Nonpharmacologic Treatment of Ventricular Heart Failure

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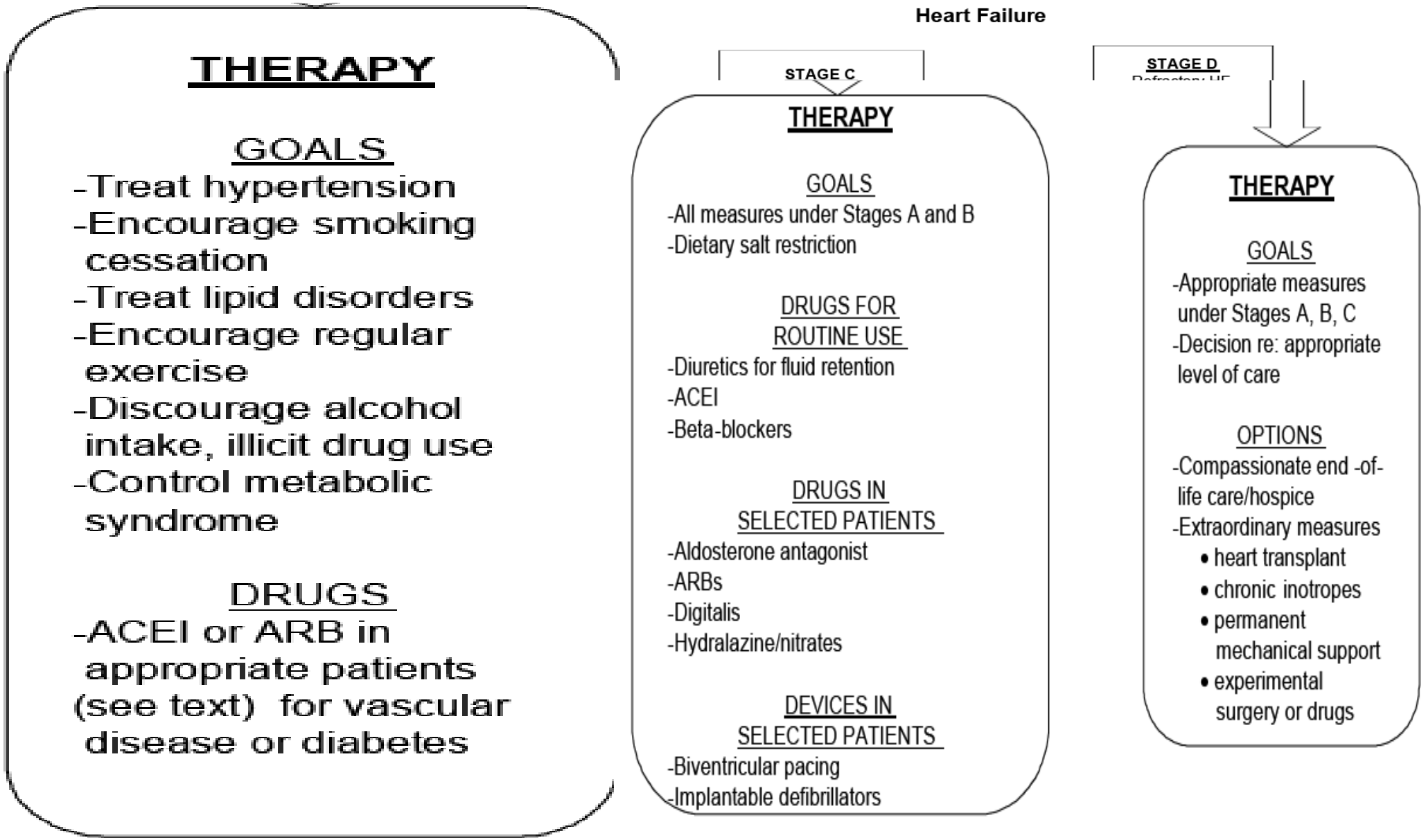
2001 AHA Guideline for CHF
- Symptomatic LV dysfunction

- Class I : Diuretics, ACE inhibitor, beta-blocker, Digoxin
- Class IIa: Spironolactone, **Exercise training**, Angiotensin receptor blocker
- Class IIb: anticoagulation
- Class III: **Routine use of nutrition supplements**(coenzyme Q, carnitine, taurine etc)



d therapy by stage. FHx CM indicates family history of cardiomyopathy; ACEI, angiotensin converting enzyme inhibitors; and ARB, angiotensin receptor blocker.

Non-pharmacologic Treatment

- Nutritional support
- Exercise therapy
- Cell based therapy
- Device therapy (CRT &/or ICD)
- Interventional treatment
- Mechanical devices
- Surgical strategy or Transplantation

Non-pharmacologic Treatment

- Nutritional support
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- Cardiac resynchronization therapy
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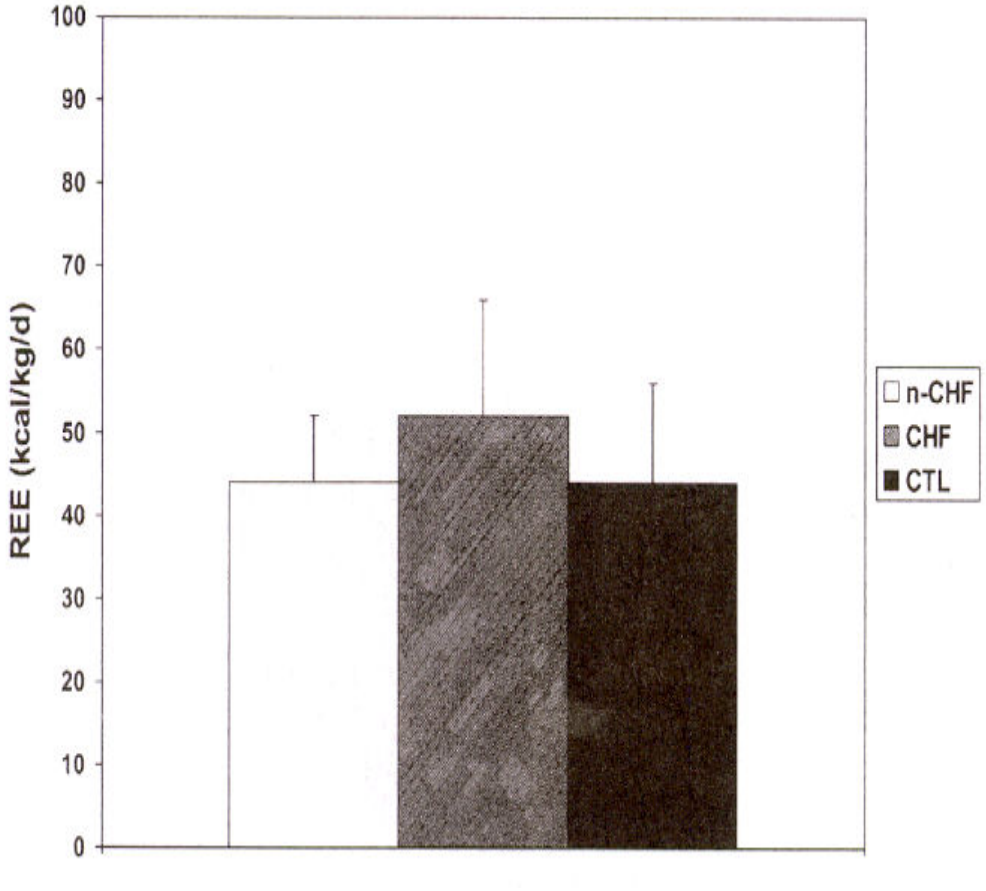
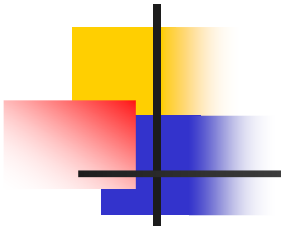
Nutritional Support in CHF

- Energy intake less than energy expenditure
- Substantial malabsorption of nutrient intake
- Infants with CHF had a trend toward the highest REE
- Energy expenditure in specific defects with CHF and dietary intervention ?



Nutritional support in CHF

- Malnutrition in up to 50% in severe CHF
  - Depletion of lean body mass including vital organ
  - Reduction of contractility and compliance
- Sufficient calories and protein to allow for normal growth & to prevent the breakdown of lean body mass
- Caloric density have to be increased to 30kcal/oz(=100kcal/100ml) or greater due to restricted fluid intake and use of diuretic therapy



- Increasing caloric content may

- augment the respiratory quotient if calories are added in the form of glucose polymer

- Patient with CHF are prone to the development of contraction alkalosis
- Combined effect my lead to inadequate ventilation or loss of calories due to excessive use of respiratory muscle

Etiology of Growth Failure: Multifactorial

- Intrauterine growth retardation
- Nutritional
- Hemodynamic disturbance
- Gene alteration
- Role of Endocrine factor such as IGF-I

Table 1. Types of support and timing of metabolic and nutritional support.

Type of support	Timing and condition
Metabolic	
GIK	First 48 h of myocardial infarction. CABG (perioperative) [7–9]
Glutamine	Angina pectoris, CABG (intraoperative) [10]
Taurine	Congestive heart failure [11]
Antioxidant	
Selenium	Cardiopulmonary bypass [12]
	Acute and chronic heart failure [13,14]
Manganese	Cardiopulmonary bypass [15]
Coenzyme Q10	Early postoperative
	Acute ischaemia [11]
Nutritional	
Vitamin B_1	Chronic and acute heart failure [16•]
Folic acid	Reduction of homocysteine [17,18]
ω-3 fatty acids	Acute and chronic heart failure [19••]

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A Few controlled exercise studies in patients with CHD

- A number of patients are physically inactive due to overprotection
- Maximal exercise capacity can be improved after a period of physical training
- Risk of physical exercise is very low
- Only a few defect associated with SCD

Exercise Intolerance in Adult Congenital Heart Disease

Comparative Severity, Correlates, and Prognostic Implication

Gerhard-Paul Diller, MD; Konstantinos Dimopoulos, MD; Darlington Okonko, BSc, MRCP; Wei Li, MD, PhD; Sonya V. Babu-Narayan, MRCP; Craig S. Broberg, MD; Bengt Johansson, MD, PhD; Beatriz Bouzas, MD; Michael J. Mullen, MD, MRCP; Philip A. Poole-Wilson, MD, FRCP; Darrel P. Francis, MA, MRCP; Michael A. Gatzoulis, MD, PhD

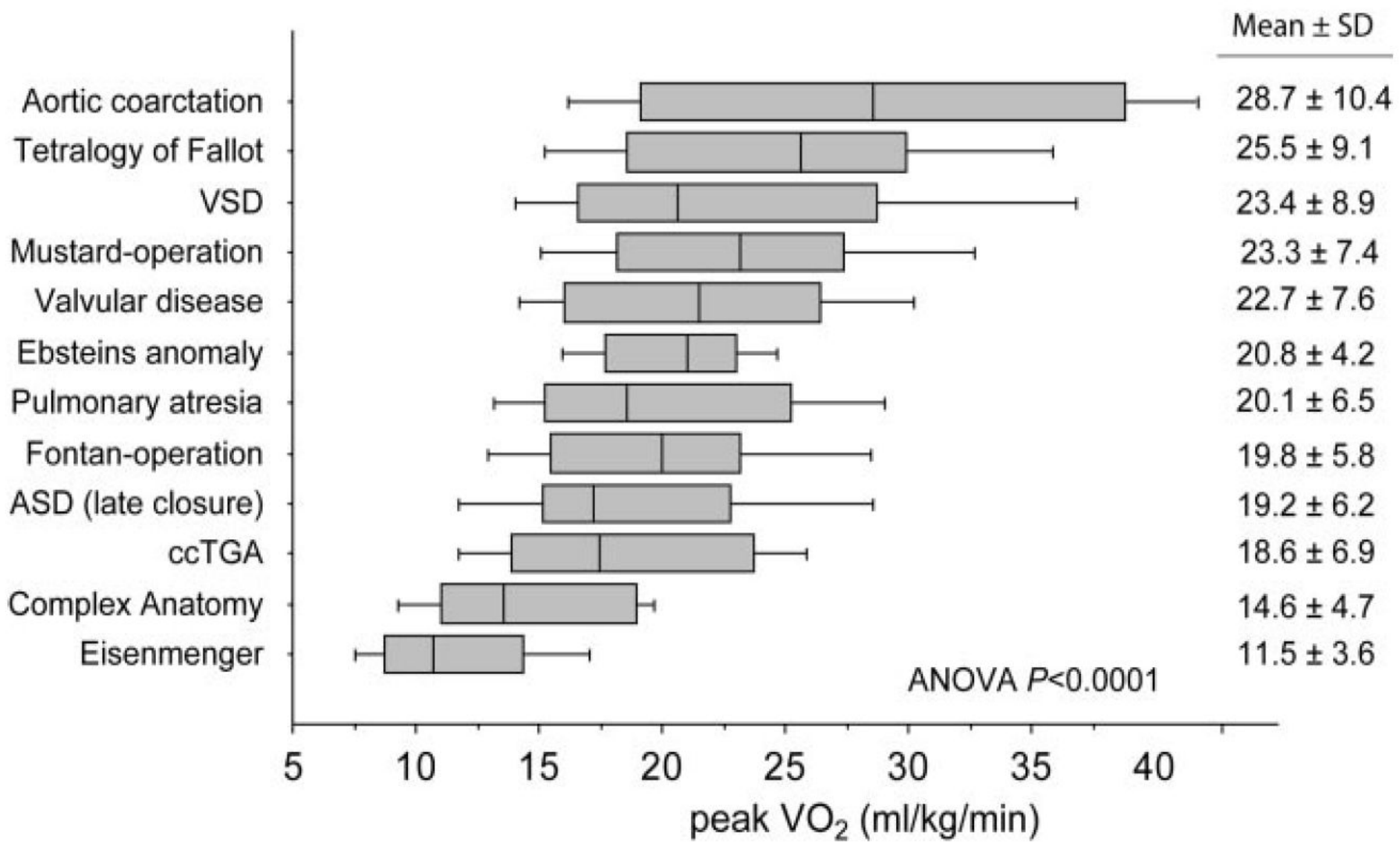
Background—Although some patients with adult congenital heart disease (ACHD) report limitations in exercise capacity, we hypothesized that depressed exercise capacity may be more widespread than superficially evident during clinical consultation and could be a means of assessing risk.

