The paced electrocardiogram cannot be used to identify left and right ventricular pacing sites in cardiac resynchronization therapy: validation by cardiac computed tomography

Anders Sommer*, Mads Brix Kronborg, Christoffer Tobias Witt, Bjarne Linde Nørgaard, and Jens Cosedis Nielsen

Department of Cardiology, Aarhus University Hospital, Skejby, Brendstrupgaardsvej 100, Aarhus N DK-8200, Denmark

Received 30 June 2014; accepted after revision 17 October 2014; online publish-ahead-of-print 5 December 2014

Aims
Paced electrocardiogram characteristics to confirm left ventricular (LV) and right ventricular (RV) pacing sites in cardiac resynchronization therapy (CRT) have not been validated with accurate knowledge of pacing lead positions. We aimed to evaluate the ability of the paced QRS morphology to differentiate between various LV and RV lead positions using cardiac computed tomography (CT) as the reference for LV and RV pacing site.

Methods and results
Ninety-seven CRT patients were included. The QRS morphology was evaluated during forced LV-only and RV-only pacing. Pacing lead positions were assessed in a standard LV 16-segment model and a simplistic RV 6-segment model using cardiac CT. Ten patients with LV lead displacement or a LV pacing site outside the non-apical free wall were excluded from the analysis of the LV paced QRS complex. Pacing within the LV free wall was associated with a superior and a right-axis deviation (P = 0.02 and 0.04, respectively). Pacing from basal LV segments mainly produced a late (V5 or later) precordial QRS transition as compared with mid-LV pacing (P = 0.001). No significant associations were found between RV pacing site and QRS axis or precordial transition. Different QRS morphologies were observed during single-chamber pacing from identical LV or RV myocardial segments.

Conclusion
Weak associations exist between LV and RV pacing sites and the paced QRS axis. None of the paced QRS characteristics can be used to reliably confirm specific LV and RV pacing sites in CRT patients.

Keywords
Electrocardiogram • Cardiac computed tomography • Right ventricular lead position • Left ventricular pacing site • Cardiac resynchronization therapy

Introduction
Cardiac resynchronization therapy (CRT) is an established treatment in selected heart failure patients exhibiting depressed left ventricular (LV) function, an electrocardiogram (ECG) with prolonged QRS duration, and who remain symptomatic despite optimal medical therapy.1 However, using the current selection criteria, a significant proportion of patients do not benefit from CRT.2,3 The LV pacing site is an important determinant of response to CRT, whereas the impact of the right ventricular (RV) lead position on clinical outcome remains controversial although a non-apical RV pacing site has been shown to increase the risk of ventricular tachyarrhythmias.4–10

Procedural biplane fluoroscopy or chest radiography is routinely used to evaluate pacing lead position in CRT.11 However, the accuracy of these imaging modalities to determine the exact LV and RV lead positions is modest when compared with cardiac computed tomography (CT).12,13 It has been suggested that the QRS morphology of the paced ECG may be helpful to confirm LV and RV lead positions both intra- and post-operatively.14–17 However, the paced ECG patterns for different LV and RV pacing sites have never been evaluated applying an imaging modality capable of displaying the exact pacing
What’s new?

- The left ventricular (LV) pacing site is an important determinant of response to cardiac resynchronization therapy (CRT), whereas the impact of the right ventricular (RV) lead position on clinical outcome remains controversial.
- Procedural biplane fluoroscopy or chest radiography is routinely used to determine LV and RV lead positions, but the accuracy of these imaging modalities is modest.
- We evaluated the ability of the paced QRS morphology to differentiate between cardiac CT verified LV and RV pacing sites.
- Different QRS-axis deviations and precordial transition patterns were observed during single-chamber pacing from identical cardiac CT verified LV or RV myocardial segments.
- No paced QRS characteristic can reliably confirm specific LV and RV pacing sites in CRT.

