Results

Baseline characteristics:

One hundred and seventy patients were included from Mar. 2011 to September 2011 (mean age 68.8 ± 9.7 years, 38 females and 132 males). Ninety-one patients had non-ischemic cardiomyopathy (NICM) and 79 patients had ischemic cardiomyopathy (ICM), of whom 43 patients had previous myocardial infarction. Most patients with ICM underwent previous revascularization (60 patients underwent previous revascularization). Table 3.1, 3.2 and 3.3 summarize the baseline data of our patients.

Baseline condition	Mean/n(%)
Age, years	68.8±9.7
Male/female ,n	132/38
NYHA II ,n	23 (13.5%)
NYHA III ,n	136 (80%)
Ambulatory NYHA IV ,n	11 (6.5%)
6 min walk distance, m	275±105
NICM, n	91(53.5%)
ICM, n	79 (46.5%)
Previous MI, n	43 (25.3%)
Previous revascularization, n	60 (35.3%)
BMI , Kg/m^2	29.2±5.1
Use of medications (% of patients)	
B blockers	94%
ACEi/ARB	95%
MRA[a]	64%
Diuretics	92%
Statins	69%
Digoxin	41%
Antiarrhythmic drugs, %	29%
Comorbidities	
Hypertension , n	139 (81.7%)
DM, n	86 (50.5%)
COPD[b], n	44(25.8%)
BMI>30, n	55 (32.3%)
History of renal disease, n	56 (32.9%)
Anemia[c], n	102 (60%)
S. Creatinine, mg/dl	1.2 ± 0.4
Blood urea nitrogen, mg/dl	9.5±6.4
eGFR, $ml/min/1.73m^2$	53.7±10.3
Hb level, gm/dl	9.9±7.1

Blood Glucose, mg/dl	136±59

Table 3.1: Baseline characteristics; clinical data

[a]MRA: Mineralocorticoid Receptor Antagonists [b] COPD: Chronic obstructive pulmonary disease (based on specialized respiratory assessment and use of specific medications). [c]Anemia (defined as hemoglobin (Hb) level < 11g/dl).

Baseline ECG	**Mean/n(%)**
QRS duration	
Mean , msec	145±25
≥120 msec	145 (85.3%)
≥150 msec	89 (52.3%)
≤120 msec	25 (14.7%)
QRS morphology	
LBBB	94 (55.3%)
RBBB	4 (2.3%)
IVCD[a]	47 (27.6)
Normal	25 (14.7%)
Rhythm	
History of AF	51(30%)
SR at CRT implantation	147 (86.5%)
AF at CRT implantation[b]	32 (13.5%)

Table 3.2: Baseline characteristics; ECG

[a]IVCD: Intraventricular conduction delay. [b]AV nodal ablation was done in 8 patients

Baseline echocardiography	**Mean/n(%)**
LV dimensions, volumes and EF	
LVIDd (mm)	67.58±9.2
LVIDs (mm)	55.71±10.3
LVEDV (ml)	205.72±66.4
LVESV (ml)	148.30±56
LVEF %	28.31±7.2%
Mitral regurgitation	
No/mild	114 (67%)
Moderate	41(24%)
Severe	15 (9%)
Diastolic dysfunction	
No/grade I	51 (35%)
Grade II	53 (36%)
Grade III	43 (29%)
Right ventricle	
TAPSE (mm)	18.16±3.8
RVd (mm)	34.15±5.9
No PHT	51 (30%)

PAP 35-50 mmhg	76 (44.7%)
PAP >50 mmhg	43 (25.3%)
Markers of dyssynchrony	
SPWMD (msec)	120.73±63.3
FT/RR %	37.64±8.3%
LVPEI (msec)	128.50±30.4
IVD (msec)	39.28±25.9
Ts- sep-lat (msec)	63.22±34.7

Table 3.3: Baseline characteristics; echocardiography

CRT implantation Procedure:

CRT-P was implanted in 65 patients and CRT-D was implanted in 105 patients. The right atrial lead was inserted in the right atrial appendage in all cases. The right ventricular lead was inserted at the right ventricular apex in all cases. The left ventricular lead position was assessed in LAO and RAO views to assess its short axis and long axis position (figure 3.1). The position of the LV lead was based on the anatomy of the coronary sinus. The lateral, posterolateral and posterior position were considered optimal short axis positions. long axis positions other than apical were considered optimal. The LV lead position was considered to be optimal when it is optimal in both long axis and short axis (table 3.4).

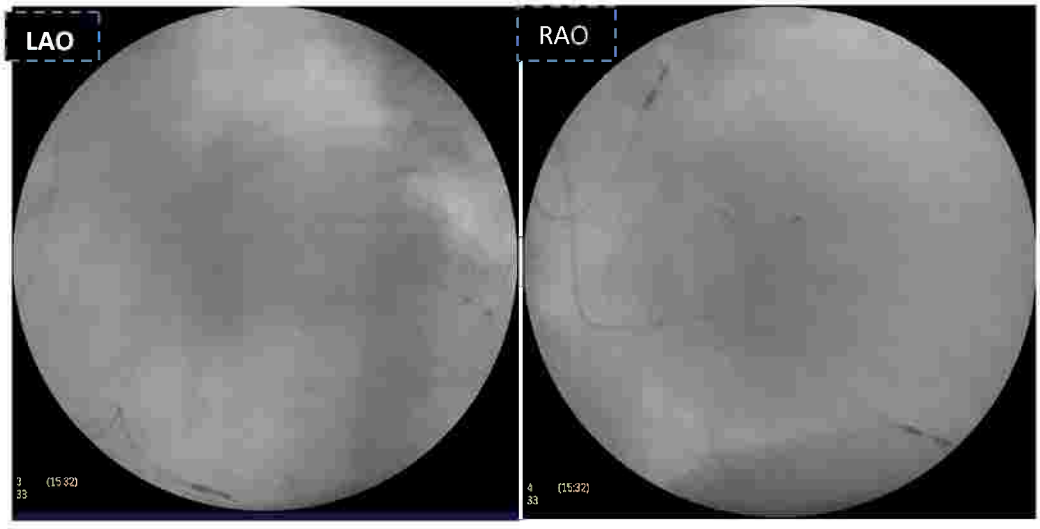


Figure 3.1: Example LV lead position in basal lateral wall.

LV lead short axis position	n (%)
Lateral	52 (30.6%)
Posterolateral	69 (40.5)

Posterior	39 (22.9)
Anterolateral	7 (4%)
Anterior	3 (1.8%)
Optimal short axis position	160 (94.1%)
LV lead long axis position	
Basal	17 (10%)
midwall	144 (84.7%)
Apical	9 (5.3%)
Optimal long axis position	161(94.7%)
Optimal LV lead position	153 (90%)

Table 3.4: LV lead position

Device optimization using device based automated algorithms was performed at the same implantation day in 57.6% of patients. Biventricular pacing percent was 96±7% at 1 day post-implantation and was 95±7% at 6 months post-implantation. Device optimization was done again at 6 months for the nonresponders.

CRT response:

Among the study population, 114 patients (67.1%) responded to CRT implantation (defined as improvement of NYHA functional class by ≥ 1 NYHA class, increased 6.MWD ≥ 10% and reduction of LVESV ≥ 15% at 6 months after CRT implantation), 56 patients didn't respond. Improvement of NYHA class was maintained 1 year after CRT implantation. Mortalities and hospitalization related to unprovoked worsening of heart failure during the 1st 6 months postimplantation was considered CRT-nonresponse.

At 6 months follow-up, CRT implantation was associated with significant improvement in LV dimensions, functions and markers of LV dyssynchrony. CRT implantation was associated with improvement in NYHA class and 6 min walk distance. CRT was also associated with significant improvement in LV diastolic dysfunction and the grade of mitral regurgitation. There were no significant effects on renal function and RV diastolic dimension and function. Tables 3.5 and 3.6 summarize different effects of CRT implantation.

Parameter	Baseline (n=170)	6 months (n=170)	P
LVIDd (mm)	67.91±8.7	64.95±8.9	0.001

LVIDs (mm)	57.02±8.8	52.42±9.9	0.0001
LVEDV (ml)	208.39±69.1	185.46±61.3	0.0001
LVESV (ml)	150.50±58.6	121.57±55.4	0.0001
LVEF %	28.38±7.2%	35.46±9.3%	0.0001
Grade of Mitral regurgitation	1.46±0.7	1.21±0.7	0.032
Grade of diastolic dysfunction	1.89±0.8	1.54±0.8	0.026
TAPSE (mm)	18.27±4.2	18.55±4.3	0.535
RVd (mm)	33.77±4.8	34.55±4.7	0.118
Grade of tricuspid regurgitation	1.07±0.7	1.07±0.7	1.0
SPWMD (msec)	144±86.4	31.33±43.7	0.0001
FT/RR %	35.71±9.1%	44.71±5.6%	0.004
LVPEI (msec)	124.79±20.1	110.57±33.2	0.059
IVD (msec)	23.83±26.6	15.83±13.3	0.375
Ts- sep-lat (msec)	67.69±36.6	35.38±19.4	0.007

Table 3.5: Effects of CRT; echocardiography

Parameter	Baseline n=170	6 months n=170	P
NYHA class	2.91±0.4	2.18±0.6	0.0001
6 min walk distance (m)	275±105	414±111	0.001
Creatinine mg/dl	1.27±0.4	1.3±0.6	0.245
B urea Nitrogen mg/dl	10.7±7.8	9.8±4.8	0.318
eGFR ml/min/1.73m^2	53±10.6	52.3±13.1	0.589

Table 3.6: Effect of CRT on clinical status and renal function

Clinical events:

There were 27 heart failure related hospitalizations. The rehospitalization rate was significantly higher among nonresponders compared to the responders (19 (33.9%) vs 8 (7%), P= 0.00001). There were also 15 heart failure related mortalities (11 (19.6%) nonresponder vs 4(3.5%) responders, P=0.0001).

