

MALIGNANT ARRHYTHMIAS / SUDDEN CARDIAC DEATH

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SCD

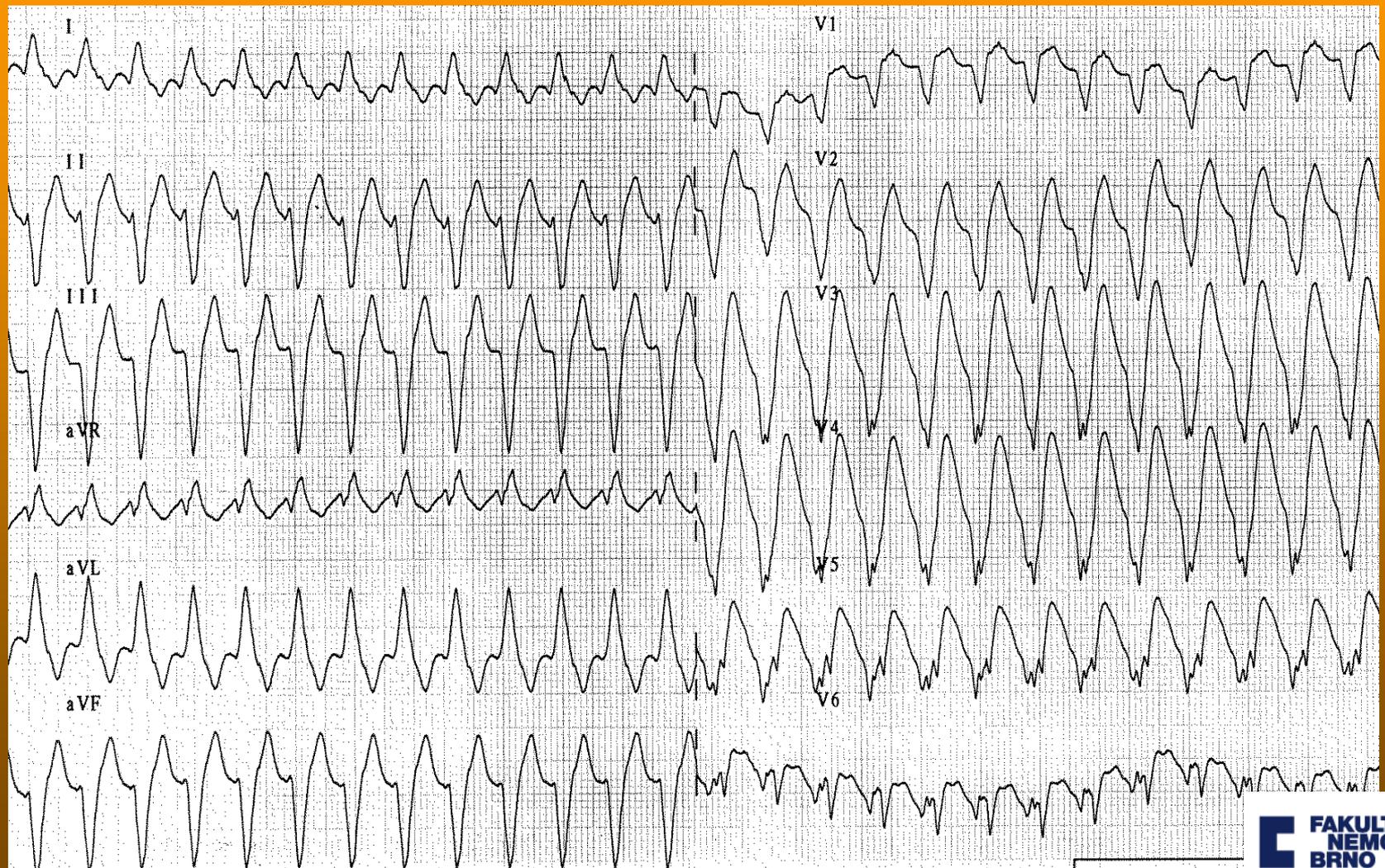
- sudden unexpected death caused by loss of heart function (1 hour time window)
- sudden collapse
- no pulse
- no breathing
- loss of consciousness



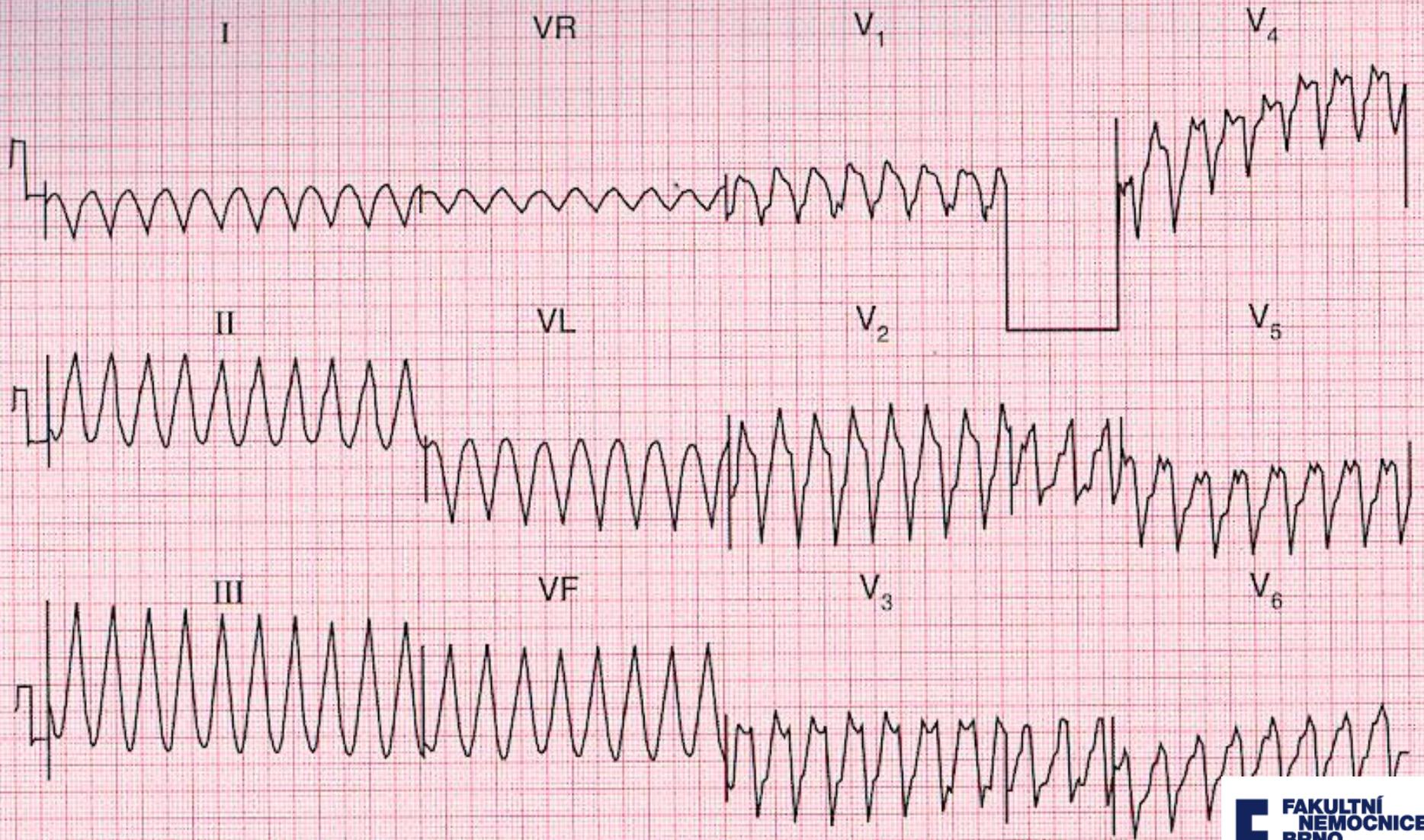
RBBB VT



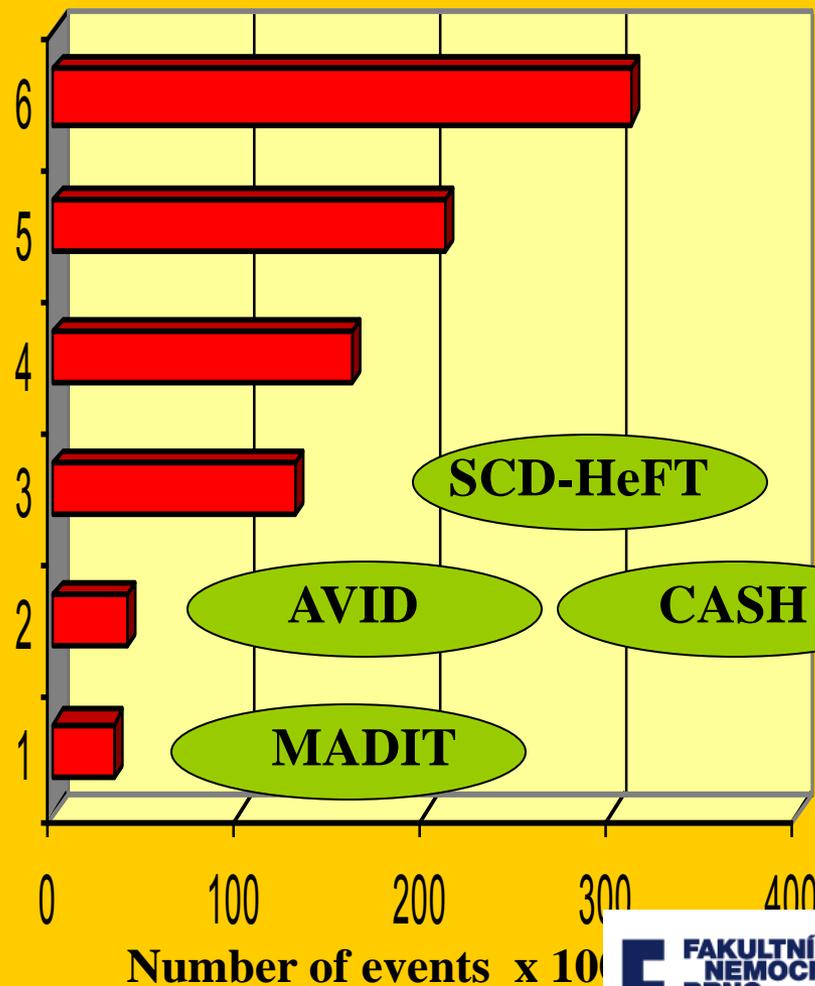
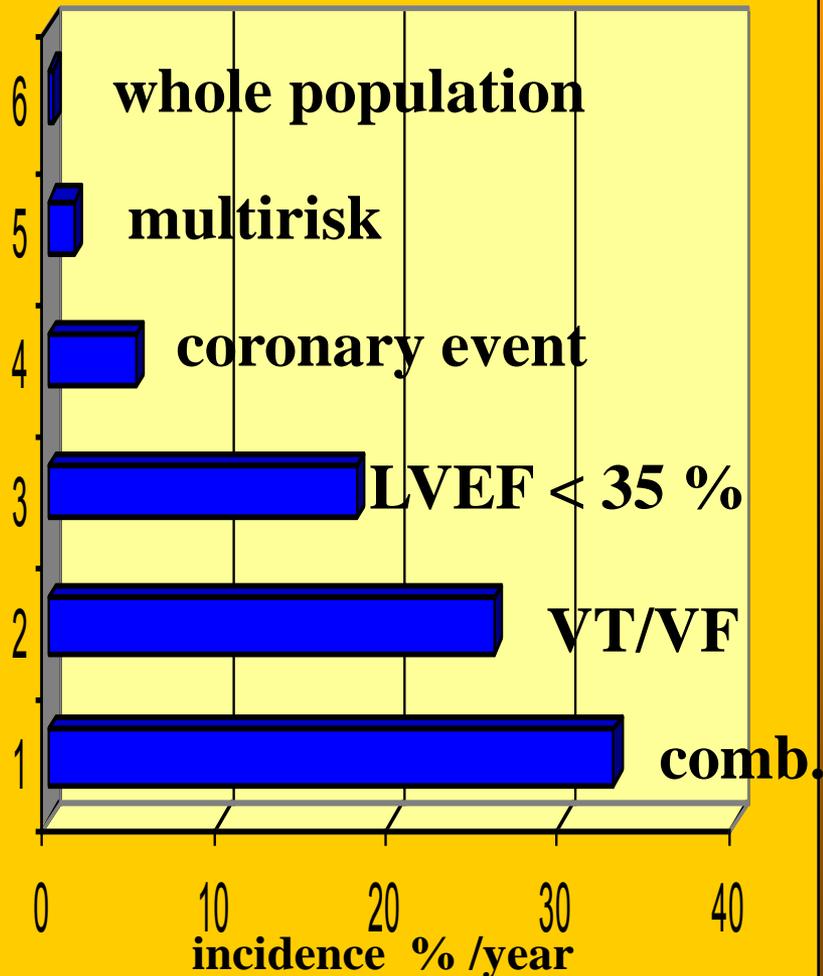
LBBB VT



FVT

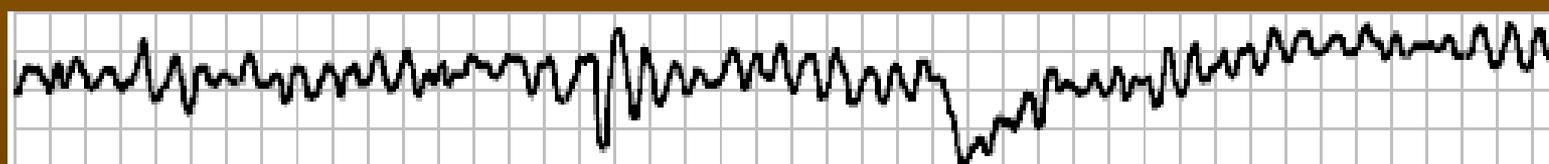
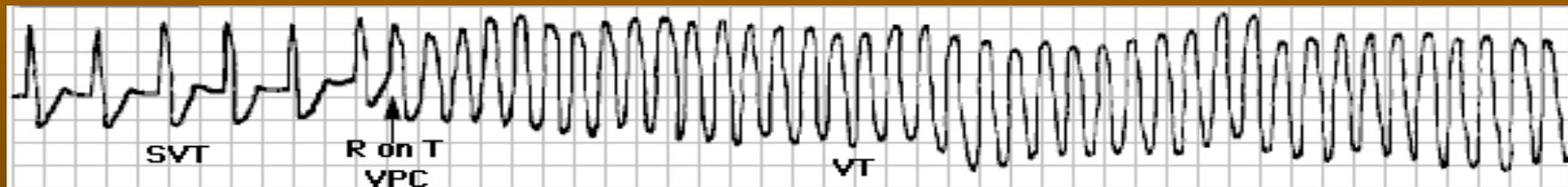
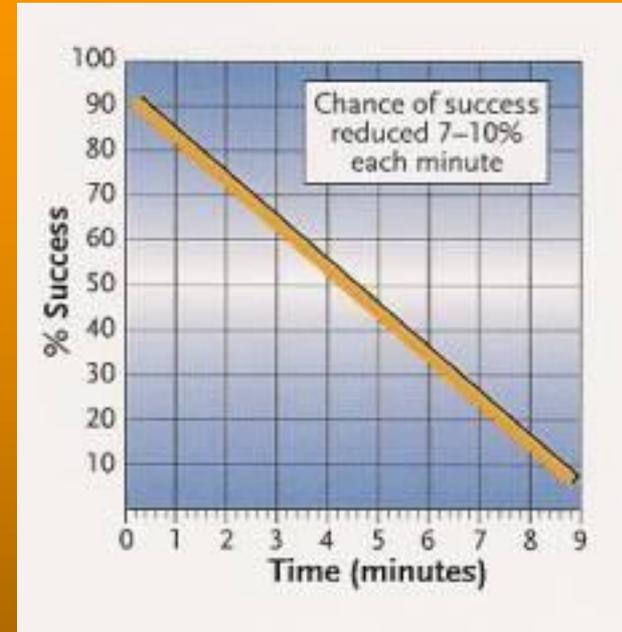


INCIDENCE / SCD TOTAL NUMBER



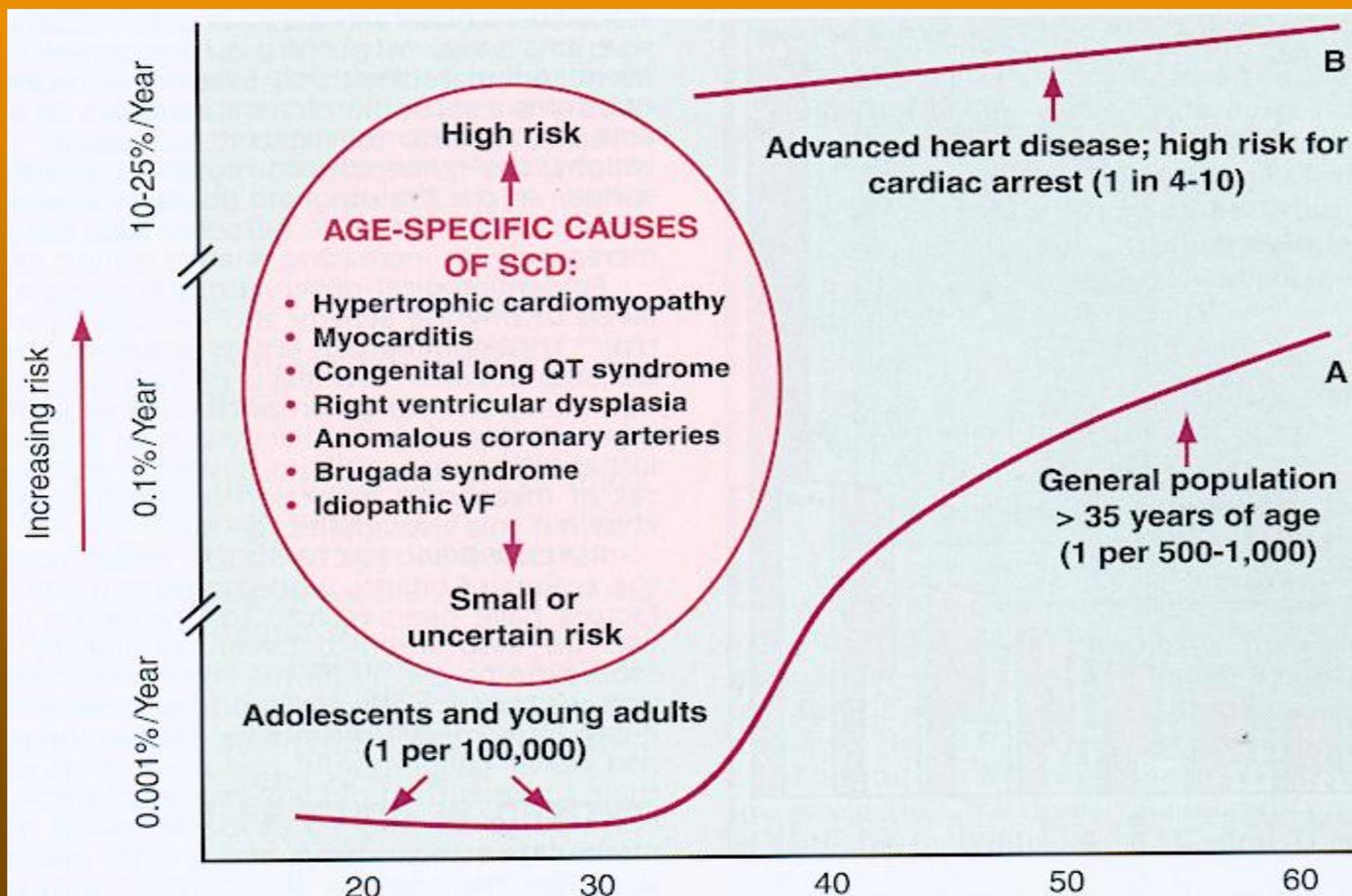
SCD - STATISTICS

SCD USA	200-400.000/ year Gillum R.F., Circul 1989
SCD EU	2.500/day Pisa Z., Sudden death 1980
Malignant arrhythmias	80 - 90% Gillum R.F., Pisa
Efficacy of CPR	10-15%



