Vectorcardiography as a Tool for Easy Optimization of Cardiac Resynchronization Therapy in Canine Left Bundle Branch Block Hearts

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Background—In cardiac resynchronization therapy (CRT), optimization of left ventricular (LV) stimulation timing is often time consuming. We hypothesized that the QRS vector in the vectorcardiogram (VCG) reflects electric interventricular dyssynchrony, and that the QRS vector amplitude (VAQRS), halfway between that during left bundle branch block (LBBB) and LV pacing, reflects optimal resynchronization, and can be used for easy optimization of CRT.

Methods and Results—In 24 canine hearts with LBBB (12 acute, 6 with heart failure, and 6 with myocardial infarction), the LV was paced over a wide range of atrioventricular (AV) delays. Surface ECGs were recorded from the limb leads, and VAQRS was calculated in the frontal plane. Mechanical interventricular dyssynchrony (MIVD) was determined as the time delay between upslopes of LV and right ventricular pressure curves, and systolic function was assessed as LV dP/dtmax. VAQRS and MIVD were highly correlated ($r=0.94$). The VAQRS halfway between that during LV pacing with short AV delay and intrinsic LBBB activation accurately predicted the optimal AV delay for LV pacing (1 ms; 95% CI, −5 to 8 ms). Increase in LV dP/dt max at the VCG predicted AV delay was only slightly lower than the highest observed ∆LV dP/dt max (−2.7%; 95% CI, −3.6 to −1.8%). Inability to reach the halfway value of VAQRS during simultaneous biventricular pacing (53% of cases) was associated with suboptimal hemodynamic response, which could be corrected by sequential pacing.

Conclusions—The VAQRS reflects electric interventricular dyssynchrony and accurately predicts optimal timing of LV stimulation in canine LBBB hearts. Therefore, VCG may be useful as a reliable and easy tool for individual optimization of CRT. (Circ Arrhythm Electrophysiol. 2012;5:544-552.)

Key Words: bundle branch block • cardiac resynchronization therapy • pacing • AV optimization • vectorcardiography

In patients with left bundle branch block (LBBB) and heart failure, cardiac resynchronization therapy (CRT) aims to accomplish a more synchronous ventricular electric activation and contraction pattern by left ventricular (LV) based pacing, thereby improving LV systolic function.

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In applying CRT, optimizing the atrioventricular (AV) delay is important, because it determines the coupling between atrial and ventricular contraction, the dyssynchrony between the right ventricle (RV) and LV (interventricular dyssynchrony), as well as the dyssynchrony within the LV (intraventricular dyssynchrony). Current procedures for AV delay optimization use assessment of diastolic filling patterns and systolic function by echocardiography (mitral inflow or aortic outflow measurements) or invasive LV pressure measurements. Disadvantages of these procedures are that they are time consuming, complicated, expensive, and may even be inaccurate.1

In a previous study we have shown that optimal LV systolic function can be predicted using mechanical interventricular dyssynchrony (MIVD).2 In that study, performed in canine LBBB hearts as well as in CRT patients, a MIVD value halfway between its minimal (LV pacing with short AV delay) and maximal value (during LBBB or RV pacing) coincided with optimal systolic function.2 We hypothesized that the QRS vector derived from the surface ECG reflects electric interventricular dyssynchrony and that, similar to the MIVD measurements, vectorcardiography (VCG) could be used to optimize LV stimulation timing in CRT.

In the present study it was our aim to investigate whether VCG can be used as a relatively easy and noninvasive method...
for optimization of timing of LV stimulation in CRT. We used the well-established canine model of LBBB, induced by radiofrequency ablation, alone or in combination with heart failure (induced by rapid pacing) or chronic myocardial infarction. 1

Methods

Animal handling was performed according to the Dutch Law on Animal Experimentation and the European Directive for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (86/609/EU). The protocol was approved by the Animal Experimental Committee of Maastricht University.