Methods

Patients

Between April 2011 and December 2012, we included 97 patients with chronic heart failure receiving a CRT pacemaker or a CRT-implantable cardioverter defibrillator at the Department of Cardiology, Aarhus University Hospital, Skejby. All patients were in New York Heart Association functional class II–IV, had LV ejection fraction (EF) < 35%, and a pre-implant ECG with QRS duration > 120 ms and left bundle branch block. Patients with a recent myocardial infarction (within 3 months), those who were pregnant or lactating, and those with severe renal dysfunction (estimated glomerular filtration rate < 30 mL/min) were excluded.

Transvenous CRT implantation using commercially available leads and devices was performed in all patients. Only active fixation RV leads were used. The LV leads were bipolar or quadripolar programmed in a bipolar configuration. No lead revisions were performed between the implant procedure and cardiac CT.

All patients were enrolled in an on-going randomized study evaluating the clinical impact of imaging-guided LV lead placement in CRT. Cardiac CT was performed according to the specific study protocol.

Echocardiographic LV EF, end-diastolic, and end-systolic volumes were assessed prior to CRT implant and at 6-month follow-up using Simpson’s biplane method (Vivid E9, GE Medical Systems).

All patients gave informed written consent before the implant procedure. The Central Denmark Regional Committee on health research ethics and the Danish Data Protection Agency approved the study.

Electrocardiogram acquisition and analysis

Before the CRT implant procedure, a standard supine 12-lead ECG (50 mm/s, 10 mm/mV) was recorded during intrinsic rhythm. After final lead positioning, a forced LV-only and RV-only pacing ECG was acquired (both V00-mode, 90 b.p.m., and an output < 3.5 V at 0.5 ms). A dedicated LV lead bipolar pacing configuration was used without cross-chamber pacing to avoid RV anodal capture. All ECGs were stored for offline analysis. Two reviewers without knowledge of the patients’ clinical lead position in three dimensions (3D). The aim of the current study was to evaluate simple characteristics of the paced QRS complex to differentiate between various LV and RV lead positions in CRT using cardiac CT as the reference for LV and RV pacing sites.

Cardiac computed tomography protocol and analysis

Cardiac CT was performed 6 months after CRT implantation using a second-generation dual-source CT scanner (Siemens Somatom Definition Flash, Siemens Healthcare). Computed tomography scanner settings and protocol were described previously. During breath hold, a contrast-enhanced [70 mL (Optiray® 350 mg/mL, Covidien)] helical retrospective ECG-gated scan timed according to contrast filling of both the RV and LV cavity was performed. The median (25th; 75th percentile) estimated radiation dose was 4.9 (3.8; 6.8) mSv. Pacing sites were determined using axial, multiplanar, and 3D volume-rendered reconstructions. Images were analysed using commercially available software (Syngo.via, Siemens Healthcare). Excellent intra- and interobserver agreements for the assessment of LV and RV lead positions by cardiac CT have previously been reported.

Assessment of left ventricular pacing site

The LV pacing site was determined using the standardized LV segmentation dividing the LV myocardium into 16 segments. The LV long axis was divided into equal thirds: basal, mid-LV, and apical. The LV short axis was divided into opposing segments: anterior, anterolateral, inferolateral, inferior, septal, and anteroseptal (Figure 1).

We excluded 10 patients from the analysis of the LV paced QRS complex. Five patients exhibited macroscopically visible LV lead displacement between the procedural fluoroscopy and follow-up cardiac CT. As previously described, this was determined by comparing the procedural fluoroscopy and a volume-rendered 3D cardiac CT reconstruction in the same right anterior oblique projection. Four of these patients showed LV lead movement within the coronary sinus tributary and small LV lead threshold changes (0.4 V at 1 ms; 0.1 V at 0.5 ms; 0.3 V at 0.4 ms; and 0.1 V at 0.4 ms, respectively). One patient demonstrated LV lead displacement to the CS without LV lead capture at follow-up. They were excluded to compare procedural acquired paced ECGs with CT images only in patients without visible LV lead movement between the procedure and the follow-up cardiac CT. Furthermore, a small minority of five patients had a LV pacing site outside the non-apical free wall: two patients had an apical LV pacing site producing a right inferior and a left inferior QRS axis, two patients had an anterior LV pacing site creating a
LV paced right inferior and a left inferior QRS axis, and one patient had an inferior LV pacing site producing a right superior-axis deviation, respectively. They were excluded from further analysis to evaluate only the paced ECG in patients with a pacing site within the non-apical anterolateral or inferolateral LV free wall. Achieving a LV pacing site in selected myocardial segments within this region is often considered optimal for CRT benefit.4,5,7