SCD x AGE

- incidence 1SCD/100.000 inhabitants < 35 (x 1/1000 u population > 35)
- acute forms CAD 20-39 - 76% SCD *Kuller et al. JAMA 1966,198:158*



PARIS PROSPECTIVE STUDY

- 7.079 man, age 43-52 years (1967-1972), follow-up 23 years

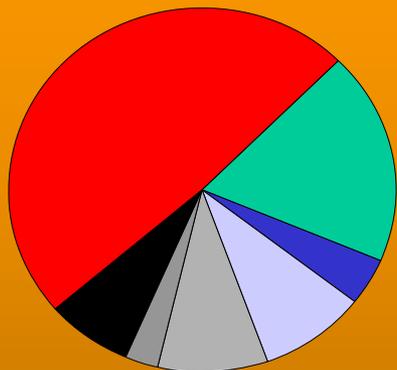
•Jouven X et al. *Circulation*. 1999;99:1978-1983

TABLE 4. Adjusted RRs Associated With Sudden Death and Fatal Myocardial Infarction Before the Age of 65 Years in the Paris Prospective Study I by Multivariate Analysis

Variables	Sudden Death at <65 y		Fatal Myocardial Infarction at <65 y	
	RR (95% CI)	P	RR (95% CI)	P
Age at entry	0.96 (0.77–1.21)	NS	1.11 (0.87–1.41)	NS
Body mass index	1.29 (1.03–1.52)	0.04	0.87 (0.70–1.08)	NS
Tobacco consumption	1.39 (1.17–1.66)	0.0002	1.29 (1.09–1.55)	0.003
Diabetic status	2.64 (1.26–5.53)	0.01	0.96 (0.30–3.07)	NS
Heart rate	1.14 (0.98–1.38)	NS	1.21 (1.00–1.45)	0.04
Systolic arterial pressure	1.23 (1.01–1.51)	0.005	1.46 (1.22–1.75)	0.0001
Cholesterol	1.40 (1.13–1.65)	0.001	1.25 (1.00–1.52)	0.05
Triglycerides	0.98 (0.80–1.22)	0.93	1.06 (0.86–1.31)	NS
Parental myocardial infarction and death at <65 y	1.73 (0.42–7.14)	NS	3.42 (1.22–9.54)	0.0
Parental sudden death at <65 y	2.00 (1.02–3.90)	0.04	0.70 (0.26–1.81)	NS

MUSTT

Schéma studie



ICHS, EFLK pod 40%, NSKT
2202 pacientů
PSK

PSK pozit.
704 p. (35%)
RANDOMIZACE

PSK neg.
1435 p.(65%)

no AA
353

EP guided terapie
351

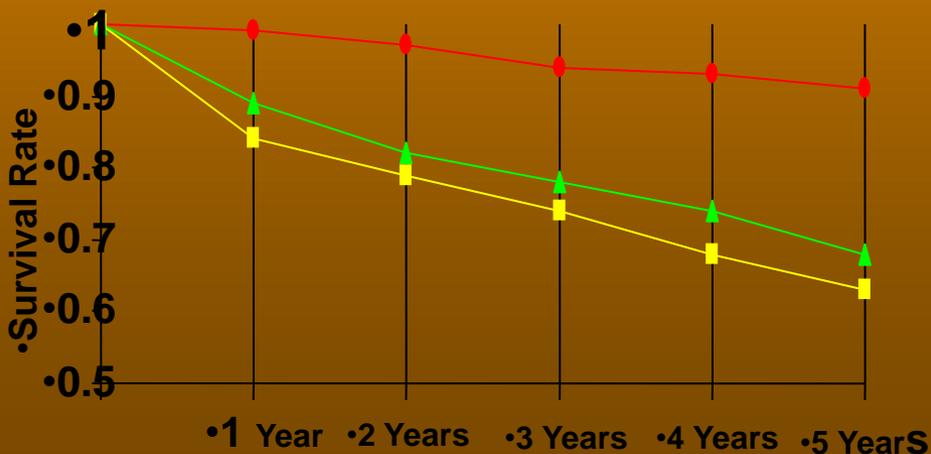
účinná AA
190

neúčinná AA
161

ICD

redukce TM
-50%

redukce TM
-27%



MORTALITY – HISTORY x TODAY

Original Articles

Changing Characteristics and Mode of Death Associated With Chronic Heart Failure Caused by Left Ventricular Systolic Dysfunction

A Study Across Therapeutic Eras

Table 2. Use of Heart Failure Therapies

	Historic (n=281)	Contemporary (n=357)	P
Drug			
Angiotensin-converting enzyme inhibitor, % (n)	83 (233)	89 (317)	0.02
Mean dose, ramipril equivalent, mg	3.4 (0.2)	5.1 (0.2)	<0.001
β-Adrenoceptor blocker, % (n)	8.5 (24)	80 (284)	<0.001
Aldosterone antagonists, % (n)	0	36 (129)	<0.001
Statin, % (n)	3.2 (9)	58 (204)	<0.001
Amiodarone, % (n)	16 (46)	13 (45)	0.18
Digoxin, % (n)	21 (60)	13 (46)	0.004
Loop diuretic, % (n)	98 (274)	72 (254)	<0.001
Mean dose, furosemide equivalent, mg	79 (3.9)	47 (2.7)	<0.001
Device-based therapies			
ICD, % (n)	0	16 (57)	<0.001
CRT, % (n)	0	23 (82)	<0.001

Table 1. Patient Characteristics Within Historic and Contemporary Cohorts

	Historic Cohort (n=281)	Contemporary Cohort (n=357)	P
Age, y	62 (0.6)	66 (0.7)	<0.001
Male sex, % (n)	81 (227)	71 (254)	0.005
Ischemic etiology, % (n)	79 (221)	62 (222)	<0.001
NYHA class, % (n)			<0.001
I	1 (3)	25 (91)	
II	51 (144)	43 (153)	
III	46 (129)	29 (103)	
IV	2 (5)	3 (9)	
Nonfasting glucose, mmol/L	5.0 (4.6–5.8)	5.2 (4.8–5.8)	0.29
Sodium, mmol/L	140 (0.2)	140 (0.2)	0.39
Potassium, mmol/L	4.3 (0.03)	4.4 (0.02)	0.004
eGFR, mL/kg per minute	58 (1.1)	56 (0.9)	0.08
Cardiothoracic ratio	0.54 (0.004)	0.55 (0.003)	0.13
LV end-diastolic dimension, mm	65 (0.6)	60 (0.5)	<0.001
LV end-systolic dimension, mm	56 (0.6)	49 (0.6)	<0.001
LV ejection fraction, %	30 (0.5)	31 (0.5)	0.44
QRS maximum, ms	138 (1.9)	134 (1.8)	0.09
QRS maximum >120 ms, % (n)	70 (168)	58 (176)	0.003
QTc maximum, ms	502 (3.1)	471 (2.3)	<0.001
QTc dispersion, ms	82 (61–104)	30 (19–43)	<0.001
LV hypertrophy on ECG, % (n)	9 (22)	20 (60)	0.001

•SCD 33,6% x 12,7%

MORTALITY – HISTORY x TODAY

•SCD 33,6% x 12,7%

Original Articles

Changing Characteristics and Mode of Death Associated With Chronic Heart Failure Caused by Left Ventricular Systolic Dysfunction

A Study Across Therapeutic Eras

Richard M. Cubbon, MRCP, PhD; Christopher P. Gale, MRCP, PhD; Lorraine C. Kearney, BSc; Clyde B. Schechter, FACPM, MD; W. Paul Brooksby, FRCP, MD; Jim Nolan, FRCP, MD; Keith A.A. Fox, FRCP, MD; Adil Rajwani, MRCP, PhD; Wazir Baig, FRCP, MD; David Groves, PhD; Pauline Barlow, BSc; Anthony C. Fisher, MD; Phillip D. Batin, FRCP, MD; Matthew B. Kahn, MRCP; Azfar G. Zaman, FRCP, MD; Ajay M. Shah, FRCP, MD; Jon A. Byrne, MRCP, MD; Steven J. Lindsay, FRCP, MD; Robert J. Sapsford, FRCP, MD; Stephen B. Wheatcroft, MRCP, PhD; Klaus K. Witte, MRCP, MD; Mark T. Kearney, FRCP, MD

Background—Therapies for patients with chronic heart failure caused by left ventricular systolic dysfunction have advanced substantially over recent decades. The cumulative effect of these therapies on mortality, mode of death, symptoms, and clinical characteristics has yet to be defined.

Methods and Results—This study was a comparison of 2 prospective cohort studies of outpatients with chronic heart failure caused by left ventricular systolic dysfunction performed between 1993 and 1995 (historic cohort: n=281) and 2006 and 2009 (contemporary cohort: n=357). In the historic cohort, 83% were prescribed angiotensin-converting enzyme inhibitors and 8.5% were prescribed β -adrenoceptor antagonists, compared with 89% and 80%, respectively, in the contemporary cohort. Mortality rates over the first year of follow-up declined from 12.5% to 7.8% between eras ($P=0.04$), and sudden death contributed less to contemporary mortality (33.6% versus 12.7%; $P<0.001$). New York Heart Association class declined between eras ($P<0.001$). QTc dispersion across the chest leads declined from 85 ms (SD, 2) to 34 ms (SD, 1) and left ventricular end-diastolic dimensions declined from 65 mm (SD, 0.6) to 59 mm (SD, 0.5) (both $P<0.001$).

led by an improvement in
(*Circ Heart Fail.* 2011;4:

Changing Characteristics and Mode of Death Associated With Chronic Heart Failure Caused by Left Ventricular Systolic Dysfunction

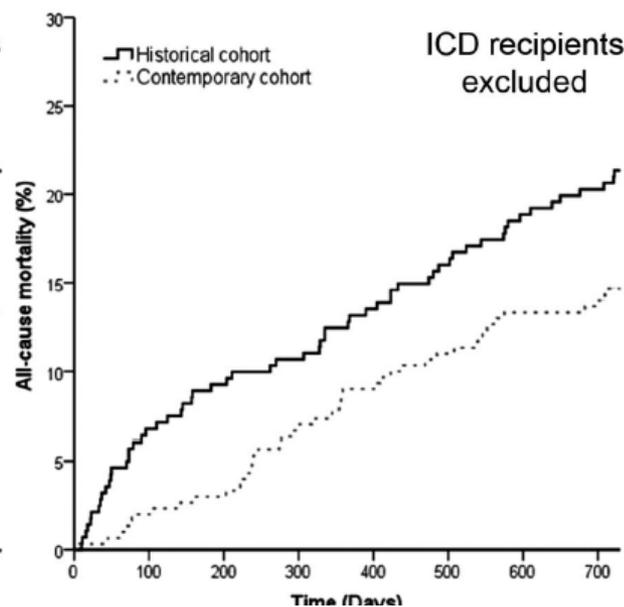
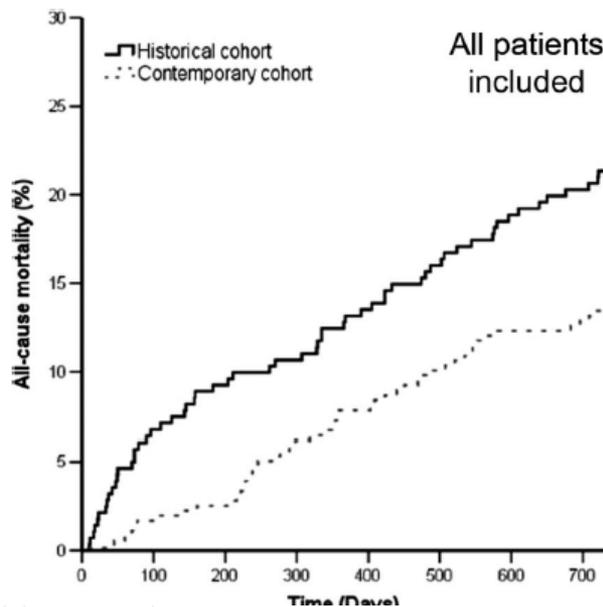
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Table 3. Mode of Death

	Historic	Contemporary	P
All patients			
Sudden death	34 (43)	13 (9)	$\chi^2 < 0.001$ Across groups
Progressive heart failure	41 (53)	37 (26)	
Other cardiovascular	14 (18)	7.0 (5)	
Noncardiovascular	11 (14)	41 (29)	
Unclassifiable	0	2.8 (2)	
ICD recipients excluded			
Sudden death	34 (43)	8.1 (5)	$\chi^2 < 0.001$ Across groups
Progressive heart failure	41 (53)	37 (23)	
Other cardiovascular	14 (18)	8.1 (5)	
Noncardiovascular	11 (14)	44 (27)	
Unclassifiable	0	3.2 (2)	

Cubbon et al Changing Chronic He



Conclusions—Survival has significantly improved in patients with chronic heart failure caused by left ventricular systolic dysfunction over the past 15 years; furthermore, sudden death makes a much smaller contribution to mortality, and noncardiac mortality is a correspondingly greater contribution. This has been accompanied by an improvement in symptoms and some markers of adverse electric and structural left ventricular remodeling. (*Circ Heart Fail.* 2011;4:396-403.)

SCD/ PP

- •ACEI (SOLVD - 23% NSS, V-HeFT - 31% NSS, CHFSTAT -52% NSS)
 - • ACEI therapy – lower risk of SCD
 - • more than 50% pts. treated ACEI can profit from SCD prophylaxis
- •amiodaron (CHFSTAT,CAMIAT, EMIAT)
 - •Do not prolong survival with LV dysfunction
- •BB (CIBIS II, BEST, MERIT-HF)
 - • downgrade risk of SCD
 - • prolong survival of pts with CHF

SCD PREVENTION

- OPT + revascularization CAD
- ICD / CRT ICD
- RFA
- Heart transplant
- Surgery of CHF (MVP, aneurysmectomy)

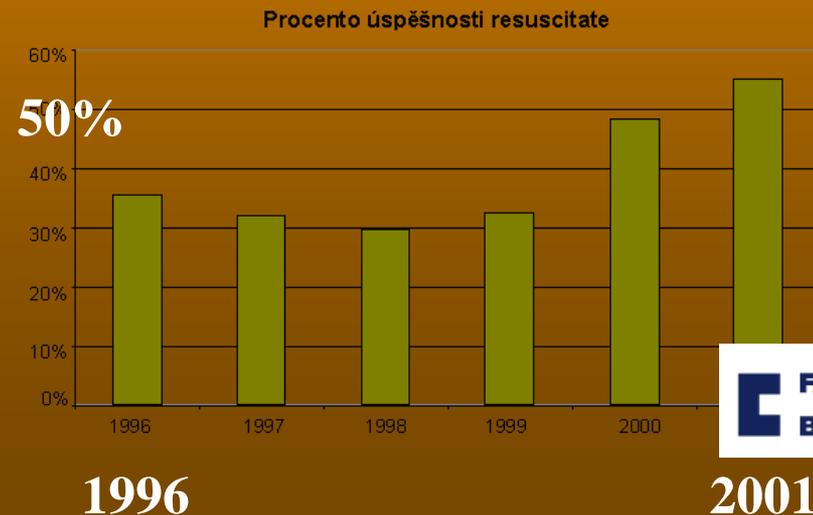


Profile of resuscitated patient

Who?

