

Four years follow-up of epicardial left ventricular pacing by mini-thoracotomy for cardiac resynchronisation therapy in congestive heart failure (four cases)

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Abstract

Background and aim: To establish whether left ventricular (LV) pacing by mini-thoracotomy is a safe and feasible procedure after failed transvenous cardiac resynchronisation therapy (CRT), we described four cases of patients who demonstrated congestive heart failure (CHF), had transvenous LV lead implantation failures, and underwent a mini-left-lateral thoracotomy and implantation of an epicardial LV lead.

Methods: After a mean follow-up 45 ± 3.5 months, the haemodynamic benefits of CRT were apparent in four patients.

Results: Mean LV ejection fraction increased from $28.4 \pm 6.5\%$ to $44.5 \pm 13.7\%$ ($p = 0.024$), in association with a reduction of LV end-systolic diameters from 62.3 ± 10.3 mm to 53.0 ± 13.11 mm ($p = 0.029$). QRS width decreased from 162.5 ± 23.6 ms to 147.5 ± 18.9 ms ($p = 0.014$). New York Heart Association values significantly improved before and after the procedure.

Conclusions: These results suggest that epicardial LV pacing by mini-thoracotomy for CRT in CHF is feasible and can bring satisfactory long-term results.

Key words: thoracotomy, epicardial left ventricular lead, cardiac resynchronisation therapy, heart failure

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INTRODUCTION

Cardiac resynchronisation therapy (CRT) is indicated in patients with New York Heart Association (NYHA) functional class III or IV, heart failure (HF), in sinus rhythm, with a QRS width of ≥ 120 ms, left bundle branch block (LBBB) QRS morphology, and an ejection fraction (EF) $\leq 35\%$ [1]. For CRT, a left ventricular (LV) pacing electrode is usually placed by catheterisation of the coronary sinus (CS), and is associated with prolonged procedure times and extensive fluoroscopy with reported implantation failure in 5–10% of cases [2–4]. Perhaps the most important limitation of CS catheterisation is that only a limited number of sites can be reached on the LV wall because of the anatomy of the cardiac venous system.

Direct surgical epicardial LV lead placement overcomes these limitations and provides the potential to pace the optimal target site [5, 6]. In this study, we described four cases of patients demonstrating depressed systolic LV function and

congestive HF who had transvenous LV lead implantation failures and underwent a mini-left-lateral thoracotomy and implantation of an epicardial LV lead.

METHODS

Cases introduction

Case 1. A 60-year-old man was diagnosed with dilated cardiomyopathy and HF in 2008 after chest discomfort and shortness of breath during activity for two years. His clinical characteristics are shown in Table 1. In November 2008 he underwent CRT-D implantation. Coronary vein sinus angiography showed that the beginning part of cardiac lateral vein bent significantly, and no other branches could be chosen (Fig. 1). After several operations, wires still could not enter the lateral vein, so transvenous LV lead implantation was abandoned. After implanted right atrial and right ventricular leads using standard transvenous lead models in a cardiac catheterisa-

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Table 1. Baseline clinical characteristics and pharmacological treatment

	Case 1	Case 2	Case 3	Case 4
Sex	Male	Male	Male	Male
Age [years]	60	58	57	67
LVEDD [mm]	75	70	85	57
LVESD [mm]	68	58	73	50
Mitral regurgitation	Severe	Mild	Moderate	Moderate
Ejection fraction [%]	20	34.8	26.9	32
NYHA	III	III	IV	IV
ECG	SR, CLBBB	SB, CLBBB	SR, CLBBB	AF, VVI pacing
Ventricular tachycardia	No	No	Yes	Yes
QRS width [ms]	180	180	130	160*
Medications	ARB, BB, digitalis, spironolactone	ACEI, BB	ARB, BB, aspirin, clopidogrel, digitalis, spironolactone	ACEI, BB, spironolactone
Cause of CHF	Non-ischaeamic	Non-ischaeamic	Ischaemic	Non-ischaeamic
Indications for surgical intervention	CS anatomy	CS anatomy	CS anatomy Lead dislodgment	CS anatomy High pacing threshold