Differences between responders and nonresponders:

Compared to nonresponders, responders had significantly wider baseline QRS duration, lower BMI, lower baseline serum creatinine level, smaller baseline RV diastolic dimension and greater TAPSE value. There was a significant gender difference between responders and nonresponders. Responders were less likely to have PAP>50 mmhg at baseline, less likely to have grade III diastolic dysfunction, more likely to have NICM, more likely to have LBBB morphology in ECG, less likely to have AF at implantation time, less likely to have history of renal disease and less likely to have COPD than nonresponders.

There were no baseline differences in the presence and severity of mitral regurgitation. Baseline NYHA functional class was not different between responders and nonresponders. There were no significant differences in age, BUN, eGFR, blood glucose, hemoglobin level, BiV pacing percent, baseline left ventricular dimensions, volumes, ejection fraction and the measured echocardiographic parameters of LV dyssynchrony between responders and nonresponders. Table 3.7, 3.8 summarize the baseline differences between responders and nonresponders.

Parameter	**Responder n = 114 Mean**	**Nonresponder n= 56 Mean**	**P**
Age (years)	68.8±10.3	68.9±8.2	0.9
BMI (kg/m^2)	28.6±4.7	30.4±5.5	0.04
Creatinine (mg/dl)	1.17±0.4	1.35±0.4	0.007
B. urea nitrogen (mg/dl)	9.2±6.5	10.2±6.2	0.3
eGFR (ml/min/1.73m^2)	54.5±9.6	52.4±11.5	0.25
Bl. Glucose (mg/dl)	131±48.	149±62	0.07
Hb level (gm/dl)	10.3±8.7	9.3±1.3	0.2
QRS duration (msec)	152±24.9	130.7±24	0.0001
BiV pacing%	95±4	94±4	0.7
LVIDd ;mm	67.7±8.9	67.3±9.8	0.8
LVIDs ; mm	55.4±10.2	56.3±10.6	0.6
LVEDV ; ml	205.7±66.3	205.6±67.5	0.9
LVESV; ml	147.7±54.3	149.5±60.3	0.8
LVEF %	28.3±6.7%	28.2±8.2%	0.9
SPWMD; msec	126.2±66	110±56.9	0.09
TAPSE; mm	18.9±3.7	16.6±3.5	0.001
RVd; mm	33±6.3	36.3±4.3	0.001
FT/RR %	37.2±7.9%	38.4±9.2%	0.4
LVPEI; msec	131.9±31.2	122±28.2	0.4
IVD; msec	40.6±26.8	36.3±23.6	0.3

Ts-sep-lat; msec	66±32.1	57.2±39.7	0.1

Table 3.6: Responders vs. nonresponders baseline characteristics

Parameter	Responders n= 114	Nonresponders n=56	P value
Females	33 (28.9%)	5(8.9%)	0.002
NICM	73 (64%)	18 (32.1%)	0.0001
LBBB	78 (68.4%)	16 (28.6%)	0.00001
AF at implantation	8 (7%)	15 (26.8%)	0.001
History of renal disease	26 (22.8%)	30 (53.6%)	0.0001
COPD	21 (18.4%)	23 (41.1%)	0.002
Grade III diastolic dysfunction	17 (14.9%)	16 (28.6%)	0.034
PAP>50 mmhg	18 (15.8%)	17 (30.4%)	0.027
HF hospitalizations	19 (33.9%)	8 (7%)	0.00001
HF mortalities	11 (19.6%)	4 (3.5)	0.0001

Table 3.7: Responders vs. nonresponders baseline characteristisc and events.

Predictors of CRT response:

Gender:

Females have been more likely to respond to CRT than males (84% vs. 61% respectively, P=0.003). This difference was not significant in multinominal regression analysis (OR 3.3; 95% CI 0.8-13.2, P= 0.09).

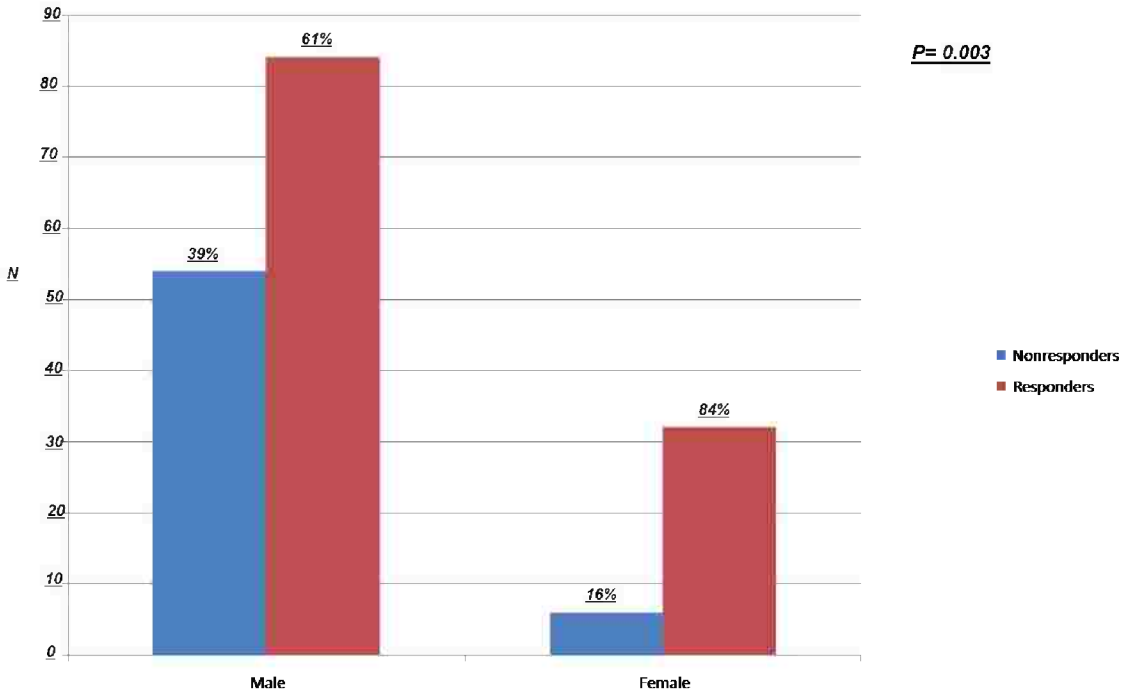


Figure 3.6: Gender difference in response

Baseline NYHA class and response:

Patients with NYHA class II and III at baseline did well after CRT implantation (response rate 69.6 and 68.4 respectively). The response rate in patients with baseline ambulatory NYHA IV was worse than other patients (45.5% response rate), but was not statistically significant due to small number of patients in NYHA IV included (11 patients).

Baseline NYHA	Nonresponders n=56	Responders n= 114	P
NYHA II	7 (30.4%)	16 (69.6%)	0.278
NYHA III	43 (31.6%)	93 (68.4%)	
NYHA VI	6 (54.5%)	5 (45.5%)	

Table 3.9: Baseline NYHA and response

Comorbid conditions:

The presence of lung disease in the form of significant chronic obstructive pulmonary disease (COPD) was an independent predictor of nonresponse to CRT (OR 0.26; 95%CI 0.08-0.8, P=0.022). The CRT response rate in patients with COPD was 47.7% vs. 73.8% response rate in patient without COPD, P= 0.002. History of renal disease was also an independent predictor of nonresponse (OR 0.14; 95% CI 0.04-0.44, P= 0.001). The responders had significantly lower baseline serum creatinine level. However there were no significant differences in CRT response between patients with eGFR above or below 60 ml/min/1.73m^2. There was a non-significant trend that non-hypertensive respond to CRT better than hypertensive and non-diabetic patients respond to CRT better than diabetic patients. The CRT response rate didn't differ significantly between obese and non-obese patients.