CAD (1. manifestation)	64 - 90 % (25 %)
64 years old man	81 %
MI in anamn.	45%

Cobb et al, Circulation, 1992



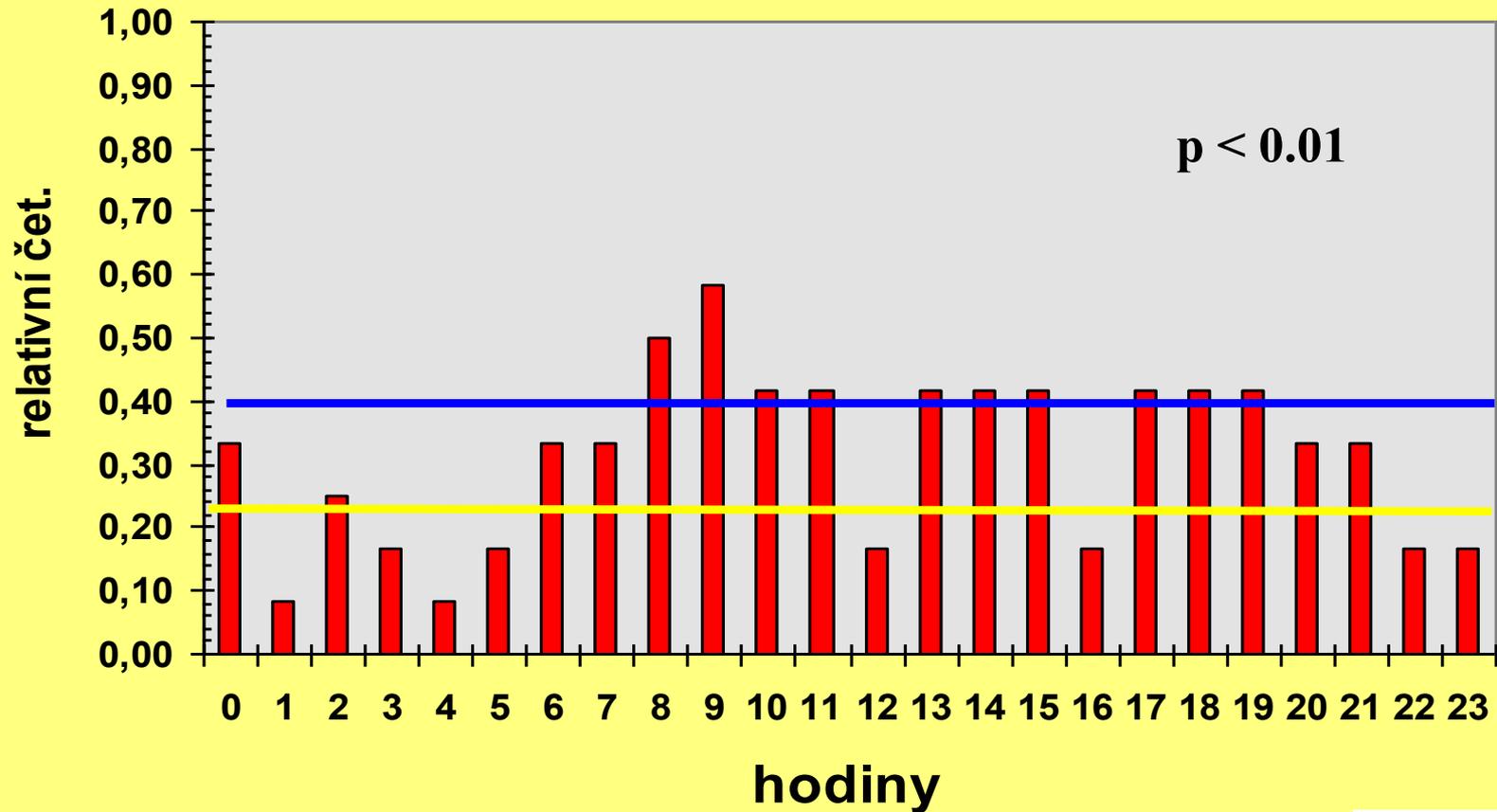
CIRCADIAN OCCURENCE

N = 72

When?

n = 506

Denní doba - souhrn 1

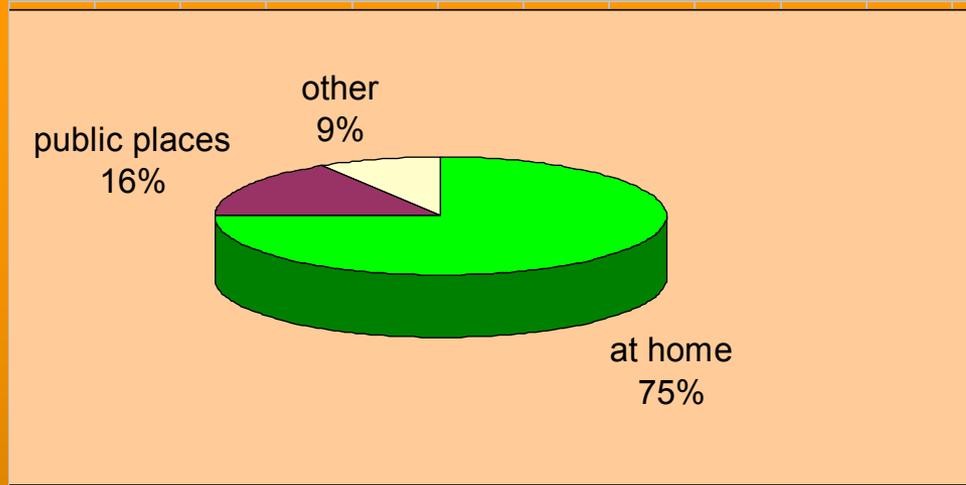


M.Kozak et al., PACE 2003

Where?

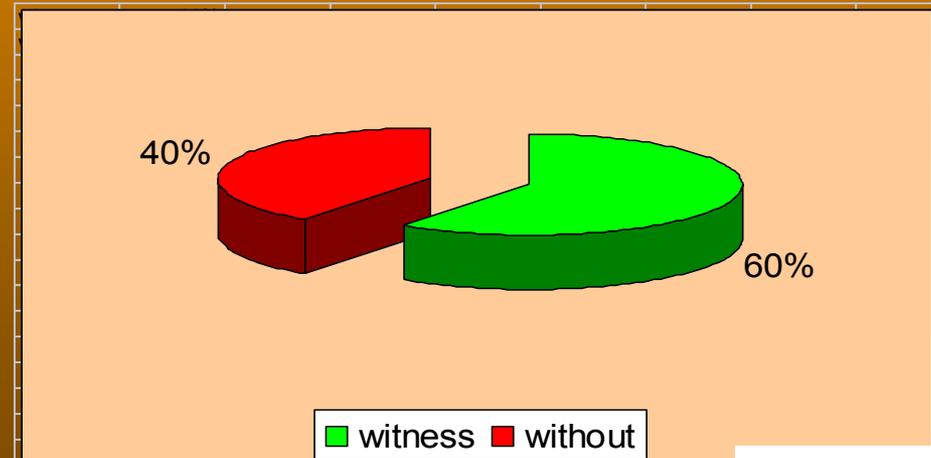
Incidence => 0.03 (30 places = 1 CA)

- airports
- industrial zones
- golf clubs
- fitcenters
- casinas



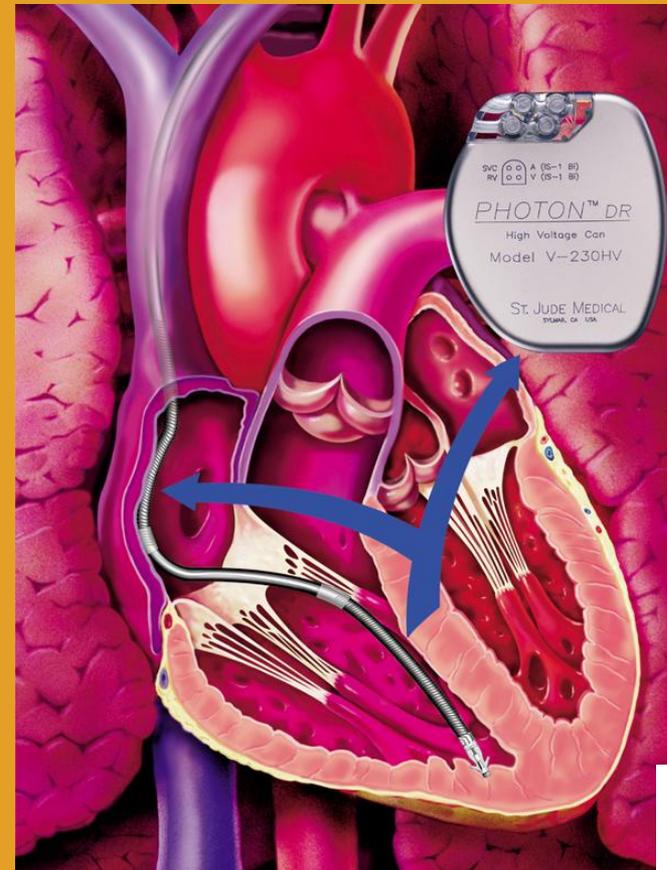
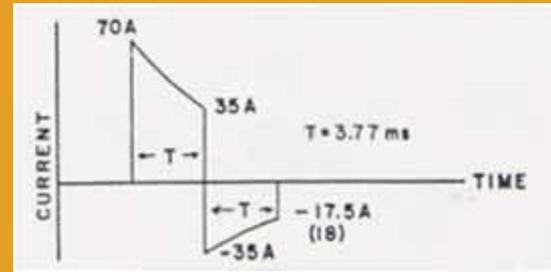
Atkins et al. Prehospital Disaster Med 1996, 11:47-49

Becker et al. Circulation. 1998,97:2106-2109

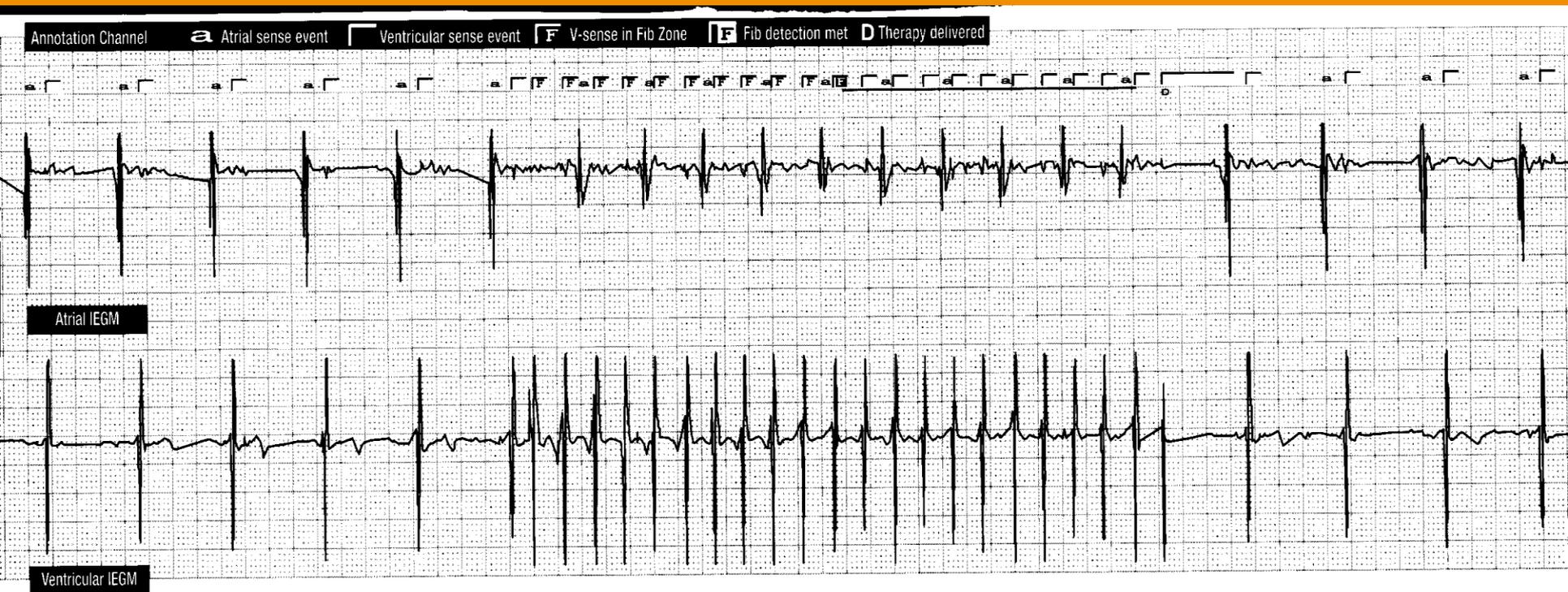


ICD IN SECONDARY PREVENTION OF SCD

ČR -75/1mil



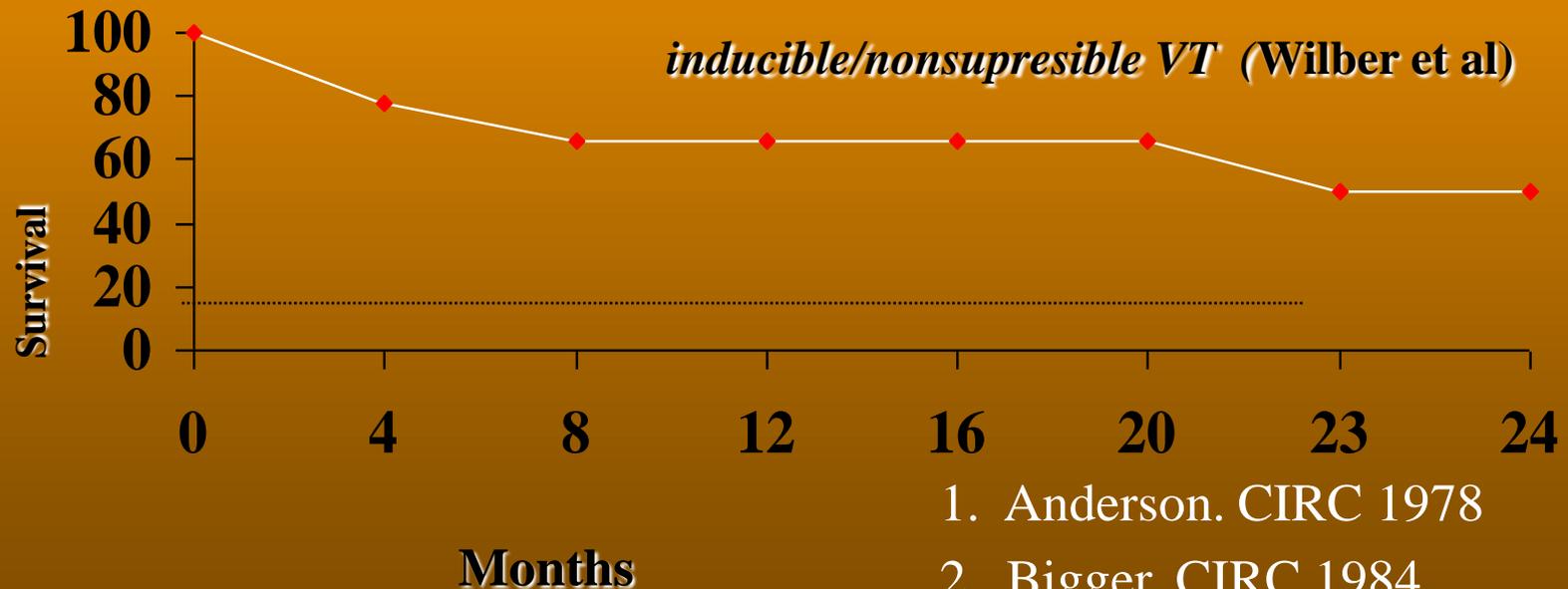
ICD IN SECONDARY PREVENTION OF SCD



ICD - PRIMARY PREVENTION

CAD + NSVT

- MI + NSVT + LV dysf. = 2 year mortality > 30%
- MI + NSVT + LV dysf. + EPS + = 50%



1. Anderson. CIRC 1978
2. Bigger. CIRC 1984
3. Buxton. Am J C 19
4. Wilber CIRC 1990

For which patient?

- A number of previous ICD studies* indicate patients are remarkably similar with respect to:
 - Age
 - Left Ventricular Ejection Fraction
 - Percentage with Coronary Artery Disease
 - NYHA classification

Prophylactic patient is not different

*Sources: Moss, A, et al; *N Engl J Med* 1996; 335: 1933-40

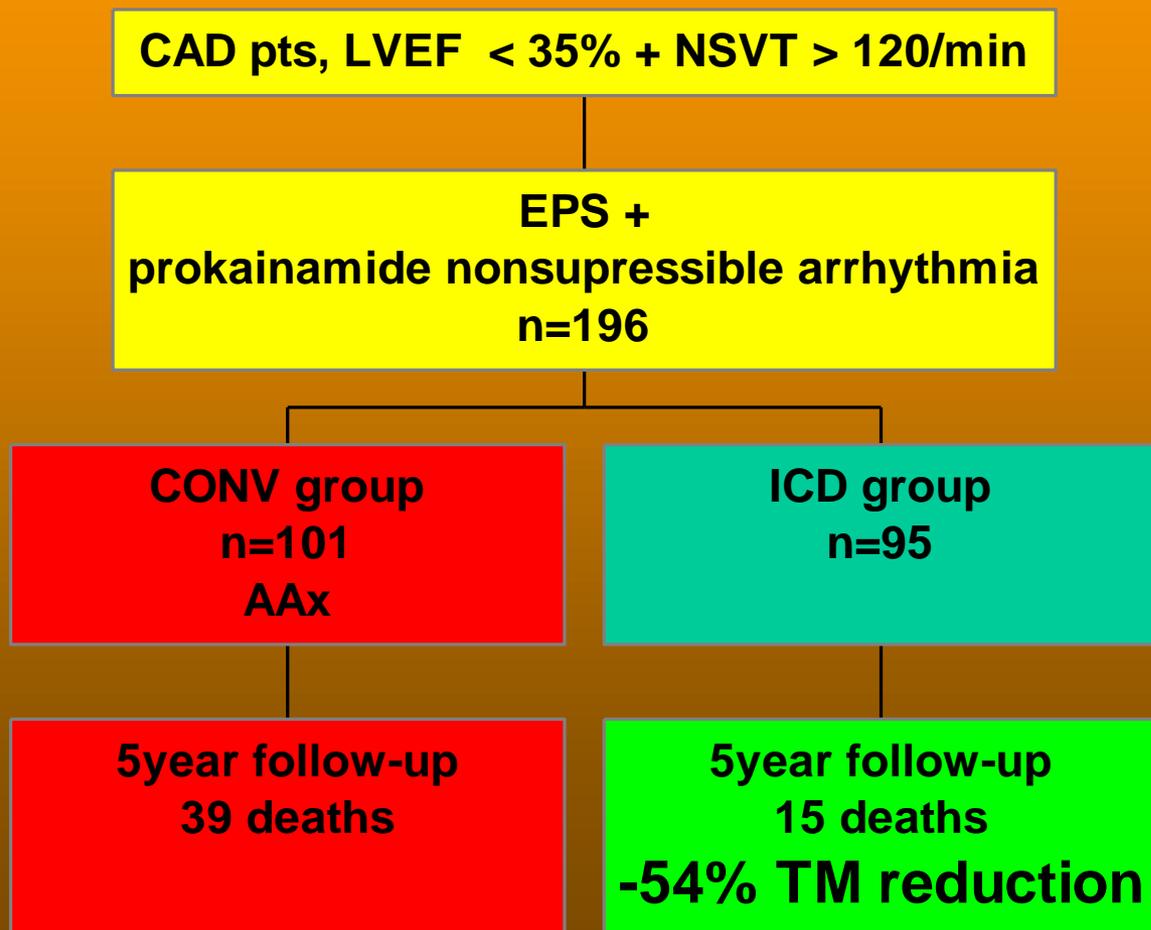
Buxton, A, et al; *N Engl J Med* 1999; 341: 1882-90

AVID Investigators; *N Engl J Med* 1997; 337: 1576-83

ICD IN PRIMARY PREVENTION OF SCD

MADIT

Schema of study



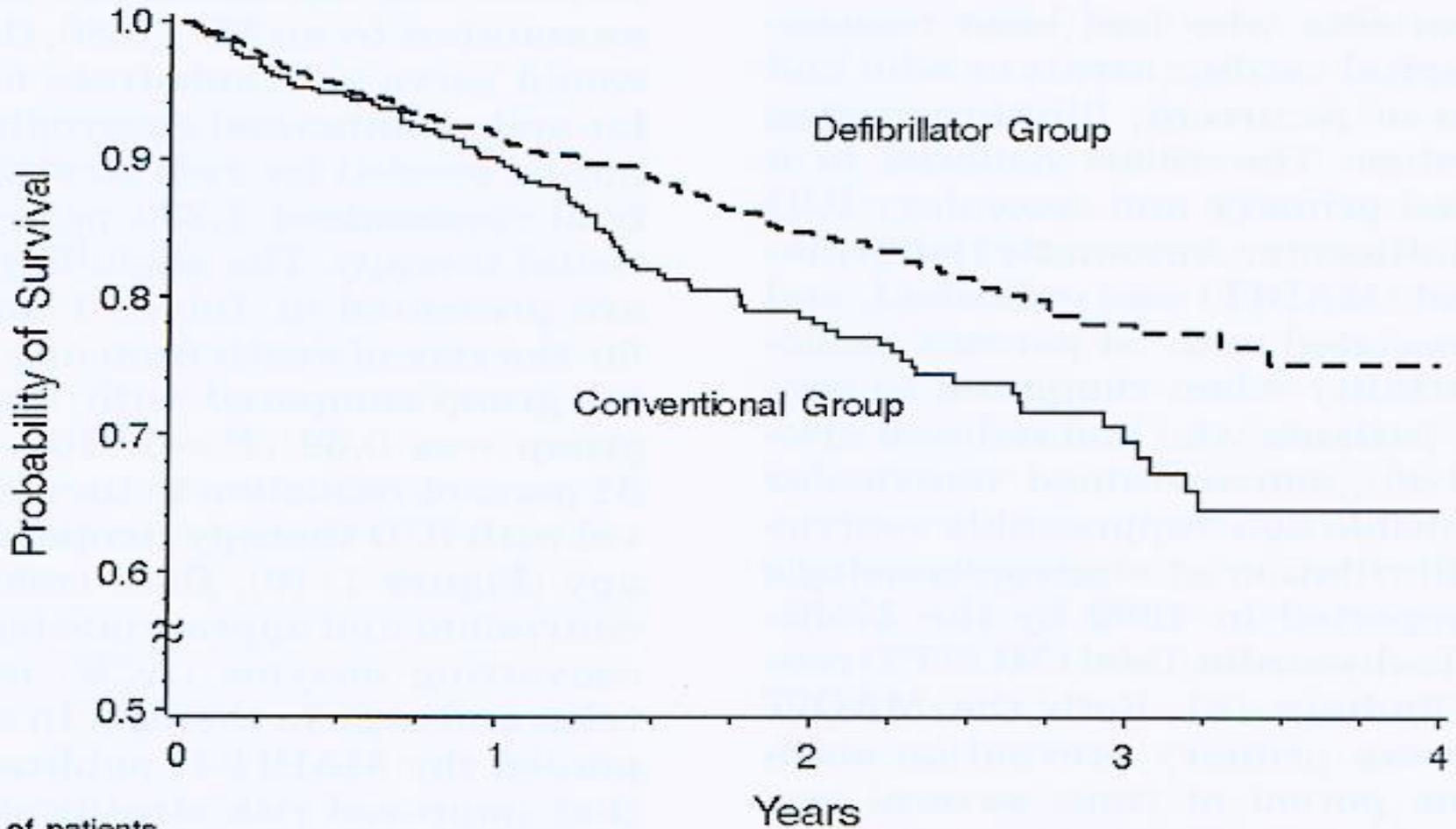
ICD - PRIMARY PREVENTION

- Documented episodes of NSVT in CAD post MI patients and LVEF < 0.35, sustained VT inducible in EPS.



