CRT Non Responders (What is wrong?)

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Introduction

• CRT is non pharmacological treatment of drug refractory Heart failure, NYHA class II – IV with prolonged QRS duration and depressed ejection fraction.

• Clinical trials shows that CRT reduce heart failure hospitalization, improve quality of life, reverse LV remodeling and decrease mortality.
However
30% of patients are non responder.

Responders

- **Subjective** (clinical):
  - improved NYHA class
  - improved 6 minute walk test
  - >10% quality of life

- **Objective**
  - **Acute**: Hemodynamic parameters (CO, LV Dp/Dt max)
  - **Chronic**: LV Reverse remodeling
  - increase EF 10 %
  - decrease LVESV 15 %
Outcome measures

- Reduction in heart failure hospitalization
- Reduction in all cause mortality and morbidity

Non Responders

Unchanged or worsening of

- Clinical
- Echocardiographic parameters
- Unprovoked worsening of heart failure
Non responders

What is wrong?

- Pre – Implantation.
- During implantation.
- Post implantation.
Pre Implantation

Pre implantation

- Patient selection: (MADIT CRT, REVERSE, COMPANION)
  - 1- female gender respond more
  - 2- Non ischemic cardiomyopathy respond more.
  - 3- Associated co morbidities (renal, hepatic)
  - 4-ECG
    - LBBB respond more than RBBB and IVCD
    - Wider QRS >150 msec respond
Echo parameters:
- marked LV dilatation (LVEDV > 240 ml)
- Poor EF less than 20%
- severe MR
- Absent dysnchrony by different echo parameters.
- Posterolateral scar (better by MRI)

During Implantation
- LV lead position
- Multisite pacing
LV lead positioning

- Conventional site:
  - Posterolateral vein
  - Basal (not apical)

Figure 1: Schematic representation of coronary sinus anatomy.
Conventional Vs targeted lead position

- **Mechanical targeting** (latest mechanical Activation)

- **Electrical activation** (QLV)
Mechanical targeting

• Targeting The latest area of mechanical activation

  Target Study
  • Targeted LV lead has more favorable reverse remodeling than conventional positioning

  STARTER Study
  • Showed reduction in heart failure hospitalization with targeted LV lead
Electrical Targeting

- Comprehensive mapping of electrical activation in patients with LBBB shows significant heterogeneity in the location of line of functional block.

- QLV: measures electrical activation between QRS onset on surface ECG to local activation of LV lead.
Multi site Pacing

- Multisite stimulation has emerged as a way of potentially overcoming non-response.

  - This may be achieved by the use of multiple leads placed within the coronary sinus and its tributaries (dual-vein pacing)
  - or more recently by the use of multi-polar (quadripolar) left ventricular pacing leads which can deliver pacing
Multiple Leads

- The concept of MSP using multiple leads is based on the hypothesis that pacing at multiple points within the ventricles will improve cardiac resynchronization.

- Two different pacing modalities have been proposed using multiple leads:
  - the first using two RV leads and one LV lead,
  - the second using one RV lead and two LV leads inserted in the two separate tributaries of the CS.
Quadripolar leads

- Quadripolar leads offer evident advantages by offering more pacing configurations (up to 17 vs. up to 6).

- which may alleviate the problem of high thresholds, avoid PNS, or at least offer an adequate safety margin (LV vs. phrenic nerve capture)
Post Implantation

- **I-** Non cardiac

- **II-** Cardiac (Non Device)

- **III-** Cardiac (Device related)
**I- Non cardiac**

- Aneamia
- Thyroid disorders
- Sleep Apnea
- Drug toxicity
- Renal problems
- Depression
- Pulmonary disease

**II - Cardiac ( non device )**

- Residual ischemia
- RT sided heart failure
- Volume status
- Arrhythmia ( PVCs, VT, AF )
- Non Optimized medical therapy
Arrhythmia and non responders

Atrial fibrillation

- Attacks of AF with rapid ventricular response lead to decrease BIV Pacing percentage and Loss of atrial kick.

Management

- Rhythm control: Amiodarone
- Rate control: digitalis, BB
- Triggering Mode
- AVN Ablation
Ventricular tachycardia

- Scar related VT
- BBRVT (bundle branch re-entry VT)

Management

- Medical therapy: Amiodarone
- Upgrade to CRT-D
- Ablation (Right bundle ablation with BBRVT
  And ablation for scar related VT)

High PVC burden > 10,000 /24h

Impair BIV pacing

- Check first atrial undersensing
- Medical therapy: Amiodarone
- Ablation
III- Cardiac (Device related)

- AV interval Optimization
- V-V optimization
- BIV pacing less than 99%
  - Fusion and pseudo fusion
  - Atrial undersensing (high PVC burden)
  - Arrhythmia
  - Lead malcapture
  - Anodal stimulation

AV interval optimization

- Default AV Interval (110-120 msec)

- To allow BIV pacing before intrinsic AV conduction occur

- BUT this could lead to ???
Short AV interval
lead to premature valve closure
Truncation of A wave

The AV delay is too short. The atria do not fully contribute to ventricular filling because of early closing of MV. The A wave is truncated when measuring the mitral inflow by pulsed wave doppler.

The AV delay is adequate. No truncation of A wave and E and A wave are separated.

The AV delay is too long. MV remains open during ventricular contraction and pre-systolic mitral regurgitation is present.

Lengthen AV interval?

- Loss of CRT BIV pacing due to intrinsic AV conduction and possible fusion.
- Diastolic MR
The AV delay is too short. The atria do not fully contribute to ventricular filling because of early closing of MV. The A wave is truncated when measuring the mitral inflow by pulsed wave doppler.

The AV delay is adequate. No truncation of A wave and E and A wave are separated.

The AV delay is too long. MV remains open during ventricular systole and pre-systolic mitral regurgitation is present.

Merged E and A
AV too long

AV optimisation

Truncated A wave
AV too short
Iterative method

- Must be done using ECG guided to verify that Optimum AV interval not Lead to intrinsic AV conduction or Fusion.

- Give drug lead to AVN delay
V- V Optimization

- Default
  Simultaneous LV – RV pacing

- LV Latency: time between LV lead discharge and actual LV capture.

- SO ,, pre excite LV by LV Latency time to allow simultaneous LV – RV capture

Echo V- V Optimization

- Echo base LVOT VTI measurements

- M mode and doppler method
Fusion and Pseudofusion

**Fusion**: ventricle being activated by spontaneous activation and paced impulse

**Pseudofusion**: superimposition of inefficient pacemaker spike on spontaneous QRS
Fusion
Pseudofusion

Fusion and pseudofusion:

• Give false impression of Biv Pacing more than 99% during programming

• So holter study is recommended and meticulous examination of QRS morphology is warranted
Anodal stimulation

For LV pacing in CRT, if the configuration is LVTip to RV ring there is possibility of anodal capture at the RV end (This configuration is sometimes necessary to avoid phrenic nerve capture)

This leads to three wavefronts starting on the ventricles
1. From RV tip CATHODAL, RV Paced
2. From LV tip CATHODAL, LV Paced
3. From RV ring ANODAL, LV Paced

RV ring anodal capture from RV pacing does not happen practically

There is an ongoing debate as to whether this leads to suboptimal CRT response
Role of holter study

- Detect fusion and pseudofusion
- Detect burden of PVCs
- Detect arrhythmia burden
Conclusion

- Problem of Non Responders
  - Could be preventable
  - Could be manageable
  - So choose well, and operate well, and Optimize well

Thank you