Experimental Setup

The experiments were performed on 24 adult mongrel dogs of either sex and of unknown age. Animals were divided into a group with only LBBB (n=12), a group with LBBB and heart failure (LBBB+HF, n=6), and a group with LBBB and myocardial infarction (LBBB+MI, n=6). All interventions and measurements were performed after percutaneous induction. All anesthesia was maintained by ventilation with O2 and N2O (1:2), in combination with continuous infusion of midazolam (0.25 mg/kg/h iv) and sufentanyl (3 µg/kg/h iv). 4

In all animals, LBBB was induced under closed chest conditions by transvascular radiofrequency ablation. 5 In the LBBB+HF group, a pacemaker (Medtronic InSyncIII) was implanted during the same procedure with a standard pacing lead transvenously positioned in the RV apex. After a week of recovery from LBBB induction and pacemaker implantation, the heart was paced at a rate of 220 beats per minute for 4 weeks to induce systolic LV dysfunction. Parasternal echocardiographic recordings were obtained once a week to monitor LV function and dimensions.

In the LBBB+MI group, transmural MI was created in the territory of either the left anterior descending (LAD, n=3) or left circumflex (LCX, n=3) coronary artery by embolization using a suspension of polyvinyl alcohol foam particles. 6 Four weeks after embolization, the animals were anesthetized again and LBBB was induced, followed by the measurements a week later.

For the pacing protocol and measurements (in the LBBB group directly after induction of LBBB, in the LBBB+HF group 4 weeks after starting rapid pacing, and in the LBBB+MI group 5 weeks after inducing myocardial infarction), thoracotomy was performed with the animal laying on its right side and epicardial LV pacing electrodes were positioned at prespecified wall regions in all animals. Right atrial and RV pacing leads, as well as LV (via carotid artery) and RV 7F pressure catheters (CD-Leycom) were positioned transvenously.

Pacing Protocol

For both LV and biventricular (BiV) pacing, atrial pacing was performed at a constant rate (approximately 10% above intrinsic rhythm) during the entire protocol. In the LV pacing protocol (n=18; 6 animals of each group), single site LV pacing was performed at 7 different LV wall regions per animal; basal anterior, basal lateral, basal posterior, mid anterior, mid lateral, mid posterior, and apical. To achieve various degrees of fusion between LV pacing induced activation and intrinsic activation via the right bundle branch, a wide range of paced AV delays (30–250 ms) were selected using intervals of 20 ms.

In addition, we performed BiV pacing with a short AV delay in all animals of the LV pacing protocol. In the BiV pacing protocol (n=6; LBBB only), simultaneous BiV pacing (RV apex + LV posterolateral wall) was performed at AV delays of 50 to 230 ms using 20 ms intervals. Subsequently, sequential BiV pacing was performed using VV intervals from –100 ms (LV first) to +100 ms (RV first) at each animal’s optimal AV according to LV pressure measurements.

Baseline measurements were performed during atrial pacing and were repeated at each ventricular pacing site to correct for baseline drift. Recording of measurements commenced 10 seconds after initiation of LV or BiV pacing or switching of AV and VV interval to achieve hemodynamic stability, and continued for approximately 1 minute.

Measurements of Vectorcardiography, Mechanical Interventricular Dyssynchrony, and Hemodynamics

Surface ECGs were recorded from the limb lead electrodes, and the QRS vector was plotted in the frontal plane using custom MATLAB software (MathWorks). The QRS vector angle and amplitude were calculated by averaging each parameter over 5 sequential heart beats, where QRS vector angle was the angle at the maximum QRS vector amplitude (VAmax). Values of the VAmax were regarded as negative when the vector pointed upwards (angle from 0 to –180 degrees). QRS duration was defined as the interval from the beginning of the pacing stimulus to the beginning of the Q-wave (during baseline and LV pacing at long AV delays when the pacing stimulus is located within the QRS complex) to the terminal component of the QRS complex.

Mechanical interventricular dyssynchrony was determined as the time delay between up-slopes of simultaneously recorded LV and RV pressure curves. 7 A negative value of MIVD indicates an earlier LV than RV pressure rise.