Comorbidity		**Nonresponders n=56**	**Responders n= 114**	**P**
Lung disease	no COPD	33 (26.2%)	93 (73.8%)	0.002
	COPD	23 (52.3%)	21 (47.7%)	
Renal disease	no history of renal disease	26 (22.8%)	88 (77.2%)	0.0001
	history of renal disease	30 (53.6%)	26 (46.4%)	
eGFR	≥ 60 ml/min/1.73m^2	25(29.4%)	60 (70.6%)	0.09
	< 60 ml/min/1.73m^2	31(36%)	54(64%)	
Hypertension	No HTN	6 (19.4%)	25 (80.6%)	0.07
	HTN	50 (36.0%)	89 (64.0%)	
Diabetes	Non-diabetic	22 (26.2%)	62 (73.8%)	0.06
	Diabetic	34 (39.5%)	52 (60.5%)	
Obesity	BMI <30	32 (30.7%)	71 (69.3%)	0.378
	BMI ≥30	23 (34.4%)	44 (65.6%)	

Table 3.10: Co-morbid conditions and CRT response

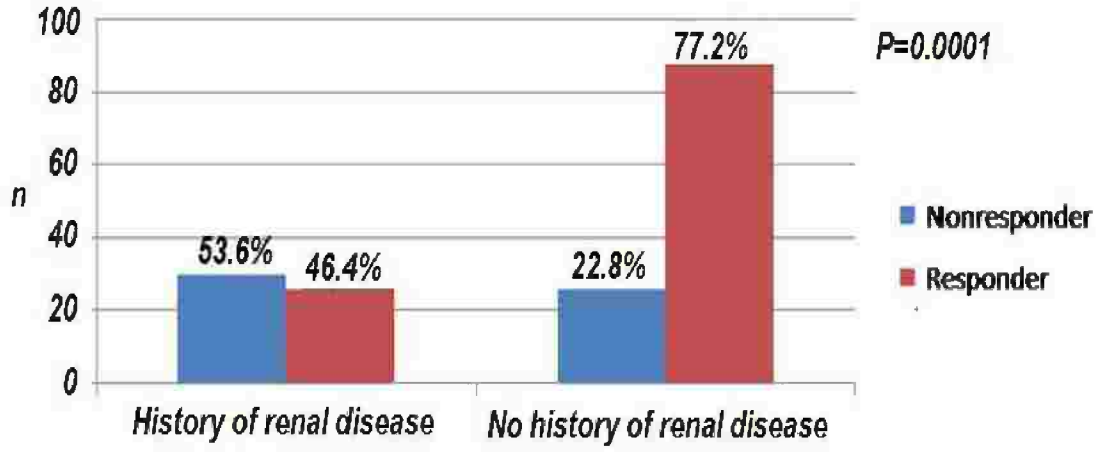


Figure 3.3: Renal disease and CRT response

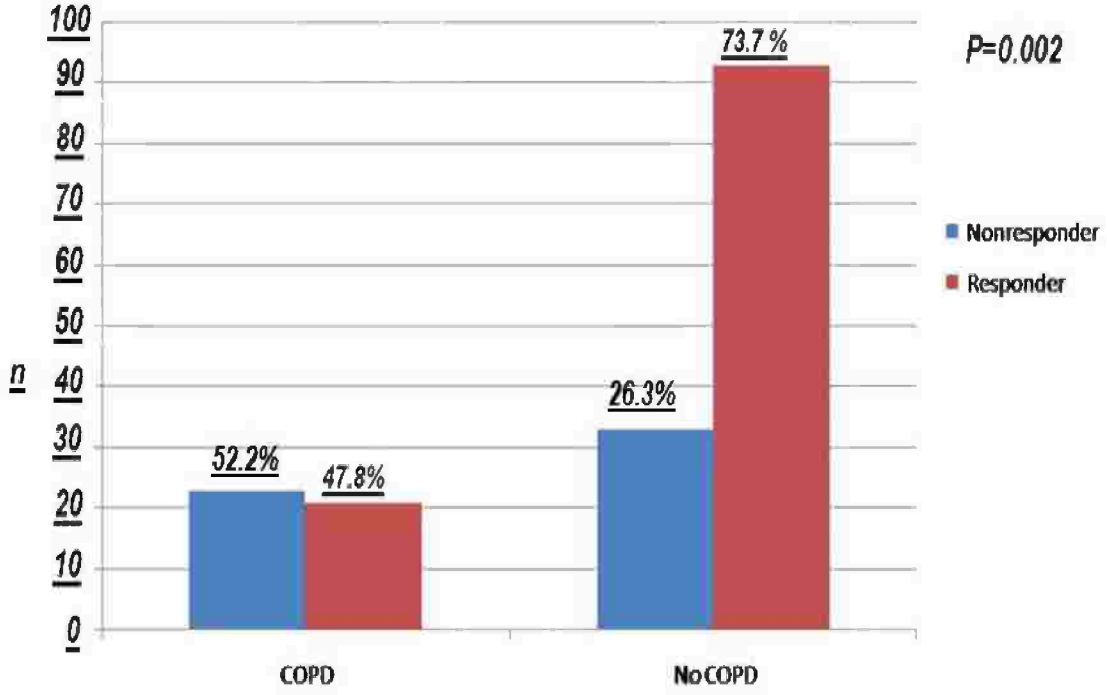


Figure 3.4: COPD and CRT response

ECG:

Patients LBBB were more likely to respond to CRT than patients without LBBB (83% vs. 47.4%, P=0.0001). However, LBBB morphology was not an independent predictor of response in multinominal regression (OR 2; 95% CI 0.75-5.4, P = 0.16).

QRS duration (QRSd) ≥ 150 msec was the strongest independent pre-implantation predictor of response (OR 9; 95% CI 3.1-27.8, P< 0.001). CRT response was documented in 84.5% of patients with baseline QRSd≥ 150 msec vs. only 50% of those with QRSd < 150 msec, P<0.001. There was no significant difference between patients with 150> QRSd ≥ 120 msec and those with QRSd < 120 msec as regards CRT response (52.5% vs. 44% respectively, P= 0.4). AF at time of CRT implantation was an independent predictor of nonresponse (OR 0.17; 95% CI 0.04-0.66, P=0.01).

The CRT response rate was much better in patients with SR during CRT implantation than patients with AF (72.1% vs. 34.8% respectively, P <0.0001. There was a significant difference in BiV pacing % between Patients with AF and patients with sinus rhythm at 1day postimplantation (91.2 ± 15 % vs. 96.9 + 5.7% respectively, P= 0.001) and at 3 months postimplantation (92.4 + 10 % vs. 96.4 + 6.8 respectively, P = 0.01).

ECG		**Nonresponders n= 56**	**Responders n= 114**	**P**
QRS morphology	No LBBB	40 (52.6%)	36 (47.4%)	0.0001
	LBBB	16 (17.0%)	78 (83.0%)	
QRSd msec	QRS <120	14 (56.0%)	11 (44.0%)	0.4
	120≤QRS<150	29 (47.5%)	32 (52.5%)	
QRSd msec	QRS≥150	13 (15.5%)	71 (84.5%)	0.0001
	QRS<150	43 (50%)	43 (50%)	
IO rhythm	SR	41 (27.9%)	106 (72.1%)	0.0001
	AF	15 (65.2%)	8 (34.8%)	