Assessment of right ventricular lead position

Evaluation of the RV lead tip position was performed using a simplistic segmentation dividing the RV long-axis cavity into equal thirds (basal, mid, and apical region). Subsequently, the RV lead position was evaluated in the short axis according to a six-segment model (two basal segments (basal septum and free wall), two mid-RV segments (mid septum and free wall), and two apical segments (apical septum including RV apex and apical free wall)) (Figure 1).

Statistics

Normally distributed continuous variables are presented as mean ± SD and were compared using Student’s t-test. Continuous variables not normally distributed were compared using a Wilcoxon’s rank-sum test. Proportions were compared by a two-sample test of proportions. A Pearson’s χ² or Fisher’s exact test wherever appropriate was used to compare categorical variables. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated to assess the ability of the paced QRS axis to identify lead positions between two categories of binary variables. Values of P < 0.05 were considered statistically significant. Commercially available software (Stata version 12, StataCorp) was used for statistical analysis.

Results

Study population

Clinical characteristics of the 97 patients are presented in Table 1. All patients received optimal medical therapy at maximum tolerated dosages. A significant increase in LV EF and reduction in LV volumes were observed at 6-month follow-up (Table 1).

Left ventricular paced QRS duration and axis

Similar LV paced QRS duration was found for patients with an anterolateral or inferolateral pacing site (197 ± 23 and 198 ± 21 ms; P = 0.80) and with a basal or mid-LV lead position (196 ± 21 and 201 ± 22 ms; P = 0.29), respectively.

The distribution of the paced QRS-axis deviations according to LV pacing site is illustrated in Table 2. Pacing from a LV non-apical free wall position produced a superior axis in 51 (59%) patients and an inferior axis in 36 (41%) patients (P = 0.02) while right- and left-axis deviations were observed in 50 (57%) and 37 (43%) patients (P = 0.04), respectively (Figure 2). An inferolateral LV pacing site was associated with a superior and a right-axis deviation (both P = 0.01), respectively. No significant associations were found between a basal

Table 1  Patient characteristics

| Description                                             | Value                |
|---------------------------------------------------------|----------------------|
| Age (years)                                             | 70 ± 9               |
| Female                                                  | 23 (23)              |
| Ischaemic heart failure                                 | 46 (47)              |
| Atrial fibrillation                                     | 27 (28)              |
| NYHA functional class II/III/IV                         | 43 (44)/50 (52)/4 (4) |
| Medical therapy                                         |                      |
| β-Blockers                                              | 89 (92)              |
| ACEI/ARB-II                                             | 89 (92)              |
| Diuretics                                               | 66 (68)              |
| Spironolactone                                          | 45 (46)              |
| Pre-implant QRS width (ms)                              | 171 ± 23             |
| Pre-implant LV end-diastolic volume (mL)                | 256 ± 70             |
| Pre-implant LV end-systolic volume (mL)                 | 197 ± 59             |
| Pre-implant LV EF (%)                                   | 23 ± 5               |
| Pre-implant creatinine (μmol/L)                         | 101 ± 31             |
| Pre-implant estimated glomerular filtration rate (mL/min) | 64 ± 18              |
| Received a CRT-implantable cardioverter defibrillator   | 62 (64)              |
| Follow-up LV end-diastolic volume (mL)                  | 204 ± 70             |
| Follow-up LV end-systolic volume (mL)                   | 135 ± 61             |
| Follow-up LV EF (%)                                     | 36 ± 10              |

Values are mean ± SD or n (%). ACEI/ARB-II, angiotensin-converting enzyme inhibitor/angiotensin receptor blockers II; CRT, cardiac resynchronization therapy; EF, ejection fraction; LV, left ventricular; NYHA, New York Heart Association.