The hemodynamic response was evaluated by calculating the average maximal rate of LV pressure rise (LV dp/dtmax) of 10 beats (includes at least 1 respiratory cycle) acquired by the 7F catheter tip manometer.

Statistical Analysis

Statistical analyses were performed using SPSS software version 18 (SPSS Inc.). Continuous variables were presented as mean ± standard deviation (SD), or as median and interquartile range (25th to 75th percentile) in case of a non-normal distribution (histogram). A linear mixed model with random intercept was used to account for repeated measurements (7 different pacing sites) per animal and missing data. Independent samples t tests (95% CI) were used to compare group means. Comparison between the predicted and observed optimal AV delay was performed by paired samples t tests. Agreement between predicted and observed measurements also was evaluated by plotting the difference between predicted and observed values as a function of the observed value, and calculating limits of agreement (defined as mean±1.96 SD for normal distributed data and median, with the 95th percentile for non-normal distributed data).

Correlations were evaluated with Pearson correlation coefficient. Differences were considered significant at a p-value level of 0.05 (1-tailed for the difference in predicted and observed LV dp/dtmax because the predicted LV dp/dtmax could only be equal or lower than the observed LV dp/dtmax). Bonferroni correction was used for post hoc comparisons between groups.

Results

Of the attempted 7 (pacing sites)x18 (experiments)=126 measurements in the LV pacing protocol, 112 were successfully acquired with similar amounts of measurements per group. The unsuccessful data acquisition in 14 cases related to hemodynamic instability or technical problems. In the BiV pacing protocol all 6 sets of measurements were successfully acquired for both simultaneous and sequential pacing.

In the LBBB+HF group, 4 weeks of rapid RV pacing caused a decrease in LV ejection fraction from 54±8% to 15±2%, when LV end systolic and end diastolic volumes increased from 29±13 mL to 83±45 mL, and from 76±21 mL to 102±55 mL, respectively. In the LBBB+MI group, myocardial infarction was transmural in all hearts and infarct size (determined post
mortem by triphenyltetrazolium chloride staining) was 21±6% (range 16%–32%) of total LV wall mass.

**Baseline Parameters**
During baseline atrial pacing, QRS duration, heart rate, and MIVD did not differ between the LBBB, LBBB+HF, and LBBB+MI groups of the LV pacing protocol (Table 1). However, baseline maximal vector amplitude was significantly larger and LV dP/dtmax was significantly lower in the LBBB+HF group than in the LBBB (P=0.017 and P=0.001, respectively) and LBBB+MI (P=0.004 and P=0.002, respectively) groups.

**Effects of Left Ventricular Pacing on Vectorcardiography, ECG, and Mechanical Interventricular Dyssynchrony**
During intrinsic conduction (LBBB), the angle of the QRS vector was 88±11 degrees (Figure 1). During LV pacing with short AV delay, this angle differed between LV pacing sites. On average, pacing at an anterior site resulted in a vector angle of 141±57 degrees, whereas this angle was –104±41 degrees for pacing at posterior sites, –92±32 degrees for pacing at anterior sites, and –94±16 degrees for pacing at the LV apex (Figure 1).

Starting from LV pacing with short AV delay, stepwise lengthening of AV delay gradually changed the shape of the QRS complex in the ECG (Figure 2, upper row) and, correspondingly, the shape of the VCG. Stepwise lengthening of the AV delay initially caused a reduction in the VAQRS and subsequently an increase in almost the opposite direction, where it finally reached its baseline value (Figure 2, middle row). This was observed in all groups and for all pacing sites, but the angle shift differed between pacing sites, with smaller angle shifts for the anterior sites, as can be observed in Figure 1.

Single site LV pacing with a short AV delay resulted in negative values of MIVD, indicating an earlier LV than RV contraction. When lengthening AV delays, MIVD increased and became positive, reaching baseline LBBB values during pacing with long AV delays (Figure 2, lower row).