*Right ventricular VVI pacing ECG; LVEDD — left ventricular end-diastolic diameter; LVESD — left ventricular end-systolic diameter; NYHA — New York Heart Association functional class; SR — sinus rhythm; SB — sinus bradycardia; AF — atrial fibrillation; CLBBB — complete left bundle branch block; ACEI — angiotensin-converting enzyme inhibitor; BB — beta-blocker; ARB — angiotensin-receptor blockers; CS — coronary sinus; CHF — congestive heart failure

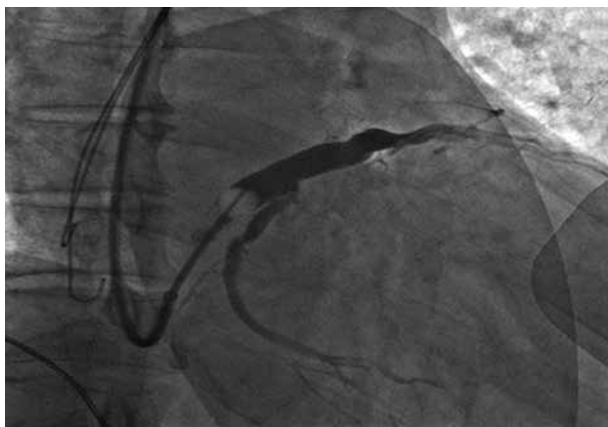


Figure 1. Coronary vein sinus angiography showed that the beginning part of the cardiac lateral vein bent significantly, and no other branches could be chosen

tion lab, the patient was transferred to the operation room to undergo a mini-left-lateral thoracotomy and implantation of an epicardial LV lead.

Case 2. A 58-year-old man was admitted to hospital in 2008 for complaint of shortness of breath during activity for ten years and bradycardia for four months. His diagnosis was dilated cardiomyopathy, sick sinus syndrome, and left heart dysfunction. The baseline clinical characteristics are shown in Table 1. He underwent CRT-D implantation in December 2008. Coronary vein sinus angiography showed

CS vein twist, accompanied by the initial segment myocardial bridge, resulting in the fact that the LV lead could not reach a satisfactory position. So transvenous LV lead implantation failed. The patient was transferred to the operation room to undergo a mini-left-lateral thoracotomy and implantation of an epicardial LV lead.

Case 3. A 57-year-old man had a history of hypertension for ten years and diabetes mellitus for three years. He was admitted to hospital in 2009 for complaint of dyspnoea during activity for ten months, worsening for seven hours, and unconsciousness for six hours. The patient had sudden unconsciousness when he arrived at the hospital. Electrocardiogram (ECG) monitor showed ventricular tachycardia and cardiac arrest. After urgent C-reactive protein, the patient regained consciousness. ECG showed sinus rhythm, LV hypertrophy, ST segment and T wave changes, complete LBBB, and ventricular premature beats. Echocardiography showed LV wall thickening, LV systolic activity weakened, below the level of papillary muscle, and the LV posterior inferior wall contraction activity weakened to zero (the other parameters shown in Table 1). Coronary angiography showed severe stenosis of three vessels, chronic occlusion in the middle of the right coronary artery lateral branch, and two stents implanted. The patient was diagnosed with coronary atherosclerotic heart disease, cardiogenic syncope and HF. So the patient had the indication of CRT-D implantation. In May 2009, he underwent CRT-D implantation. But pacing thresholds were unacceptably high ($> 3 \text{ V}/0.5 \text{ ms}$) when the LV lead was placed in the cardiac

lateral vein. When the LV lead was adjusted to the branch of the cardiac posterior vein, obvious diaphragmatic stimulation appeared with pacing. When we changed LV lead to the distal part of the posterior vein, the electrical parameters were good with no diaphragmatic stimulation; however, the lead was easily dislocated. As a result, transvenous LV lead implantation failed. The patient underwent a mini-left-lateral thoracotomy and implantation of an epicardial LV lead after 25 days because of severe cardiac dysfunction with ischaemic cardiomyopathy.