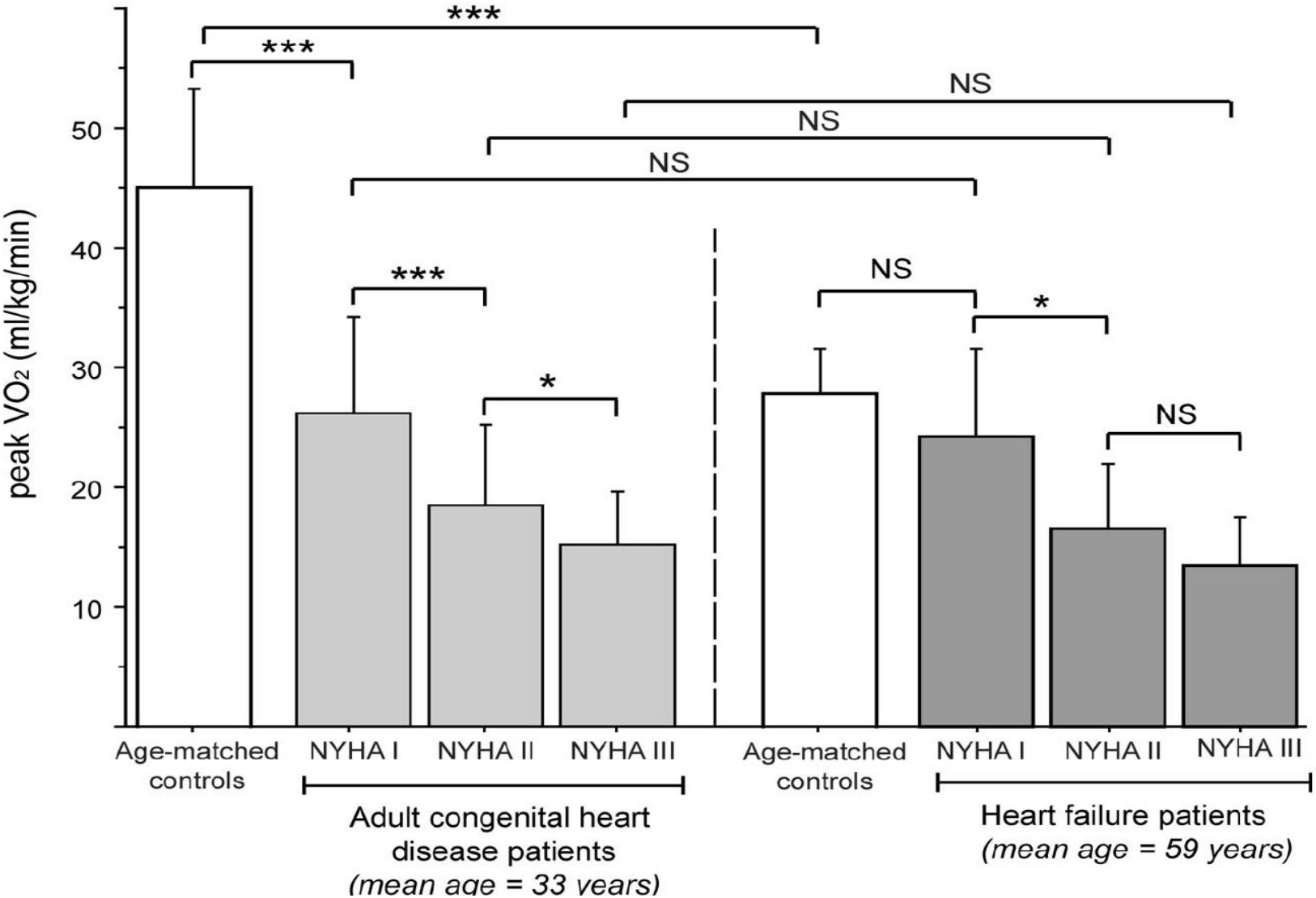
Methods and Results—Cardiopulmonary exercise testing was performed in 335 consecutive ACHD patients (age, 33±13 years), 40 non–congenital heart failure patients (age, 58±15 years), and 11 young (age, 29±5 years) and 12 older (age, 59±9 years) healthy subjects. Peak oxygen consumption (peak $\dot{V}O_2$) was reduced in ACHD patients compared with healthy subjects of similar age (21.7±8.5 versus 45.1±8.6; $P<0.001$). No significant difference in peak $\dot{V}O_2$ was found

10 months, 62 patients (18.5%) were hospitalized or had died. On multivariable Cox analysis, peak $\dot{V}O_2$ predicted hospitalization or death (hazard ratio, 0.937; $P=0.01$) and was related to the frequency and duration of hospitalization ($P=0.01$ for each).

Conclusions—Exercise capacity is depressed in ACHD patients (even in allegedly asymptomatic patients) on a par with chronic heart failure subjects. Lack of heart rate response to exercise, pulmonary arterial hypertension, and impaired pulmonary function are important correlates of exercise capacity, as is underlying cardiac anatomy. Poor exercise capacity identifies ACHD patients at risk for hospitalization or death. (***Circulation*. 2005;112:828-835.**)

Key Words: exercise test ■ heart defects, congenital ■ heart failure ■ prognosis ■ survival


Mean ± SD
Aortic coarctation
Tetralogy of Fallot
VSD
Mustard-operation
Valvular disease
Ebsteins anomaly
Pulmonary atresia
Fontan-operation
ASD (late closure)
ccTGA
Complex Anatomy
Eisenmenger
28.7 ± 10.4
25.5 ± 9.1
23.4 ± 8.9
23.3 ± 7.4
22.7 ± 7.6
20.8 ± 4.2
20.1 ± 6.5
19.8 ± 5.8
19.2 ± 6.2
18.6 ± 6.9
14.6 ± 4.7
11.5 ± 3.6
ANOVA P<0.0001
5
10
15
20
25
30
35
40
peak VO2 (ml/kg/min)


peak VO2 (ml/kg/min)
50
40
30
20
10
***
***
NS
NS
NS
***
*
NS
*
NS
Age-matched controls
NYHA I
NYHA II
NYHA III
Adult congenital heart disease patients
(mean age = 33 years)
Age-matched controls
NYHA I
NYHA II
NYHA III
Heart failure patients
(mean age = 59 years)

Kaplan-Meier plots for combined end point of hospitalization or death

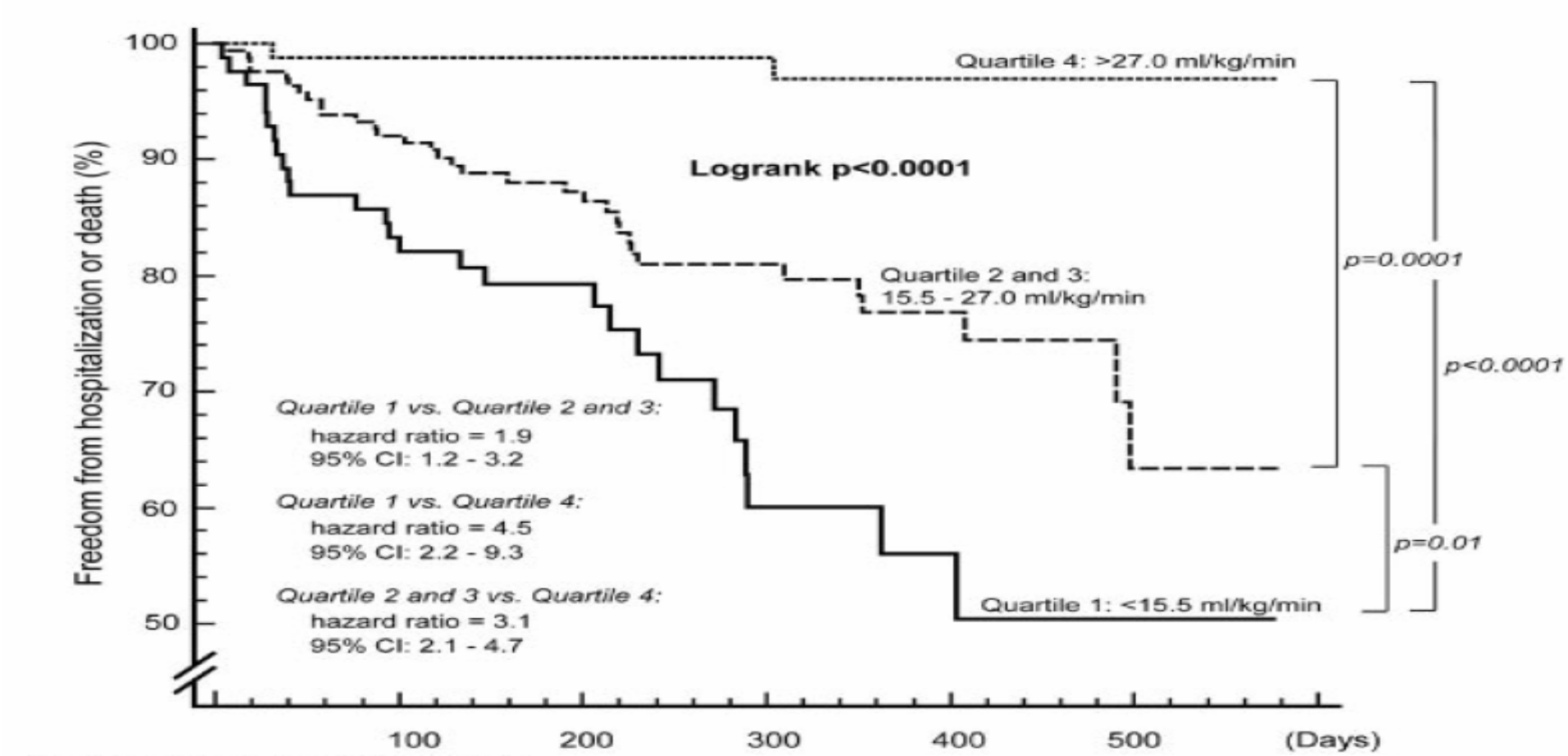


TABLE 5. Significant Predictors of Hospitalization or Death on Cox Proportional-Hazards Analysis

	P	Hazard Ratio	95% CI for Hazard Ratio
Single-variable analysis			
NYHA class	<0.001	2.556	1.790–3.652
Peak $\dot{V}o_2$	<0.001	0.908	0.873–0.943
Diagnosis	0.04	. . .	. . .
Peak heart rate	<0.001	0.985	0.976–0.991
Age at surgery	0.04	1.018	1.000–1.036
Multivariable analysis			
NYHA class	0.002	2.150	1.317–3.486
Peak $\dot{V}o_2$	0.01	0.937	0.890–0.986

Hazard ratio refers to unit increase in NYHA class, peak $\dot{V}o_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$), heart rate (bpm), and age at surgery (years).

Evaluation of children with CHD

- Formal exercise testing with continuous measurement of gas exchanges
- Maximal exercise performance – maximal workload, maximal O2 uptake(gold standard to assess aerobic fitness, not motivated), maximal exercise endurance time, 6 minute work test
- Ventilatory anaerobic threshold, HR based measurement(limited)
- Oxygen uptake kinetics

Evaluation and risk stratification

- Latest diagnostic testing and surgical report
- NYHA
- Physical examination and BP
- Standard 12 lead ECG
- Imaging studies

Table 1 Eligibility for non-restricted participation in competitive sports in congenital heart disease patients

Eligible	Not eligible
I Surgical procedure	Uncorrected or palliative corrected
Fully corrected (anatomically)	Significant lesions not operated
	Univentricular hearts
	Mustard, Senning or Rastelli corrected TGA
	Arterio-pulmonal shunts
II Medical history	
Satisfactory	Abnormal
NYHA class I	Symptoms of severe palpitations/syncope
	Exercise-induced symptoms (dyspnoea, angina, palpitations, syncope)
	NYHA class II or higher
III Physical examination	
Satisfactory	Abnormal
	Hypertension
	Hepatomegaly, raised venous pressure
IV ECG/Holter	
Satisfactory	Abnormal
	Ischemia (coronary anomaly, TGA-switch)
	QRS-duration (Fallot)
	Significant hypertrophy
	Significant arrhythmia
V Morphology/haemodynamic	
Satisfactory	Abnormal
	Significant rest-lesion
	Mean transvalvular gradient of aorta $\geq$ 20 mmHg
	Peak transvalvular gradient of the pulmonary artery of $>$50 mmHg
	Significant hypertrophy
	Significant myocardial dysfunction
	Pulmonary hypertension
VI Maximal ergospirometry	
Satisfactory	Abnormal
Values within normal range	Chest pain or syncope
	Significant arrhythmia
	Ischemia on ECG

Exercise recommendation in patients with CHD

- Impossible to predict how much energy will be expended for different pathologies
- Exercise intensity of sports- low, moderate, high dynamic with al low, moderate and high static components
- Talk test- safe exercise intensity, children should exercise at an intensity level at which they still able to talk to their peer or parents during exercise.

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Position Paper

Recommendations for participation in competitive and leisure sports in patients with congenital heart disease: a consensus document

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Background Physical activity is important for patients with congenital heart disease. The aim of this paper is to provide a consensus document for participation in competitive or leisure sport activity in children and adults with congenital heart disease.

Methods The recommendations are based on expert consensus meetings, personal experience of the contributing authors and an updated review of the literature regarding exercise performance and risk stratification in patients with congenital heart disease.

Results Physical performance and exercise tolerance is close to normal in patients with simple lesions with successful repair or no need for therapy. Most patients with complex lesions have some degree of residual disease, making them less suitable for participation in competitive sport.