**Correlation Between Mechanical Interventricular Dyssynchrony and QRS Vector Amplitude**
While stepwise lengthening of the AV delay during LV pacing, values for MIVD and VAQRS changed in parallel, which resulted in a strong correlation between these 2 variables, as indicated by the example presented in Figures 3A and B.

For all LV pacing sites and models combined, the median Pearson correlation coefficient was r=0.94 (interquartile range [IR], 0.88–0.97; Figure 4). Poorer correlations between VAQRS and MIVD were observed for anterior pacing sites in all groups and for the sites where pacing was performed near the infarcted area in the LBBB+MI group (Figure 4; anterolateral pacing sites for LAD infarction and posterior pacing sites for LCX infarction).

Correlation between VAQRS and QRS duration during LV pacing was poor for all models; median r=−0.38 (IR, −0.69–−0.01; data not shown).

**Prediction of the Optimal Atrioventricular Delay With Vectorcardiography**
Compared with baseline (atrial pacing), LV dP/dtmax increased with LV pacing at the optimal AV delay by a mean of 12% (95% CI, 5%–20%) for the LBBB group, 18% (95% CI,
11%–26%) for the LBBB+HF group, and 11% (95% CI, 3%–18%) for the LBBB+MI group.

A typical example of LV pacing at the basal lateral level in a canine heart with LBBB+HF illustrates and supports the concept presented in the methods, that the optimal AV delay coincides with a value of the \( V_{AQRS} \) halfway between its minimal and maximal value, that is during LV pacing with short AV delay and LBBB, respectively (Figures 3C and D).

Combined data of all animals and LV pacing sites also showed that for \( V_{AQRS} \) and for MIVD the values at the optimal AV delay were halfway in between that of LV pacing with short AV delay and LBBB (Figure 5).

Overall, there was no significant difference between the VCG predicted and the observed AV delay at which LV \( dp/dt_{max} \) had increased most (1 ms; 95% CI, –5 to 8 ms; \( P = 0.675 \); Figure 6, panels A and E). The Pearson correlation coefficient of the linear relation between VCG predicted and observed optimal AV delay was \( r = 0.75 \) (\( P < 0.001 \)) for all groups and pacing sites combined. Comparing different LV pacing sites, the correlation coefficients were lower for anterior sites (\( r = 0.59 \)) than for lateral (\( r = 0.82 \)), posterior (\( r = 0.85 \)), and apical sites (\( r = 0.82 \)).

Relative increase (compared with baseline LBBB) in LV \( dp/dt_{max} \) (\( \Delta LV \ dp/dt_{max} \)) at the VCG predicted AV delay was only slightly lower than the highest observed \( \Delta LV \ dp/dt_{max} \).

Figure 2. ECG (lead aVF; upper panel), vectorcardiogram (VCG, frontal plane; middle panel), and normalized pressure curves of the right (RV) and left ventricle (LV; lower panel), while stepwise increasing AV delay during LV pacing. Contribution of intrinsic conduction and loss of contribution by LV pacing can be recognized by changing QRS morphology in the ECG, changing QRS vector amplitude in the VCG, and changing values of mechanical interventricular dyssynchrony (MIVD) as measured by the time difference between upstrokes of RV and LV pressure curves. A negative value of MIVD indicates an earlier LV than RV pressure rise.