*p < 0.0001 vs. pre-implant value.
or mid-LV pacing site and superior-to-inferior or right- to left-axis deviations.

The ability of different QRS-axis deviations to distinguish between regional LV lead positions is illustrated in Table 4.

### Left ventricular paced precordial QRS pattern

During LV pacing all patients had a net positive QRS complex in V1 and V2. In leads V3, V4, and V5, a positive QRS complex was seen in 85 (98%), 79 (91%), and 47 (54%) patients, respectively. Positive concordance was observed in 12 (14%) patients. The median (25th; 75th percentile) precordial QRS transition was V5 (V4; V5). Pacing from a basal vs. a mid-LV lead position produced a QRS transition in V5 or later in 36 (69%) and 11 (31%) patients ($P = 0.001$), respectively. Anterolateral and inferolateral wall LV pacing resulted in a QRS transition in V5 or later in 20 (57%) and 27 (52%) patients ($P = 0.63$), respectively.

### Right ventricular paced QRS duration and axis

The RV paced QRS duration was similar for patients with a septal or free wall (184 ± 22 vs. 191 ± 19 ms; $P = 0.16$) and with a mid-RV or apical RV lead position (183 ± 19 vs. 190 ± 22 ms; $P = 0.11$), respectively.

The distribution of RV lead positions according to paced QRS-axis deviation is shown in Table 3. No significant associations existed between RV pacing site and QRS-axis deviation (Figure 3). The diagnostic performance of RV paced QRS-axis deviations to distinguish between regional RV lead positions is presented in Table 4.

### Right ventricular paced precordial QRS pattern

A net negative QRS complex in V1 was observed in 37 (95%) and 55 (95%) patients with a mid-RV or apical RV lead position ($P = 0.99$) and in 48 (92%) and 44 (98%) patients with a septal or free wall RV lead position ($P = 0.22$), respectively. Five (5%) patients demonstrated a positive QRS complex in V1 (two patients with mid-RV septal pacing sites, two patients with apical septal pacing sites, and one patient with an apical free wall pacing site). The median (25th; 75th percentile) RV paced QRS transition was V4 (V4; V5). Pacing from a mid-RV vs. an apical or a septal vs. a free wall position produced a QRS transition at V4 or later in 33 (85%) vs. 52 (90%) patients ($P = 0.46$) and in 43 (83%) vs. 42 (93%) patients ($P = 0.11$), respectively.

### Notching of the QRS in inferior leads during right ventricular pacing

Inferior QRS notching was present in 25 (64%) and 34 (59%) patients with a mid-RV and apical RV lead position ($P = 0.59$) and in 31 (60%) and 28 (62%) patients with a septal and free wall RV lead position, respectively ($P = 0.81$).

---

**Table 2** Association between left ventricular lead position and paced QRS axis

| LV lead position | QRS axis | Right | Left | Superior | Inferior |
|------------------|----------|-------|------|----------|---------|
| Basal AL (n = 21) |          | 9 (43)| 12 (57)| 10 (48)  | 11 (52) |
| Basal IL (n = 31) |          | 22 (71)| 9 (29)| 22 (71)  | 9 (29)  |
| Mid-LV AL (n = 14)|          | 5 (36)| 9 (64)| 5 (36)   | 9 (64)  |
| Mid-LV IL (n = 21)|          | 14 (67)| 7 (33)| 14 (67)  | 7 (33)  |
| Total (n = 87)   |          | 50 (57)| 37 (43)| 51 (59)  | 36 (41) |

Values are n (row %). AL, anterolateral; IL, inferolateral; LV, left ventricular.