Conclusion Regular exercise at recommended levels can be performed and should be encouraged in all patients with congenital heart disease. Many can attend sports with no restrictions. Special concern should be given to those patients with a significant ventricular dysfunction or recent history or risk of arrhythmia. *Eur J Cardiovasc Prev Rehabil* 13:293–299

Table 2 Recommendations for sport participation in congenital heart diseases

Lesion	Recommendation
ASD (closed or non-significant or PFO)	No restrictions Scuba diving should be avoided in those with a remaining shunt, due to the risk of paradoxical embolism
VSD (closed or non-significant)	No restrictions
PDA (closed or non-significant)	No restrictions
AVSD (successfully repaired)	No restrictions
Moderate MVR	Low to moderate dynamic and static sports
PAPVC/TAPVC (successfully repaired)	No restrictions
Pulmonary stenosis (mild)	No restrictions
Moderate	Low to moderate dynamic and static sports
Aortic stenosis (mild)	Low to moderate dynamic and static sports
Moderate	Low dynamic and static sports No competitive sport if left ventricular dysfunction or symptoms
CoA (successfully repaired)	No restrictions[a]
TOF (successfully repaired)	Low to moderate dynamic and static sports[a]
Residual disease	Low dynamic and static sports[a]
TGA	
asoTGA (successfully repaired)	No restrictions
iarTGA, ccTGA	Low to moderate dynamic and low static sports[b]
Ebstein anomaly	Low to moderate dynamic and low static sports[b]
Univentricular hearts/Fontan circulation	Low to moderate dynamic and low static sports[b]
	Low dynamic sports[b]
Eisenmenger's syndrome	No restrictions
Congenital coronary artery anomalies	
Successfully repaired	

Classification of sports (based on peak dynamic and static components during competition)

	A. Low dynamic	B. Moderate dynamic	C. High dynamic
I. Low static	Billiards Bowling Cricket Golf Riflery	Baseball Softball Table tennis Tennis (doubles) Volleyball	Badminton Cross-country skiing (classic) Field hockey[a] Orienteering Race walking Racquetball Running (long distance) Soccer Tennis (singles)
II. Moderate static	Archery Auto racing[a,b] Diving[a,b] Equestrian[a,b] Motorcycling[a,b]	Fencing Field events (jumping) Figure skating[a] Football (American)[a] Rodeoing[a,b] Rugby[a] Running (sprint) Surfing[a,b] Synchronized swimming[b]	Basketball[a] Ice hockey[a] Cross-country skiing (skating) Football (Australian)[a] Lacrosse[a] Running (middle distance) Swimming Team handball
III. High static	Bobsledding[a,b] Field events (throwing)[a,b] Gymnastics[a,b] Karate/judo[a] Luge[a,b] Sailing Rock climbing[a,b] Waterskiing[a,b] Weight lifting[a,b] Windsurfing[a,b]	Body building[a,b] Downhill skiing[a,b] Wrestling[a]	Boxing[a] Canoeing/kayaking Cycling[a,b] Decathlon Rowing Speed skating

Non-pharmacologic Treatment

- Nutritional support
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- Interventional treatment
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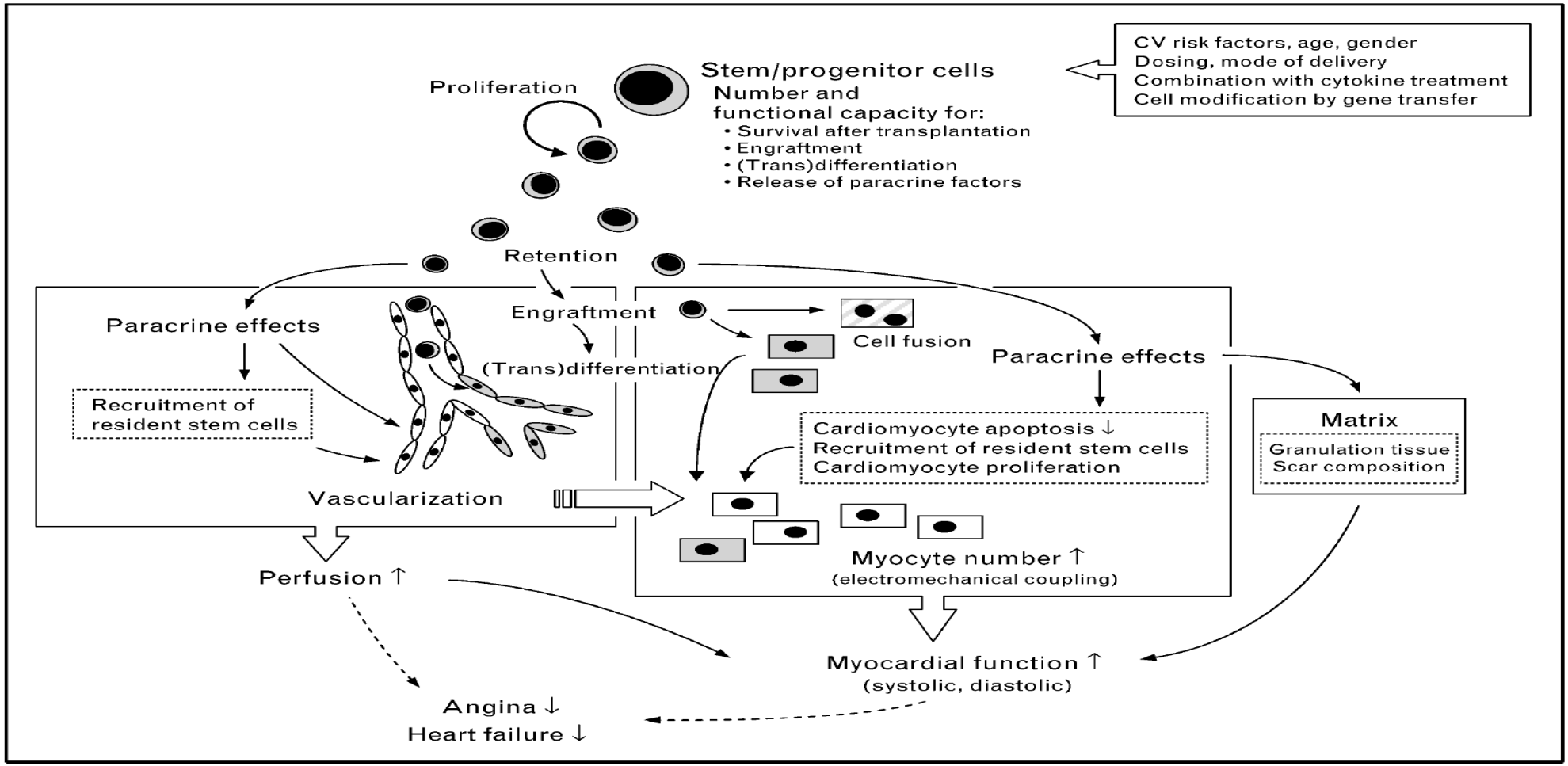
Working hypothesis of therapeutic stem cell transplantation for myocardial regeneration



Cell based therapy

- Stem cell can exert beneficial effects on the failing heart by
  - Transdifferentiation into cardiac cell type
  - Providing a source of cardio-protective paracrine factors
- Replacement of scar tissue by viable myocardim should improve cardiac function and inhibit remodeling in CHF

Table 1 Cell-based therapy for heart failure: potential donor cells, advantages, limitations and practicability issues

Cell type	Cell source	Ex-vivo expansion possible	Capacity for transdifferentiation into:		Paracrine effects	Clinical data suggest	
			Cardiomyocytes	Vessels		Safety	Efficacy
Skeletal myoblasts	Skeletal muscle biopsy	Yes	No	No	Uncertain	Uncertain	Yes
Unfractionated bone marrow cells	Bone marrow	No	Probably no	Probably yes	Yes	Yes	Yes
Endothelial progenitor cells	Bone marrow Peripheral blood	Yes	Uncertain	Yes	Yes	Yes	No
Mesenchymal stem cells	Bone marrow, and other mesenchymal tissues	Yes	Yes	Uncertain	Yes	n.d.	n.d.
Resident cardiac stem and progenitor cells	Cardiac muscle biopsy?	Yes	Yes	Yes	Uncertain	n.d.	n.d.
Embryonic stem cells	Allogeneic cell lines Autologous cell line: 'therapeutic cloning'?	Yes	Yes	Yes	Uncertain	n.d.	n.d.

n.d. denotes not determined. All clinical data were derived from non-randomized trials. See the text for details and references.

Table 2 Cell-based therapy for heart failure: early clinical trial experience

Study	*n*	LVEF	Cell type	Time after AMI	Delivery	Reported outcome
Menasche *et al.* [29]	10 treated; no controls	24 ± 4%	Myoblasts	3–228 months	Transepicardial (during CABG)	Regional wall motion ↑ Global LVEF ↑
Herreros *et al.* [30]	11 treated; no controls	36 ± 8%	Myoblasts	3–168 months	Transepicardial (during CABG)	Regional wall motion ↑ Global LVEF ↑ Viability in infarct area ↑
Siminiak *et al.* [31]	10 treated; no controls	25–40%	Myoblasts	4–108 months	Transepicardial (during CABG)	Regional wall motion ↑ Global LVEF ↑
Chachques *et al.* [32]	20 treated; no controls	28 ± 3%	Myoblasts	Not reported	Transepicardial (during CABG)	Regional wall motion ↑ Global LVEF ↑ Viability in infarct area ↑
Smits *et al.* [33]	5 treated; no controls	36 ± 11%	Myoblasts	24–132 months	Transendocardial (guided by EMM)	Regional wall motion ↑ Global LVEF ↑
Siminiak *et al.* [27]	10 treated; no controls	30–51%	Myoblasts	5–96 months	Transcoronary vein	Global LVEF ↑
Dib *et al.* [34••]	24 treated; no controls	15–43%	Myoblasts	Not reported	Transepicardial (during CABG)	Global LVEF ↑ Viability in infarct area ↑
Stamm *et al.* [36,37]	12 treated; no controls	36 ± 11%	$CD133^+$	3–12 weeks	Transepicardial (during CABG)	Global LVEF ↑ Perfusion ↑
Strauer *et al.* [39••]	18 treated; 18 controls	52 ± 9%	Unfractionated BMCs	5–102 months	Intracoronary	Regional wall motion ↑ Global LVEF ↑ Viability in infarct area ↑
Assmus *et al.* [38]	86 treated; 16 controls	40 ± 11%	Unfractionated BMCs Endothelial progenitor cells	3–144 months	Intracoronary	Global LVEF ↑ (only in BMC group)

n denotes number of patients; AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; LVEF, left ventricular ejection fraction; EMM, electromechanical mapping; $CD133^+$, bone marrow-derived CD133-positive cells; BMC, bone marrow cell.

Safety concern of cell based therapy

- Abnormal cellular differentiation
- Coronary obstruction, accelerated atherosclerosis
- Myoblast graft may represent an arrhythmogenic substrate, esp. early after cell injection
  - Transplanted myoblast fuse with cardiomyocyte

  - generating spatial heterogenecity of Ca signaling at the graft-host interface
  - Tissue injury and local inflammation

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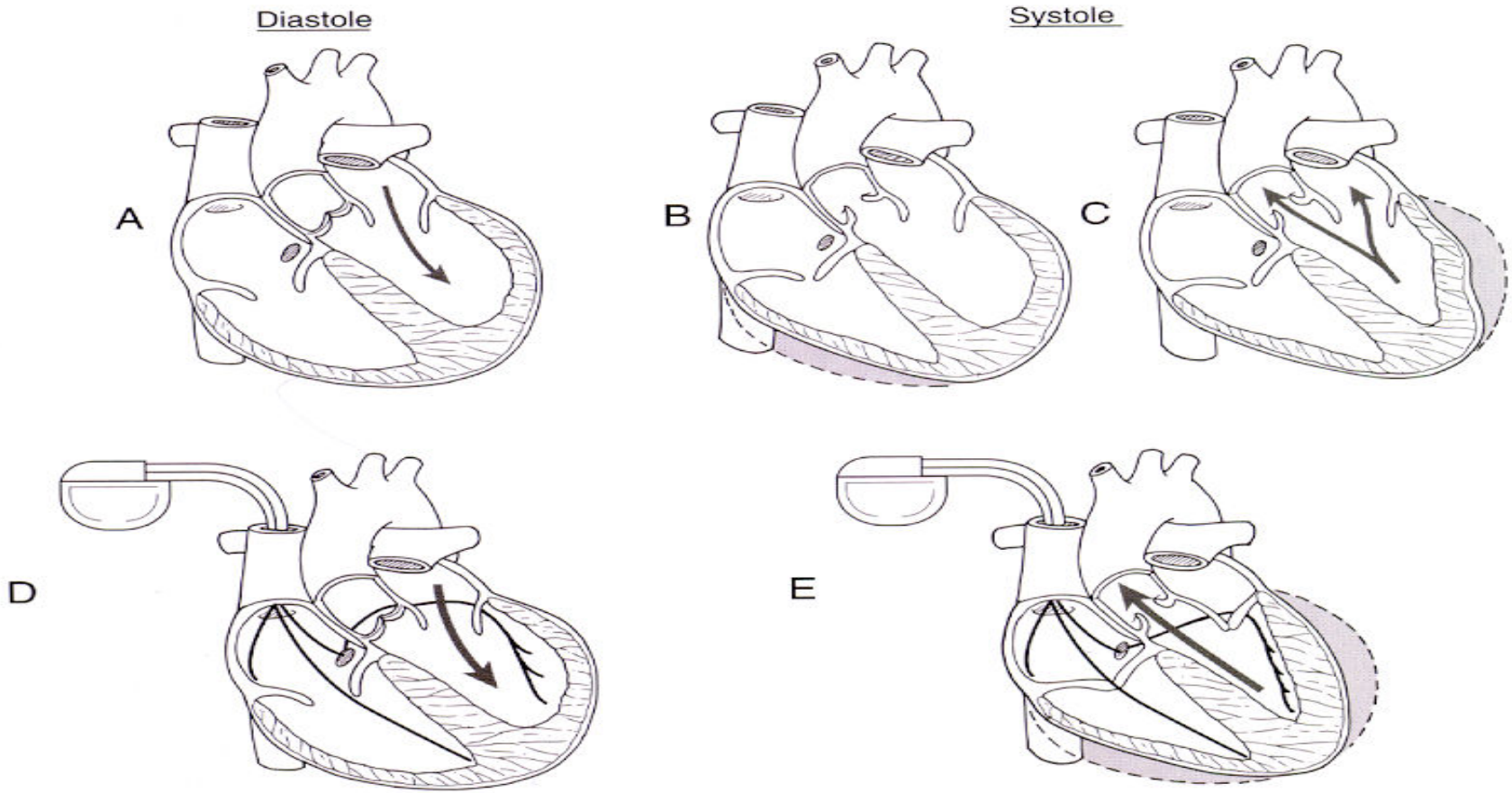
Pathophysiology of ventricular dyssynchrony and mechanism of action for cardiac resynchronization



Rationale for CRT

- Advanced CHF(adult) , evidence of conduction delay, most often LBBB
- Significant delay uneven activation with region of early and late activation
- Dyskinetic activation increase wall stress, decrease dP /dT, increase LVEDP, impair systolic function
- Dyssynchrony between LV & RV &

  Sepal dyskinesis

Summary of Cardiac Resynchronization Therapy Trials

Study	Design	Patients	Results
COMPANION[30]	Multicenter, prospective, randomized, controlled	1600 patients (terminated early) with DCM, NYHA classes III/IV, IVCD with 3 arms: drug therapy only, drug + CRT, drug + CRT/ICD	Combined all-cause mortality and hospitalizations decreased 20% in device arms; 40% reduction in total mortality with combined CRT/ICD
PATH-CHF[8]	Single-blinded, randomized, crossover, controlled	42 patients with DCM, NYHA classes III/IV, IVCD	Improved exercise tolerance (6-min walk), QOL, and NYHA class
MIRACLE[9]	Prospective, randomized, double-blinded, parallel, controlled	453 patients with DCM, NYHA classes III/IV, IVCD	Improved exercise capacity, NYHA class, QOL, LVEF, and LVEDD; decreased hospitalizations
MUSTIC[26]	Randomized, crossover	131 patients with DCM, NYHA class III, IVCD, with sinus rhythm and atrial fibrillation	Improved exercise capacity, NYHA class, and QOL; decreased hospitalizations. Improvement slightly less in atrial fibrillation group
InSync[10]	Prospective, multicenter	117 patients with DCM, NYHA classes III/IV, IVCD	Improved exercise capacity, QOL, NYHA class, QRS duration, LVEF, and LVEDD
Ventak CHF[29]	Prospective, blinded, randomized	32 patients with DCM, NYHA classes II/III/IV, IVCD with indications for ICD placement	Significant decrease in appropriate therapy for ventricular arrhythmias with CRT versus no CRT