Table 3.11: ECG and response

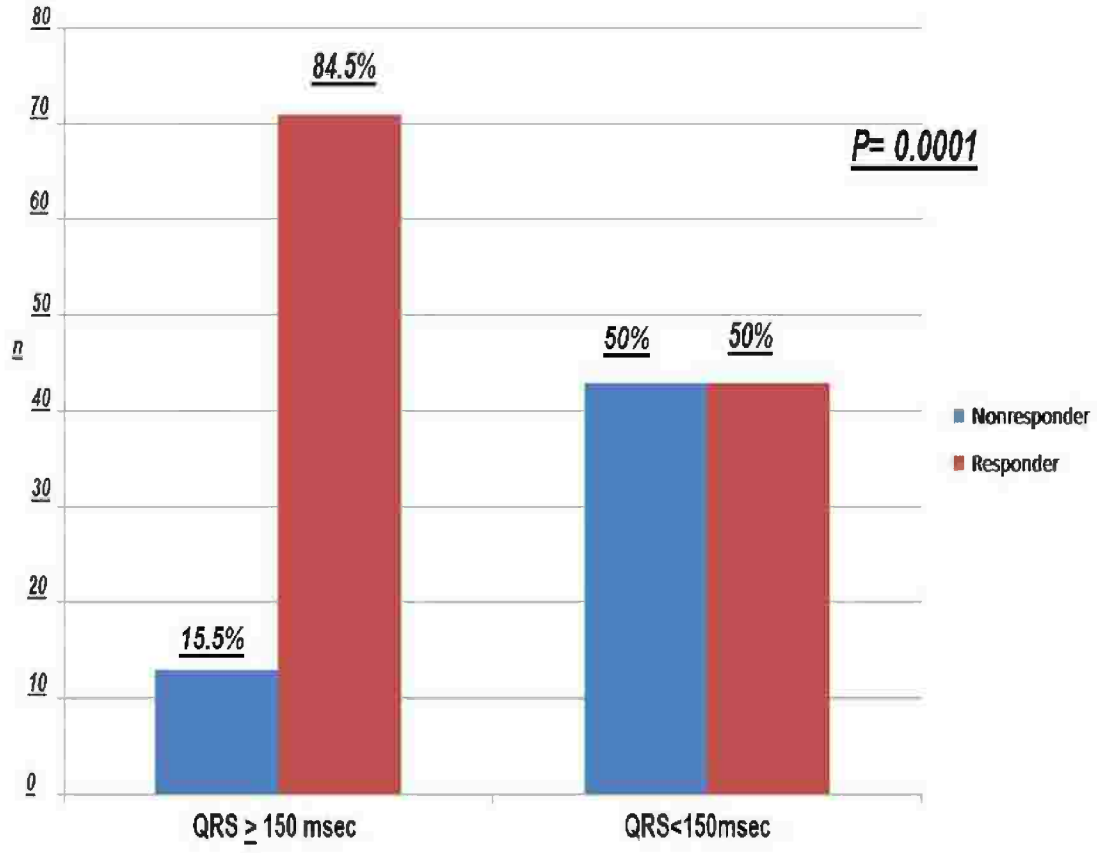


Figure 3.5: QRS duration and response

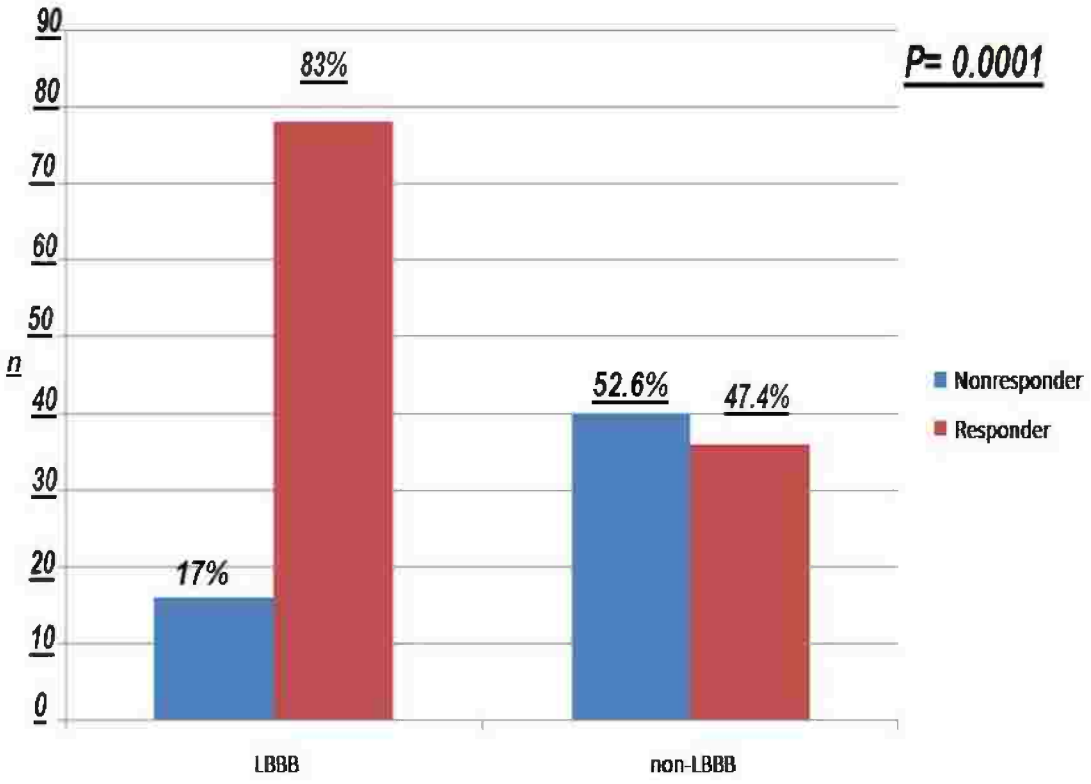


Figure 3.6:QRS morphology and response

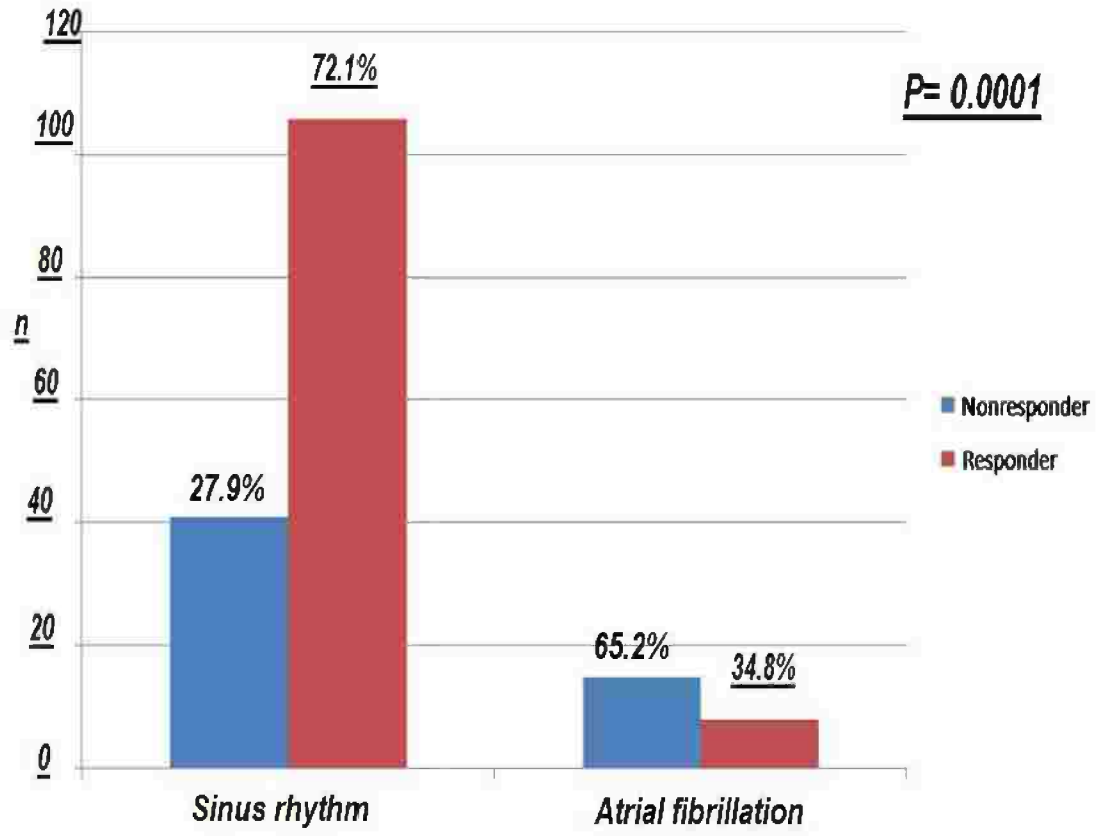


Figure 3.7: Rhythm at CRT implantation and CRT response

Echocardiography:

The presence of RV dysfunction defined as TAPSE <15 mm was an independent predictor of nonresponse in multinominal logistic regression (OR 3.4; 95% CI 1.1-10.4, P=0.036). Patients with baseline TAPSE≥15 mm had better response rate than those with baseline TAPSE < 15 mm (76.1% vs. 50.8% respectively, P=0.001).

Baseline RV dilatation was associated with CRT nonresponse; 57.1% of patients with dilated RV (RVd>35mm) were responders vs. 75.3% of those with RVd≤35mm, P=0.012. Presence of more than mild pulmonary hypertension was associated with reduced response rate to CRT. The response rate was 78%, 71% and 51% for no PHT, mild PHT, more than mild PHT respectively, P= 0.027. Both RV dilatation and PHT were not significant predictors of CRT response in multinominal logistic regression analysis.

Fifty-one patients had grade I diastolic dysfunction, 74% of them responded to CRT. Fifty-three patients had grade II diastolic dysfunction, 70% of them responded to CRT. Forty-three patents had grade III diastolic dysfunction, 51% of them responded to CRT. Patients with grade III diastolic dysfunction were less likely to respond to CRT compared to patients with less than grade III diastolic dysfunction (51.5% vs. 70.8% respectively, P=0.034). The probability of CRT response was not related to the severity of baseline mitral regurgitation or the left ventricular dimensions, volumes nor function.

As regards the echocardiographic markers of dys-synchrony, septal-posterior wall motion delay (SPWMD), interventricular delay (IVD) and the delay between time to peak systolic velocity in ejection phase at basal septal and basal lateral segments (Ts-septal-lateral) were significant predictors of CRT response in univariate analysis. The CRT response was 77% in patients with SPWMD≥130 msec vs. 58% of those with SPWMD<130 msec, P= 0.02. The CRT response rate was 74% when Ts-septal-lateral ≥ 60 msec vs. 63% when Ts-septal–lateral <60 msec, P= 0.043. Also, 74% of patients with IVD≥ 40 msec were responders vs. 63% of patients with IVD <40 msec, P= 0.04. However, in multinominal logistic regression analysis, none of them proved to be an independent predictor of CRT response.

Baseline echocardiography		**Nonresponders n=56**	**Responders n= 114**	**P**
RV function	TAPSE <15 mm	30 (49.2%)	31 (50.8%)	0.001
	TAPSE≥15 mm	26 (23.9%)	83 (76.1%)	
Right side dilatation	RVd<35 mm	23 (24.7%)	70 (75.3%)	0.012
	RVd≥35 mm	33 (42.9%)	44 (57.1%)	
PHT	PAP≤50 mmhg	39 (28.9%)	96 (71.1%)	0.027
	PAP>50 mmhg	17 (48.6%)	18 (51.4%)	
Diastolic dysfunction	Less than grade III diastolic dysfunction	30 (29.2%)	73 (70.8%)	0.034
	Grade III diastolic dysfunction	21 (48.8%)	22 (51.2%)	

Mitral regurgitation	No/mild	40 (35%)	74 (65%)	0.8
	Moderate/severe	16 (29%)	40 (71%)	
Tricuspid regurgitation	No/mild	41 (30%)	92 (70%)	0.4
	Moderate/severe	15 (39%)	22 (61%)	
LVIDd	<65 mm	30 (35.5%)	53 (64.5%)	0.8
	≥ 65mm	26 (32.1%)	61 (67.9%)	
LVIDs	<55mm	26 (35%)	51 (65%)	0.9
	≥55mm	30 (34%)	63 (66%)	
LVFT/RR%	<40%	21 (30%)	50 (70%)	0.4
	≥40%	35 (35%)	64 (65%)	
IVD	<40msec	30 (37%)	49 (63%)	0.04
	≥40msec	26 (26%)	65 (74%)	
LVPEI	<140msec	36 (37%)	66 (63%)	0.5
	≥140msec	20 (31%)	48 (69%)	
SPWMD	<130msec	35 (42%)	49 (58%)	0.02
	≥130msec	21 (23%)	65 (77%)	
Ts-septal-lateral	<60msec	32 (37%)	54 (63%)	0.04
	≥60msec	24 (26%)	60 (74%)	

Table 3.12: Echocardiography and CRT response

Etiology and CRT response:

Non-ischemic cardiomyopathy was an independent predictor of response (OR 3.1; 95% CI 1.1-8.4, P= 0.028). The CRT response rate among patients with N-ICM was 80.2% vs. 51.9% in patients with ischemic cardiomyopathy, P<0.0001. Patients with ICM without previous infarction were not significantly different from those who experienced previous myocardial infarction. Previous revascularization did not induce significant difference in response rate among ischemic patients.