---

**Figure 2** Example of two different patients with a basal anterolateral LV pacing site (arrow) demonstrating a LV paced right inferior (left) and a left superior QRS-axis deviation (right) deviation, respectively. Both patients had a precordial transition in V5.
Impact of heart failure aetiology on the paced QRS complex

Among the 51 (53%) patients with non-ischaemic heart failure, we found a significant association between a free wall RV pacing site and an inferior QRS-axis deviation ($P = 0.03$) [sensitivity 83% (95% confidence interval (CI): 61–95%), specificity 46% (95% CI: 28–66%), PPV 56% (95% CI: 38–73%), and NPV 76% (95% CI: 50–93%)]. This association was not seen in the 46 (47%) patients with ischaemic heart failure ($P = 0.78$) [sensitivity 50% (95% CI: 28–78%), specificity 46% (95% CI: 26–67%), PPV 46% (95% CI: 26–67%), and NPV 50% (95% CI: 28–78%)]. During LV pacing, the 40 (46%) patients with ischaemic heart failure showed a significant association between an anterolateral pacing site and a left QRS axis ($P = 0.03$) [sensitivity 68% (95% CI: 43–87%), specificity 67% (95% CI: 43–85%), PPV 65% (95% CI: 41–85%), and NPV 70% (95% CI: 45–88%)]. This association did not reach significance in patients with non-ischaemic heart failure ($P = 0.16$) [sensitivity 50% (95% CI: 25–75%), specificity 71% (95% CI: 52–86%), PPV 47% (95% CI: 23–72%), and NPV 73% (95% CI: 54–88%)].

No other associations between LV and RV pacing sites and paced QRS axis or precordial transition pattern were found in patients with non-ischaemic or ischaemic heart failure. Irrespective of the heart failure aetiology, no associations between RV pacing site and inferior QRS notching were observed.

**Discussion**

In the present study, we evaluated simple paced QRS characteristics applicable in the routine clinical setting for confirming LV and RV pacing sites in CRT using cardiac CT as a reference for exact lead position. We found weak associations between the paced QRS axis and pacing sites and none of the paced QRS characteristics could be used to reliably confirm specific LV and RV pacing sites.

Along with device interrogation, the paced ECG has been applied to ensure adequate device function and provide an understanding of lead positions in CRT patients. The LV pacing site is an important determinant of response to CRT. Several studies have demonstrated an improved clinical outcome with LV pacing according to a pre-implant selected optimal LV myocardial segment; frequently within the non-apical anterolateral and inferolateral free wall. The optimal RV pacing site remains controversial. The paced QRS is readily available during the implantation and could be valuable if reflecting specific pacing sites. Several ECG markers of the paced QRS complex to confirm LV and RV pacing sites in CRT have been proposed using fluoroscopy, electroanatomical mapping, or chest X-ray as reference for LV and RV lead positions.

Commonly, a LV paced QRS axis pointing towards the right inferior or right superior quadrant and a net positive QRS in V1–V3 is reported to confirm pacing from the LV anterolateral and inferolateral free wall. Furthermore, a basal pacing site has been associated with a later precordial QRS transition than

**Table 3** Association between RV lead position and paced QRS axis

| QRS axis | Right | Left | Superior | Inferior |
|----------|-------|------|----------|---------|
| RV lead position |       |      |          |         |
| Mid-RV septum ($n = 16$) | 5 (31) | 11 (69) | 9 (56) | 7 (44) |
| Mid-RV free wall ($n = 23$) | 9 (39) | 14 (61) | 6 (26) | 17 (74) |
| Apical septum ($n = 36$) | 18 (50) | 18 (50) | 15 (42) | 21 (58) |
| Apical free wall ($n = 22$) | 9 (42) | 13 (58) | 9 (42) | 13 (58) |
| Total ($n = 97$) | 41 (42) | 56 (58) | 39 (40) | 58 (60) |

Values are n (row %).

