defined by intrinsic high compliance, a characteristic essential for buffering preload changes with minimal pressure elevation [12]. Loss of this compliance, often signaled by a dominant Y descent on JVP, signifies impaired RV diastolic function, which has been linked to HF development and poor prognosis [1–5, 13–16]. While recent studies have suggested a direct link between AF and impaired RV diastolic function [17–19], the prevalence and nature of this relationship, particularly in the elderly, remained unclear. In our elderly cohort (mean age > 70 years), a dominant Y descent was nearly universal (98 %), a rate substantially higher than that reported in younger populations [20], suggesting that age-related RV stiffening [1, 4], when compounded by AF, critically impairs ventricular distensibility. Our central finding, however, was that this dysfunction appears remarkably reversible. While previously described in only a single case report [7], this is the first longitudinal study to systematically document that CA can restore normal JVP waveforms, even in elderly patients.

The pattern of recovery offered profound insights into the underlying pathophysiology. Patients with delayed improvement (beyond 48 h) typically had a history of HF with concomitant loop diuretic use and elevated right atrial pressure. This suggests that resolving significant residual right-sided congestion required more time after sinus rhythm restoration, consistent with reports that increased right atrial pressure can itself augment the Y descent and reflect poor right heart compliance [5]. In stark contrast, failure to improve was uniformly associated with a history of open-heart surgery and a significantly longer AF duration. This finding points compellingly toward irreversible structural remodeling, such as pericardial adhesions, decreased RV contractility [16, 21, 22], and fibrotic changes in the right heart chambers [19, 23–26], as the limiting factor for recovery. Thus, while AF-induced RV dysfunction is reversible, the potential for improvement is ultimately dictated by the degree of pre-existing, permanent structural change.

Clinical implications

These findings offer a compelling physiological explanation for the benefits of rhythm control demonstrated in major trials like CASTLE-AF [27] and EAST-AFNET4 [28]. We propose that the reversal of RV diastolic dysfunction which can otherwise impair left-sided filling and reduce cardiac output [29] is a key mechanism through which CA improves HF outcomes. This framework also helps explain the vexing clinical problem of why rhythm control yields

Table 2 Patient characteristics in the immediately and late modified groups

Variables	Immediately modified group n = 47		Late modified group n = 9		*P*-value
Age, years	68	(± 9)	73	(± 5)	0.14
Male, %	30	(64)	4	(44)	0.27
BMI, kg/m^2	24	(± 4)	23	(± 6)	0.68
Systolic BP, mmHg	123	(± 19)	120	(± 22)	0.72
Heart rate, beats/min	82	(± 16)	93	(± 19)	0.06
AF duration, months	2	(0–3)	2	(0–7)	0.81
Paroxysmal AF, %	17		2		0.40
Hypertension, %	35	(75)	5	(56)	0.25
Diabetes mellitus, %	10	(21)	2	(22)	0.95
Ischemic heart disease, %	4	(9)	2	(22)	0.22
Previous open-heart surgery, %	0	(0)	0	(0)	N/A
HF, %	13	(27)	5	(56)	0.03
Class I antiarrhythmic drugs, %	16	(34)	4	(44)	0.55
Amiodarone, %	4	(8)	1	(11)	0.80
β-Blockers, %	23	(49)	6	(67)	0.33
ACE-I/ARBs, %	19	(40)	3	(33)	0.69
MRAs, %	8	(17)	2	(22)	0.71
Loop diuretics, %	8	(17)	5	(56)	0.01
DOACs, %	37	(79)	8	(89)	0.48
Warfarin, %	2	(4)	1	(11)	0.40
eGFR, mL/min/1.73 m^2	60	(± 14)	51	(± 20)	0.20
Hemoglobin, g/dL	13.8	(± 1.9)	14.0	(± 2.7)	0.42
BNP, pg/mL	126	(57–232)	149	(79–671)	0.36
LAVI, mL/m^2	48	(± 13)	55	(± 21)	0.30
LVEF, %	60	(51–65)	62	(51–64)	0.59
E wave of mitral inflow, cm/s	78	(62–93)	97	(82–104)	0.03
Average mitral E/e' ratio	9	(7–12)	13	(7–17)	0.08
TRPG, mmHg	20	(17–27)	25	(20–34)	0.11
Estimated RAP mmHg	3	(3–3)	3	(3–15)	0.03
Moderate or severe TR, %	3	(6)	0	(0)	0.44

Abbreviations are as in **Table 1**.

inconsistent results in patients with established structural heart disease, such as HF with preserved ejection fraction, where the degree of myocardial fibrosis varies widely [30, 31]. The dominant Y descent may therefore serve as a simple, non-invasive marker to identify patients for whom hemodynamics are still salvageable by rhythm control therapy.