Case 4. A 67-year-old man was admitted to hospital in 2009 for complaint of shortness of breath during activity for two years and worsening for one day. He had a history of atrial fibrillation (AF) and third-degree atrioventricular block, and he had been implanted with a single-chamber pacemaker (VVI) 20 years previously. Echocardiography showed LV systolic activity weakened and right heart enlargement (the other parameters shown in Table 1). His diagnosis was AF and third-degree atrioventricular block, HF (NYHA IV), and pacemaker implantation state. He had indications for implantation of a CRT-D (recommendation IIa, level of evidence A). In July 2009 he underwent CRT-D implantation, but the LV lead could not enter the cardiac lateral-posterior vein. Therefore, we chose the middle cardiac vein through multi-site testing, with pacing thresholds > 4.5 V/0.4 ms, and with visible diaphragmatic stimulation. Because no other vascular could be chosen, we had to give up transvenous LV lead implantation and suggested a mini-left-lateral thoracotomy and implantation of an epicardial LV lead.

Informed consent was obtained from all four patients.

Operative course and follow-up

Transvenous approach via CS and its tributaries. The percutaneous procedures were performed using local anaesthesia and antibiotic prophylaxis. The procedures were carried out under fluoroscopic control. Right atrial and right ventricular pacing were established using standard transvenous lead models with insertion techniques through the left axillary vein or subclavian vein. No atrial lead was implanted in one chronic AF patient. The LV lead was positioned as far as possible within the venous system, preferably into a lateral or postero-lateral venous tributary to obtain the longest possible interventricular conduction time. In case of implant failure, the LV lead connector was temporarily capped in order to allow future LV epicardial lead connection. All device pockets were located in the left infraclavicular area.

Limited thoracotomy. In the operation room, under general anaesthesia, and with single, right-lung ventilation using a double-lumen endotracheal tube, the patient was placed in the supine position with left chest elevated 30–40°. A left lateral, mid-axillary mini-thoracotomy (8 cm) was performed at the side of the fourth intercostal space, and the pericardium was opened anterior to the phrenic nerve. This procedure

ensured sufficient access to expose the atrioventricular groove and LV lateral posterior wall. A unipolar, epicardial, steroid lead (Capsure-Epi Models 4965, Medtronic Inc., Minneapolis, MN, USA) was attached to the target area (between the left circumflex artery and obtuse marginal branch, below the left atrial appendage). The lead was sutured in the selected position when electrical parameters were within the following ranges: impedance $> 200 \Omega$ and $< 2000 \Omega$, sensing (peak-to-peak amplitude of R-wave) greater than 5 mV, and pacing threshold measured at 0.4 ms less than 2.0 V. Once the electrode was sewn in the correct position, 10-V stimulation was delivered in order to assess the presence of phrenic nerve stimulation. The connector was brought through the third intercostal space and tunnelled subcutaneously to the CRT-D device, in the pocket previously created in the left pectoral area. The pericardium was partially closed. A small pleural drain (19 French Blake drain, Ethicon) was inserted followed by standard wound closure. Successful deployment of the epicardial electrode was accomplished in all of the four patients. Epicardial procedure time (skin-to-skin) was 51 ± 28 min. No surgical complications occurred. Optimal lateral position, close to the obtuse marginal branch of the circumflex coronary artery, was achieved for all patients.

Patient follow-up. Clinical status was evaluated before implantation and after three, six, nine, 12, 24, 36, and 48 months. Each visit included pacemaker parameters, ECG recording, NYHA classification, echocardiographic evaluation, electrical measurements, and pharmacological therapy optimisation. QRS durations were automatically measured as the maximum of leads II, V₁, and V₆; LV end-diastolic diameter and LV end-systolic diameter were determined using M-mode echocardiography, under two-dimensional guidance in the parasternal long-axis view.

Statistical analysis

Means \pm standard deviations were used for normally distributed variables. To assess intra-group changes in measurements from baseline to follow-up, paired t-test was used for normally distributed variables. A p value < 0.05 was considered statistically significant.

RESULTS

Baseline clinical characteristics and pharmacological treatment of the four patients are listed in Table 1. Last follow-up data are shown in Table 2. Electrical parameters evaluated between baseline and follow-up in the thoracotomy group are listed in Table 3.