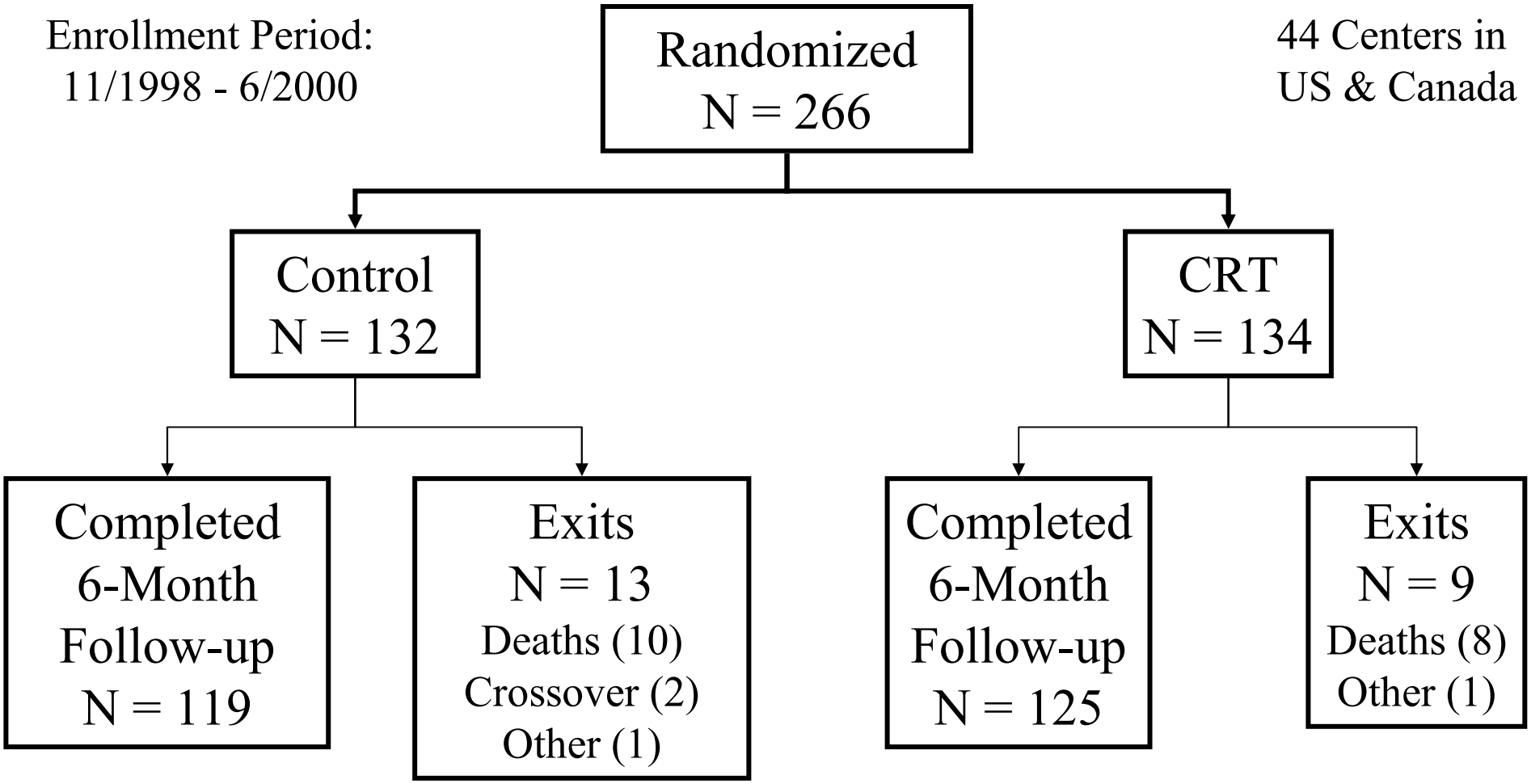
CRT, cardiac resynchronization therapy; DCM, dilated cardiomyopathy; ICD, implantable cardioverter defibrillator; IVCD, intraventricular conduction delay; LVEDD, left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; QOL, quality of life.

MIRACLE

Study Population

- ≥ 18 years of age
- NYHA Functional Class III or IV
- QRS duration ≥ 130 msec
- LVEF ≤ 35% by echocardiography
- LVEDD ≥ 55 mm (echo measure)
- Stable HF medical regimen for ≥ 1-month
  - ACE-I or substitute, if tolerated
  - β-blocker - stable regimen for ≥ 3-months