RV, right ventricular.
In agreement with these criteria, we found the majority of patients with a free wall LV pacing site having a superior and a right QRS-axis deviation and a net positive QRS in the precordial leads V1–V3, with basal pacing sites demonstrating the latest precordial QRS transition. However, a large percentage of anterolateral LV pacing sites produced a left-axis deviation, mainly in patients with ischaemic heart failure. A leftward axis during free wall LV pacing has previously been reported but the reasons for this unusual axis deviation remain unclear.19 Varying the epicardial LV pacing output has been shown to influence the LV electrical activation sequence possibly by RV anodal capture or by capturing a larger myocardial area.22 Using dedicated LV bipolar pacing, we avoided RV anodal stimulation. However, extending LV myocardial capture beyond or away from local conduction blocks and thereby altering QRS morphology may possibly explain a left-axis deviation during free wall LV pacing.

The proposed RV paced QRS criteria to confirm an apical RV lead location is the presence of a superior axis and mainly a net negative QRS in V1 while a septal RV pacing site produces a right inferior axis and a negative QRS in V1.15,16,19 Furthermore, RV free wall pacing has been reported to produce a left-axis deviation, QRS notching in the inferior leads, and a longer QRS duration than septal RV pacing in patients without heart failure.15,16 Concordantly, we recorded a net negative QRS in V1 in the majority of patients during RV pacing from all analysed RV regions. In contrast to previous reports, we found no significant associations between regional LV pacing site and paced QRS-axis deviation, precordial QRS transition, inferior QRS notching, or QRS duration in the total study population. This lack of agreement may likely be explained by different imaging methods applied for determining lead positions. We used cardiac CT. Previous studies applied fluoroscopy or electroanatomical mapping as reference for the RV pacing site.15,16,19 Fluoroscopy has been demonstrated to be inaccurate and poorly reproducible for localizing RV pacing site as compared with cardiac CT.12 In addition, electroanatomical mapping can only approximate cardiac anatomy and not illustrate detailed cardiac morphology as displayed by cardiac CT.

Despite several associations between pacing site and paced QRS characteristics, the present study demonstrates different QRS-axis deviations during single-chamber pacing from identical LV or RV myocardial segments. Accordingly, the diagnostic performance of the paced ECG to confirm specific LV and RV lead positions is low. This may be explained by a varying degree of cardiac dilatation, rotation, and positioning in the thoracic cavity in heart failure patients altering the direction of electrical activation. Also, in patients with ischaemic cardiomyopathy, scar areas may change electrical activation patterns. Hence similar cardiac anatomical pacing sites may produce different paced QRS morphologies.

### Limitations

We acknowledge the inherent limitations of a single-centre study with a moderate sample size. Nevertheless, this is the first study evaluating the diagnostic value of the paced QRS complex characteristics to distinguish between regional LV and RV pacing sites using cardiac CT as the reference for exact pacing lead position.

We applied a 6-month follow-up cardiac CT as a reference for pacing lead positions and LV remodelling was observed during follow-up. However, the anatomical relationship between the LV myocardium and the coronary sinus tributary containing the LV lead was presumably unaltered despite cardiac remodelling. We excluded patients with visible LV lead displacement from the analysis and only active fixation RV leads were used.

Our analysis of the LV paced QRS complex was limited to patients with a non-apical free wall LV lead position and the results are not applicable for other LV lead positions.

Introduction of cardiac CT into the diagnostic algorithm in CRT patients will increase their cumulative radiation exposure and should not be used routinely outside protocolled studies. In clinical practice, cardiac CT may be useful to confirm exact LV lead position and assess the presence and location of additional cardiac veins before considering lead revision. Several approaches to minimize radiation dose are applied in this study, including the use of iterative reconstruction algorithms, individual settings of tube voltage and