While our study highlights a novel mechanism, a critical next step is to validate this pathological framework. Future prospective studies must correlate the normalization of the

Table 3 Patient characteristics of patients in the modified and unmodified groups

Variables	Modified group (immediately + late) n = 56		Unmodified group n = 4		*P*-value
Age, years	69	(± 8)	69	(± 10)	0.87
Male, %	34	(61)	2	(50)	0.68
BMI, kg/m^2	24	(± 4)	25	(± 4)	0.83
Systolic BP, mmHg	122	(± 19)	109	(± 13)	0.17
Heart rate, beats/min	83	(± 17)	65	(± 11)	0.03
AF duration, months	2	(0–4)	7	(5–8)	0.02
Paroxysmal AF, %	19	(34)	0	(0)	0.16
Hypertension, %	40	(71)	2	(50)	0.36
Diabetes mellitus, %	12	(21)	1	(25)	0.87
Ischemic heart disease, %	6	(10)	0	(0)	0.49
Previous open-heart surgery, %	0	(0)	4	(100)	< 0.0001
HF, %	20	(36)	2	(50)	0.57
Class I antiarrhythmic drugs, %	20	(36)	2	(50)	0.57
Amiodarone, %	5	(9)	1	(25)	0.30
β-Blockers, %	29	(52)	2	(50)	0.95
ACE-I/ARBs, %	22	(39)	1	(25)	0.57
MRAs, %	10	(18)	2	(50)	0.18
Loop diuretics, %	13	(23)	2	(50)	0.23
DOACs, %	45	(80)	3	(75)	0.80
Warfarin, %	3	(5)	0	(0)	0.63
eGFR, mL/min/1.73 m^2	59	(± 15)	70	(± 21)	0.18
Hemoglobin, g/dL	13.8	(± 1.9)	13.7	(± 1.7)	0.83
BNP, pg/mL	126	(64–312)	93	(74–112)	0.54
LAVI, mL/m^2	49	(± 15)	38	(± 10)	0.47
LVEF, %	60	(51–65)	57	(53–61)	0.96
E wave of mitral inflow, cm/s	79	(64–97)	110	(72–123)	0.17
Average mitral E/e' ratio	9	(7–12)	11	(9–16)	0.33
TRPG, mmHg	21	(17–27)	20	(16–26)	0.75
Estimated RAP, mmHg	3	(3–3)	3	(3–8)	0.61
Moderate or severe TR, %	3	(5)	1	(25)	0.13

Abbreviations are as in **Table 1**.

JVP waveform post-CA with long-term clinical outcomes, such as HF rehospitalization and mortality. Establishing whether this simple physical finding is a true surrogate marker for improved prognosis will be essential to translate our findings into clinical practice and refine patient selection for rhythm control therapy.

Limitations

This study showed several limitations. The single-center, retrospective design and small size limited the statistical

power and generalizability. The brief follow-up period and lack of systematic right heart echocardiographic data (e.g., right atrial strain) precluded assessment of long-term JVP changes and their direct structural correlates. Furthermore, selection bias is a key consideration, as our cohort mainly comprised patients with less advanced HF. This cohort may not represent patients with advanced HF, but accurately reflects the real-world population for whom CA is indicated. Consequently, our findings require validation in larger, prospective, multicenter studies incorporating comprehensive and serial echocardiographic assessments.

Conclusion

A dominant Y descent, as a marker of impaired RV distensibility, was present in the vast majority of patients with AF. CA promptly reversed this prognostically unfavorable waveform, except in cases marked by advanced structural remodeling. This rapid restoration of RV diastolic function highlights a key physiological mechanism for the clinical benefits of rhythm control, underscoring the profound and immediate hemodynamic impact of this procedure.

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DISCLOSURE STATEMENTS

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