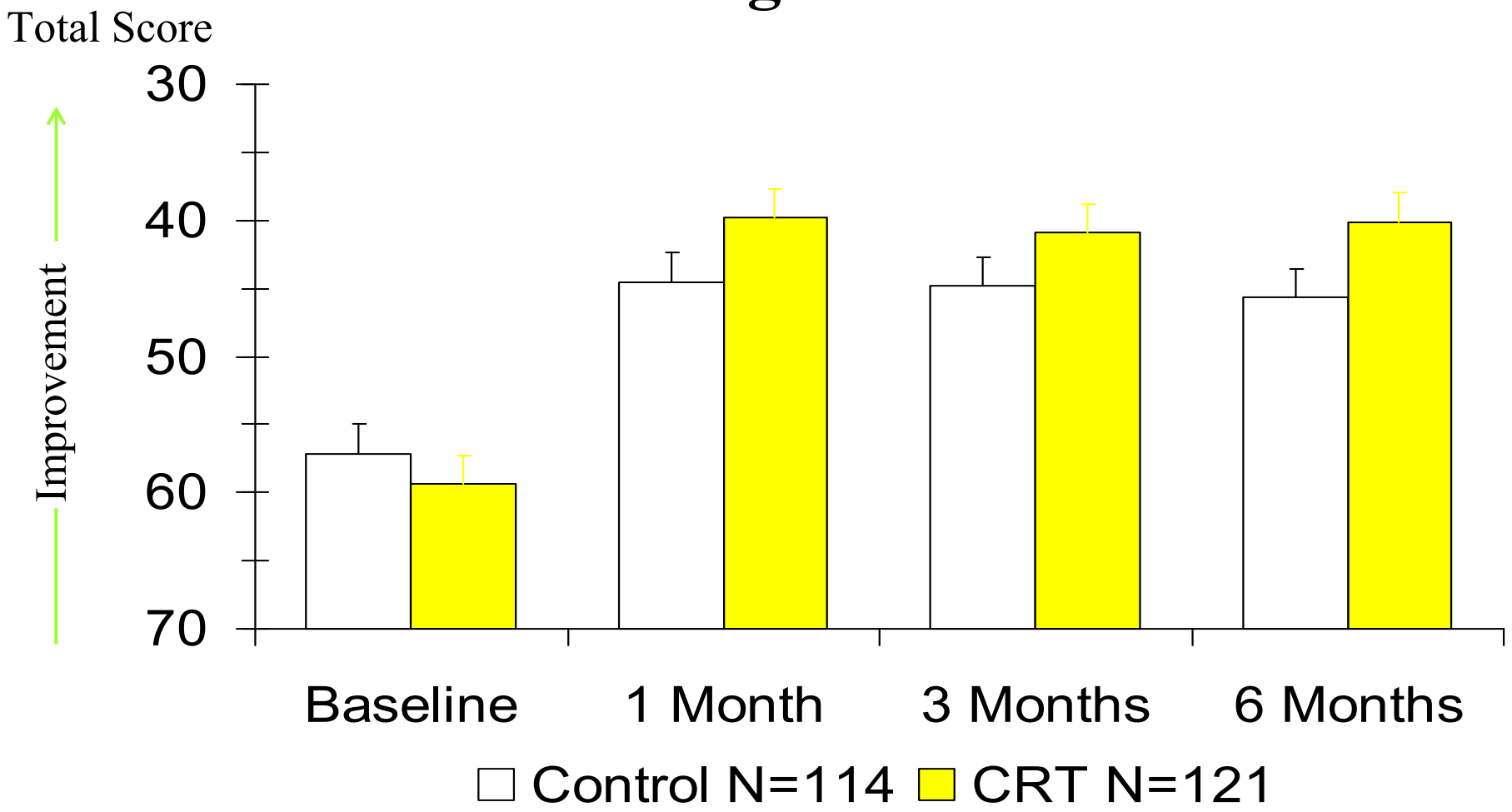
MIRACLE Study Patient Status



MIRACLE

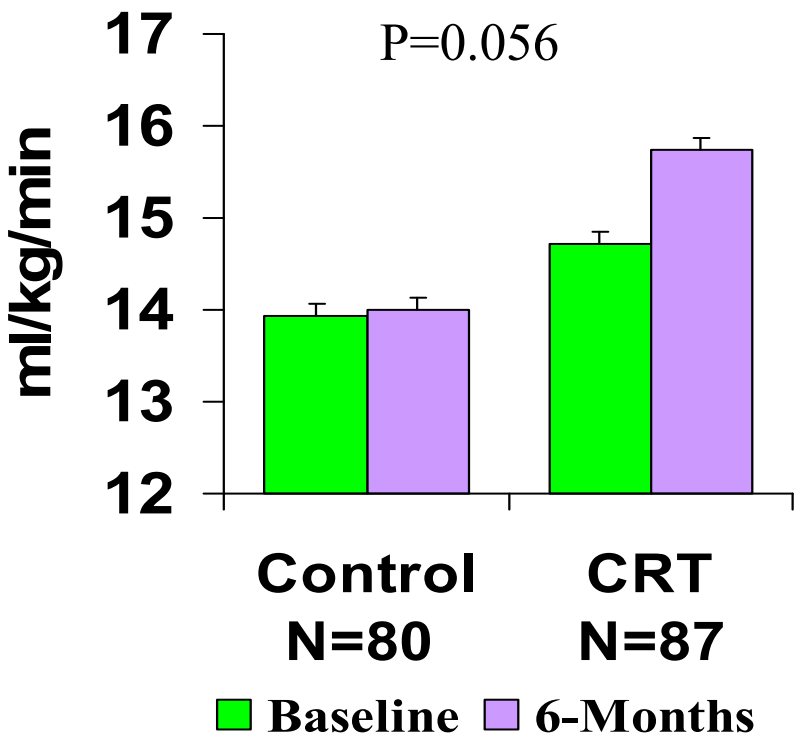
Quality of Life

Minnesota Living With Heart Failure Score

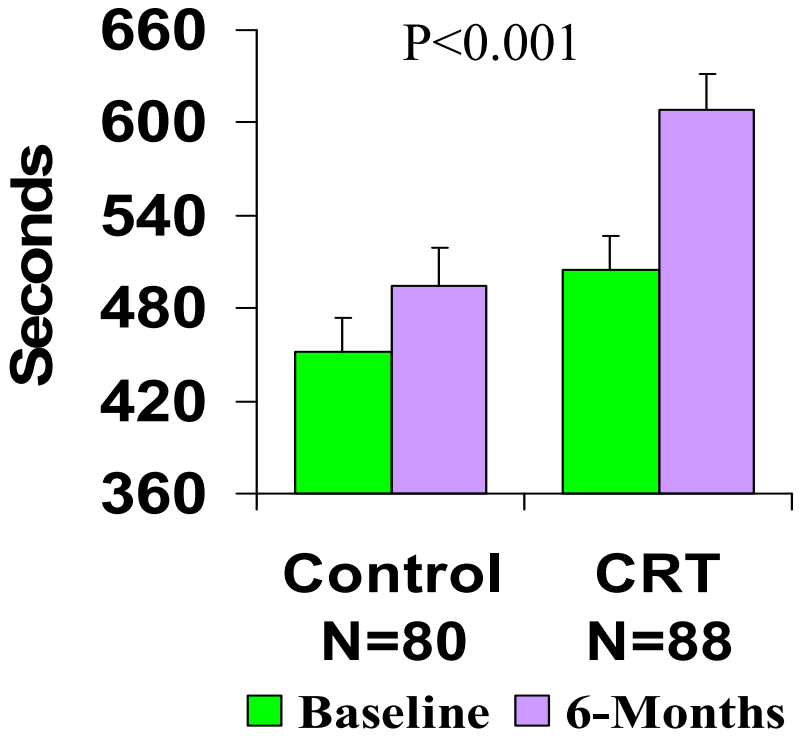


Metabolic Exercise Testing

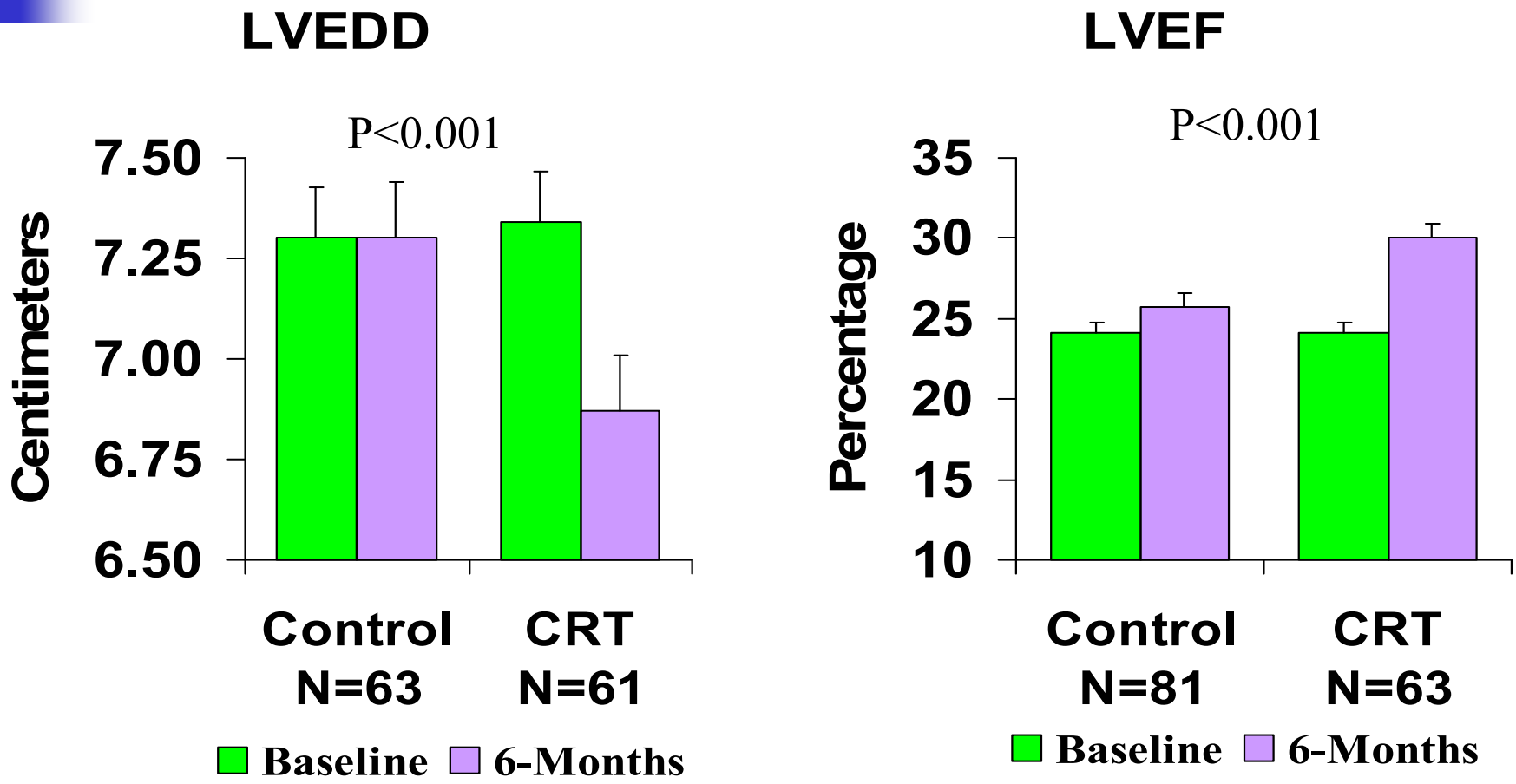
Peak VO_2



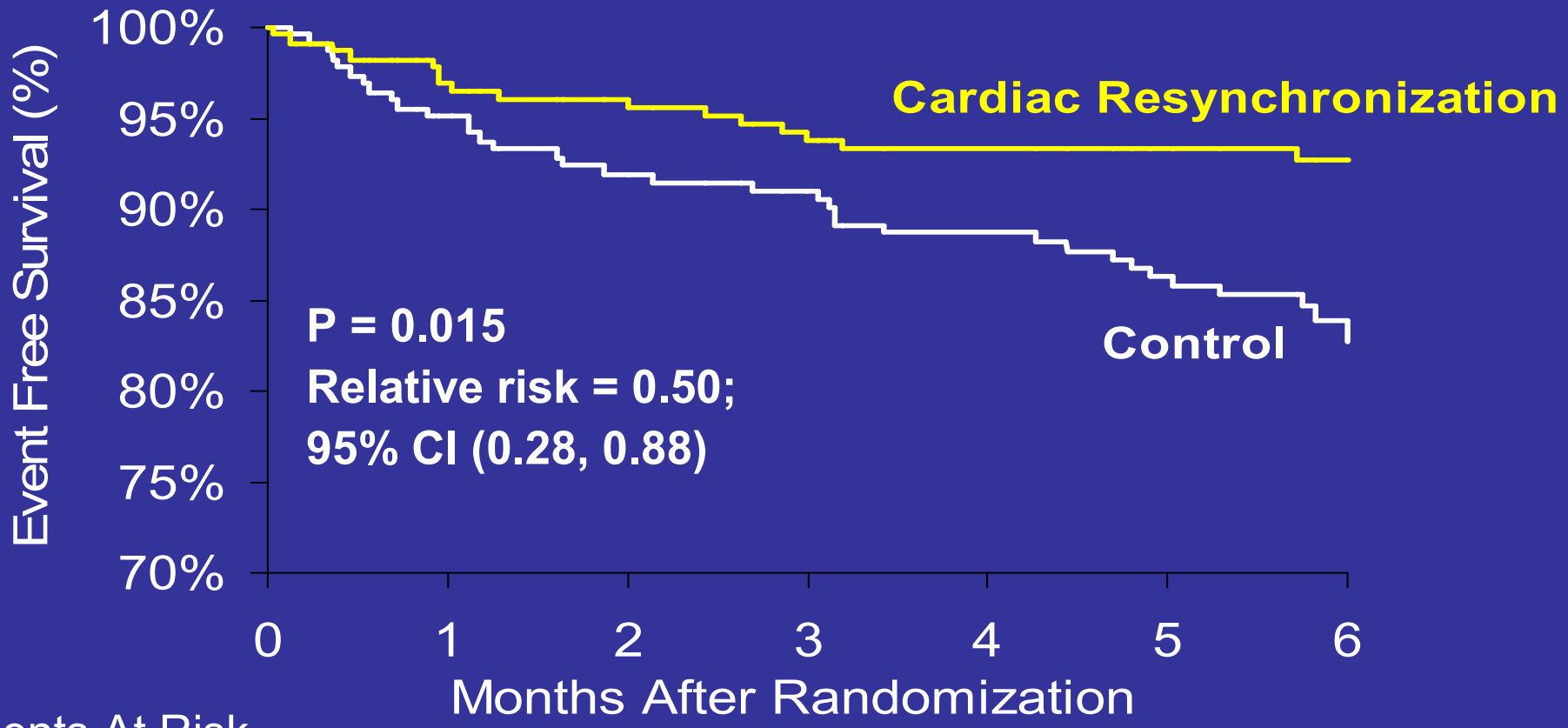
Total Exercise Time



Echocardiographic Parameters



Risk of Hospitalization for Worsening Heart Failure



Patients At Risk

Control	225	214	204	197	191	179	70
CRT	228	218	213	209	204	201	99

Abraham W, et.al. ACC 2002

COMPANION STUDY

Inclusion Criteria

NYHA Class III or IV

QRS > 120 ms

LVEF ≤ 35%, LVEDD > 60 mm

No brady or tachy device indications

History of CHF hospitalization in last 12 months

Optimal pharmacological therapy

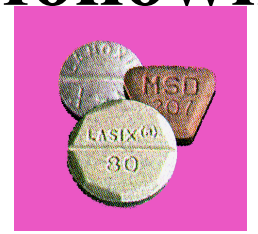
- Betablocker (3 months)
- All other HF drugs (1month)

COMPANION STUDY

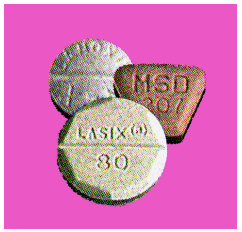
Study Design: Cohort Study

1932 patients, 1:2:2 to one of the following 3 arms:

- **Optimal pharmacological therapy (OPT)**



- **OPT + Biventricular CRT**



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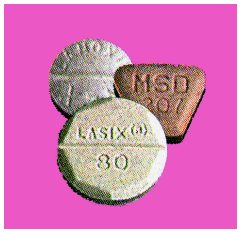
- **OPT + Biventricular CRT + ICD**



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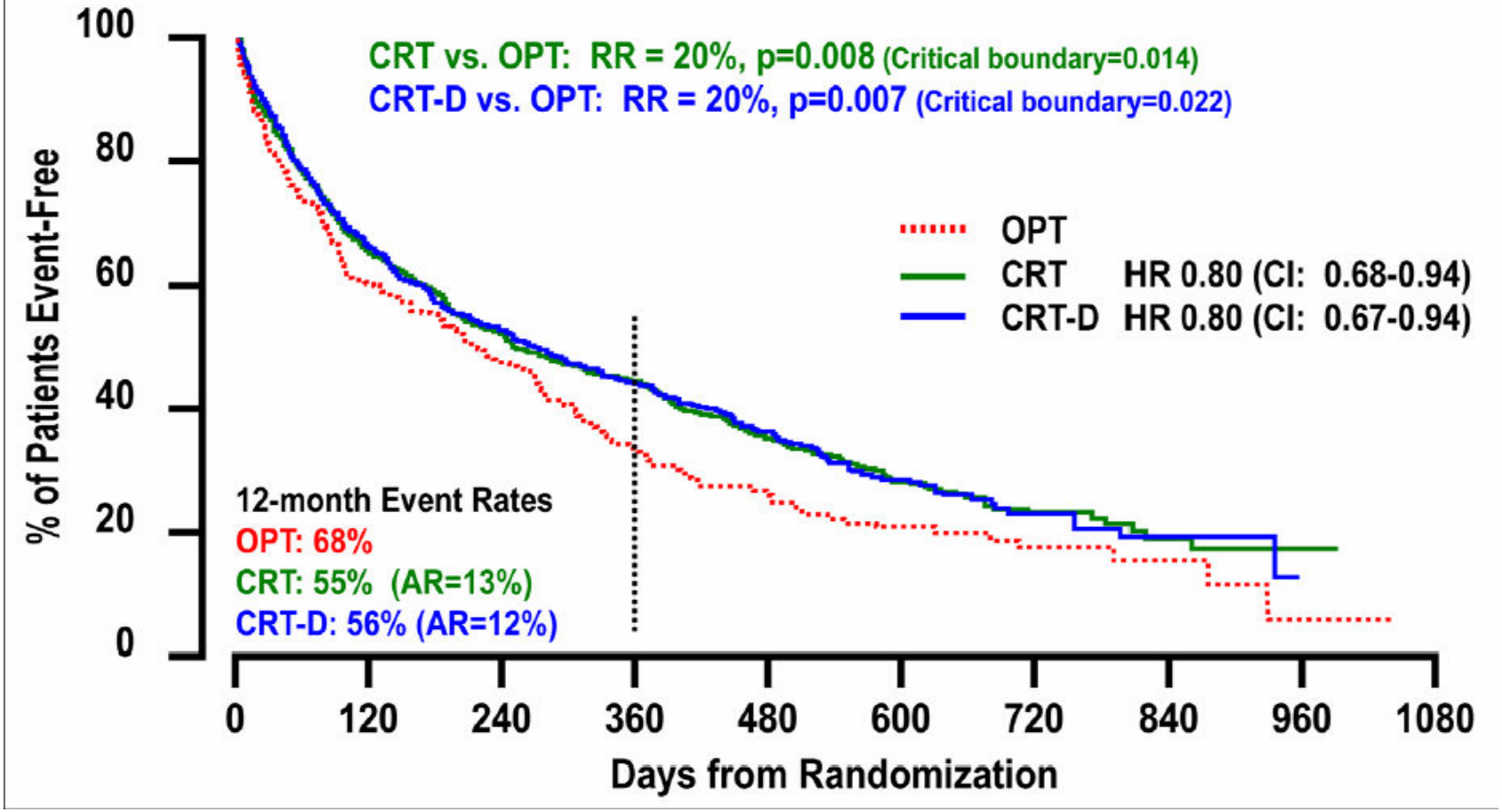
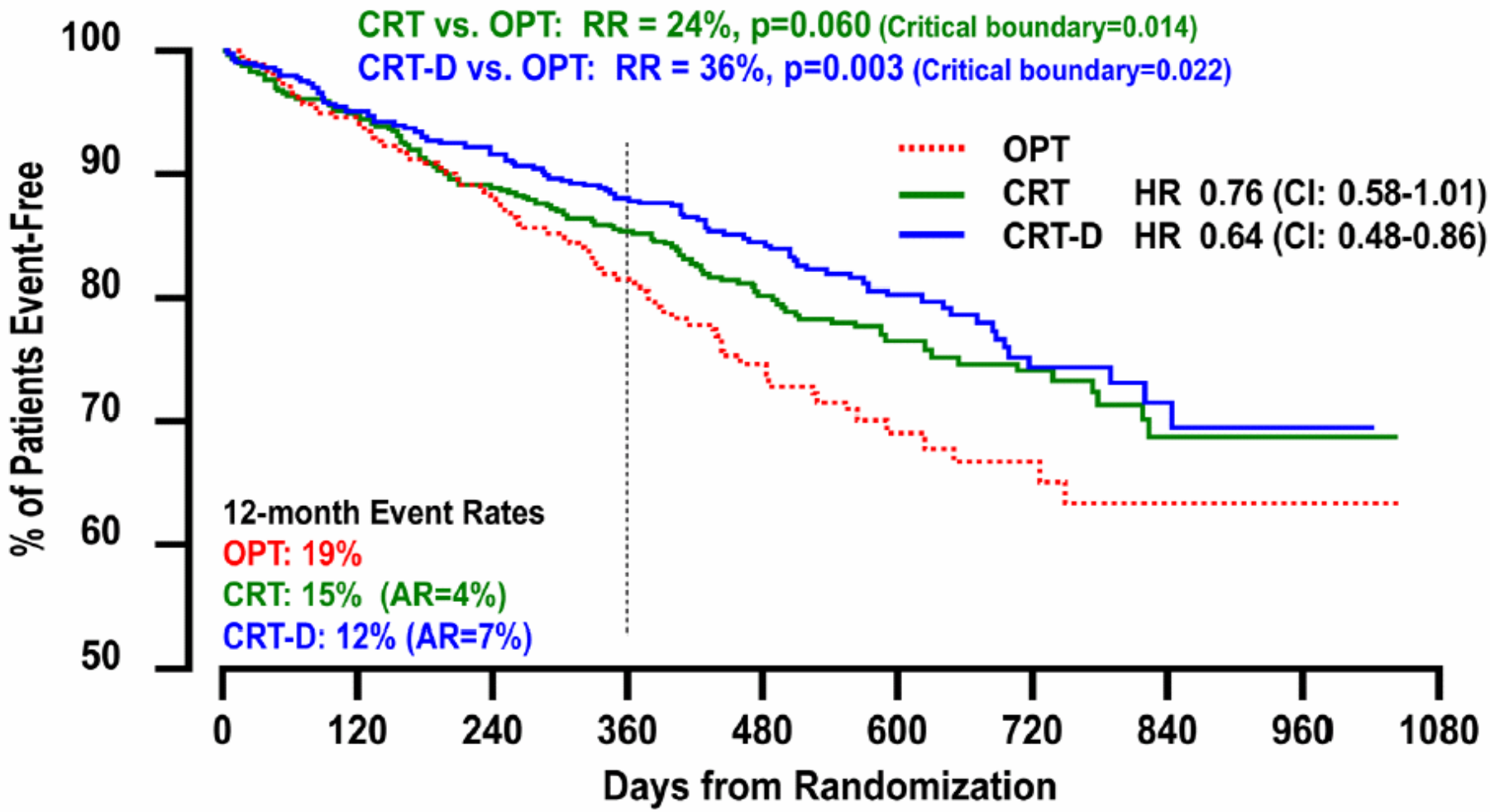
COMPANION: Primary Endpoint
Death or Any Hospitalization



COMPANION: Secondary Endpoint
All-Cause Mortality



Asynchrony, not QRS Duration Predicts Improvement

- Acute improvement can be predicted by the degree of mechanical baseline asynchrony [1,2]
- Furthermore, the improvement in function is linearly correlated with the improvement in synchrony [3]

1 Yu et al., PACE, 23:II9, 2000
2 Nelson et al., Circulation, 101:2703-9, 2001
3 Yu et a., PACE, 23:II148, 2000

Device Recommendations for CHF and Primary Prevention of Sudden Death

	CAD				NISCM	
	EVEF ≤0.35		LVEF 0.36–0.40		LVEF ≤0.35	
NYHA Class	Wide QRS	No Wide QRS	Wide QRS	No Wide QRS	Wide QRS	No Wide QRS
I	No device/EPST*	No device/EPST*	No device/EPST	No device/EPST	No device	No device
II	ICD	ICD	No device/EPST	No device/EPST	ICD	ICD
III	CRTD	ICD	No device/EPST	No device/EPST	CRTD	ICD
IV	CRT (D)	No device	No device	EPST	CRT (D)	No device

*If nonsustained VT is present and MI occurred >40 days age.

