Optimization after CRT implant

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To maximize the effect of CRT

- candidates
- The position of LV lead
- Maximal and continuous correction of AV timing
- Maximal and continuous correction of LV conduction delay
### Recommendations for cardiac resynchronization therapy implantation in patients with heart failure

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRT is recommended for symptomatic patients with HF in sinus rhythm with a QRS duration $\geq 150$ msec and LBBB QRS morphology and with LVEF $\leq 35%$ despite OMT in order to improve symptoms and reduce morbidity and mortality.</td>
<td>I</td>
<td>A</td>
<td>261–272</td>
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<td>IIa</td>
<td>B</td>
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<td>CRT is recommended for symptomatic patients with HF in sinus rhythm with a QRS duration of 130–149 msec and LBBB QRS morphology and with LVEF $\leq 35%$ despite OMT in order to improve symptoms and reduce morbidity and mortality.</td>
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<td>IIb</td>
<td>B</td>
<td>266, 273</td>
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<td>CRT rather than RV pacing is recommended for patients with HFrEF regardless of NYHA class who have an indication for ventricular pacing and high degree AV block in order to reduce morbidity. This includes patients with AF (see Section 10.1).</td>
<td>I</td>
<td>A</td>
<td>274–277</td>
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<td>CRT should be considered for patients with LVEF $\leq 35%$ in NYHA Class III–IV$^4$ despite OMT in order to improve symptoms and reduce morbidity and mortality, if they are in AF and have a QRS duration $\geq 130$ msec provided a strategy to ensure bi-ventricular capture is in place or the patient is expected to return to sinus rhythm.</td>
<td>IIa</td>
<td>B</td>
<td>275, 278–281</td>
</tr>
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<td>Patients with HFrEF who have received a conventional pacemaker or an ICD and subsequently develop worsening HF despite OMT and who have a high proportion of RV pacing may be considered for upgrade to CRT. This does not apply to patients with stable HF.</td>
<td>IIb</td>
<td>B</td>
<td>282</td>
</tr>
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<td>CRT is contra-Indicated in patients with a QRS duration $&lt; 130$ msec.</td>
<td>III</td>
<td>A</td>
<td>266, 283–285</td>
</tr>
</tbody>
</table>

**AF** = atrial fibrillation; **AV** = atrio-ventricular; **CRT** = cardiac resynchronization therapy; **HF** = heart failure; **HFrEF** = heart failure with reduced ejection fraction; **ICD** = implantable cardioverter-defibrillator; **LBBB** = left bundle branch block; **LVEF** = left ventricular ejection fraction; **NYHA** = New York Heart Association; **OMT** = optimal medical therapy; **QRS** = Q, R and S waves (combination of three of the graphical deflections); **RV** = right ventricular.

$^a$Class of recommendation.

$^b$Level of evidence.

$^c$Reference(s) supporting recommendations.

$^d$Use judgement for patients with end-stage HF who might be managed conservatively rather than with treatments to improve symptoms or prognosis.
CS anatomy
Case

◆ M/72

◆ Severe LV dysfunction despite optimal medical therapy
◆ LBBB

◆ 17년 10월 타 병원에서 CRTD implantation, 다음날 CXR에서 LV lead dislodgement
  ● 본원으로 전원됨

◆ CS angiography
  ● Too small to implant
RV pacing ECG
Initial chest PA
Chest PA after implanting epicardial LV lead
Biventricular pacing
Continuous means on every cardiac cycle for the duration of the patient’s lifetime

◆ 100% BV pacing is difficult to achieve

◆ Transient interruptions to CRT are common
  ● Up to 36%

◆ Permanent loss of CRT occurs in up to 5% of patients within 2 years

◆ The causes are diverse
  ● AF with rapid AV conduction
    ● At least 18% of all therapy interruptions
Pacing event counters underestimate

- the true magnitude of the negative effect of AF with native AV conduction

- Despite pacing event counters showing >90% BV pacing
  - 24hr Holter monitoring
    - Only 47% of pts with permanent AF had effective BV pacing (>90% BV pacing with complete V capture)
      - Ineffective (pseudofusion)
      - Incompletely effective BV pacing
Ventricular pacing must be continuous

◆ AF
  ● Beta blockers
  ● digoxin
  ● Amiodarone
  ● AV node ablation
  ● AF ablation

◆ VPCs
  ● Beta blockers
  ● Catheter ablation
Programming considerations for CRT

◆ Pacing modes
  ● Ventricular pacing must be continuous

◆ Optimal AV resynchronization

◆ Optimal ventricular resynchronization
AV optimization

◆ Echocardiography-based optimization

● Ritter or iterative methods
● Maximizing stroke volume (or volume-time integral at aortic valve)

◆ Intracardiac electrogram-based optimization

● QuickOpt (by Abbott)
● SmartDelay (by Boston Scientific)
● AdaptiveCRT (by Medtronic)

◆ Regardless of the method, 80 to 110ms
Effect of AV Delay on LV Diastolic Filling Pattern

Short AV Delay 50 ms
- A-wave truncated
- Less time for filling
- Atrial contraction against a closed Mitral valve

Long AV Delay 280 ms
- Fused A and E wave
- Less time for filling
- Pre-systolic Mitral regurgitation

Optimized AV Delay 200 ms
- Max diastolic filling time
- Mitral closure occurs at end of A-wave
Consequences of loss of AV synchrony

**Timing of AV delay influences diastolic filling**

**Too Short**
- Sub optimal filling
- A-wave is truncated by the start of ventricular systole
- Atrial contraction occurs against a closed Mitral valve with subsequent reversal of flow in the pulmonary veins