Etiology	Nonresponders	Responders	P

	n=56	n=114	
ICM	38 (48.1%)	41 (51.9%)	0.0001
N-ICM	18 (19.8%)	73 (80.2%)	
Previous infarction	23 (53.5%)	20 (46.5%)	0.2
No previous infarction	15 (41.7%)	21 (58.3%)	
Previous revascularization	29 (48.3%)	31 (51.7%)	0.9
No previous revascularization	9 (47.4%)	10 (52.6%)	

Table 3.13: Etiology and revascularization vs. CRT response

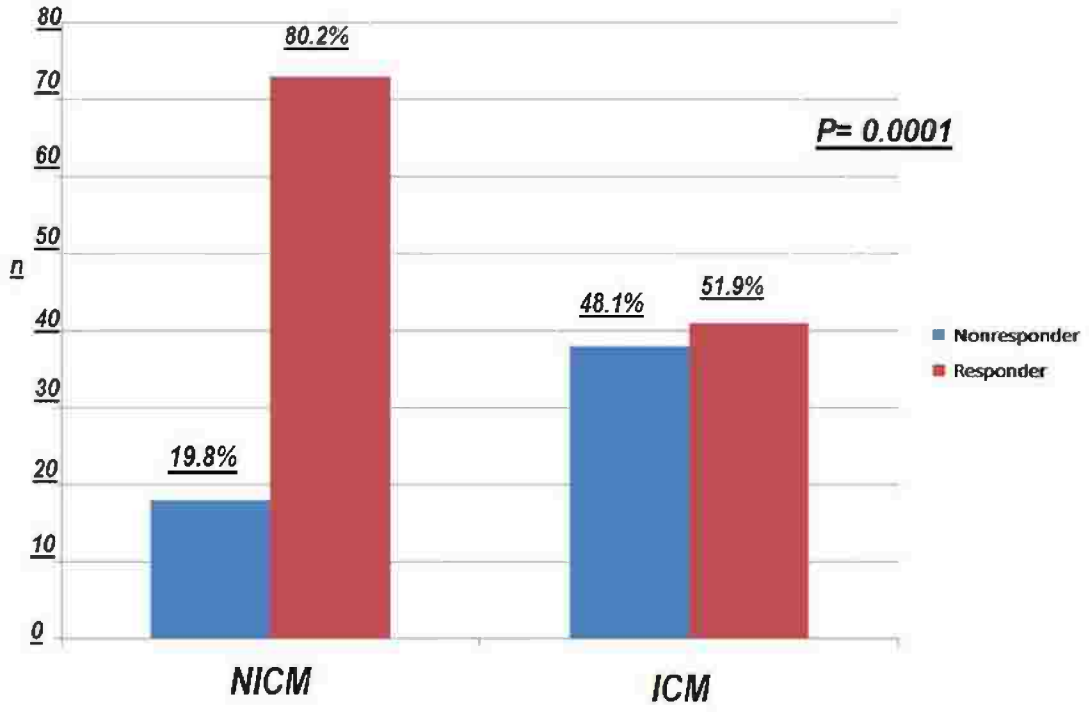


Figure 3.8: Etiology and CRT response

Device optimization and CRT response:

Device optimization using device based automated algorithms was performed at the same implantation day in 57.6% of patients. The AV delay was set empirically at 120 msec and VV delay at 0 msec in patients with no device optimization. Device optimization was done in the nonresponders at 6 months with no improvement in NYHA class at 12 months post-implantation.

There was no significant difference in CRT response rate between patients underwent device optimization and others who didn't undergo device optimization (69.4% vs. 63.9% respectively, P= 0.09).

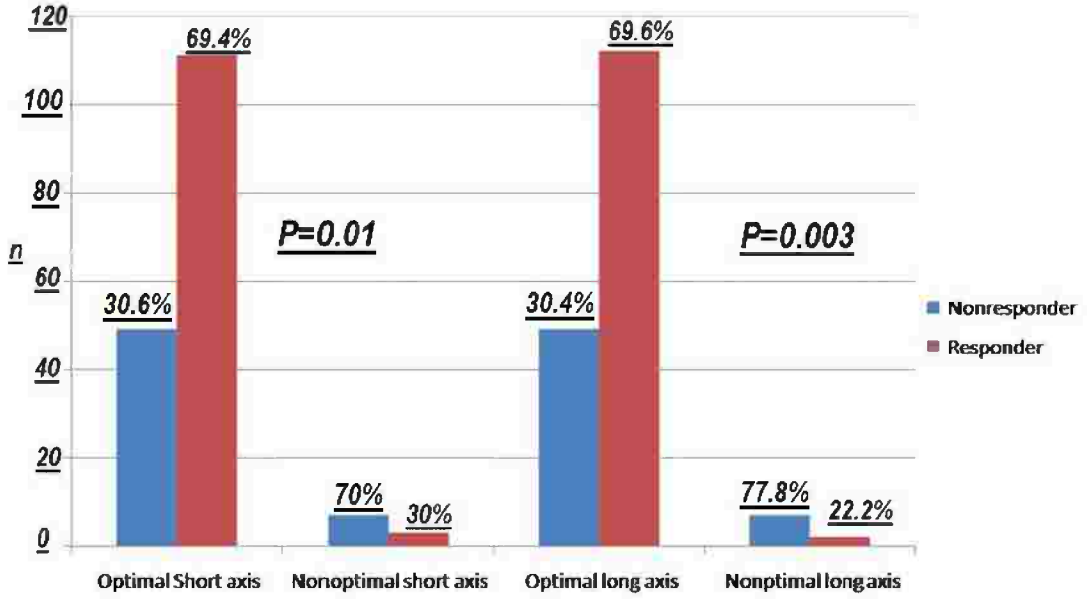
LV lead position and response:

Optimal short axis and long axis position of the LV lead was associated with improved response to CRT. Patients with LV leads in a lateral, posterior or posterolateral position compared to anterior and anterolateral positions were more likely to be responders (74.9% vs. 53.3%, P=0.005), also they had better LVEF after 6 months (37.4+10.5 vs. 28.9+11.9, P=0.002).

Patients with LV leads in the apical position compared to basal and mid cavity positions had significantly higher heart failure related mortality rates at 1 year (44.4% vs. 6.8%, P<0.001, P=0.0001) and lower LVEF after 6 months (27.1+7.8 vs. 36.8+11, P=0.007). Optimal LV lead position (combined optimal long axis and short axis position) was an independent predictor of CRT response (OR 11.3; 95% CI 2-63.8). The CRT response rate with optimal LV lead position was 71.2% vs. 29.4% with non-optimal LV lead position, P<0.0001.

LV lead position		**Nonresponders n=56**	**Responders n=114**	**P**
Short axis	Non-optimal	7 (70.0%)	3 (30.0%)	0.01
	Optimal position	49 (30.6%)	111 (69.4%)	
long axis	Non-optimal	7 (77.8%)	2 (22.2%)	0.003
	Optimal position	49 (30.4%)	112 (69.6%)	
LV lead position	Non-optimal	12 (70.6%)	5 (29.4%)	0.0001
	Optimal	44 (28.8%)	109 (71.2%)	

Table 3.14: LV lead position and CRT response



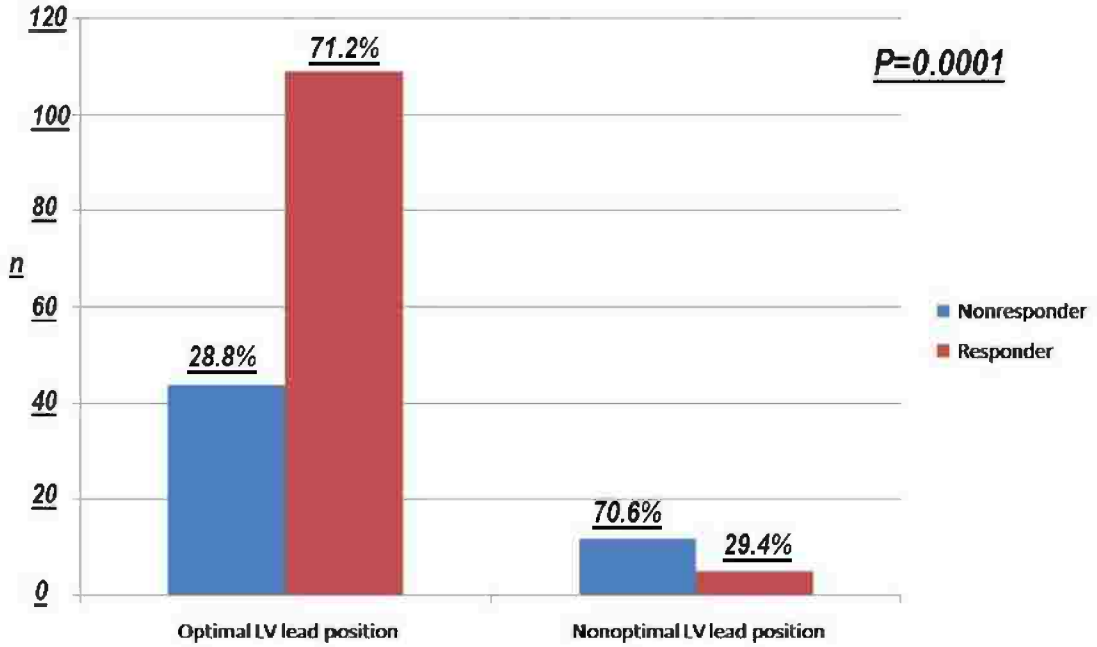


Figure 3.9: LV lead position and CRT response

New score to predict CRT response:

We developed a new simple CRTscore based on preimplantation date to predict CRT response. The CRTscore consists of maximum 9 point. Initially all significant independent preimplantation predictors of CRT response in

multinominal logistic regression analysis were included in the CRTscore according to their relative effect in the regression model to generate an initial CRTscore. It has been shown that the magnitude of benifit from CRT is highest in patients with wider QRS complex, LBBB, female gender and nonischemic cardiomyopathy (figure 3.10).[46]

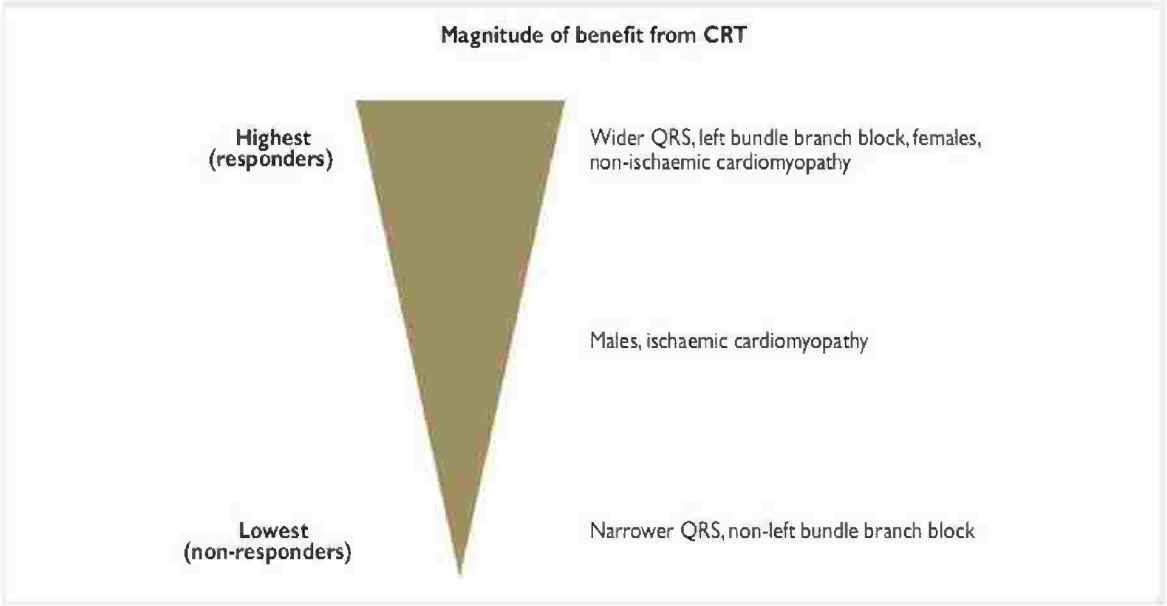


Figure 3.10: Magnitude of benefit from CRT[46]

When LBBB and female gender were added to other variables, the statistical power of the score increased, so they were included in the final CRTscore. The CRTscore includes QRS duration ≥ 150 msec, LBBB morphology of the QRS complex, non-ischemic etiology of cardiomyopathy, sinus rhythm at time of CRT implantation, preserved RV function with TAPSE ≥ 15 mm, female gender, absence of history of renal disease and finally absence of significant COPD (based on specialized respiratory assessment and use of specific medications). Each parameter was assigned to a single point except QRS duration ≥ 150 msec was assigned to 2 points. The CRTscore is the sum of all points.

Parameter	Points
QRS≥150msec	2
LBBB	1
NICM	1
Sinus rhythm	1
Female gender	1

TAPSE≥15mm	1
No history of renal disease	1
No lung disease	1
Total	9

Table 3.15: Points in the CRTscore

The CRT response rate has been markedly different according to the CRTscore. Patients with CRTscore ≥ 6 had CRT response rate of 97.5% vs. only 40.7% if CRTscore <6, P<0.001.

CRTscore	Nonresponders (n)	Responders (n)	Response rate
0	1	0	0%
1	3	0	0%
2	9	2	18.2%
3	18	6	25%
4	11	12	52.2%
5	12	17	58.6%
6	2	28	93.3%
7	0	18	100%
8	0	22	100%
9	0	9	100%
Total	56	114	67.1%

Table 3.16: CRT response according to the CRTscore

CRTscore	Nonresponders n= 56	Responders n= 114	P
<6	54 (59.3%)	37 (40.7%)	0.0001
≥6	2 (2.5%)	77 (97.5%)	