CAD indicates coronary artery disease, NISCM, nonischemic cardiomyopathy, and EPST, electrophysiology testing.

Patient selection for CRT

Current FDA Labeling

- LVEF <35%
- NYHA class III or IV
- Optimized medical program
- Normal sinus rhythm
- QRS>130msec

Patient selection for CRT

-*Predictor of response*

- 20-30% do not respond
- Increasingly recognized that QRS duration alone inadequate
- Optimal pre-implant parameters for response to CRT remain to be defined

CRT in Children and young adult with Congenital heart disease

- Acute effects of temporary epicardial CRT in 20 patients with evidence of AV or intraventricular conduction delay & need for inotropic support
- Used temporary pacing wire(RA, RV, LV) after CHD PO
- CRT resulted in improved arterial systolic, diastolic, and pulse pressure
- Correlated well with initial QRS duration and degree of QRS shortening
- Temporay epicardial CRT could be used as an adjunct in the immediate PO period in CHD patients

Janousek et al, 2001, Am J Cardiology

CRT in Children and young adult with Congenital heart disease

- Acute effect of temporary epicardial CRT at surgery in 29 patients with CHD PO,
- Patient with Single ventricle and biventricular anatomy
- Significant increase in systolic blood pressure and CI, and shortening of QRS duration
- Facilitated weaning from CPB in two patient
- ➢ Epicardial CRT could be used as an adjunct PO therapy

Zimmerman et al, 2003 Ann Thorac Surg

Electrical Resynchronization

A Novel Therapy for the Failing Right Ventricle

Anne M. Dubin, MD; Jeffrey A. Feinstein, MD; V. Mohan Reddy, MD; Frank L. Hanley, MD;
George F. Van Hare, MD; David N. Rosenthal, MD

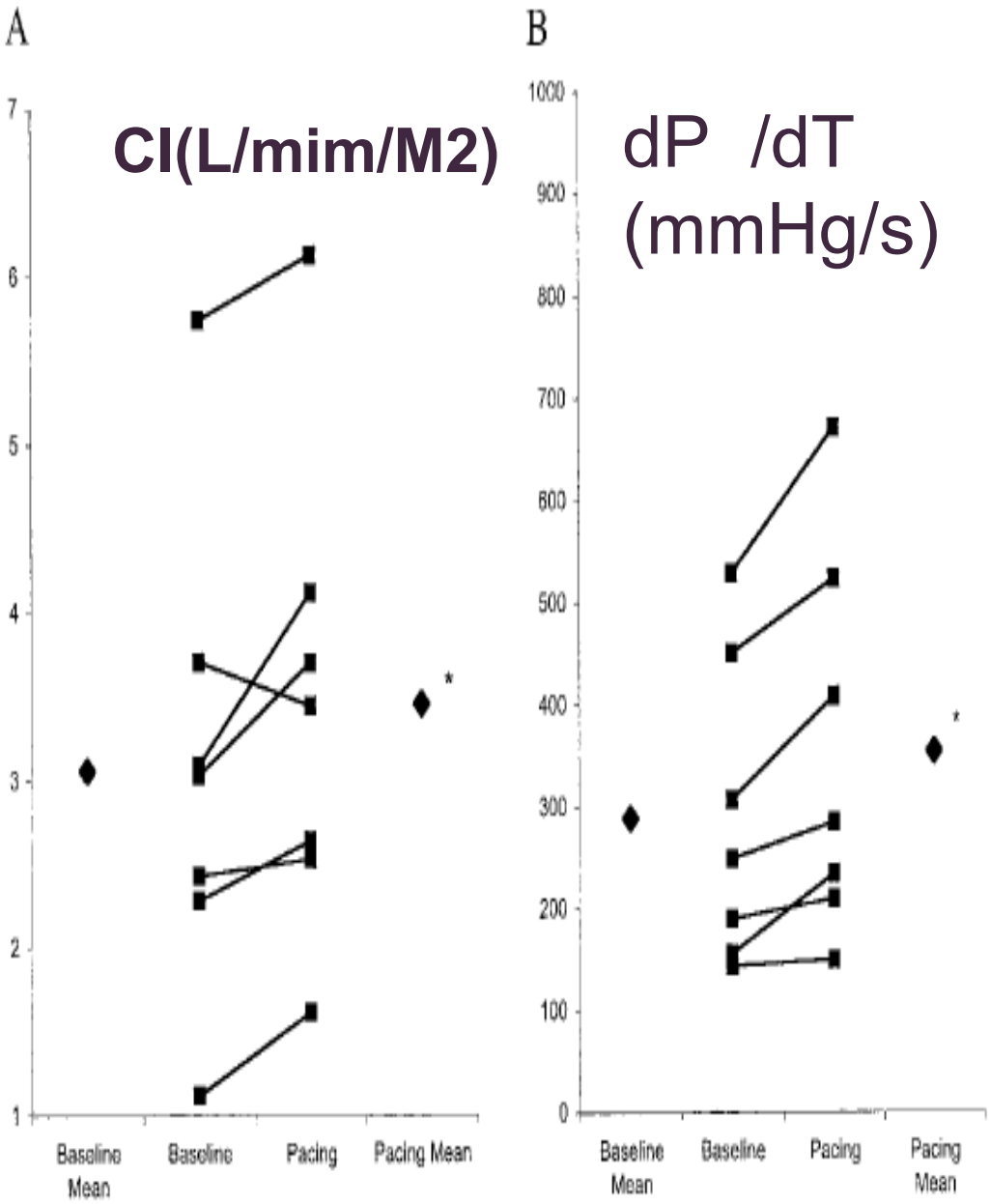
Background—Many patients with congenital heart disease develop right ventricular (RV) failure due to anatomy and prior therapy. RV problems may include right bundle-branch block (RBBB), volume loading, and chamber enlargement.

Methods and Results—We studied 7 patients with RV dysfunction and RBBB, using a predefined pacing protocol. QRS duration, cardiac index (CI), and RV dP/dt were measured in 4 different pacing states. Atrioventricular pacing improved CI and RV dP/dt_{max} and decreased QRS duration as compared with atrial pacing or sinus rhythm.

mance. RV resynchronization is a promising novel therapy for patients with RV failure. (***Circulation***. **2003;107:2287-2289.)**

Baseline Characteristics and Hemodynamic Data of Study Subjects

	Mean (SD)	Minimum	Maximum
Age, y	23.6 (18.7)	1.7	53
Height, cm	147 (42)	70	178
Weight, kg	66.5 (38)	9.3	81
Baseline cycle length, ms	828 (123)	590	1000
QRS duration, ms	166 (39)	140	200
Hemoglobin, gm/dL	13.2 (2.8)	10	17
NYHA class	2–4	2	4
Cardiac index, L/min per m^2	2.85 (1.19)	1.12	4.60
Systolic blood pressure, mm Hg	114 (23)	90	148
Diastolic blood pressure, mm Hg	69 (14)	54	88
RV peak pressure, mm Hg	54 (36)	26	130
RV/LV ratio	0.47 (0.25)	0.18	0.94
RV minimum pressure, mm Hg	4 (5)	0	12
RV end-diastolic pressure, mm Hg	14 (9)	5	28
RV dP/dt_{max}, mm Hg/s	289 (151)	149	548



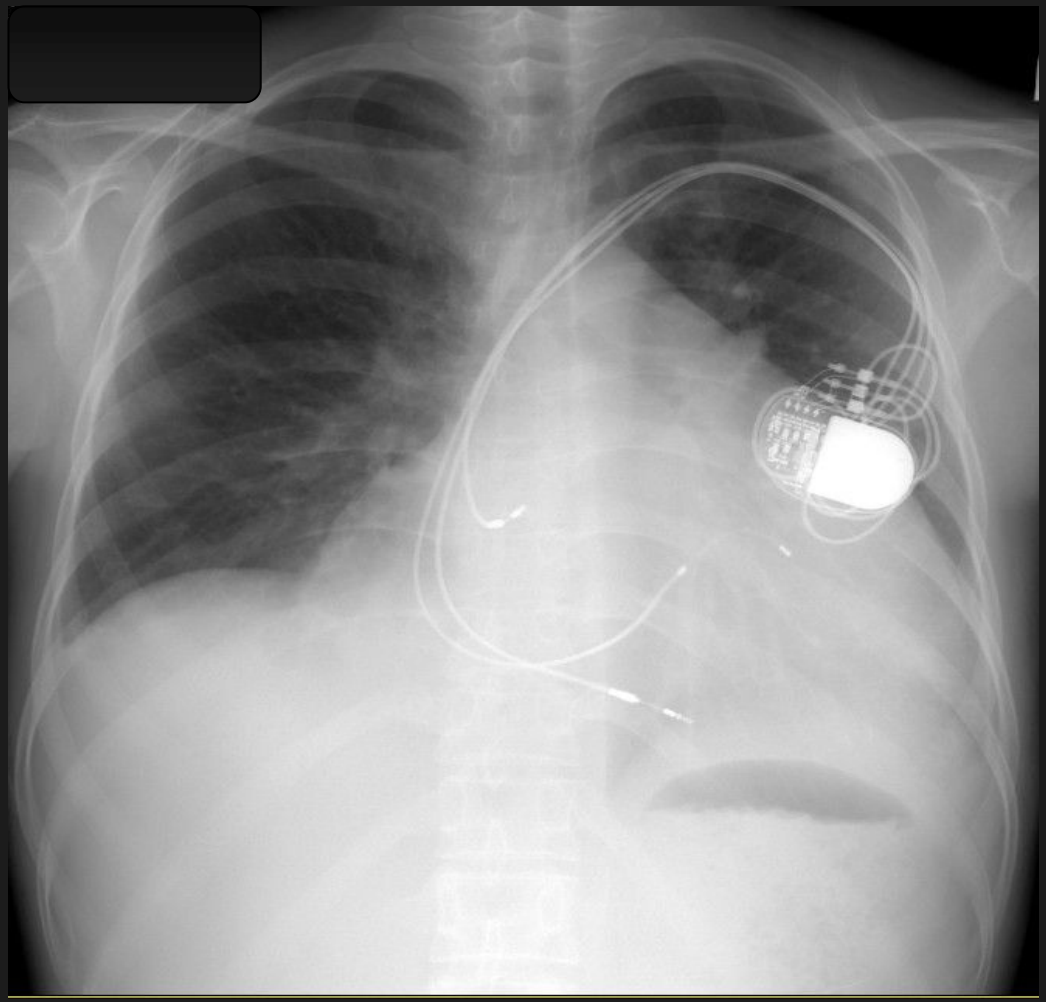
Unsolved issues with CRT

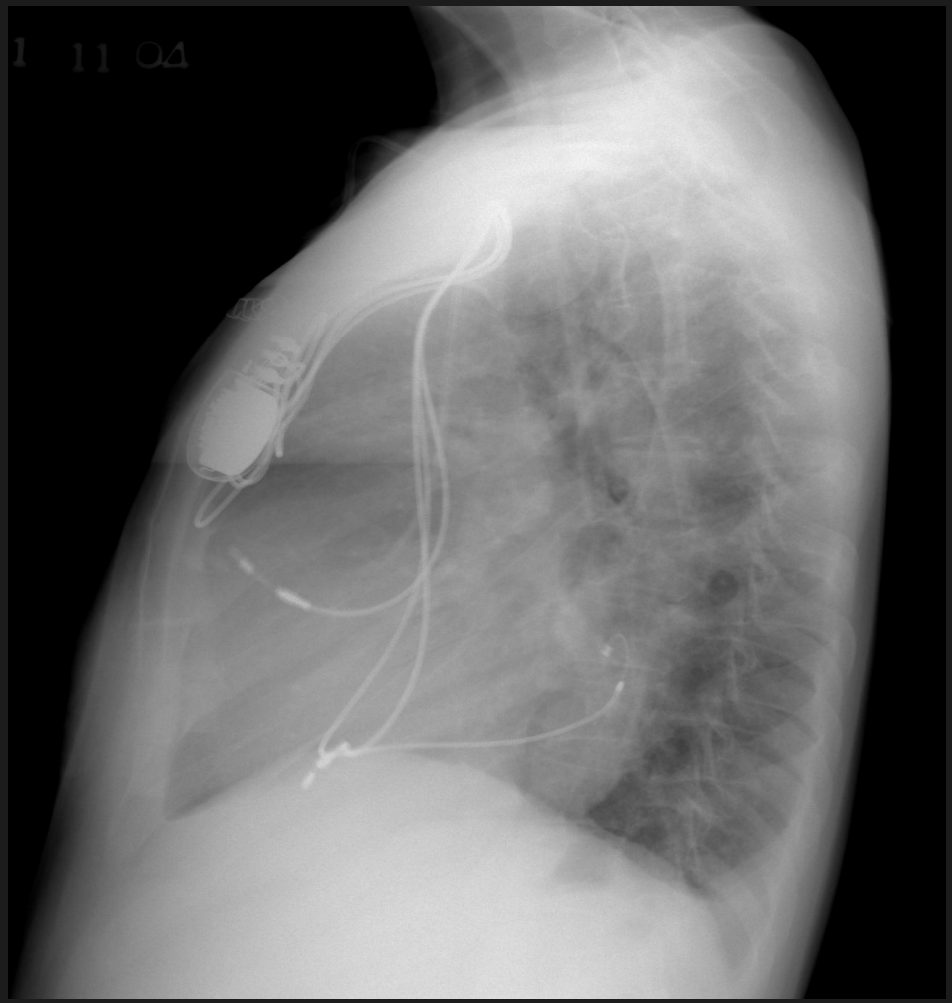
- Device placement exert a Substantial placebo response
- Assessment of mechanical asynchrony using echocariography such as TDI, strain, strain rate, tissue tracking , etc
- Uncertain whether optimization of AV delay, intraventricular resynchronization, or interventricular resynchronization
- Underlying etiology of HF(MI, scar tissue, op scar, conduit or patch)
- Extrapolation of CRT to mild(NYHA II)
- Unclear whether ICD must accompany CRT (CRTD)

Summary: Who benefits most from CRT?