### Table 4 Diagnostic performance of different QRS-axis deviations to distinguish between regional LV and RV lead positions

|                | Sensitivity | Specificity | PPV  | NPV  |
|----------------|-------------|-------------|------|------|
| LV pacing      |             |             |      |      |
| Superior axis distinguishing an inferolateral from an anterolateral pacing site | 69 (55–81) | 57 (39–74) | 71 (56–83) | 56 (38–72) |
| Rightward axis distinguishing an inferolateral from an anterolateral pacing site | 69 (55–81) | 60 (42–76) | 72 (58–76) | 57 (39–73) |
| Superior axis distinguishing a basal from a mid-LV position | 62 (47–75) | 46 (29–63) | 63 (48–76) | 44 (28–62) |
| Rightward axis distinguishing a basal from a mid-LV position | 59 (45–73) | 46 (29–63) | 62 (47–75) | 43 (27–61) |
| RV pacing      |             |             |      |      |
| Inferior axis distinguishing a free wall from a septal position | 67 (51–80) | 46 (32–61) | 52 (38–65) | 62 (45–77) |
| Leftward axis distinguishing a free wall from a septal wall position | 60 (44–74) | 44 (30–59) | 48 (35–62) | 56 (40–72) |
| Inferior axis distinguishing a mid-RV from an apical position | 62 (45–77) | 41 (29–55) | 41 (29–55) | 62 (45–77) |
| Leftward axis distinguishing an mid-RV from an apical position | 64 (47–79) | 47 (33–60) | 45 (31–59) | 66 (49–80) |

Values are proportion in % (95% confidence interval).
LV, left ventricular; NPV, negative predictive value; PPV, positive predictive value; RV, right ventricular.
current, and tube current modulation with a narrow full current window, respectively.23

Conclusion
Different QRS morphologies exist during single-chamber LV or RV pacing from identical cardiac CT verified LV or RV myocardial segments. The paced QRS characteristics are not valid for confirming specific LV and RV pacing sites in CRT patients.

Conflict of interests: J.C.S. has received speakers’ honoraria from Biotronik, Biosense Webster, St Jude Medical, and Medtronic and a research grant from Biosense Webster. M.B.K. has received speakers’ honoraria from Biotronik. B.L.N. has received research grants from Edwards Lifesciences and Siemens. All other authors have no conflicts of interest to disclose.

Funding
This work was supported by Aarhus University, the Danish Heart Foundation (grant number 11-04-R84-A3234-22641), the Danish Council for Independent Research (grant number 11-107461), Central Denmark Region (grant number 1-45-72-4-09), Eva and Henry Fränkels Foundation, and Fabrikant Karl G. Andersen’s Foundation.