Figure 3. Changes in mechanical interventricular dyssynchrony (MIVD) and QRS vector amplitude (\( VA_{QRS} \)) as a function of the paced atrioventricular (AV) delay (A), with their corresponding linear relation (B) during left ventricular (LV) pacing at the basal lateral wall in a canine heart with left bundle branch block (LBBB) and heart failure. Changes in LV \( dp/dt_{max} \) relative to baseline LBBB as a function of the paced AV delay (C) and its relation with \( VA_{QRS} \) (D). Changes in QRS duration (QRSd) as a function of the paced AV delay (E) and its relation with \( \Delta LV \ dp/dt_{max} \) (F). The AV delay at which the halfway value of \( VA_{QRS} \) during LV pacing with short AV delay and LBBB was found (solid line), resulted in maximal improvement of LV \( dp/dt_{max} \), whereas the AV delay at which QRS duration was minimal (dotted line) resulted in suboptimal improvement of LV \( dp/dt_{max} \).
Lack of reaching the halfway value of VAQRS with simultaneous BiV pacing showed a strong tendency to result in lower ∆LV dP/dt max values during BiV than during LV pacing at the VCG predicted AV delay (−2.3%; 95% CI, −4.7% to 0.1%; P=0.060). Application of sequential BiV pacing in case of unreachable halfway VAQRS during simultaneous BiV pacing resulted in achievement of the halfway VAQRS with LV pre-excitation, and corresponded with increased ∆LV dP/dt max, whereas no further increase was observed with sequential pacing if the halfway VAQRS already was achieved during simultaneous BiV pacing (Figure 8, lower panels). No significant difference was observed between the VCG predicted and the optimal VV interval (0 ms; 95% CI, −10 to 10 ms; limits of agreement ±25; P=0.175; r=0.77). Also, ∆LV dP/dt max at the VCG predicted VV interval was not significantly lower than the highest observed ∆LV dP/dt max (median 0%; IR, −0.3–0; P5th–1.6%; P=0.134).

Interbeat Variability and Signal-To-Noise Ratio

The VAQRS during all paced and nonpaced conditions was reproducible (5 beats per setting), with a mean value of 0.74 mV and a beat-to-beat variability of 0.05 mV (95% CI, 0.04–0.06). The mean signal-to-noise ratio was 40.4, indicating a high degree of certainty in predicting the same optimal AV delay repeatedly with use of the VAQRS.

Discussion

The present study in canine LBBB hearts demonstrates a linear relation between the QRS vector amplitude in the frontal plane and mechanical interventricular dyssynchrony, indicating that the VAQRS can be regarded as an index of electric interventricular dyssynchrony. In addition, the VAQRS can be used to predict the timing of LV stimulation, at which optimal LV systolic function occurs. Because the VCG is an easy, noninvasive, accurate, and reproducible

Simultaneous and Sequential Biventricular Pacing

Simultaneous BiV pacing resulted in values of VAQRS and MIVD in between those of baseline LBBB and LV pacing with short AV delay. In 53% of 68 tested cases with LV lateral or posterior wall pacing, simultaneous BiV pacing was not able to converse VAQRS to at least the halfway value (“inappropriate AV optimization”, left upper panel of Figure 8).

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measurement, these data indicate that this technique may be attractive to use for routine optimization of AV delay and VV interval in CRT.

The QRS Vector Reflects Electric Interventricular Dyssynchrony

Our data demonstrate that the VAQRS reflects electric interventricular dyssynchrony in canine LBBB hearts, irrespective of the presence of heart failure or myocardial infarction. The present study also shows that the VAQRS is superior to QRS duration in reflecting interventricular dyssynchrony. Besides for selection of CRT patients and prediction of response to CRT, QRS duration commonly is used in daily practice for optimization of timing of biventricular stimulation.8 The poor relation between VAQRS and QRS duration in the present study is corroborated by measurements using electrocardiographic imaging in patients undergoing CRT, which demonstrated that interventricular electric dyssynchrony did not correlate with QRS duration.9 In addition, mechanical interventricular dyssynchrony showed a poor correlation with QRS duration in canine LBBB hearts during LV pacing in previous studies,2,5 as well as in the present study. While QRS duration provides a good reflection of total ventricular activation time, the QRS vector presumably provides additional information about interventricular dyssynchrony. The latter can be understood from the fact that the VCG depicts integral changes in the current flow through the heart during the cardiac cycle. A similar impression could be obtained from the standard 12-lead ECG, but because the latter only projects the vector along the axis of each lead, the true vector amplitude may be missed by the ECG, while it will always be observed using VCG.