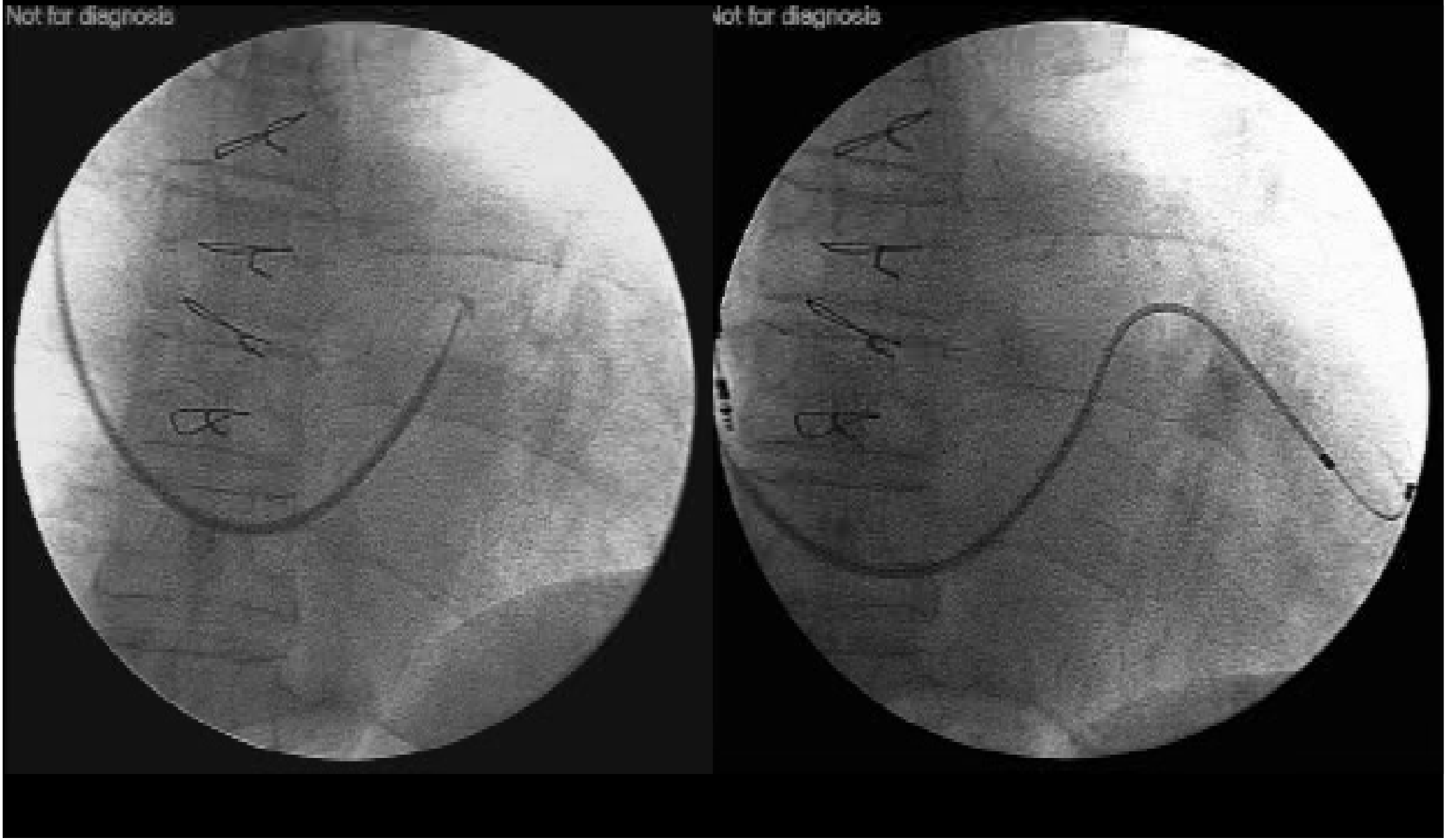
- Patients with NYHA III/IV CHF tend to show greater therapeutic benefit than those with NYHA I/II CHF
- Patients with LBBB respond consistently, whereas those with RBBB do not
- Patients with DCM tend to respond more than those with ICM
- 80% of patients with QRS > 150 ms improve their hemodynamics with CRT(mechanical synchrony and electrical synchrony are not synonymous)
- Improvements in asynchrony seem to be the determinant of the improvements obtained with CRT, and this may be independent of QRS width
- Heterogeneous nature of pediatric patients with CHD pose a special challenge in assessing the short-term and long-term efficacy of CRT