**Too Long**
- Pre-systolic Mitral regurgitation
- Fusion of E and A wave, decreasing diastolic filling time
Quick, simple approach to AV optimization

◆ A-wave is truncated, increase SAV or PAV

◆ E and A waves are fused, decrease SAV or PAV
Truncated A wave
Fused E and A waves
Optimized AV-delay
Iterative Method of AV-Optimization
Ritter Method

\[
240 + 86 = 326 \quad (A)
\]

SAV Long \quad QA Long

\[
120 + 125 = 245 \quad (B)
\]

SAV Short + QA Short

Difference (A-B)= 81 (C)

SAV short= 120 (D)

Optimal AV delay (C+D)= 201
Optimal ventricular resynchronization

- Inter & intraventricular conduction delay
- Minimization of ventricular electromechanical asynchrony
- Improvement of pump function
Stroke volume / VTI

The volume of blood ejected by the LV each beat =

**Stroke Volume** = LVOT area \( \times \) **Velocity Time Integral (VTI)**

Since LVOT is a constant, the larger VTI the larger stroke volume.

Echo: maximizing stroke volume
Optimizing VV Delay Based on Aortic VTI

- Obtain Pulsed Wave Doppler of LVOT with several different sequential ventricular paced intervals
- Select the setting yielding the largest VTI as the optimal paced interval
**VV optimization**

- **ECG-based optimization**
  - The narrowest paced QRS duration

- **Echocardiography-based optimization**
  - Minimizing LV dyssynchrony
  - Maximizing stroke volume

- **Intracardiac electrogram-based optimization**
  - QuickOpt (by Abbott)
  - LV pacing alone (AdaptiveCRT by Medtronic)
Echo: minimizing LV dyssynchrony

◆ M-mode or color M-mode
  ● Septal-to-posterior wall motion delay

◆ Color tissue Doppler (tissue synchronization imaging)
  ● Time-tissue velocity curve → time from QRS onset to Vmax (Ts)

◆ Strain
  ● Longitudinal/radial strain
  ● Global strain by automated functional imaging (AFI)

◆ Real time 3D echocardiography
  ● Time-volume curve → systolic dyssynchrony index (SDI)
Echo: maximizing stroke volume

- **Doppler**
  - TVI at aortic valve
  - $dP/dt$ of MR jet

- **Real time 3D echocardiography**
  - Ejection fraction
Interventricular timing considerations

- Fusion of ventricular activation wavefront is easily achieved and confirmed in animal models

- Not the case in the diseased, failing human ventricle
  - Conduction times of activation wavefronts initiated on opposite sides of the LV are difficult to measure
  - Timing between stimulation sites does not accurately reflect global activation
  - A readily available measure of global activation for fusion evidence does not exist
  - Propagation of paced activation wavefronts is unpredictably modified by conduction blocks and other factors
    - Fiber orientation, capture latency, etc
◆ Sequential ventricular activation during LBBB
  ● RV → LV
  ● Posterolateral LV is latest activated

◆ The location, orientation, and extent of conduction blocks display
  ● Significant interpatient variability, yielding distinct patterns of LV epicardial activation during similar activation patterns registered on the surface ECG
Conduction block

◆ Fixed or functional

◆ Fixed
  ● Due to ventricular scar

◆ Functional blocks are more difficult to understand
  ● Absence of scar
  ● Ability to shift the location of the line of block with pacing maneuvers

◆ LV pacing usually targets the posterolateral LV (latest activated region)
  ● Conduction block usually reside between BV pacing sites
Noninvasive ECG imaging of epicardial isochronal activation

A. Patient #5

Native Rhythm
QRSD: 180 ms
(Esyn: -113 ms)

Biventricular Pacing
QRSD: 150 ms
(Esyn: 20 ms)

LV stimulation (asterisk) from posterobasal free wall ended locally at basal free wall 110 ms after pacing.

Anterior line of fixed block present during SR and LV pacing. Latest activation occurred at anterior LV adjacent to line of block (138 ms).

B. Patient #3

Native Rhythm
QRSD: 180 ms
(Esyn: -93 ms)

Biventricular Pacing
QRSD: 120 ms
(Esyn: -45 ms)

LV stimulation from anterior LV.

Activation pivots around antero-lateral line of functional block and ends at posterobasal LV 117 ms later.
To maximally reduce LV conduction delay

- LV stimulation must occur distal to the line of conduction block
- LV paced activation wavefront must propagate out from the stimulation site
- R-L or L-R activation wavefront must be properly timed to generate fusion
Implication of conduction blocks during BV pacing

◆ The posterolateral is almost always the latest activated site during LBBB and should be targeted for LV pacing
◆ Other unpredictable sites (anterior wall, apex) may be latest activated in some patients

◆ Any pacing site may induce or worsen conduction block in an unpredictable manner

◆ Resynchronization of the most delayed LV region may be irrelevant if there is a large intervening zone of scar

◆ Selection of LV pacing sites based on interventricular timing recorded by local EGMs (RV, LV) may be misleading
  ● Simultaneous activation timing at widely spaced RV and LV sites does not indicate absence of mechanical delay
◆ Equivalent conduction times between widely spaced RV and LV stimulation sites do not guarantee that simultaneous BV pacing will generate global ventricular activation wavefront fusion
Sequential ventricular timing

- Separately programmable RV and LV stimulation outputs and circuitry
  - To permit timing delay between outputs by stimulation site (VV timing)

- Sequential vs simultaneous BV pacing
  - No such benefit has been recorded in randomized clinical trials
Reference

1. Cardiac electrophysiology from cell to bedside, 6th edition
2. 2016 ESC guidelines
THANK YOU FOR YOUR ATTENTION