Table 3.17: CRT response in patients with CRTscore ≥6

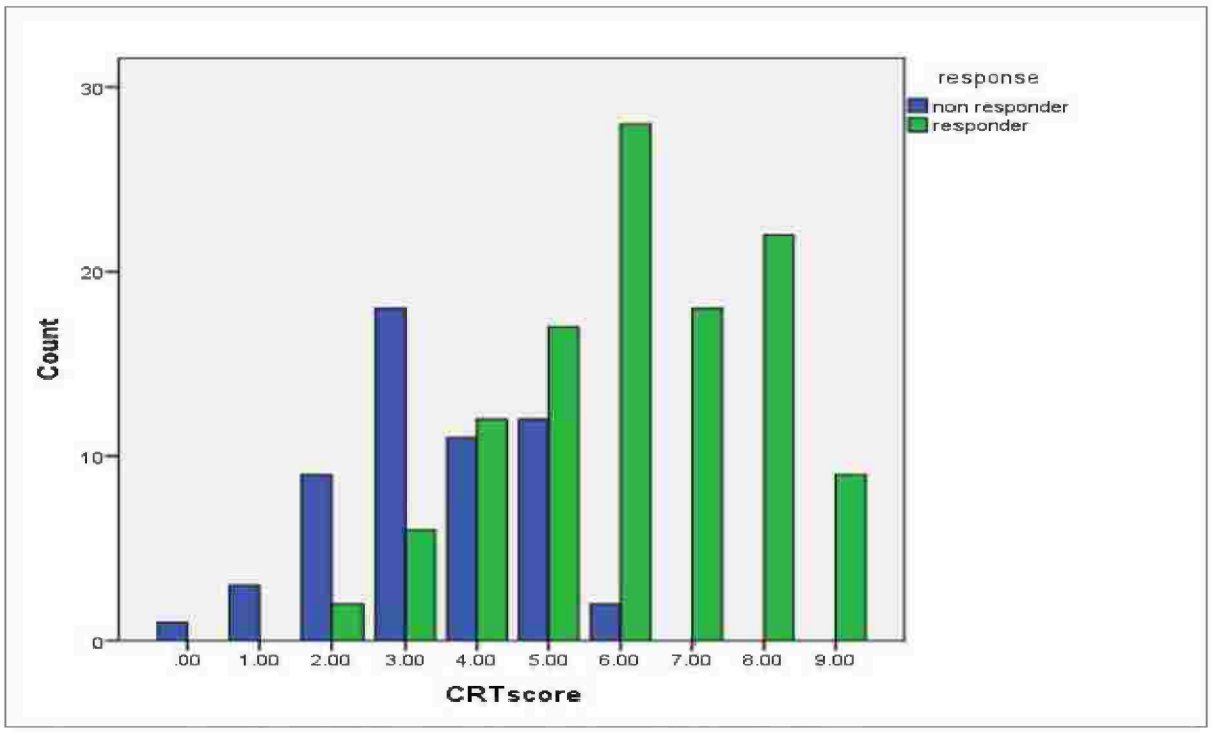


Figure 3.11: CRT response according to the CRTscore

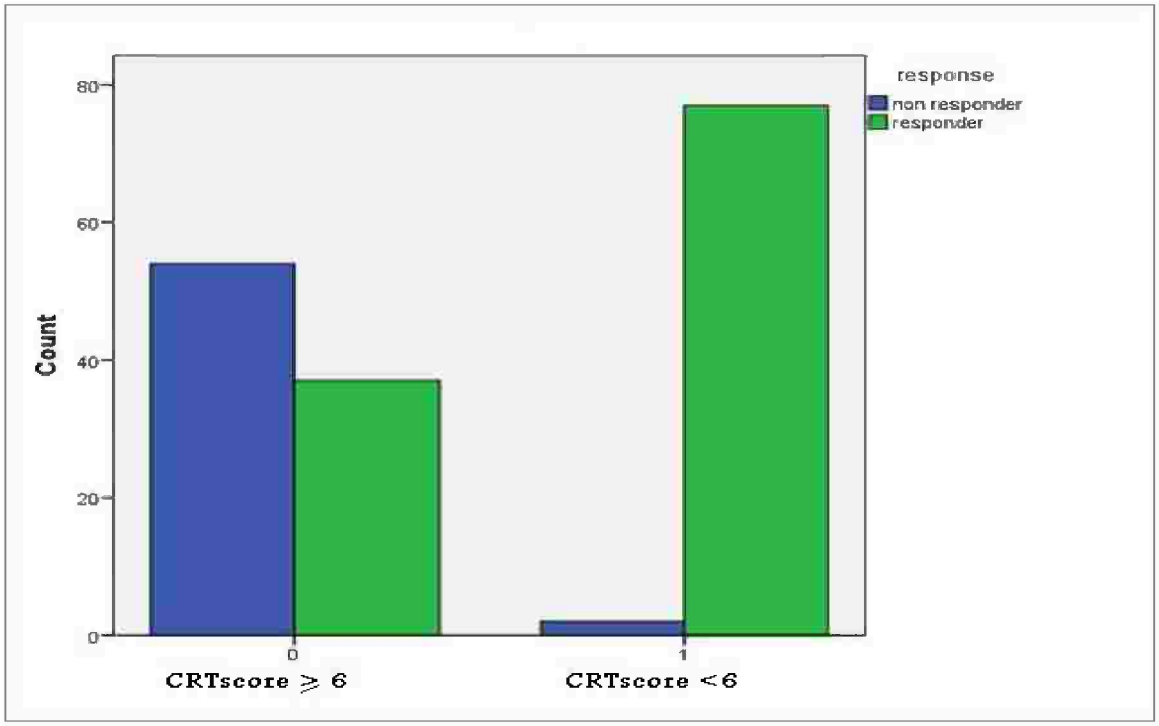


Figure 3.12: CRT response in patients with CRTscore ≥6

The CRTscore in the scope of guidelines

According to the 2013 European Society of Cardiology guidelines on cardiac pacing and CRT, 87 of our patients met class I indication for CRT implantation, 33 met class IIa indication , 25 patients met class IIb and 25 met class III indication for CRT implantation.[46]

Patients with class I indication for CRT implantation had a response rate of 85%. The CRTscore was $\geq$ 6 in 68 patients (66 (97%) responders and 2 (3%) nonresponders), while the CRTscore was less than 6 in 19 patients (8 (42.1%) responders and 11 (57.9%) nonresponders, P <0.0001).

CRTscore	Nonresponders (n)	Responders (n)	Response rate
2.00	1	0	0%
3.00	1	0	0%
4.00	3	2	40%
5.00	6	6	50%
6.00	2	20	90.9%
7.00	0	15	100%
8.00	0	22	100%
9.00	0	9	100%
Total	13	74	85.1%

Table 3.18 : CRTscore and response in patients with class I indication

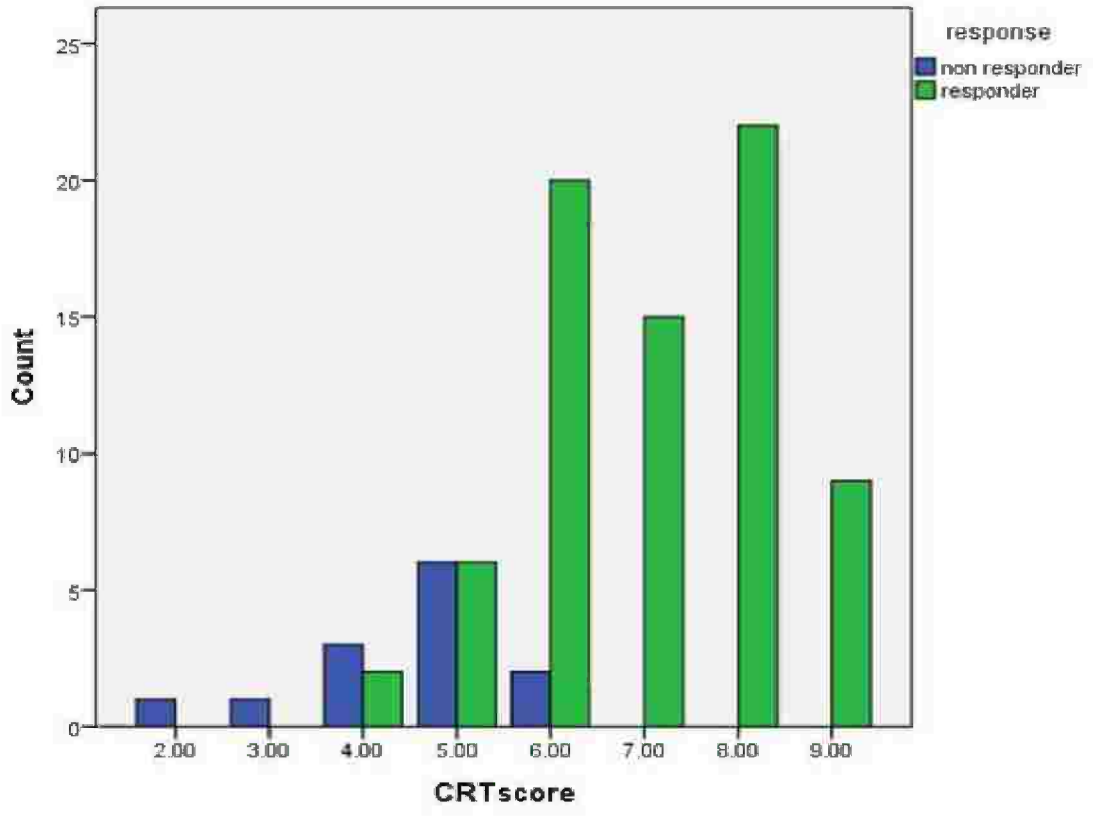


Figure 3.13 : CRTscore and response in patients with class I Indication

Patients with class IIa indication for CRT implantation had a response rate of 48.5%. The CRTscore was ≥ 6 in 9 patients (9 (100%) responders), while the CRTscore was less than 6 in 24 patients (7 (29.2%) responders and 17 (70.8%) nonresponders, P <0.0001).

CRTscore	Nonresponders (n)	Responders (n)	Response rate
0	1	0	0%
1	1	0	0%
2	2	0	0%
3	4	1	20%
4	4	2	33.3%
5	5	4	44.4%
6	0	6	100%
7	0	3	100%

Total	17	16	48.5%

Table 3.19 : CRTscore and response in patients with class IIa indication

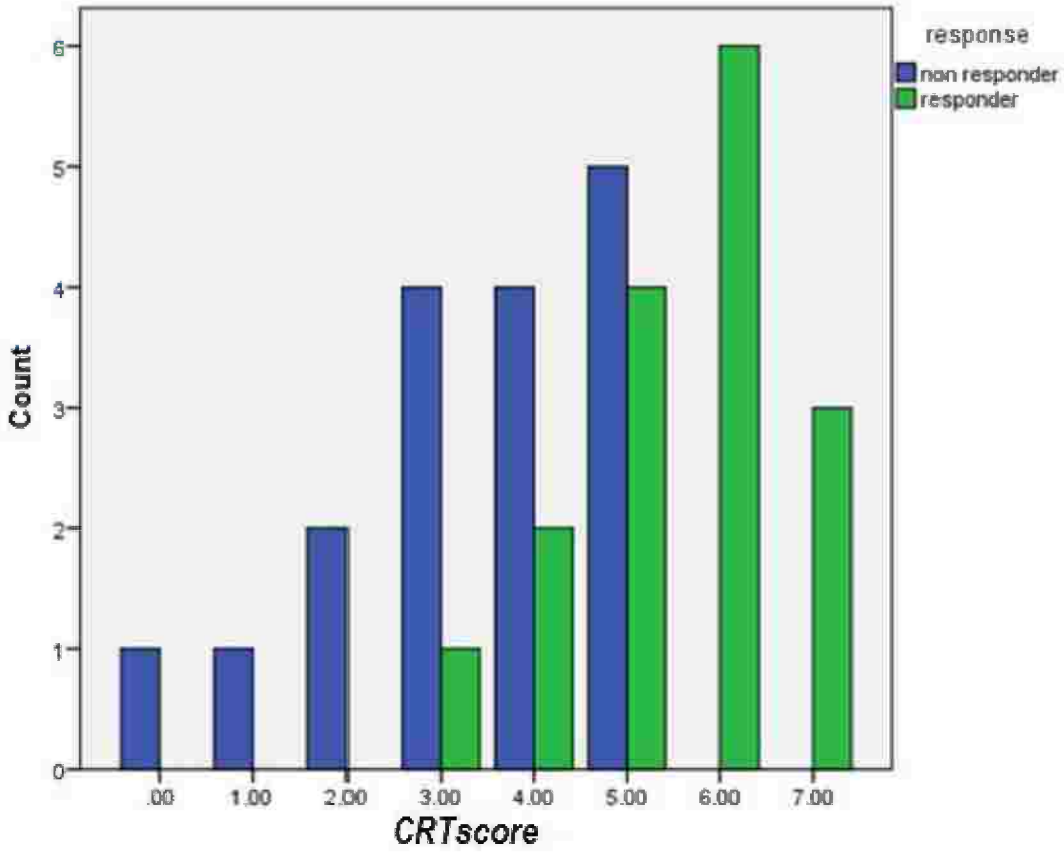


Figure 3.14 : CRTscore and response in patients with class IIa indication

Patients who met class IIb or III indication for CRT implantation had a response rate of 48%. The CRTscore was ≥ 6 in 2 patients (both are responders), while the CRTscore was less than 6 in 48 patients (22 responders and 26 nonresponders). Most of the nonresponders had a CRTscore < 4. The CRT response was better in patients with CRTscore ≥ 4 compared to those with lower CRTscore (77.3% vs 25%, P<0.001).