Biventricular Pacing

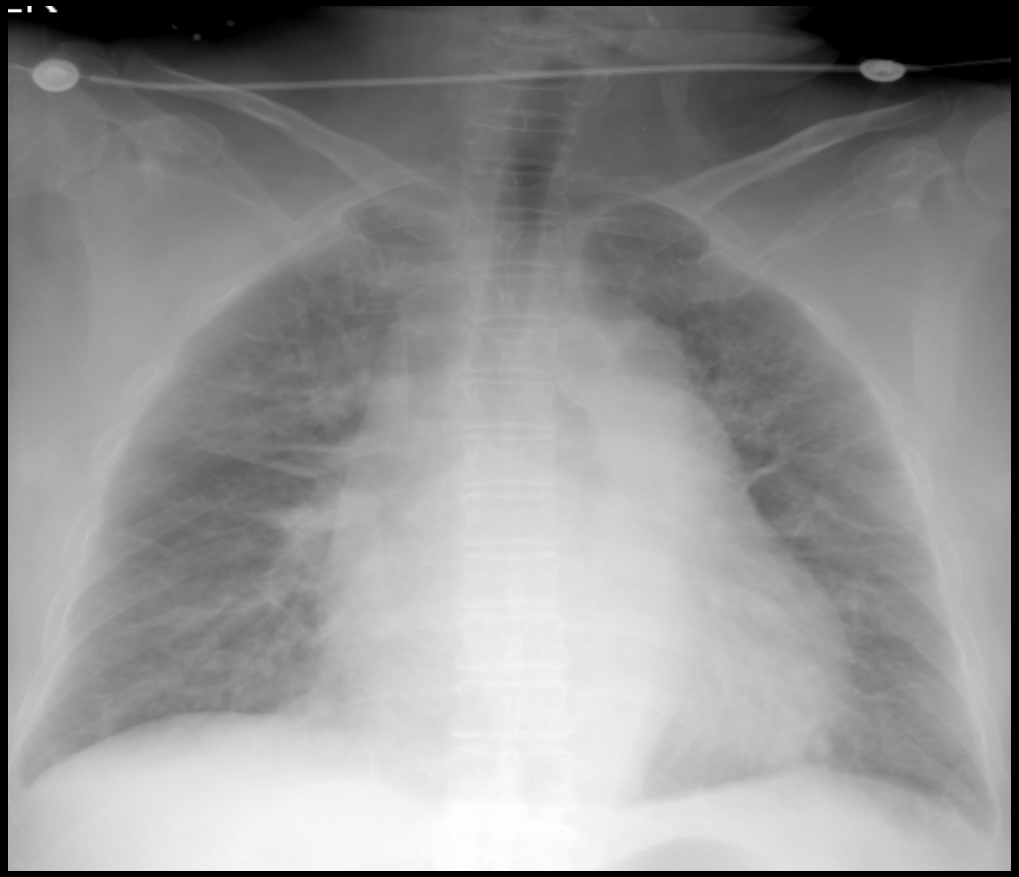




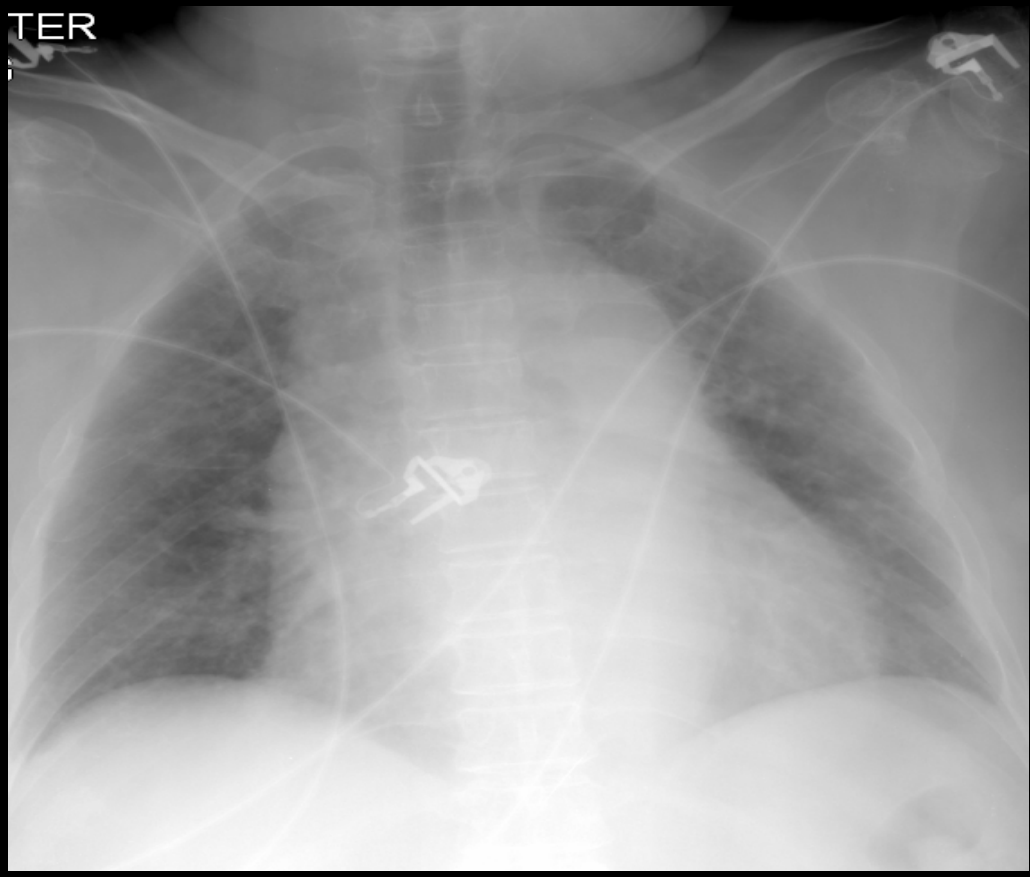
Lead introduction into coronary vein



75/F with Dyspnea (NYHA IV)
EF: 11%, Sinus rhythm, LBBB

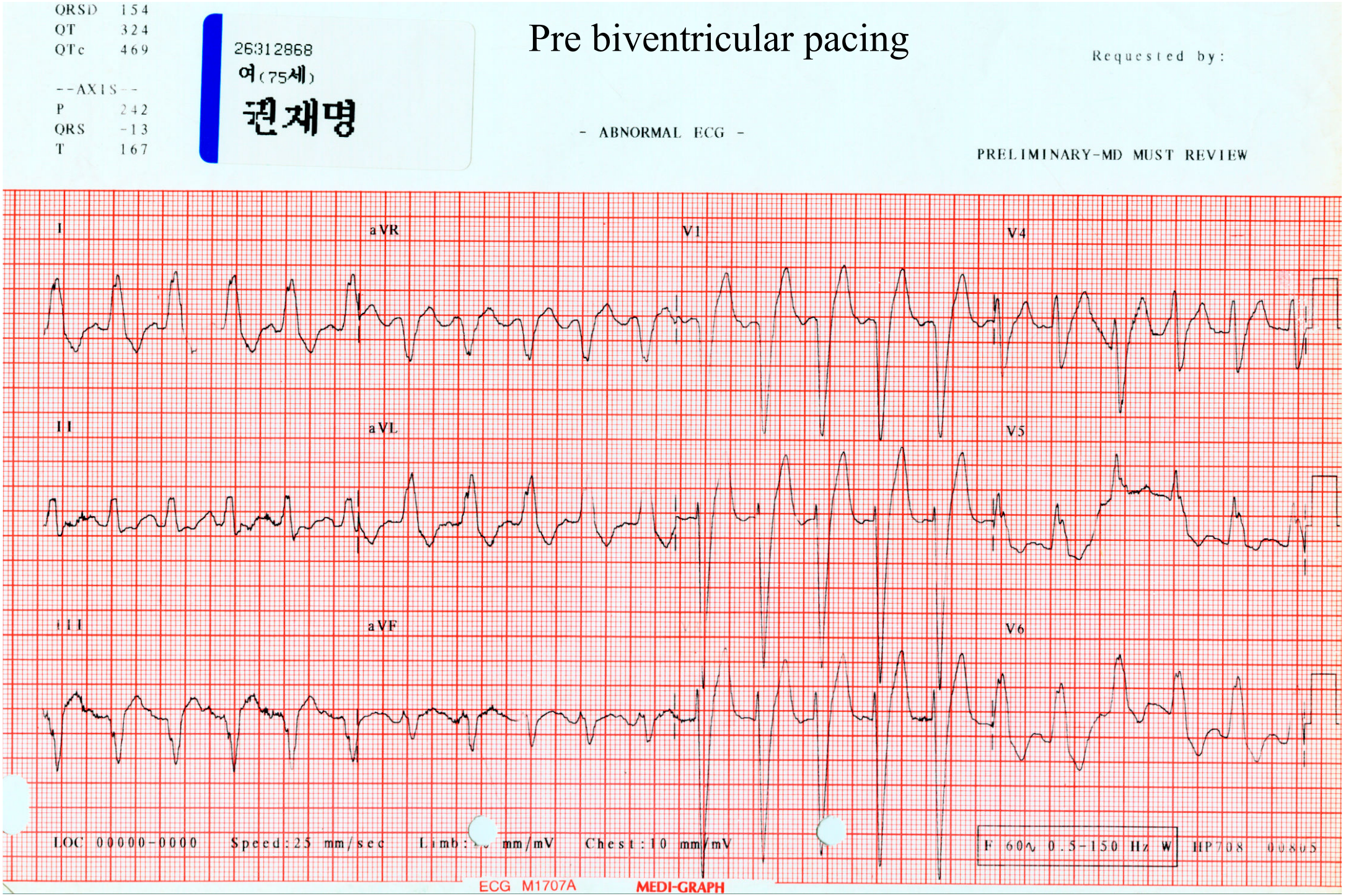


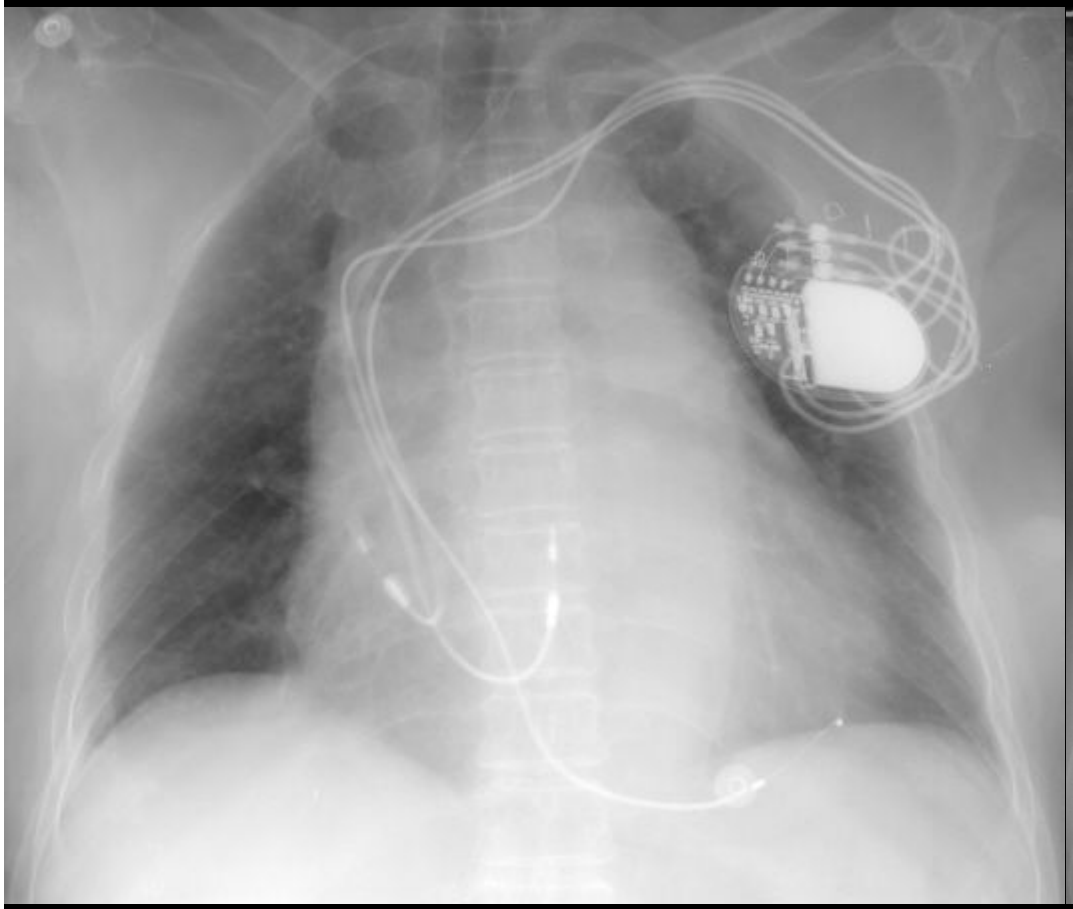
On admission



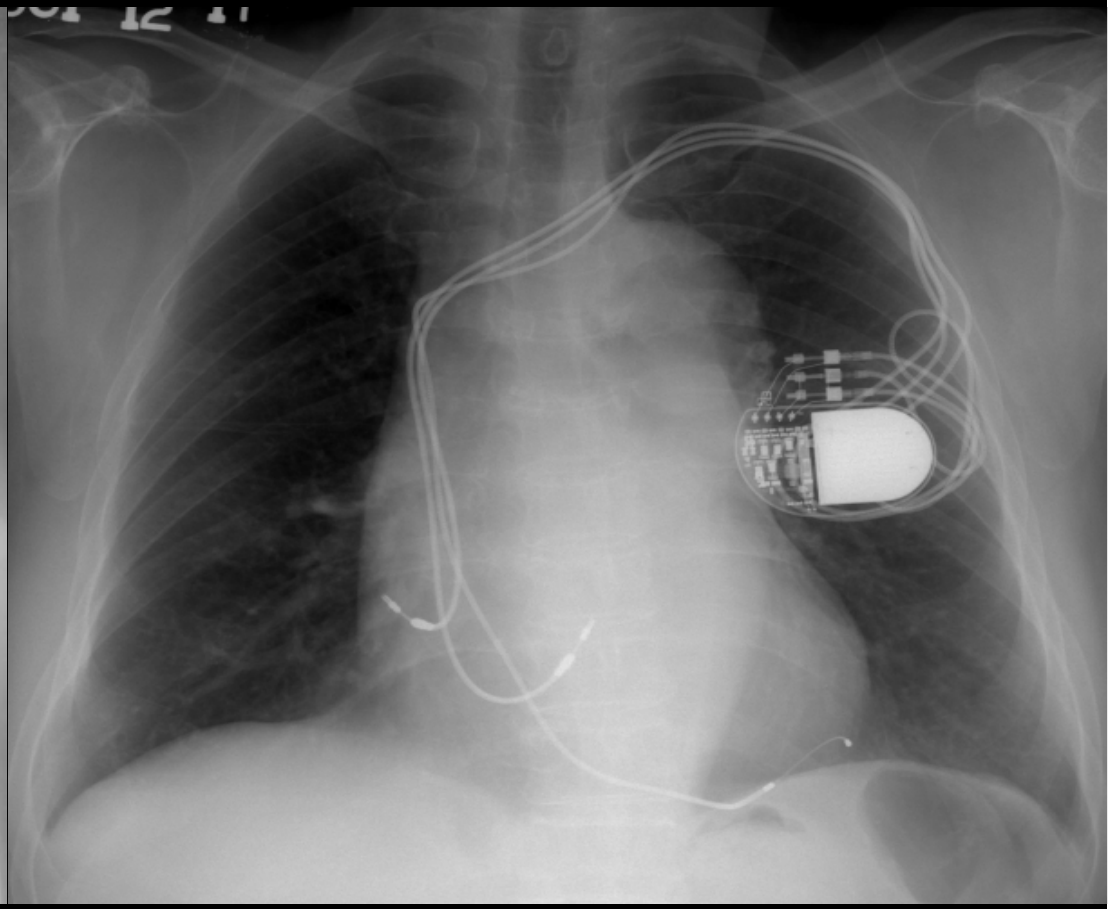
5th day in CCU

Pre biventricular pacing

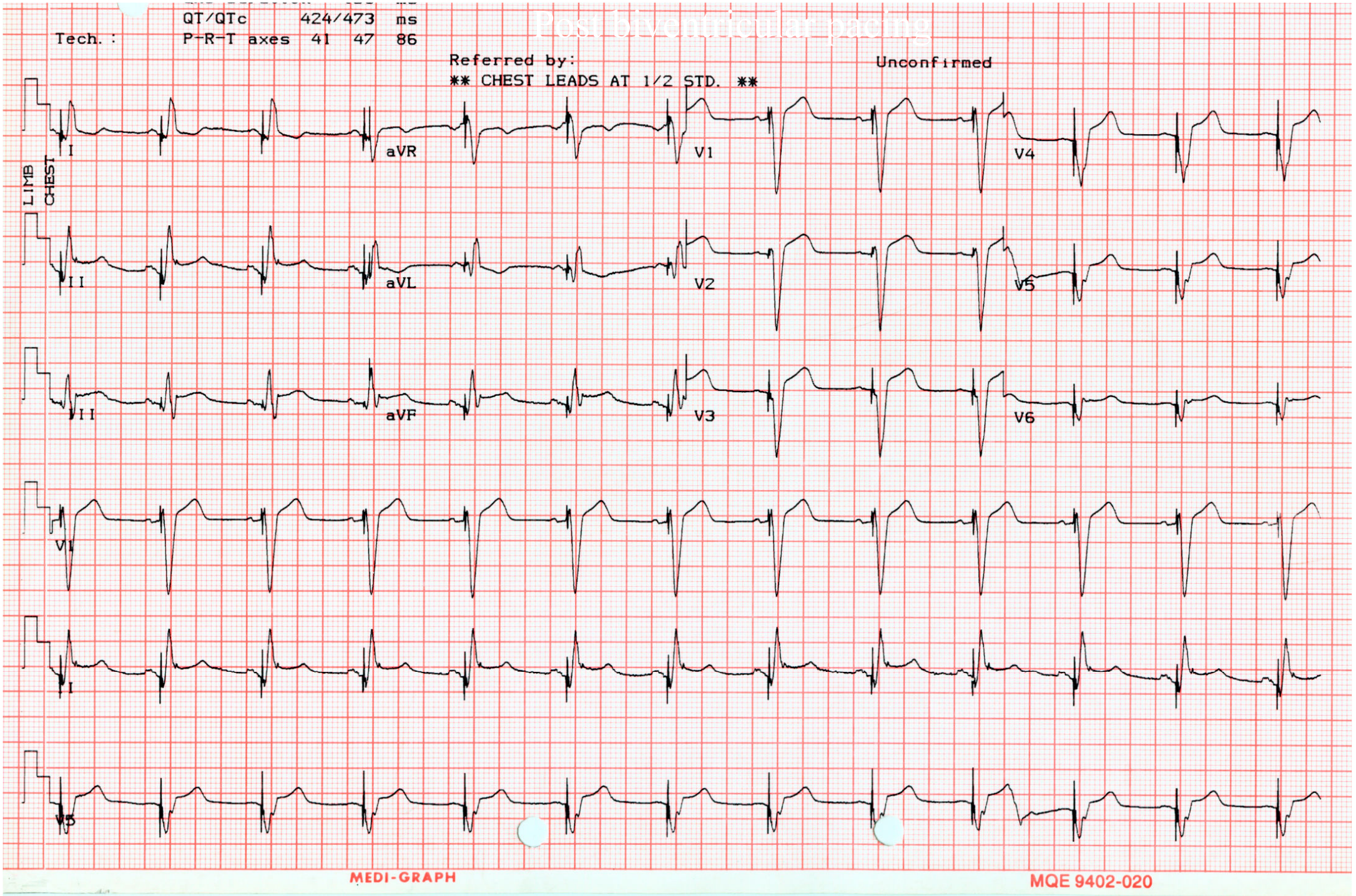


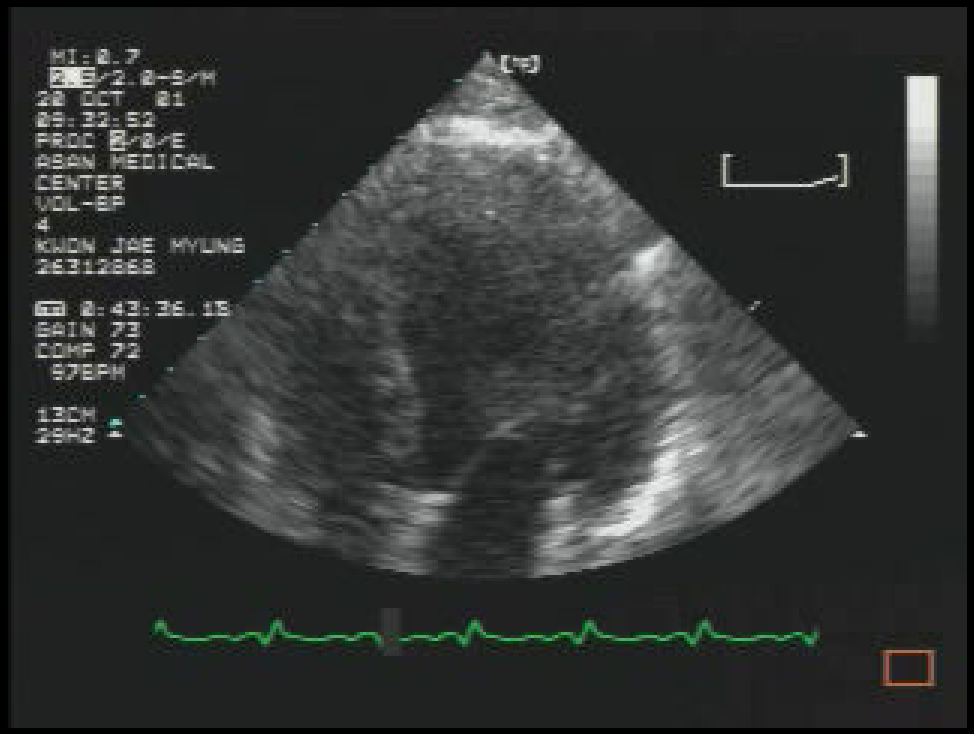


The next day after resynchronization

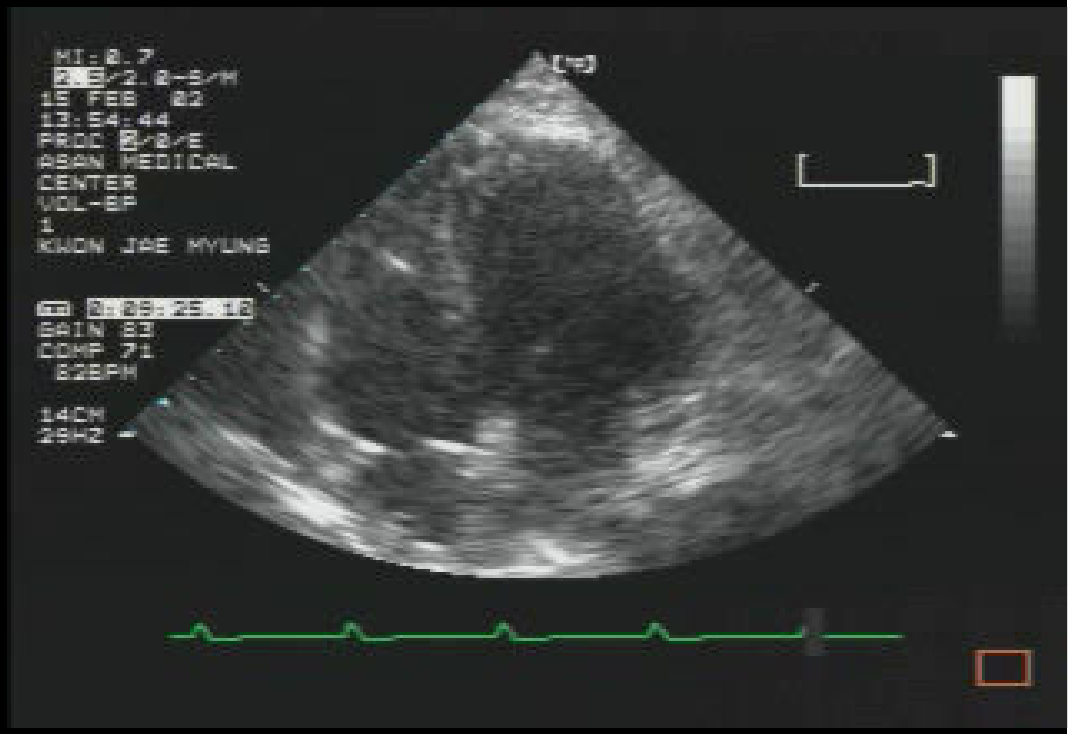


6 months after resynchronization


Post Biventricular pacing
QT/QTc 424/473 ms
P-R-T axes 41 47 86
Tech. :
Referred by:
** CHEST LEADS AT 1/2 STD. **
Unconfirmed
LIMB
CHEST
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
MEDI-GRAPH
MQE 9402-020



Before synchronization
EF=11%



6 mo after cardiac
Resynchronization, EF=46%

Summary

- A Major focus in children with HF is growth and nutrition. Supplementary nutritional support is needed.
- Patient with Pediatric CHF seemed to benefit from regular physical activity. Not all patients, however, are eligible for competitive sports.
- Cell therapy for patients with CHF is still in its infancy.Stem cell transfer to failing heart may be feasible, firm conclusion regarding efficacy cannot be drawn at this time
- There is preliminary evidence to suggest that selected patients with CHD may benefit from CRT, perhaps with RV or alternate pacing
- Multidiscipline team approach should be considered