References
1. Moss AJ, Hall WJ, Cannom DS, Klein H, Brown MW, Daubert JP et al. Cardiac-resynchronization therapy for the prevention of heart-failure events. N Engl J Med 2009; 361: 1329–38.
2. Cleland JG, Daubert JC, Erdmann E, Freemantle N, Gras D, Kappenberger L et al. The effect of cardiac resynchronization therapy on morbidity and mortality in heart failure. N Engl J Med 2005; 352: 1539–49.
3. Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA et al. 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: the task force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Europace 2013; 15: 1070–118.
4. Delgado V, van Bommel RJ, Berthin PM, Borleffs CJ, Marsen NA, Arnold CT et al. Relative merits of left ventricular dyssynchrony, left ventricular lead position, and myocardial scar to predict long-term survival of ischemic heart failure patients undergoing cardiac resynchronization therapy. Circulation 2011; 123: 70–8.
5. Khan FZ, Virdee MS, Palmer CR, Pugh PJ, O’Halloran D, Elsk C et al. Targeted left ventricular lead placement to guide cardiac resynchronization therapy: the TARGET study: a randomized, controlled trial. J Am Coll Cardiol 2013; 59: 1509–18.
6. Singh JP, Klein HU, Huang DT, Reek S, Kuniss M, Quesada A et al. Left ventricular lead position and clinical outcome in the multicenter automatic defibrillator implantation trial-cardiac resynchronization therapy (MADIT-CRT) trial. Circulation 2011; 123: 1159–66.
7. Saba S, Marek J, Schwartzman D, Jain S, Adelstein E, White P et al. Echocardiography-guided left ventricular lead placement for cardiac resynchronization therapy: results of the speckle tracking assisted resynchronization therapy for electrode region trial. Circ Heart Fail 2013; 6: 427–34.
8. Kristiansen HM, Vollan G, Hovstad T, Keiglevagen H, Faerestrand S. A randomized study of haemodynamic effects and left ventricular dyssynchrony in right ventricular apical vs. high posterior septal pacing in cardiac resynchronization therapy. Eur J Heart Fail 2012; 14: 506–14.
9. Kutyifa V, Bloch Thomsen PE, Huang DT, Rasera S, Tompkins C, Jons C et al. Impact of the right ventricular lead position on clinical outcome and on the incidence of ventricular tachyarrhythmias in patients with CRT-D. Heart Rhythm 2013; 10: 1770–7.
10. Reddauxcha L, Cihak R, Bytserkji J, Vancura V, Fridl P, Haskova L et al. Optimization of right ventricular lead position in cardiac resynchronization therapy. Eur J Heart Fail 2006; 8: 609–14.
11. Kumar P, Blends D, Nandigam V, Moore SA, Heist EK, Singh JP. Assessment of the post-implant final left ventricular lead position: a comparative study between radiographic and angiographic modalities. J Interv Card Electrophysiol 2010; 29: 17–22.
12. Sommer A, Kronborg MB, Norgaard BL, Gerdes C, Mortensen PT, Nielsen JC. Left and right ventricular lead positions are imprecisely determined by fluoroscopy in cardiac resynchronization therapy: a comparison with cardiac computed tomography. Europace 2014; 16: 1334–41.
13. Rickard J, Ingelino C, Sraw N, Wilkof BL, Grimm RA, Schoenhagen P et al. Chest radiography is a poor predictor of left ventricular lead position in patients undergoing cardiac resynchronization therapy: comparison with multidetector computer tomography. J Interv Card Electrophysiol 2011; 32: 59–65.
14. Jastrzebski M, Fijorek K, Czarnecka D. Electrocardiographic patterns during left ventricular epicardial pacing. Pacing Clin Electrophysiol 2012; 35: 1361–8.
15. McGavigan AD, Roberts-Thomson KC, Hillock RJ, Stevenson IH, Mond HG. Right ventricular outflow tract pacing: radiographic and electrocardiographic correlates of lead position. Pacing Clin Electrophysiol 2006; 29: 1063–8.
16. Hayes DL, Aravatham SJ, Friedman PA. Cardiac pacing, defibrillation, and resynchronization: A clinical approach. 3rd ed. Chichester, West Sussex: Wiley-Blackwell, 2013. pp. 235–318.
17. van Deursen CJ, Blauw Y, Wijtens ML, Debie L, Wecke L, Crijsen H et al. The value of the 12-lead ECG for evaluation and optimization of cardiac resynchronization therapy in daily clinical practice. J Electrocardiol 2014; 47: 202–11.
18. Sommer A, Kronborg M, Poulsen S, Bottcher M, Norgaard B, Bouchelouche K et al. Empiric versus imaging guided left ventricular lead placement in cardiac resynchronization therapy (ImagingCRT): study protocol for a randomized controlled trial. Trials 2013; 14: 113.
19. Barold SS, Herweg B. Usefulness of the 12-lead electrocardiogram in the follow-up of patients with cardiac resynchronization devices. Part I. Cardio 2011; 18: 476–86.
20. Daubert JC, Saxon L, Adamson PB, Auricchio A, Berger RD, Beshe JJ et al. 2012 EHRA/HERFS expert consensus statement on cardiac resynchronization therapy in heart failure: implant and follow-up recommendations and management. Europace 2012; 14: 1236–86.
21. Cerquera MD, Weismann NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey W et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Circulation 2002; 105: 539–42.
22. Tedrow UB, Stevenson WG, Wood MA, Shepard RK, Hall K, Pellegrini CP et al. Activation sequence modification during cardiac resynchronization by manipulation of left ventricular epicardial pacing stimulus strength. Pacing Clin Electrophysiol 2007; 30: 65–9.
23. Einstein AJ, Knutti J. Cardiac imaging: does radiation matter? Eur Heart J 2012; 33: 573–8.