CRTscore	Nonresponders (n)	Responders (n)	Response rate
1	2	0	0%
2	6	2	25%
3	13	5	27.8%
4	4	8	66.7%
5	1	7	87.5%

6	0	2	100%
Total	26	24	48%

Table 3.20 : CRTscore and response in patients with class IIb/III indication

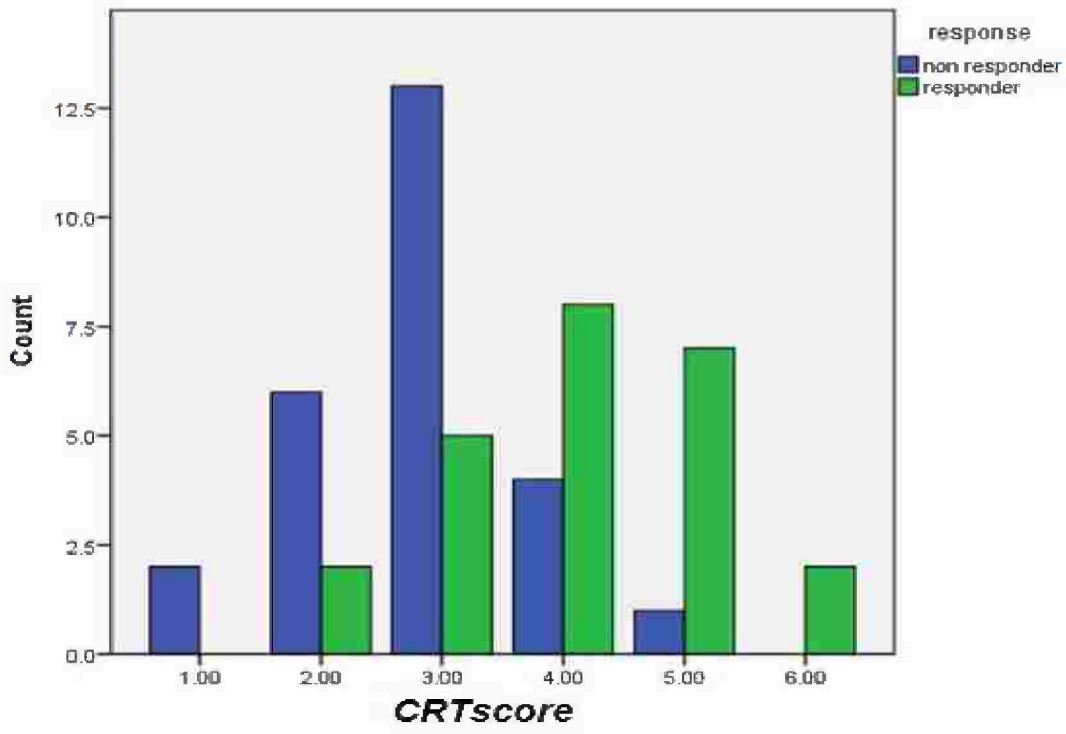


Table 3.15 : CRTscore and response in patients with class IIb/III indication

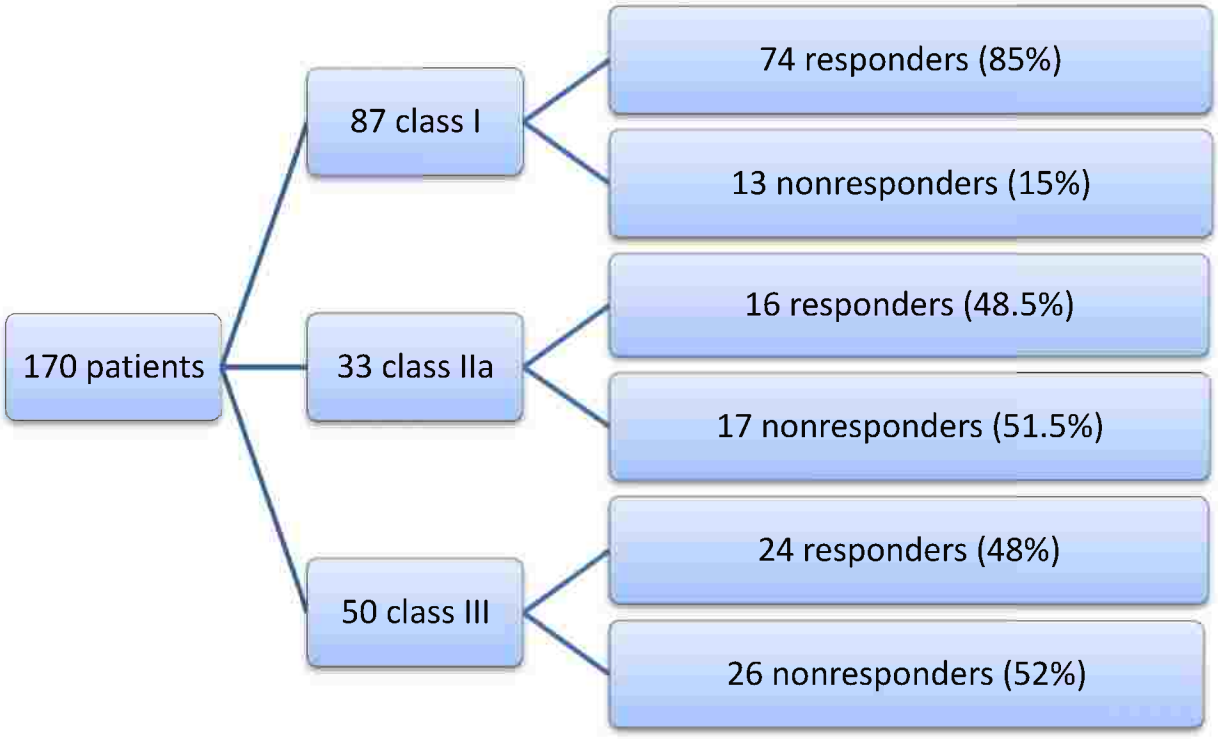


Figure 3.16: Guidelines and CRT response

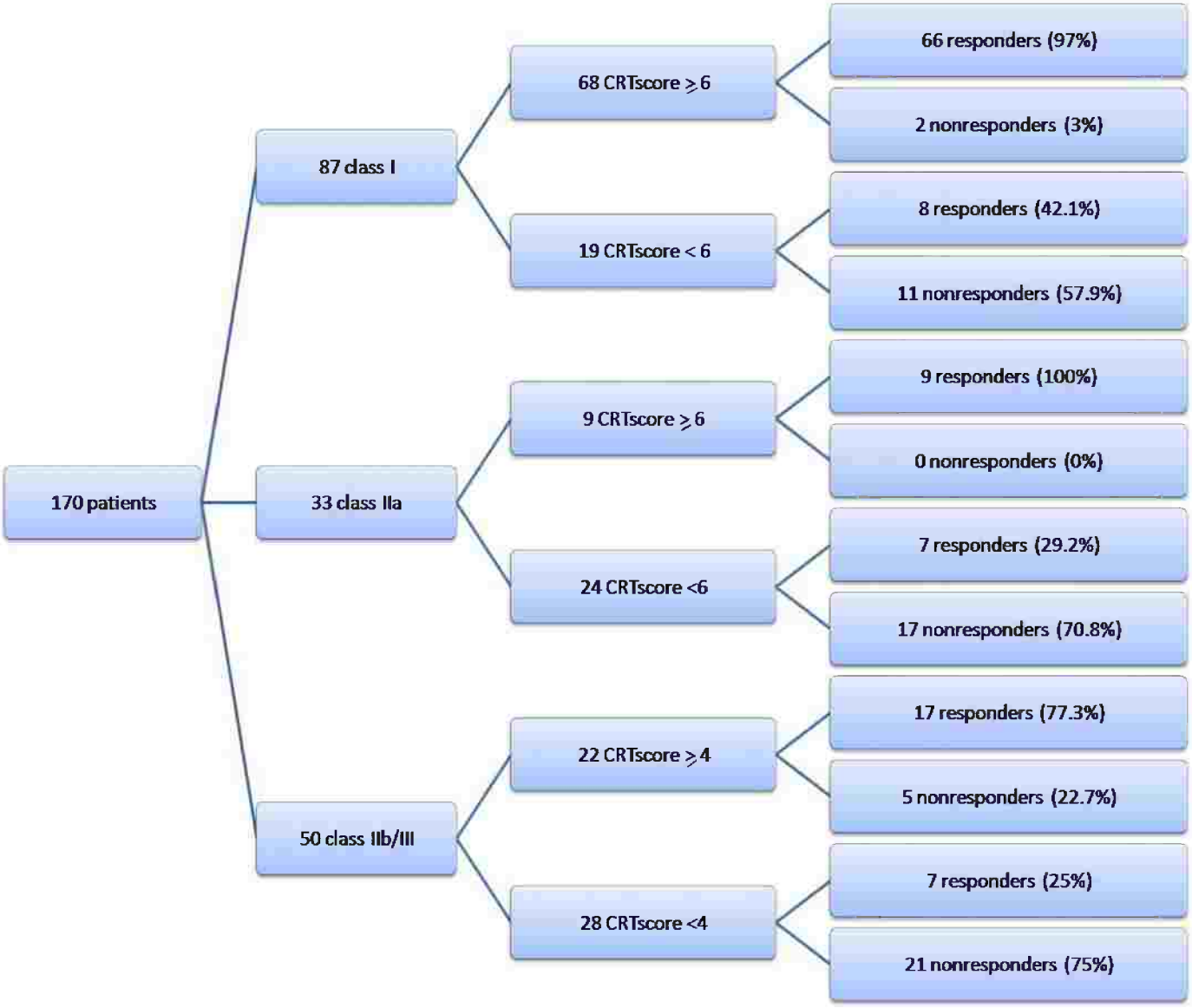


Figure 3.17: Guidelines, CRTscore and CRT response