After a mean follow-up 45 ± 3.5 months, the haemodynamic benefits of CRT were apparent in all four patients. Mean LVEF increased from $28.4 \pm 6.5\%$ to $44.5 \pm 13.7\%$ ($p = 0.024$) in association with a reduction of LV end-systolic diameter from 62.3 ± 10.3 mm to 53.0 ± 13.11 mm ($p = 0.029$). QRS width decreased from 162.5 ± 23.6 ms to 147.5 ± 18.9 ms

Table 2. Clinical and echocardiographic parameters evaluated in 45 ± 3.5 months follow-up

	Case 1	Case 2	Case 3	Case 4
Sex	M	M	M	M
LVEDD [mm]	76	65	85	58
LVESD [mm]	60	42	68	42
Mitral regurgitation	Moderate	Mild	Mild-moderate	Mild-moderate
LVEF [%]	30	59	36	52.8
NYHA	II	II	III	III
QRS width [ms]	140	160	120	140

LVEDD — left ventricular end-diastolic diameter; LVESD — left ventricular end-systolic diameter; LVEF — left ventricular ejection fraction; NYHA — New York Heart Association functional class

Table 3. Electrical parameters evaluated between baseline and follow-up in the thoracotomy group (n = 4)

Parameter	Baseline	Follow-up: 45 ± 3.5 months	P
Pacing threshold at 0.4 ms [V]	0.9 ± 0.3	1.0 ± 0.2	NS
R-wave amplitude [mV]	15.2 ± 5.0	14.2 ± 4.2	NS
Pacing impedance [Ω]	635 ± 205	622 ± 190	NS

($p = 0.014$). NYHA values significantly improved before and after the procedure (3.3 ± 0.6 ratio 2.5 ± 0.5 , $p = 0.014$). No significant differences were found in any of the electrical parameters compared to baseline (pacing threshold at 0.4 ms [V]: 0.9 ± 0.3 ratio 1.0 ± 0.2 , $p = 0.367$; R-wave amplitude [mV]: 15.2 ± 5.0 ratio 14.2 ± 4.2 , $p = 0.160$; pacing impedance [Ω]: 635 ± 205 ratio 622 ± 190 , $p = 0.184$).

No other complications occurred in the pre-discharge period. Median hospitalisation after the thoracotomy procedure was 15 (range 10–20) days. No patients experienced complications related to the CRT system during follow-up. One was re-hospitalised for HF twice, and the other three experienced no re-hospitalisations for HF.

DISCUSSION

CRT is an important treatment option for selected patients with advanced chronic HF. Limited availability of suitable CS tributary veins often increases difficulties in achieving the optimal haemodynamic response. Endocardial procedures are often time-consuming, and X-ray exposure may be sub-optimal. Post-implant lead displacement or late LV threshold increases remain concerns despite substantial progress in lead technology and implantation procedures [7].

The particular coronary vein used for the LV lead will be dependent on individual coronary venous anatomy. In one series, placement of the LV lead tip in the intended target area (namely lateral, anterolateral, or posterolateral tributaries of the CS) was achieved in only 70% of cases [8]. When the

coronary lead position is reviewed in the context of area of the latest LV myocardial activation, LV lead tip concordance to, or in the vicinity of, the region with maximal delay was seen in only 64.8% and 55.2% of patients, respectively [9, 10]. Therefore, although a feasible implant technique, the coronary venous anatomy may limit placement of the LV lead and the ability to pace a defined region of the LV for CRT via the transvenous route. Direct surgical epicardial LV lead placement may overcome these limitations and provide the potential to pace the optimal target site [11]. With the improvement of the method, in recent years, researchers have examined the approach via mini-thoracotomy for biventricular pacing as a safe and reliable technique that may be considered as an alternative [5, 6].

Mair et al. [5] compared two different operative strategies (CS vs. epicardial stimulation) for LV pacing and found no difference in early mortality between patients undergoing transvenous CRT or surgical implantation. Twenty-five (31.6%) LV lead-related complications occurred in the CS-lead group, compared with one dislodgement in the surgical epicardial group (16 patients) ($p < 0.05$). At the 18-month follow-up seven CS-leads had a threshold of > 4 V/0.5 ms vs. epicardial leads, which were under 1.1 V/0.5 ms, except for one (1.8 V/0.5 ms). Correct lead positioning (oblique marginal branch area) was achieved in all surgical epicardial placements but only in 70% with CS leads. The authors concluded that epicardial LV lead placement is a safe and reliable method for CRT, and it has advantages regarding lead-related complications and the necessity for reoperation. Puglisi et al. [6] also had no surgical complications, and optimal lateral position was achieved for all patients who experienced transvenous implantation failure and converted to surgical epicardial LV-leads. After 12 months follow-up, there were no significant differences in any of the electrical parameters between baseline and follow-up. Significant improvement was observed in functional and echocardiographic parameters. The results suggested that LV pacing via limited thoracotomy was feasible and safe, and may be a second choice to transvenous implant for CRT delivery. The EP Wire survey [12] investigated