ICD IN PRIMARY PREVENTION OF SCD

MADIT II



No. of patients	1 Year	2 Years	3 Years
Defibrillator: 742	503 (0.91)	274 (0.84)	110 (0.78)
Conventional: 490	329 (0.90)	170 (0.78)	65 (0.69)

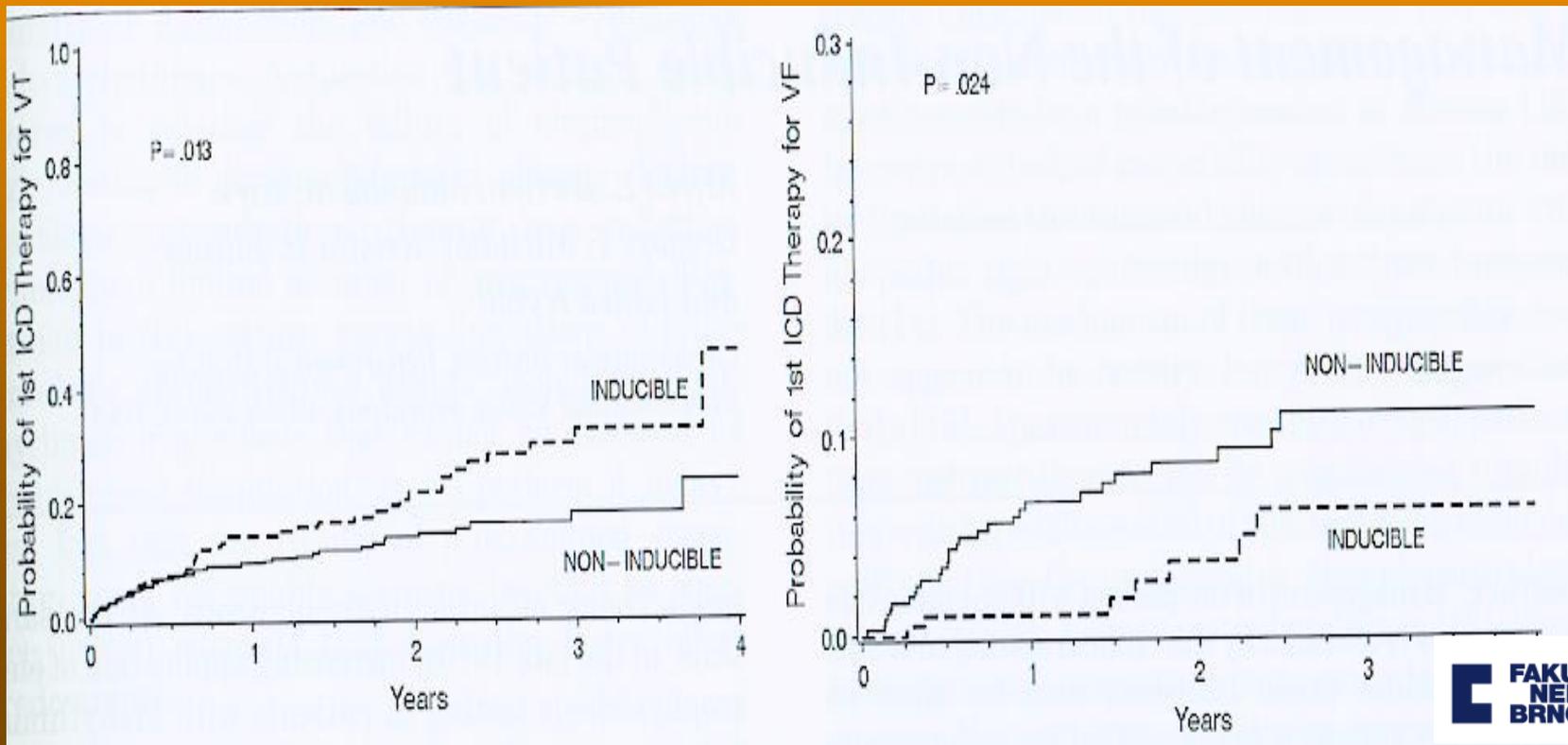


ICD IN PRIMARY PREVENTION OF SCD

- ICD group (N=742)
- CONV therapy (N=490)
- 31% reduction of deaths in ICD group
- 63% reduction of mortality - QRS > 120 ms

MADIT II

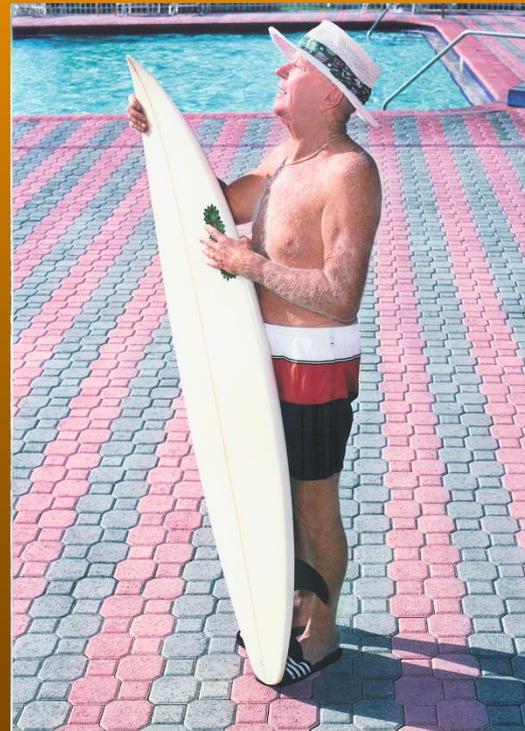
105 (14,2%) deaths
97 (19,8%)



ICD IN PRIMARY PREVENTION OF SCD

• 4.12.5.

CAD post MI, LV dysfunction, LVEF < 0.30, QRS > 120ms,
NYHA II, 6 m post IM, standard pharmacotherapy (bb)



ICD IN PRIMARY PREVENTION OF SCD - CZ

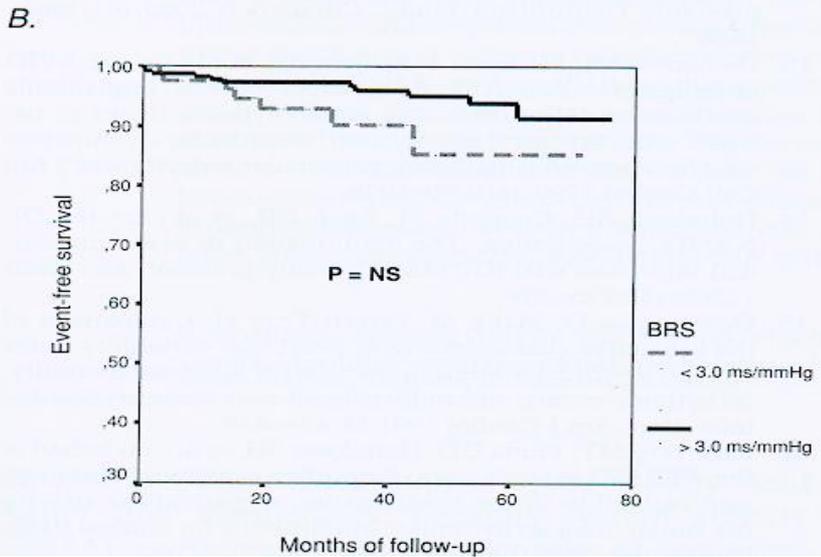
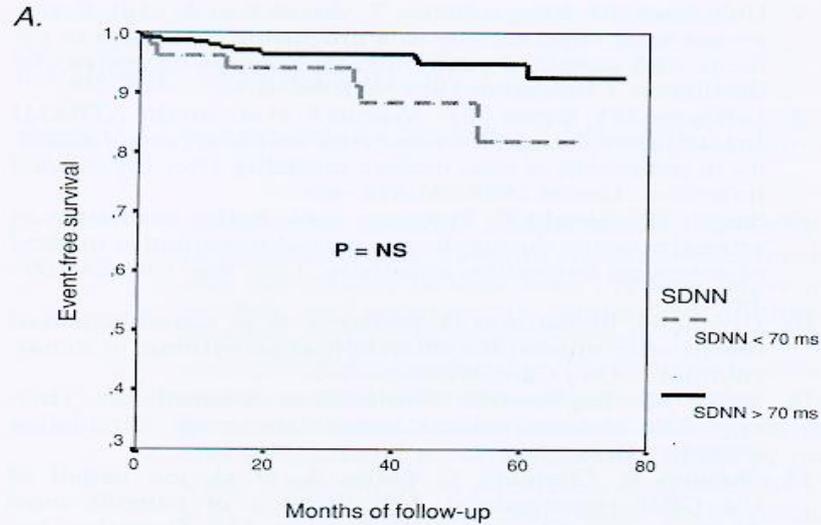
• 2010 – 2019 - 75% primary preventive implantation

• 2004	5,5%
• 2005	7%
• 2006	22,8%
• 2007	34 %
• 2008	39%
• 2009	46%

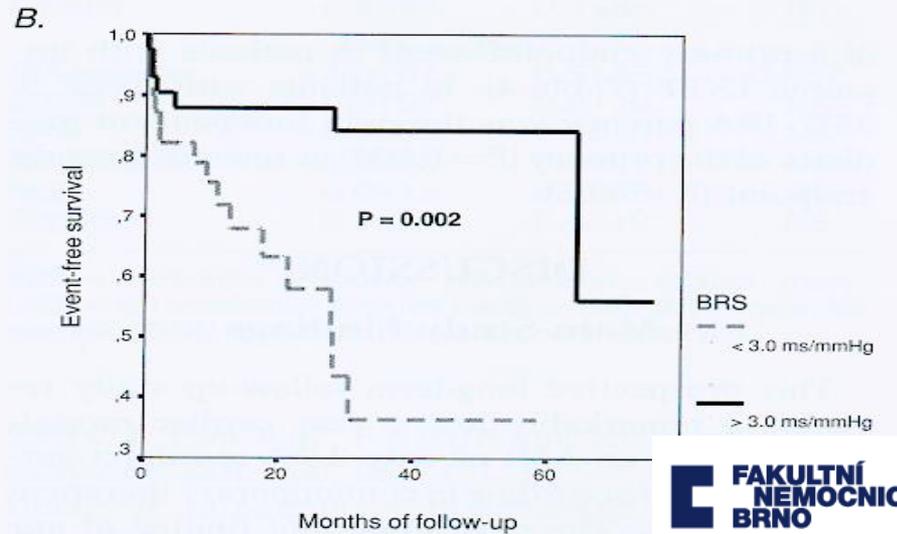
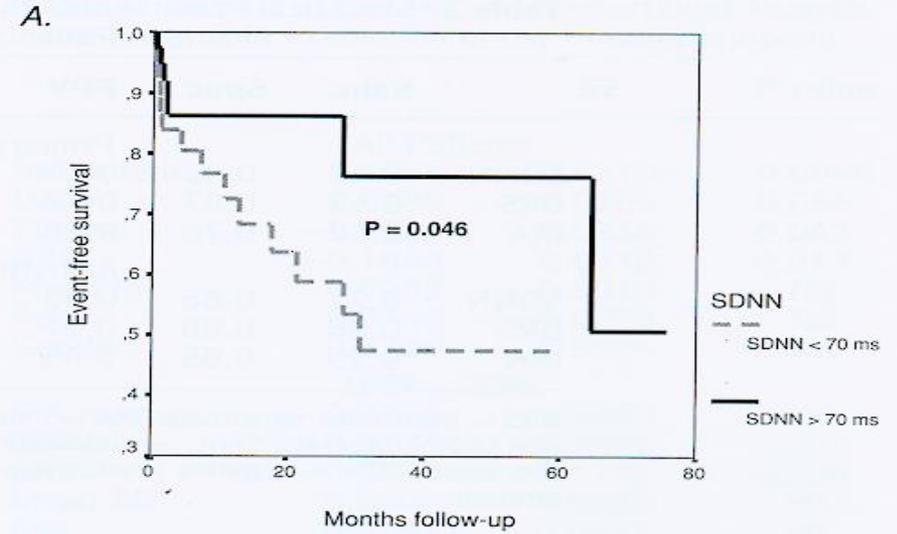


RISK STRATIFICATION

*Klingenhöben et al.,
A.N.E.2003,8(1):68-74*



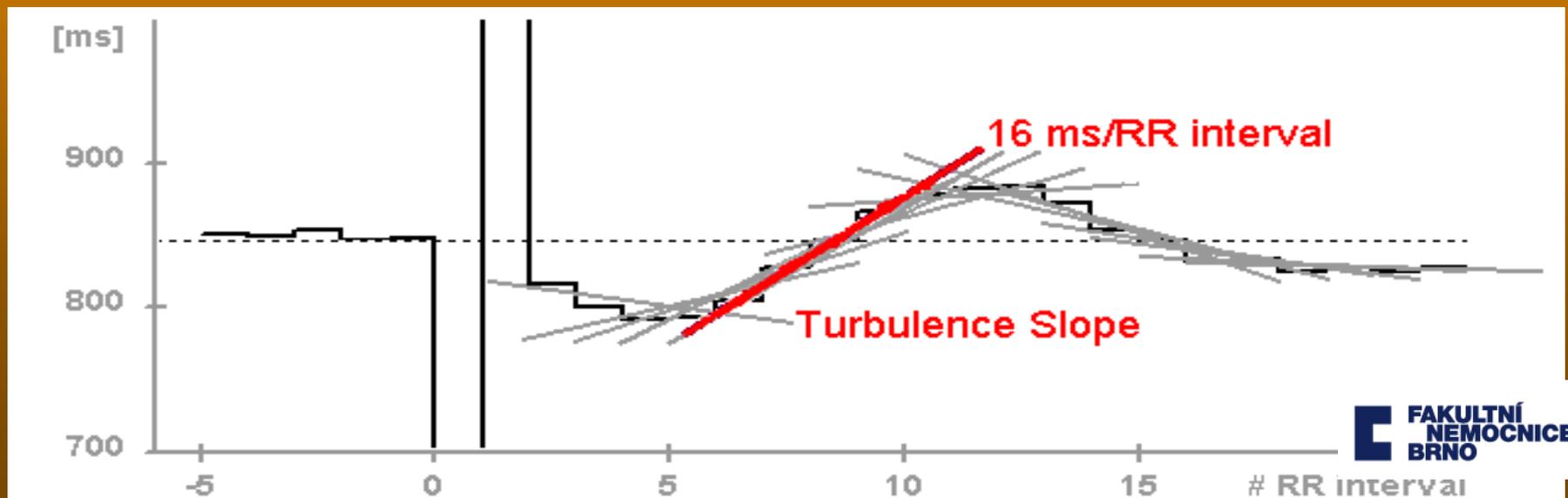
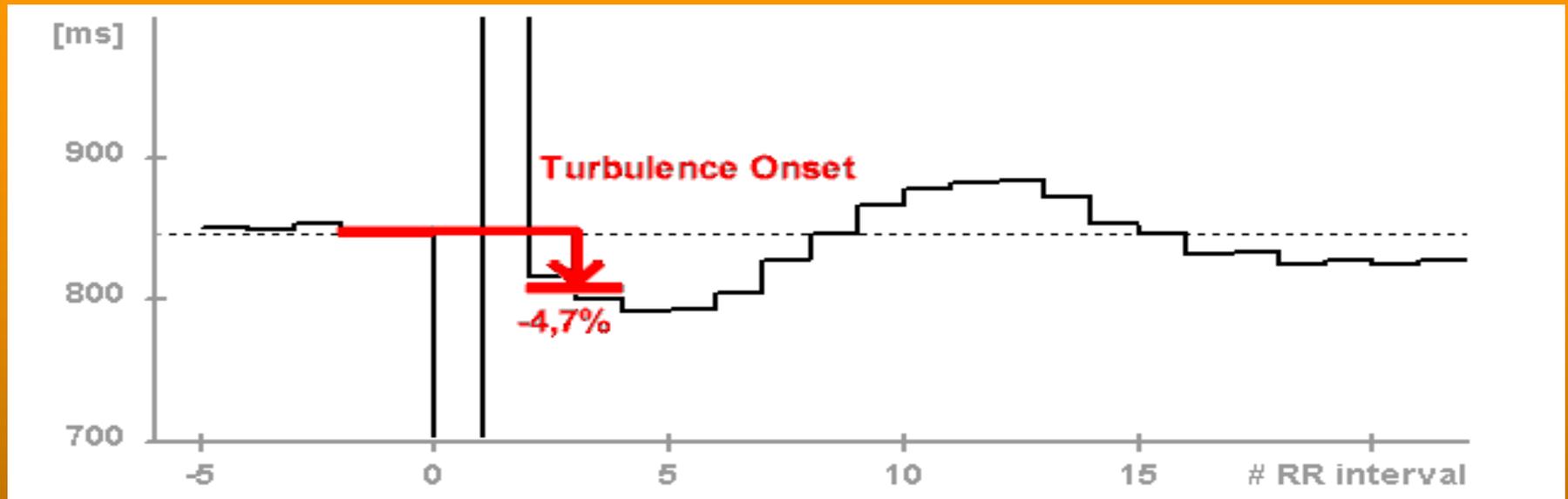
LVEF > 35%