Figure 6. Correlation between the observed atrioventricular (AV) delay resulting in maximal increase in ∆LV dP/dtmax (relative to baseline left bundle branch block), and the AV delay predicted by the “halfway value” of the QRS vector amplitude (panel A), and the AV delay predicted by narrowest QRS (panel C). The area of the dots indicates the number of observations at that specific value; clustering of data points are due to stepwise increase of AV delay using intervals of 20 ms. The observed maximal increase in ∆LV dP/dtmax plotted against the increase in ∆LV dP/dtmax at the vectorcardiogram predicted AV delay (panel B) and at the predicted AV delay with narrowest QRS (panel D). Panels E, F, G, H: observed values plotted against the difference between predicted and observed. Indicated are bias and upper and lower limits of agreement (UPL and LWL, respectively), defined as mean±1.96*SD (E, G) or median accompanied by the 5th percentile (95th percentile=zero; F, H).

Figure 7. Observed (black bars) optimal atrioventricular (AV) delay that resulted in maximal increase in ∆LV dP/dtmax per group versus the vectorcardiogram (VCG) predicted optimal AV delay (gray bars; left panel). Observed (black bars) maximal increase in ∆LV dP/dtmax per group versus the increase in ∆LV dP/dtmax at the VCG predicted AV delay (gray bars; right panel). Error bars represent 95% CI.
Individual Prediction of Optimal Left Ventricular Stimulation Timing With Vectorcardiography

Prediction of the optimal AV delay for LV pacing is based on the assumption that a VAQRS halfway between its minimal (LV pacing with short AV delay) and maximal (intrinsic activation=LBBB) value reflects optimal collision of the activation waves originating from the right bundle branch and LV pacing electrode, thus, optimal electric ventricular resynchronization. Indeed, the present study shows that this “halfway value” of VAQRS accurately predicts the AV delay at which maximal hemodynamic response was achieved with LV pacing. Moreover, this prediction is accurate in the individual animal, independent of the presence of heart failure or myocardial infarction and despite the considerable variation in optimal AV delays (50–210 ms). The VCG prediction of optimal resynchronization also proved to be applicable to AV and VV interval optimization during BiV pacing; inability to achieve the halfway VAQRS during simultaneous BiV pacing (53% of cases) was associated with suboptimal hemodynamic response, which could be corrected by VV optimization. These data are consistent with data from our previous studies, showing that optimal AV and VV interval could accurately be predicted by MIVD,7 and that inability to achieve halfway values of MIVD by simultaneous BiV pacing was related to suboptimal hemodynamic response in patients.2

The implication of the good prediction of the VCG derived VAQRS for AV optimization is that the optimal hemodynamic response in CRT, at least in our canine models, seems to primarily depend on optimal electric resynchronization rather than on optimal preload (timing of filling of the ventricles).

Our VCG prediction model was less accurate for LV anterior pacing sites. A possible explanation is that with pacing at anterior sites the electrode is so close to the right ventricle that the vector hardly changes. Furthermore, the increase in ∆LV dP/dtmax at each AV delay was mostly minor and did not show any distinctive peak when using anterior LV pacing sites, making a prediction for an optimal AV delay less accurate. The observation that LV anterior pacing displays less improvement in systolic function is in accordance with a previous clinical study,10 and this site therefore is not recommended in CRT.
The Importance of Atrioventricular and Interventricular Optimization in Cardiac Resynchronization Therapy

The present study in canine LBBB hearts, as well as other studies in animals and patients, showed a significant acute beneficial hemodynamic effect of optimizing AV coupling.\(^2,11,12\)

This hemodynamic improvement appears to coincide with a more efficient myocardial contraction, as it occurs without an increase in myocardial oxygen consumption.\(^13\)

Our VCG derived algorithm for AV and VV optimization might be compared with already applied algorithms using electrophysiological data: SmartDelay and QuickOpt. Recent clinical studies showed disappointing results with respect to hemodynamic, echocardiographic, and clinical response.\(^14-16\)

The potential performance of these intracardiac electrogram based algorithms could be explained by noise in the outcome parameter and a small effect of AV optimization. An alternative explanation could be that these algorithms use predictions of optimal fusion, based on measurements without BIV pacing (intrinsic AV delay and QRS duration in case of SmartDelay, and the interval between intrinsic activation of RV and LV leads combined with the difference between RV pacing to LV sensing, and LV pacing to RV sensing in case of QuickOpt). Instead, in our approach, measurements of LBBB and the effect of LV pacing are combined to define the degree of resynchronization and, subsequently, this preferred resynchronization parameter is traced to achieve optimal pacemaker settings.