41 centres (members of the EHRA-EP Network) in several countries across Europe, and showed that if there were no feasible lateral veins for LV lead implantation via CS, the first option was to implant an epicardial LV lead via thoracotomy (54% of centres). The possibility of direct surgical placement of the LV lead to overcome the limitations of transvenous implantation imposed by coronary venous anatomy has raised the prospect of surgical implantation beyond rescue therapy for failed transvenous implants [13].

In our study, the four cases underwent a mini-left-lateral thoracotomy and implantation of an epicardial LV lead successfully, and optimal lead position was achieved in all four patients. Although this approach is more invasive, we did not observe any surgery-related or peri-operative complications, such as pericardial effusion, LV lead dislocation, high threshold, phrenic nerve pacing, and death. The four patients had no re-intervention but had increased length of hospital admission; these results are consistent with Koos's research [14]. After about four years (45 ± 3.5 months) of observation, no significant differences were found in any of the electrical parameters between baseline and follow-up. In the biventricular pacing mode, QRS duration decreased from 162.5 ± 23.6 ms to 147.5 ± 18.9 ms. Significant improvement was observed in functional and echocardiographic parameters (LVEF increased from $28.4 \pm 6.5\%$ to $44.5 \pm 13.7\%$). All four patients' quality-of-life and exercise tolerance improved significantly, and three patients experienced no further hospitalisations during follow-up.

CONCLUSIONS

After failed transvenous CRT, a mini-thoracotomy implant allows the choice of LV catheter implantation site, obtains low-pacing thresholds, and permits avoidance of additional X-ray exposure. Surgically placed epicardial leads had good long-term results and a lower LV-related complication rate compared to CS-leads. These results suggest that a combined approach to CRT delivery, including a transvenous attempt followed by a back-up mini-thoracotomy procedure, is feasible and safe, and can bring satisfactory long-term results.

Conflict of interest: none declared

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Czteroletnia obserwacja chorych po implantacji nasierdziowej elektrody do stymulacji lewej komory przez minitorakotomię w celu terapii resynchronizującej w zastoinowej niewydolności serca (cztery przypadki)

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Streszczenie

Wstęp i cel: Celem pracy było ustalenie, czy implantacja nasierdziowej elektrody lewokomorowej przez minitorakotomię jest bezpieczną i dostępną procedurą po niepowodzeniu przeżytej terapii resynchronizującej (CRT).

Metody: Autorzy opisali 4 przypadki chorych z zastoinową niewydolnością serca (CHF), u których po niepowodzeniu przeżytej implantacji elektrody lewokomorowej wykonano lewostronną minitorakotomię i wszczepiono elektrodę nasierdziową.

Wyniki: Po obserwacji trwającej średnio $45 \pm 3,5$ miesiąca korzyści hemodynamiczne związane z CRT stwierdzono u wszystkich pacjentów. Zanotowano zwiększenie średniej frakcji wyrzutowej lewej komory z $28,4 \pm 6,5\%$ do $44,5 \pm 13,7\%$ ($p = 0,024$) z jednoczesnym zmniejszeniem wymiaru późnorozkurczowego lewej komory z $62,3 \pm 10,3$ mm do $53,0 \pm 13,11$ mm ($p = 0,029$). Szerokość zespołu QRS zmniejszyła się ze $162,5 \pm 23,6$ ms do $147,5 \pm 18,9$ ms ($p = 0,014$). Po zabiegu stwierdzono również istotną poprawę w skali *New York Heart Association* w porównaniu z wartościami sprzed zabiegu.

Wnioski: Powyższe rezultaty wskazują, że implantacja nasierdziowej elektrody do stymulacji lewej komory przez minitorakotomię w celu stosowania CRT jest metodą, która może być wykorzystywana w leczeniu chorych z CHF i może się wiązać z satysfakcjonującymi wynikami długookresowymi.

Słowa kluczowe: torakotomia, nasierdziowa elektroda lewokomorowa, terapia resynchronizująca serca, niewydolność serca

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