LVEF < 35%

RISK STRATIFICATION

HRT

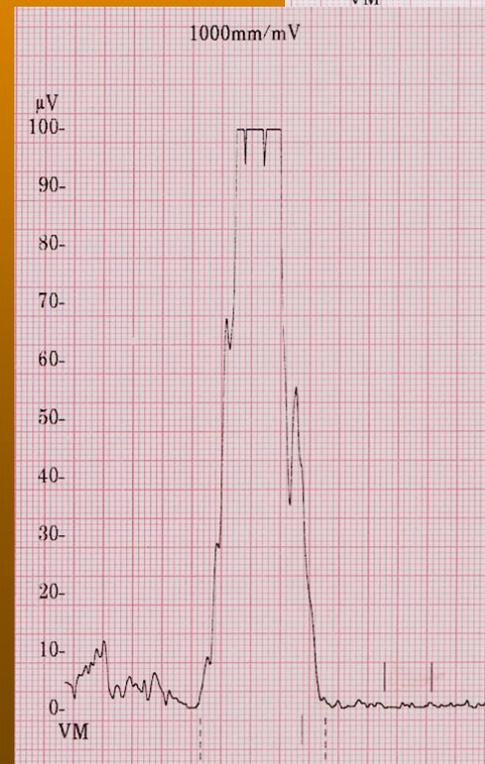
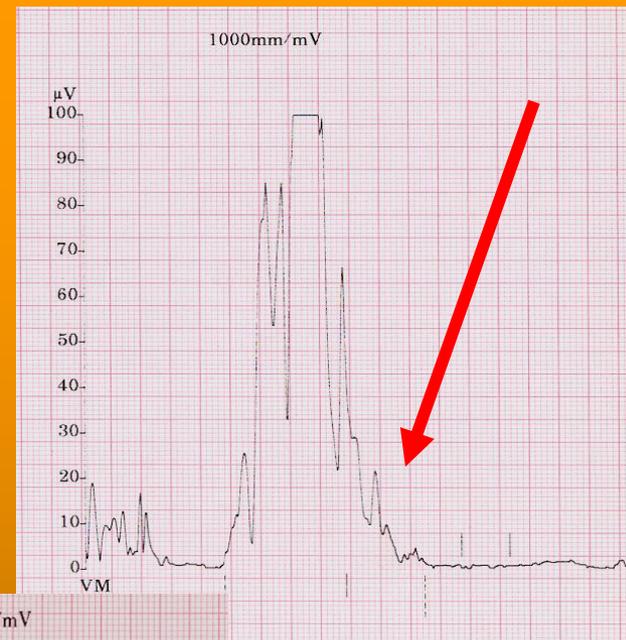


RISK STRATIFICATION

- PPV - 30%
- NPV - 96%

*Steinberg JS., Regan A.,
Sciacca R., et al., Am J Cardiol
1992;69:13-21*

*Breithardt G., Schwartzmaier
J., Borggreffe M., et al., Eur
Heart J 1983;4:487-95*

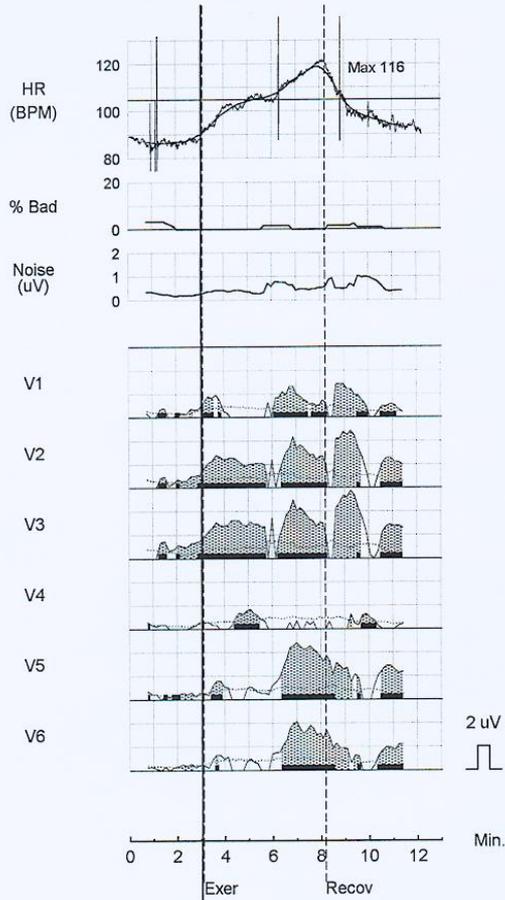


RISK STRATIFICATION

TWA

Site: ALTERNANS PRECORDIAL TREND SUMMARY

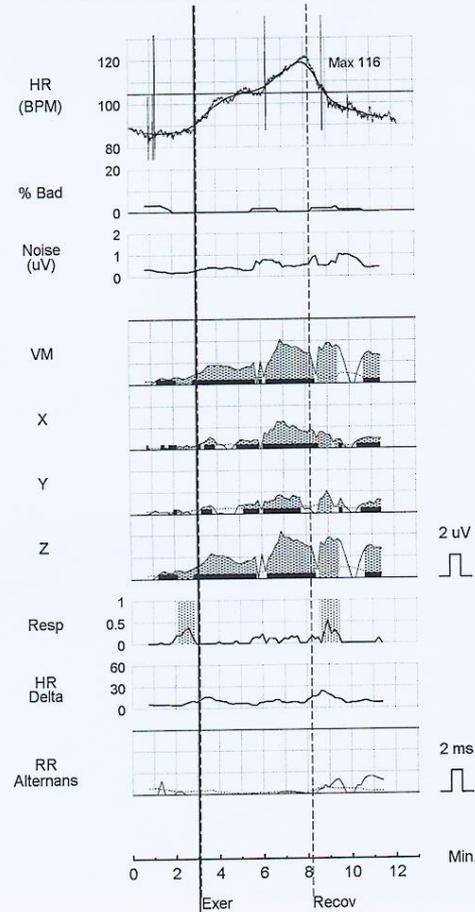
Patient : DEMO DATA Protocol : MTWA Exer
ID : 22-066



Onset HR 91
Max Neg HR 91

Site: ALTERNANS VECTOR TREND SUMMARY

Patient : DEMO DATA Protocol : MTWA Exer
ID : 22-066



Onset HR 91
Max Neg HR 91

RISK STRATIFICATION

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ACC/AHA/ESC PRACTICE GUIDELINES—EXEC

ACC/AHA/ESC 2006 Guidelines of Patients With Ventricular Arrhy Prevention of Sudden Cardiac Deat

A Report of the American College of Cardiology
Force and the European Society of Cardiology C
(Writing Committee to Develop Guidelines for I
Ventricular Arrhythmias and the Prevention of S
*Developed in Collaboration With the European E
Heart Rhythm Society*

VII. ELECTROCARDIOGRAPHIC TECHNIQUES AND MEASUREMENTS

Recommendations

Class IIa

It is reasonable to use T-wave alternans for improv-
ing the diagnosis and risk stratification of patients
with ventricular arrhythmias or who are at risk for
developing life-threatening ventricular arrhythmias.
(Level of Evidence: A)

Class IIb

ECG techniques such as signal-averaged ECG, heart
rate variability, baroflex sensitivity, and heart rate
turbulence may be useful for improving the diagnosis
and risk stratification of patients with ventricular
arrhythmias or who are at risk of developing life-
threatening ventricular arrhythmias. *(Level of Evi-
dence: B)*

ICD trials, especially Multicenter Automatic Defibrilla-
tor Implantation Trial (MADIT) II, have highlighted the
need to develop novel tools in order to identify patients at
highest risk of ventricular arrhythmias and SCD. Numerous
modalities exist at present for assessing this risk but only 2
are currently approved by the U.S. Food and Drug
istration: signal-averaged ECG and T-wave a
However, heart rate variability and baroflex sensi...
show considerable promise.

TABLE 3 Association of the Composite Autonomic Index (created by combining TO, TS, BRS, and SDNN) With FCA/NFCA Using Cox's Univariate Regression Analysis

	[FCA/NFCA]/Total	RR (95% CI)	p Value
Variables Combined Into a Single Index With Five Categories (each compared with all factors normal)			
Combined variable		—	<0.0001
All factors normal	14/552	1	—
Any 1 factor abnormal	10/228	1.71 [0.76–3.85]	0.194
Any 2 factors abnormal	4/63	2.84 [0.93–8.63]	0.066
Any 3 factors abnormal	5/40	5.16 [1.86–14.33]	0.0016
All 4 factors abnormal	5/15	16.79 [6.01–46.89]	<0.0001
Composite Autonomic Index Dichotomized to Create Four New Variables, in Which the Abnormal Category Is Compared With Rest of Study Population			
≥1 factor abnormal	24/346	2.82 [1.46–5.45]	0.002
≥2 factors abnormal	14/118	4.32 [2.23–8.36]	<0.0001
≥3 factors abnormal	10/55	5.99 [2.91–12.33]	<0.0001
All 4 factors abnormal	5/15	11.31 [4.39–29.16]	<0.0001

CONCLUSION

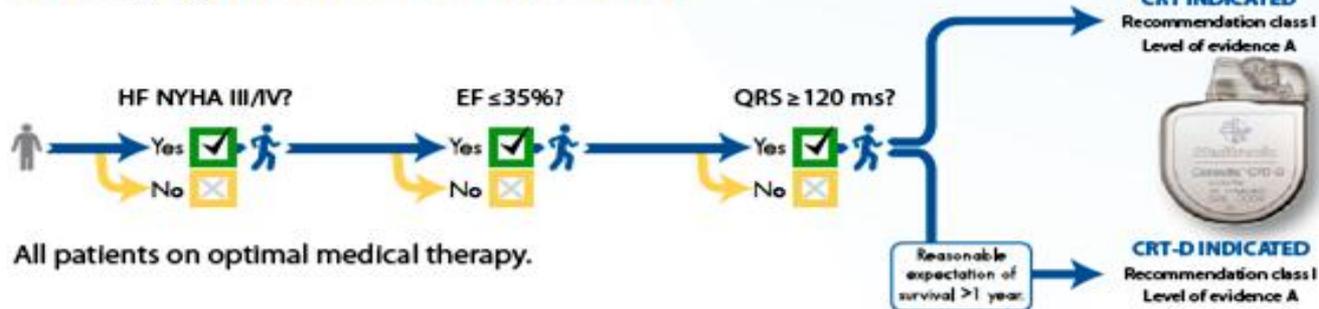
- **Prophylactic ICD patient is not different to the general ICD population**
- **NNT ratio is low and reduced in time**
- **ICD therapy is cost effective**
- **Prophylactic pts require a full featured device, just like any other pt**

CONCLUSION

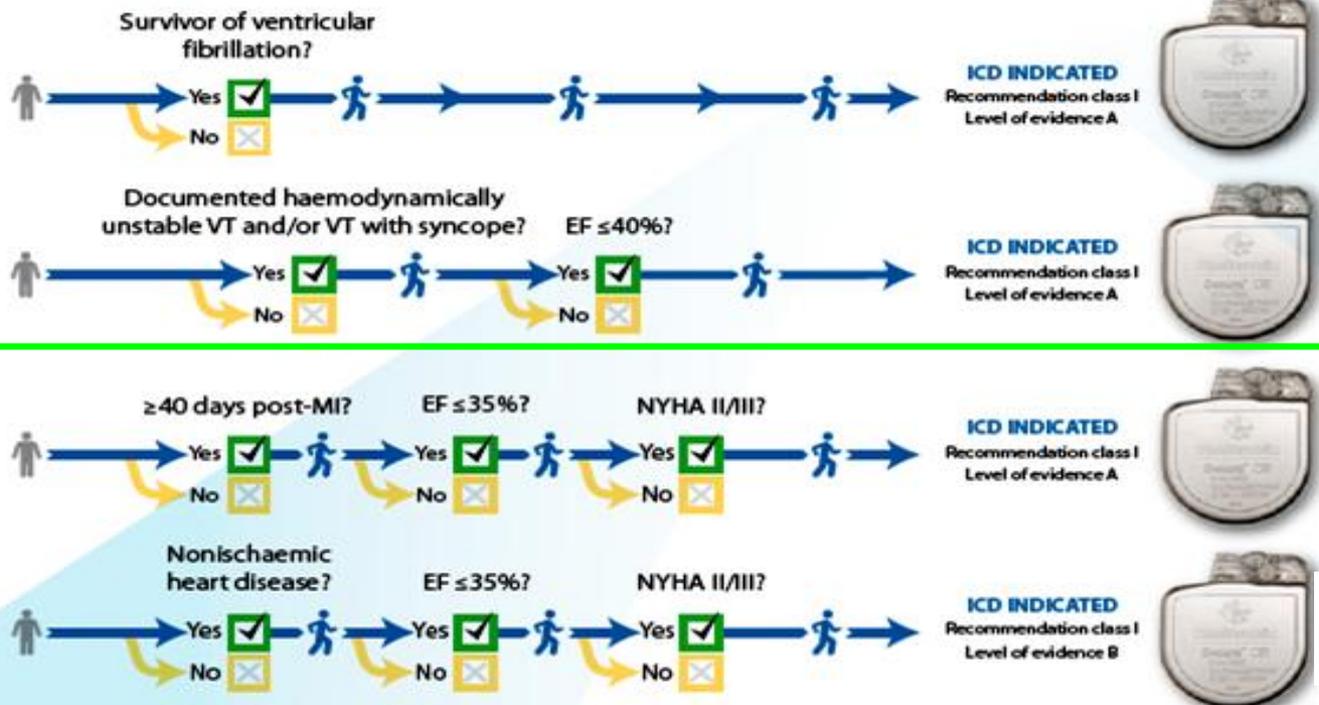
- No of PP ICD implantation is growing
- Each fifth pt in CZ is implanted from PP reasons
- The most frequent - combined indication
PP ICD + CRT

Recognising candidates for Heart Failure device therapies as indicated in ESC treatments guidelines

Identifying candidates for CRT/CRT-D



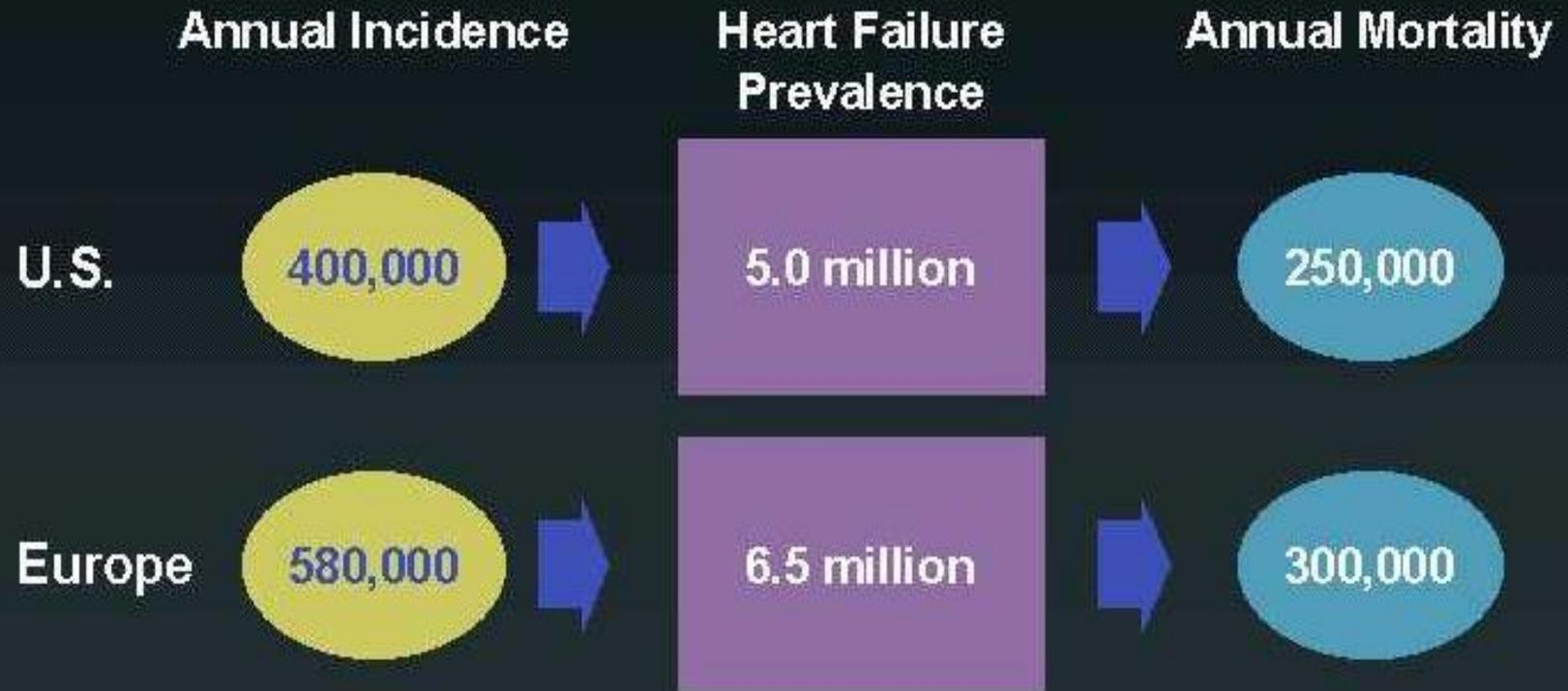
Identifying candidates for ICD



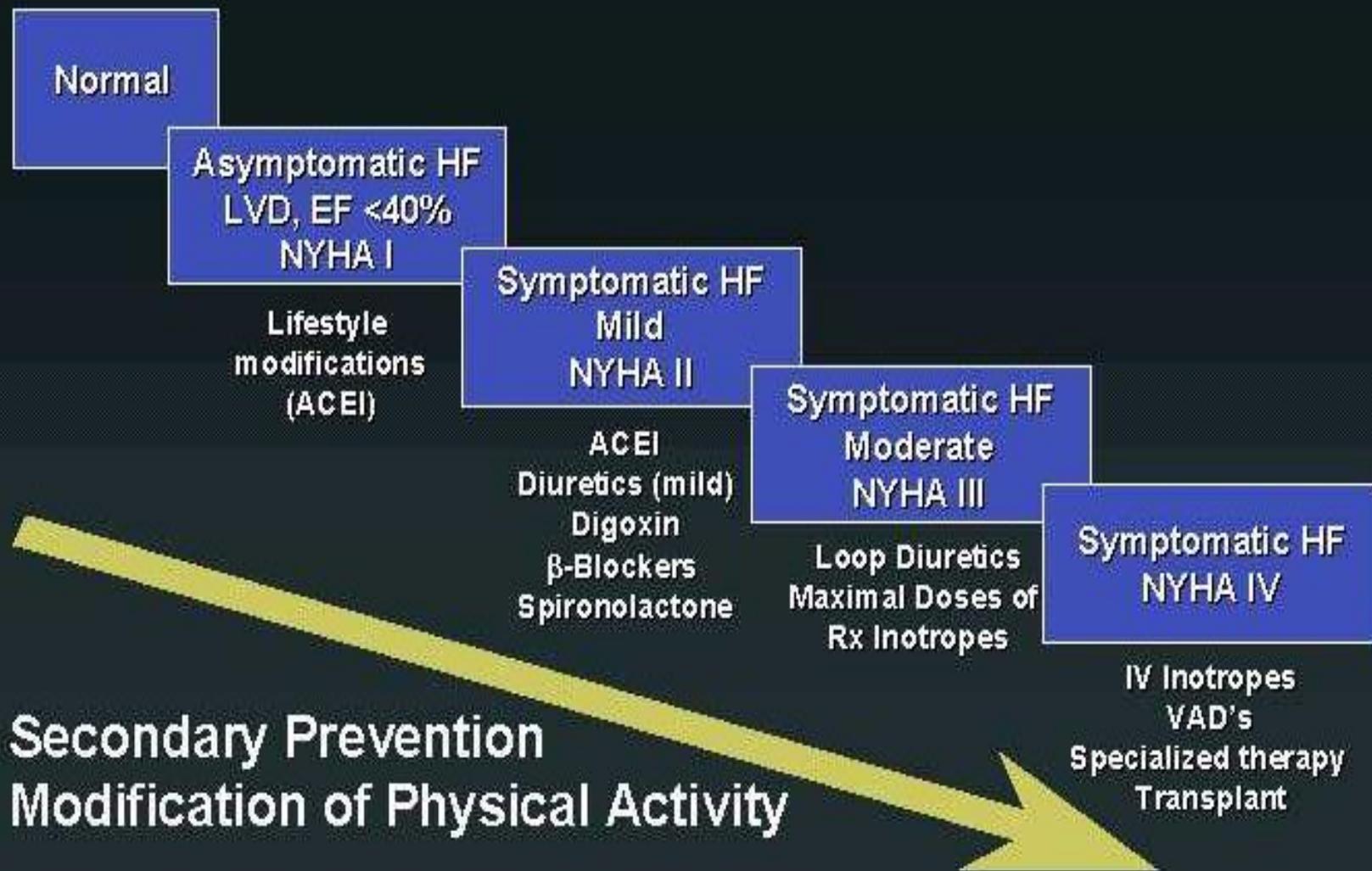
All patients on optimal medical therapy with reasonable expectation of survival > 1 year.