Potential Clinical Implications

Extrapolation of data from the experimental to the clinical situation should always be done with care. However, in an earlier publication of CRT patients from the the Pacing Therapies for Congestive Heart Failure (PATH-CHF) trial, it was demonstrated that MIVD can be used for AV delay optimization.\(^2\)

Because of the strong correlation between \(V_{AQRS}\) and MIVD, the results from the present animal study might well be applicable to the clinical situation.

Atrioventricular and VV interval optimization using VCG is relatively simple, can be performed fast, and might therefore be an attractive alternative to current optimization methods, such as echocardiography and invasive hemodynamic measurements. Only 2 measurements (LV pacing with short AV delay and LBBB) have to be applied, resulting in minimal and maximal values of the \(V_{AQRS}\) to estimate the optimal \(V_{AQRS}\) for AV and VV interval programming. Moreover, interpretation is less dependent on operator skills or technical limitations as with echocardiography.

Limitations

A limitation of this study is that we only investigated the frontal plane of the VCG. However, canine hearts are positioned almost cranio-caudally (vertically) in the thorax, resulting in a maximal vector predominantly in the frontal plane. The use of true 3-dimensional VCG in patients can be expected to result in equally reliable QRS vector analysis.

Furthermore, this study investigated the acute improvement in LV \(dP/dt_{max}\) without evaluation of the long term benefit. It is known that acute responses may not always predict chronic outcome, and optimal AV delay may differ as heart rate and cardiac loading conditions change (exercise, standing upwards, anatomic remodeling of the heart, administration of drugs).

Conclusions

In canine LBBB hearts, the \(V_{AQRS}\) in the frontal plane reflects electric interventricular dysynchrony. The value halfway between minimal (during LV pacing with short AV delay) and maximal (during intrinsic LBBB activation) \(V_{AQRS}\) predicts the AV and VV interval with optimal hemodynamic effect in CRT. Consequently, VCG could provide a reliable and easy new method for individual optimization of LV stimulation timing in CRT.

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9. Varma N, Jia P, Rady Y. Electrocardiographic imaging of patients with heart failure with left bundle branch block and response to...
Optimization of left ventricular (LV) stimulation timing in cardiac resynchronization therapy (CRT) usually is performed by echocardiography. However, this approach is time consuming, depends on operator skills, and has technical limitations. A rapid, reliable, and easy to interpret method for optimization of timing in CRT would be valuable. We hypothesized that the QRS vector of the vectorcardiogram would provide a reliable reflection of the sequence of electric activation. Consequently, a QRS vector halfway between that during LV pacing with a short atrioventricular (AV) delay and during left bundle branch block (LBBB) should reflect optimal collision of the wavefronts derived from the LV pacing site and the right bundle branch, thereby indicating the preferable setting for CRT. The present study investigated this idea in canine LBBB hearts. The results demonstrate that the QRS vector can be regarded as an index for electric interventricular dysynchrony, and that it serves as a reliable predictor of optimal LV stimulation timing in CRT, both during single site LV pacing and during (sequential) biventricular pacing. Measurements of the vectorcardiogram need to be performed during baseline LBBB and during LV pacing with a short AV delay. Subsequently, different pacemaker settings can be explored until the optimal QRS vector is achieved. Because the QRS vector can be easily, noninvasively, and accurately obtained, it is an attractive technique to use for routine optimization of AV delay and interventricular interval in CRT.
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