Heart Failure Management

A Growing Medical Challenge

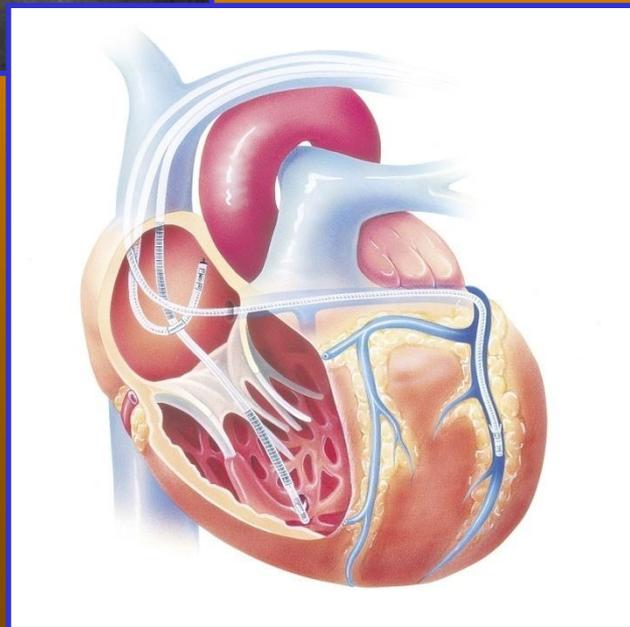
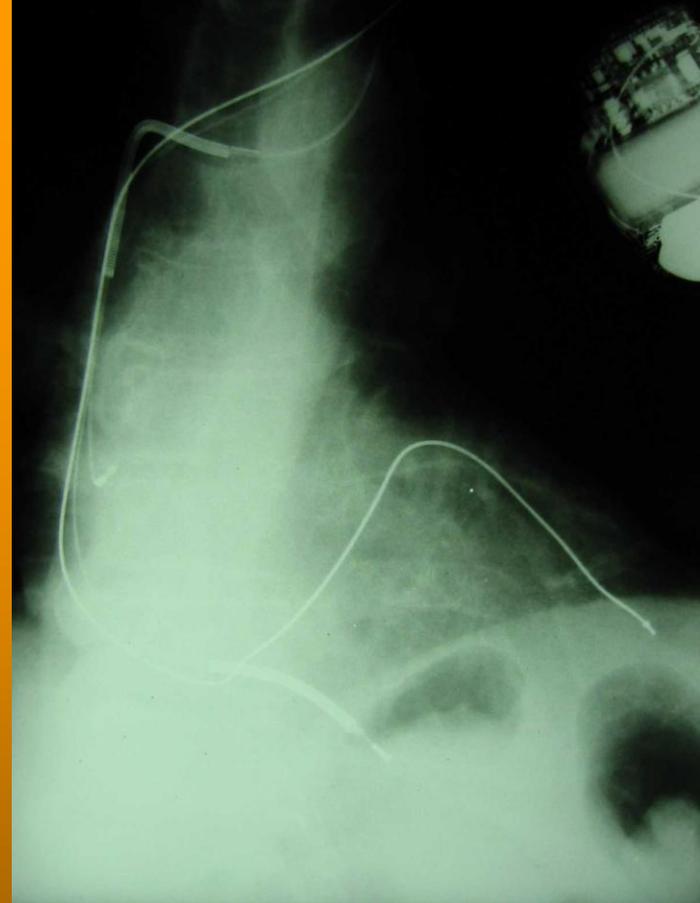
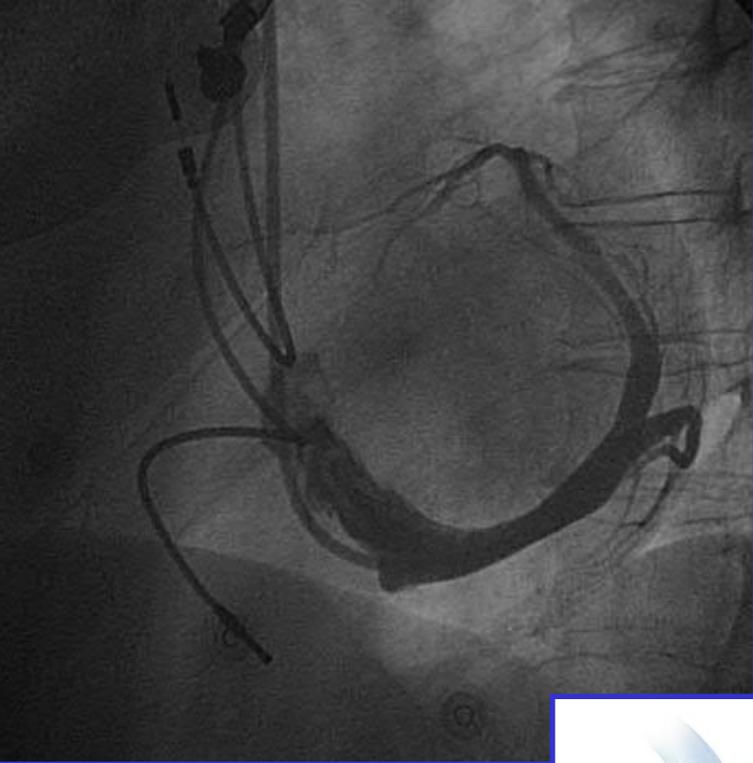


Congestive heart failure worldwide markets, clinical status and product development opportunities. *New Medicine, Inc.*, 1997:1-40.
Wilkerson Group Survey, 1998.



Bolger A, Sendón J. Chronic congestive heart failure. American Heart Association 1999.

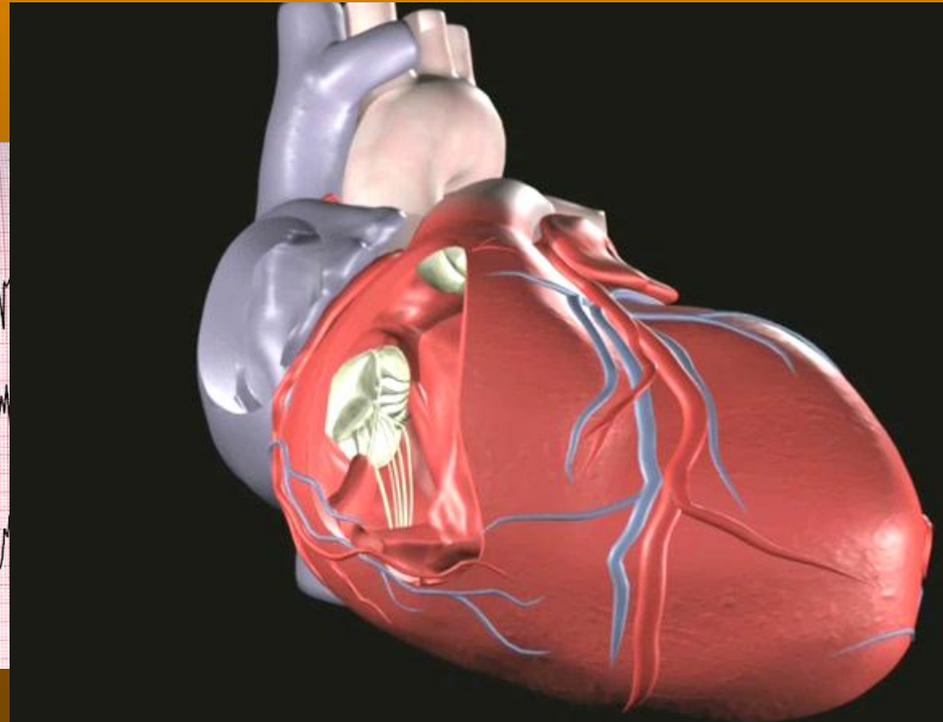
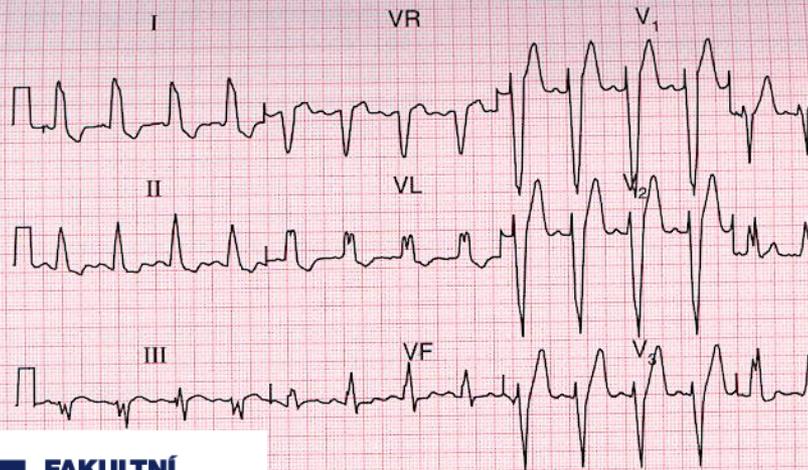
CRT



CRT INDICATION

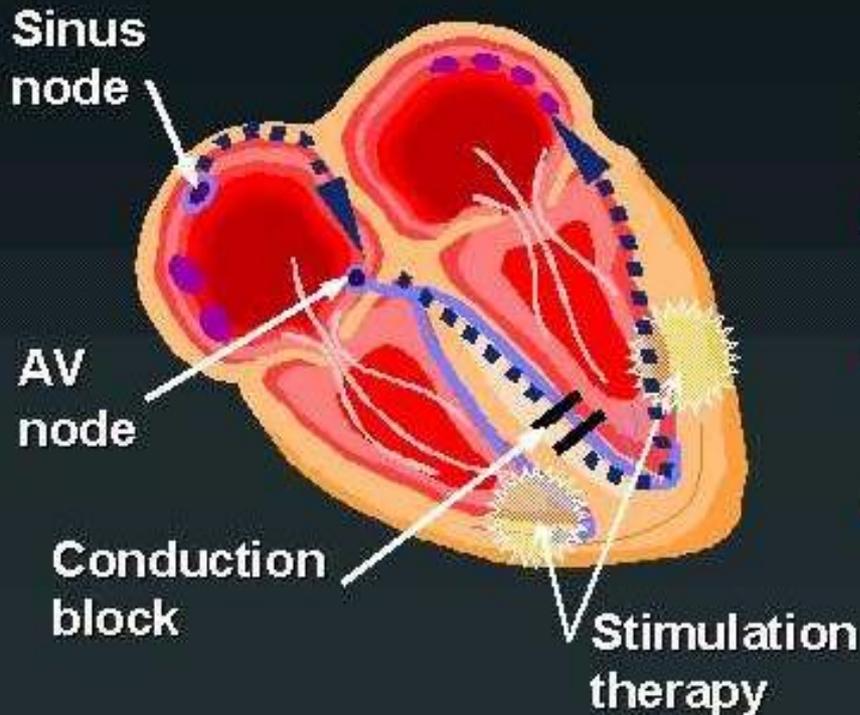
- 4.10. HF, stand. Rx 6 months (NYHA II/ III 6 m, NYHA IV)

LVEF < 35%, QRS > 150 ms, 120-150ms,
dyssynchrony



Mechanism II

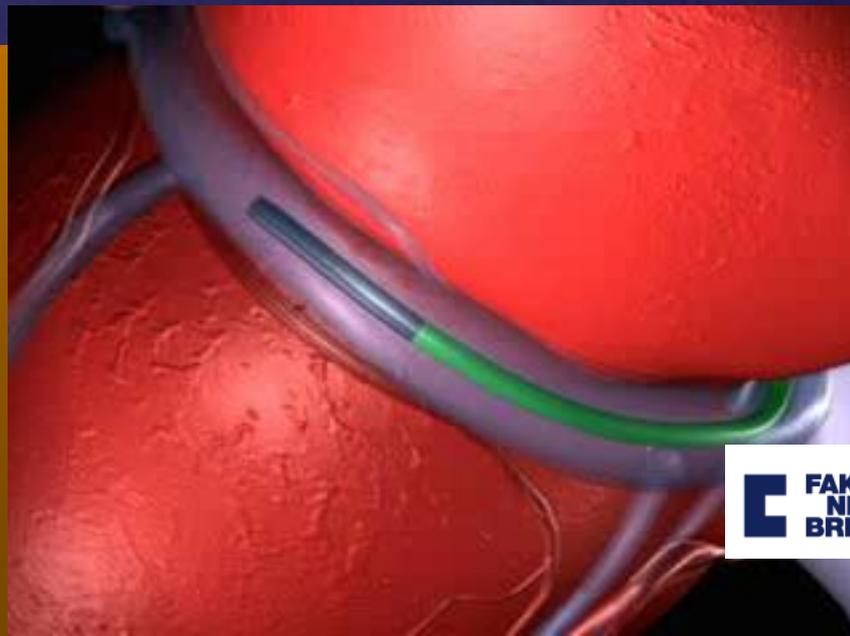
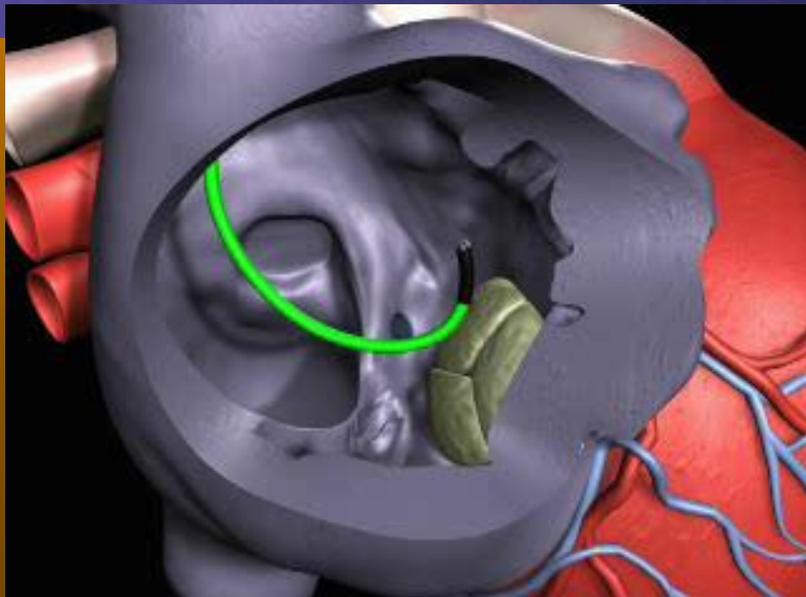
Restoring Intraventricular Coordination

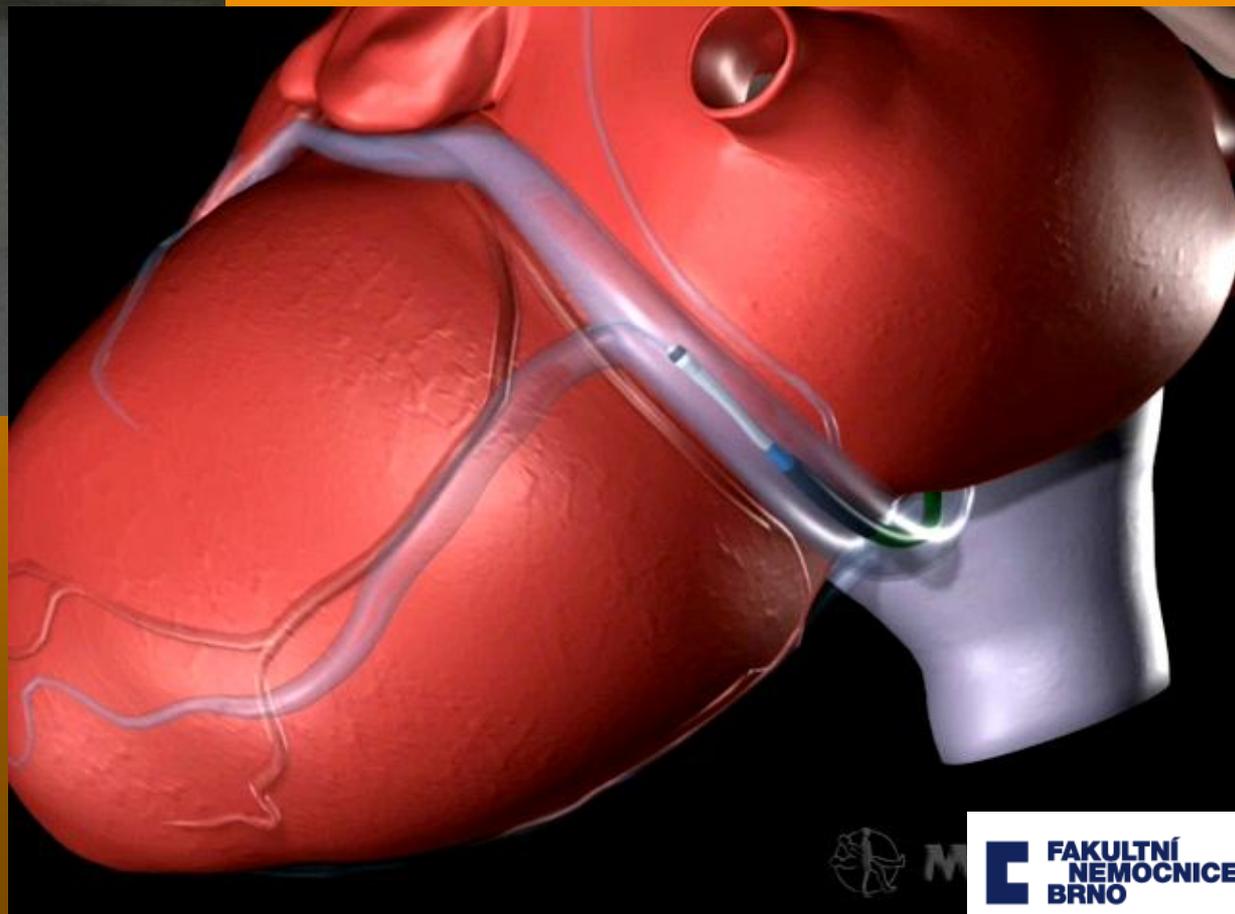
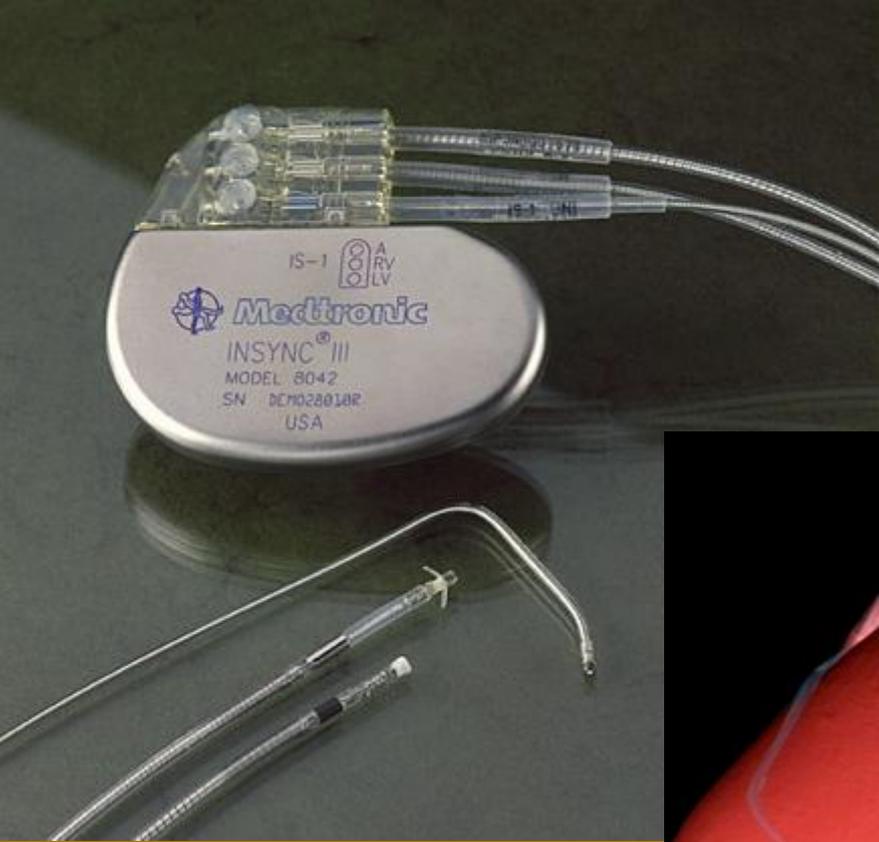


Intraventricular Activation

- Organized ventricular activation sequence
- Coordinated septal and freewall contraction
- Improved cardiac efficiency

Kass D. New dimensions in device-based therapy for heart failure—mechanisms of stimulation for heart failure. Heart Failure Society of America 1999.

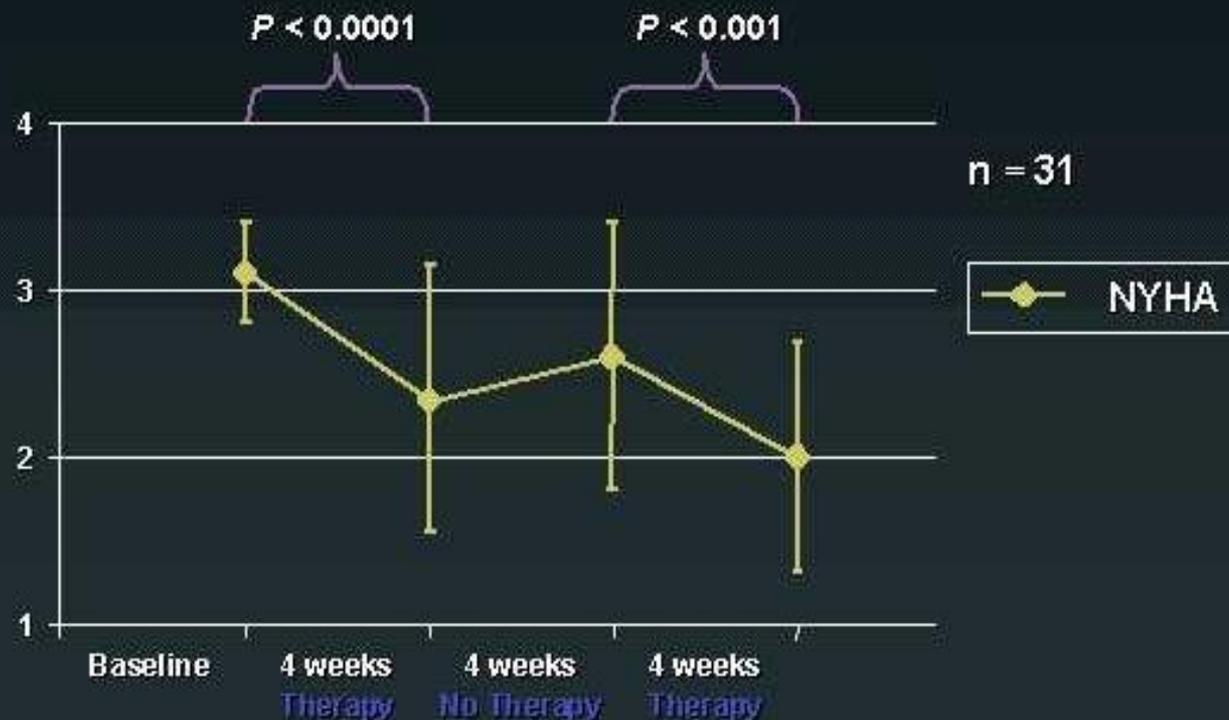




The PATH-CHF Study Results

Symptom Relief

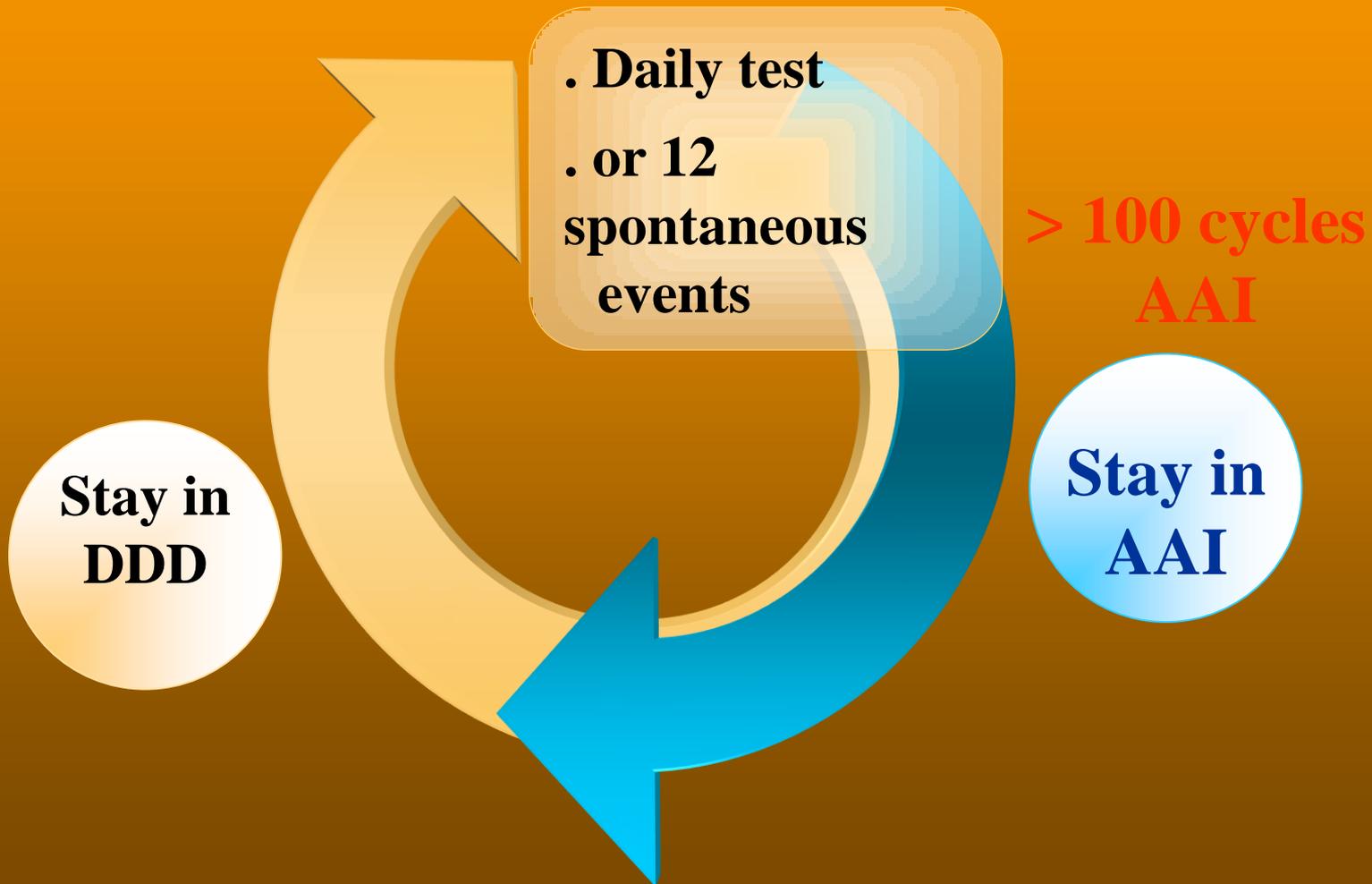
NYHA Functional Class



Auricchio A, et al. Chronic benefit as a result of pacing in congestive heart failure: results of the PATH-CHF trial. *Journal of Cardiac Failure*. 1999;5:78.



Minimalizace komorové stimulace



Patient Look system



ICD



Conexus™
activator



- Medtronic CareLink®
(2090W)
Programmer
- wireless



MEDTRONIC OPTIVOL[®]



Lower fluid = higher transthoracic impedance



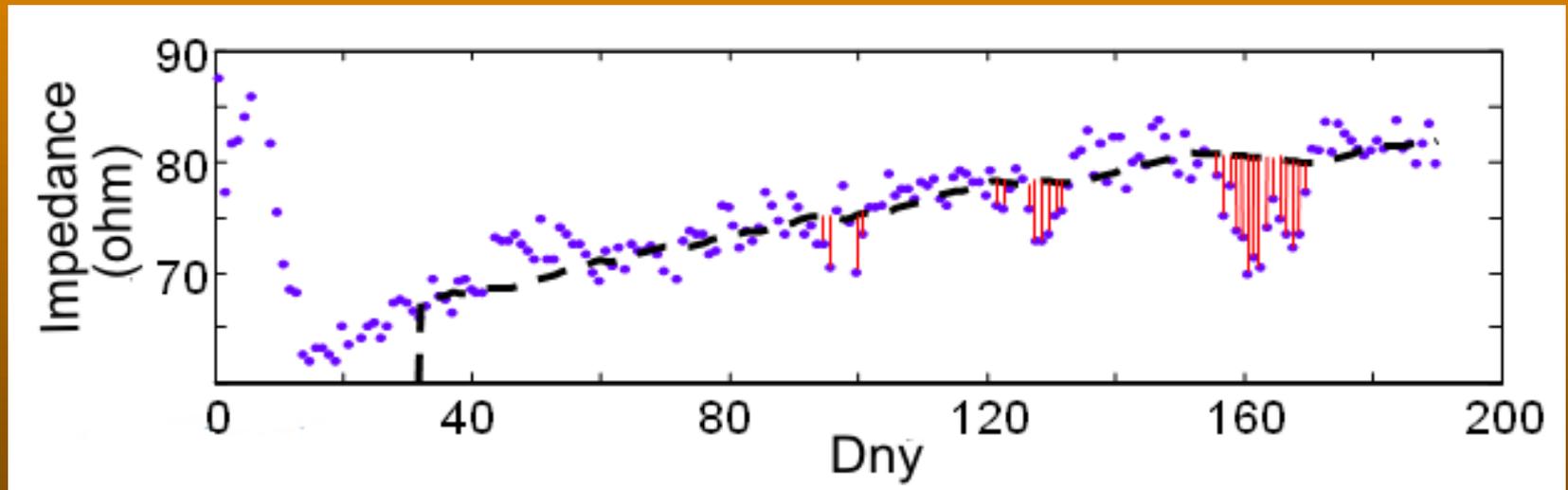
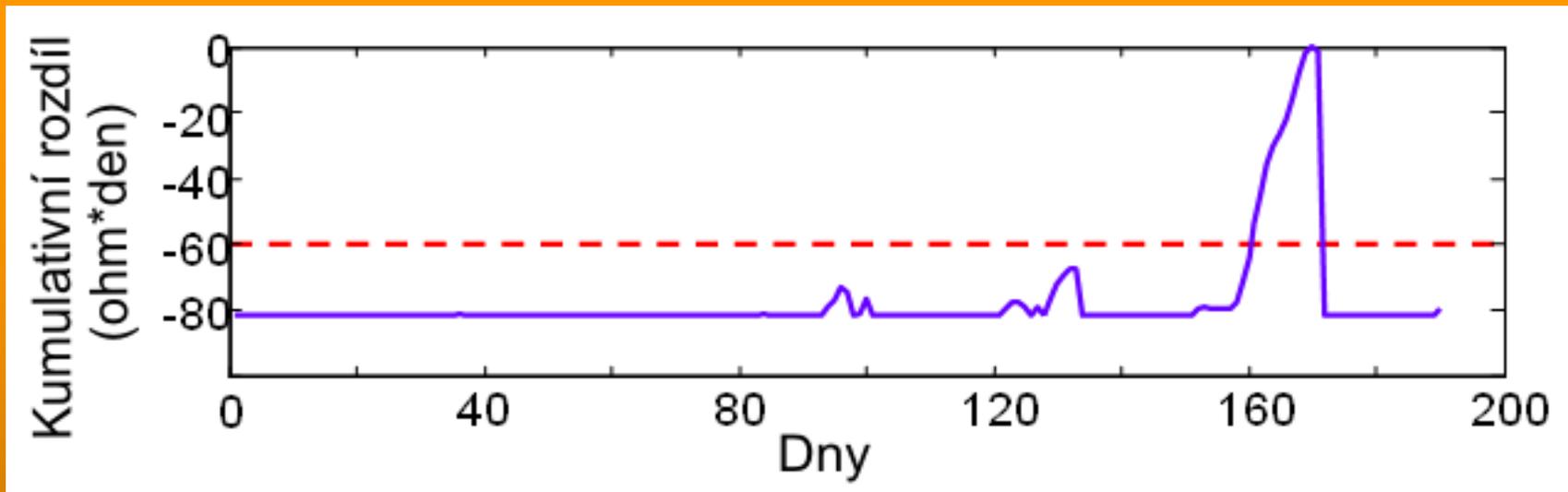
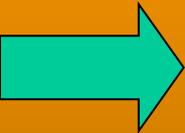
better



Higher fluid = lower transthoracic impedance



worse



UP TO DATE STATUS

2003 2004 2005 2006 2007 2008

BIO



Home Monitoring ('02)

Home Monitoring II



MDT



Directo - CareLink Programmer & RemoteView

CareLink Network ('02)

CareLink Network



STJ



HouseCall

HouseCall Plus

HouseCall Plus



GDT



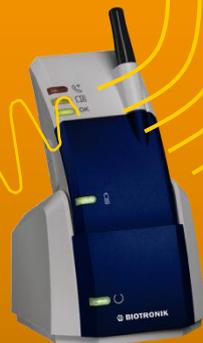
Renewal/Inductive/Frontier

Frontier



Home Monitoring

Implant with Home Monitoring



BIOTRONIK
Service Center



Patient

Physician

Cardio Report



Housecall +



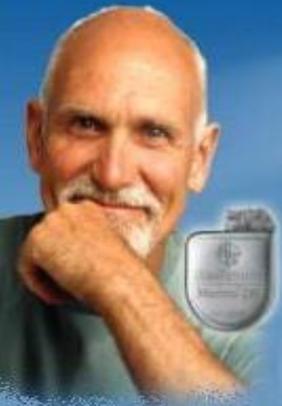
CareLink and Paceart Integration

Medtronic
CareLink®
Monitor

Medtronic
CareLink
Network

Paceart® System

Electronic
Medical Record
(EMR)



•ICD HARDWARE

Medtronic Implantable Defibrillators (1989-2000)

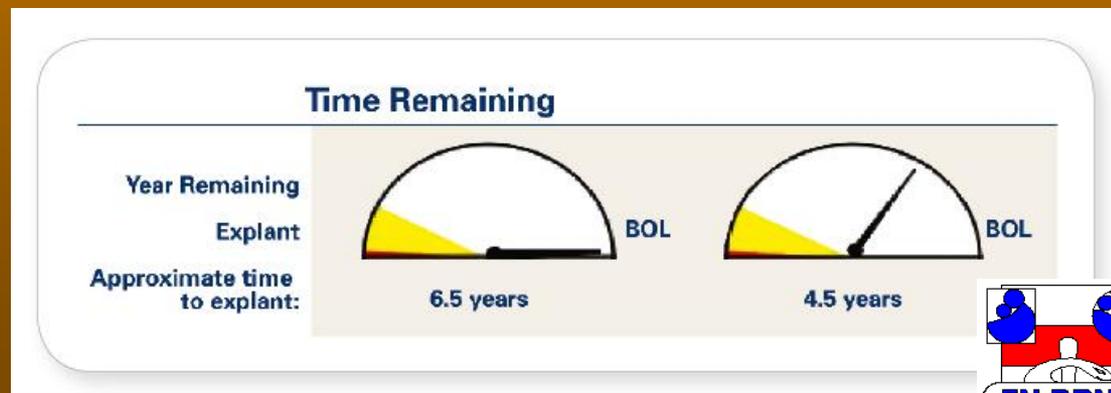
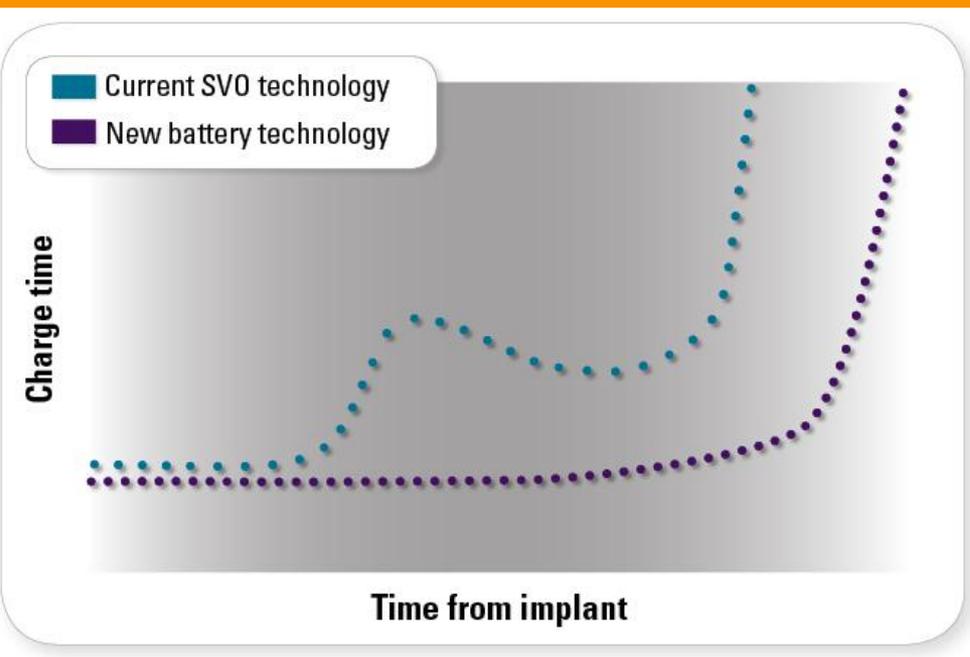


© Copyright Medtronic, Inc.

	COGNIS	TELIGEN
		
Objem (cm³)	32.5	31.5 / 30.5*
Tloušťka (mm)	9.9	9.9
Hmotnost (g)	72.0	71.0

•ICD HARDWARE

•Nová technologie baterie Li/MnO₂



•ICD HARDWARE

Summary - Lead



- Dual Shock, Bipolar Defibrillator Lead
- (One IS-1 and two DF-1 Connectors)



- Single Four-pole High / Low Voltage Connector



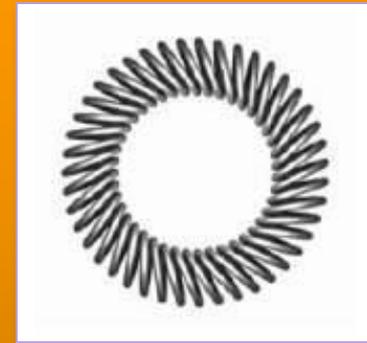
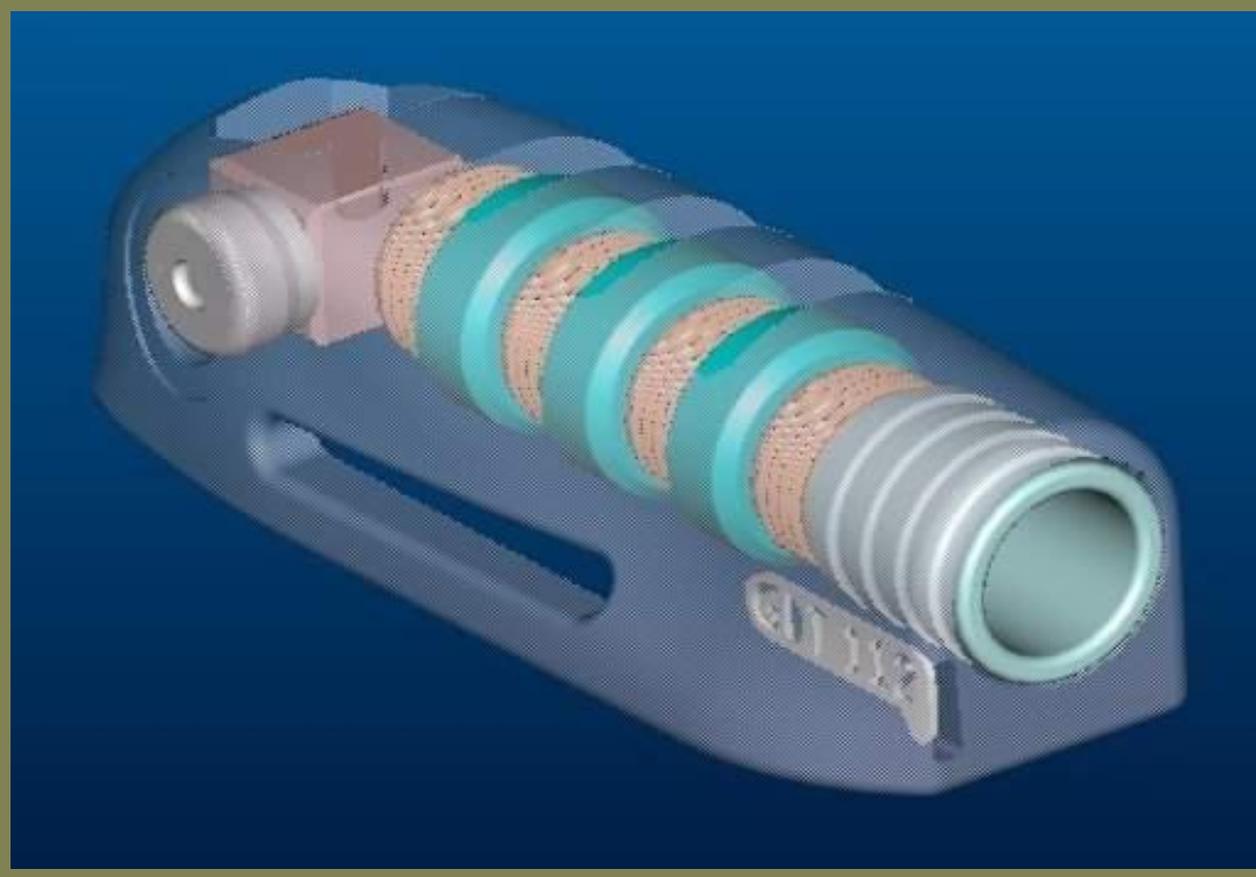
- Four-pole Brady Lead
- (Two IS-1 Connectors)



- Single Four-pole Low Voltage Connector

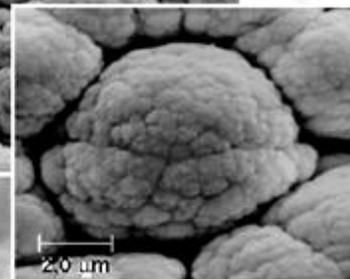
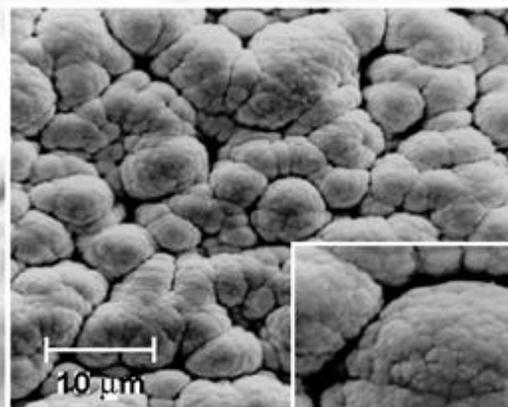
•ICD HARDWARE

IS - 4 Hlava ICD



•ICD ELEKTRODY

•Mapping capabilities

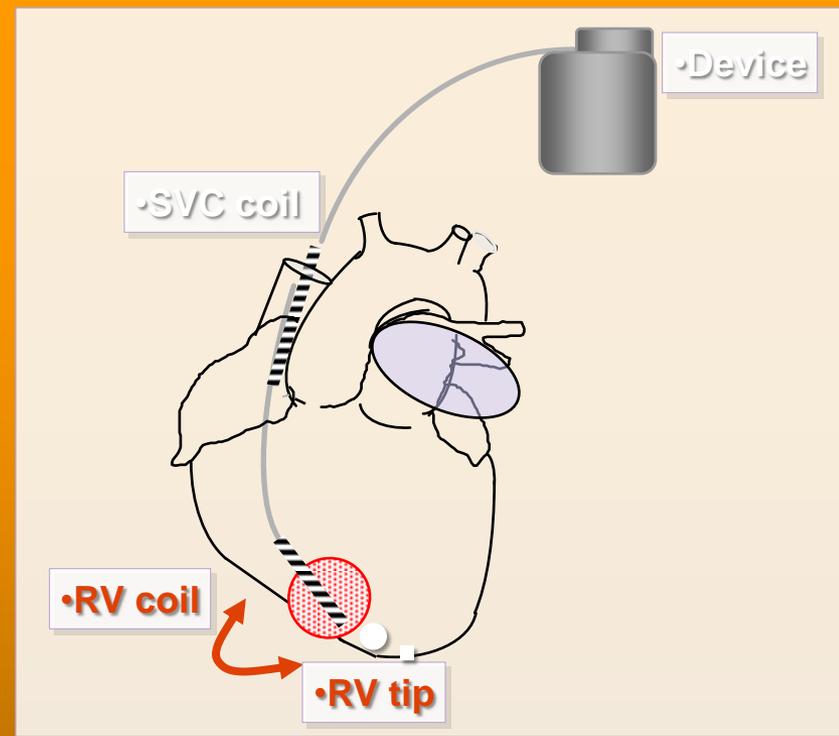


•ICD SOFTWARE

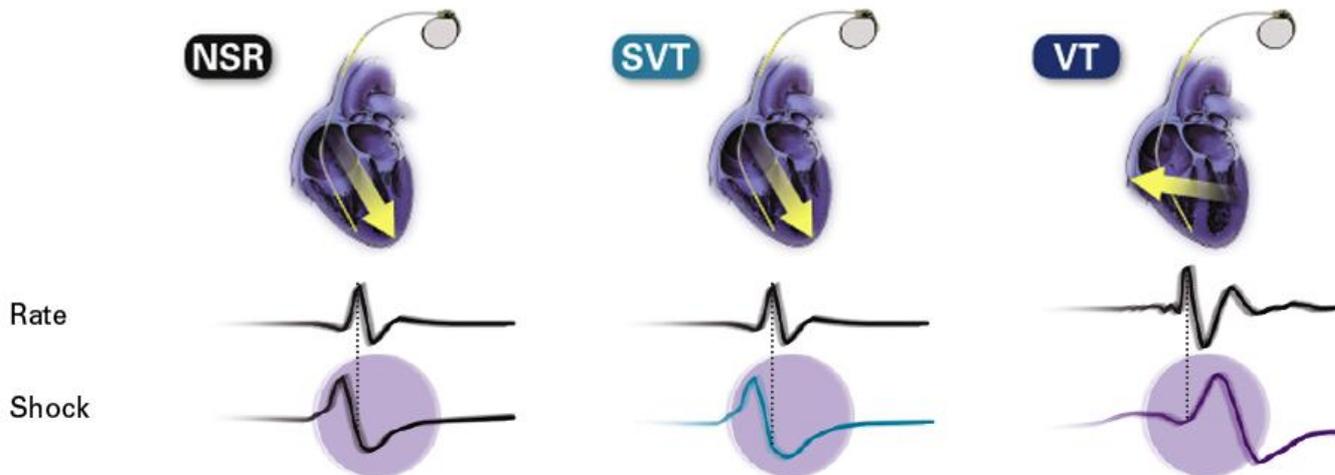
•Dynamic Noise Algorithm - D.N.A.



•ICD SOFTWARE



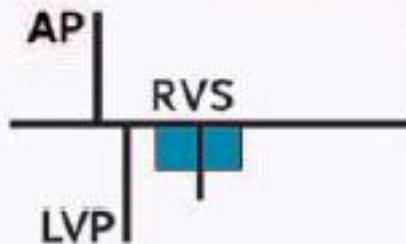
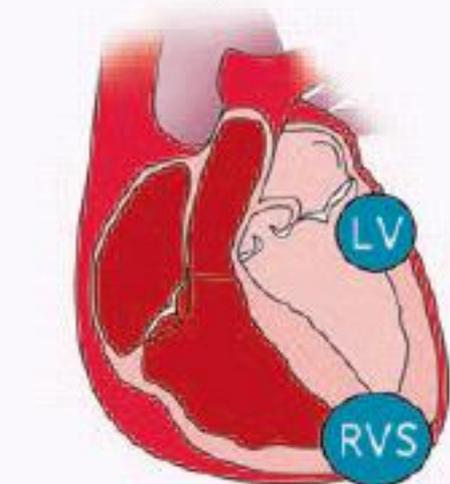
Enhanced Rhythm ID



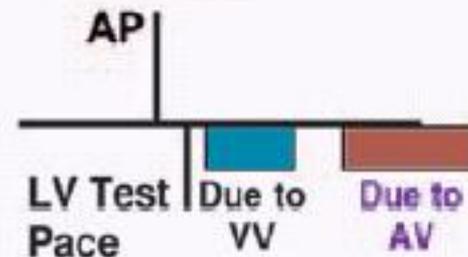
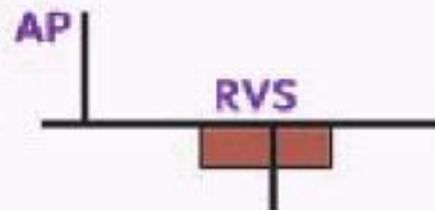
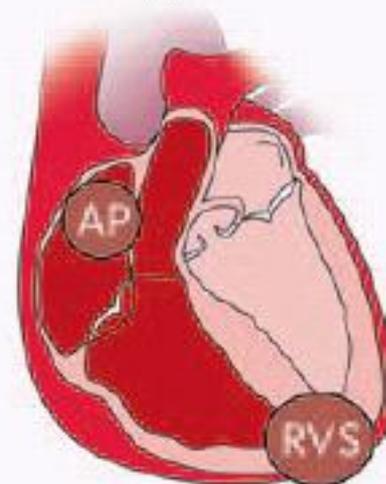
•ICD SOFTWARE

•LV capture management

Measure LVP-RVS Interval

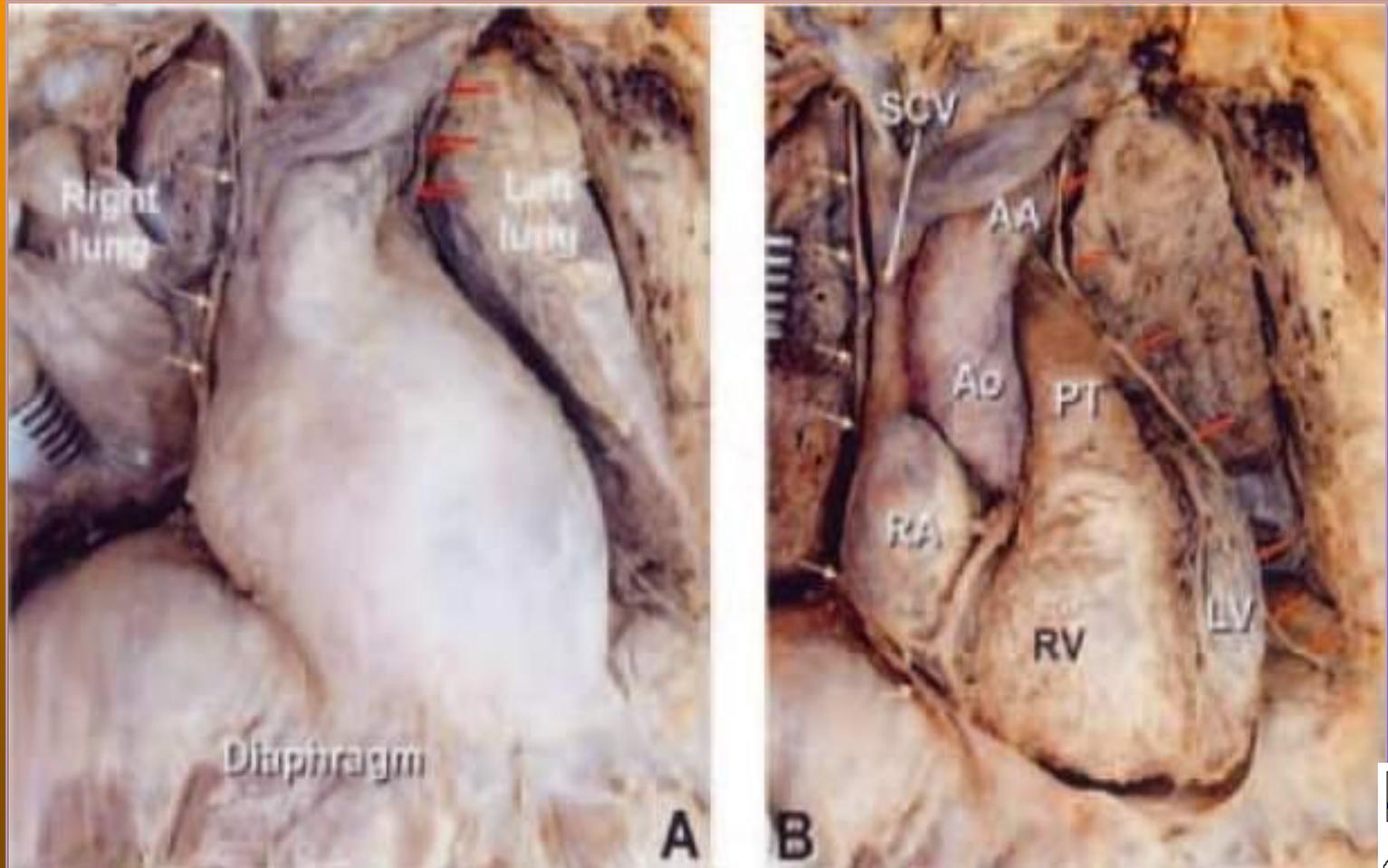


Measure AV Conduction Interval



•ICD SOFTWARE

•Anatomie nervus phrenicus



•ICD SOFTWARE

•Stimulační konfigurace v prevenci stimulace n. phrenicus

Electronic Repositioning™

1 LV Tip to LV Ring
Dedicated Bipolar Pacing Vector

2 LV Ring to LV Tip
Dedicated Bipolar Pacing Vector

3 LV Tip to RV
Extended Bipolar Pacing Vector

4 LV Ring to RV
Extended Bipolar Pacing Vector

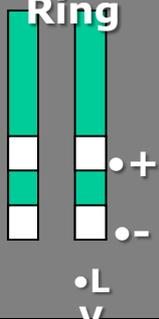
5 LV Tip to Can
Unipolar Pacing Vector

6 LV Ring to Can
Unipolar Pacing Vector

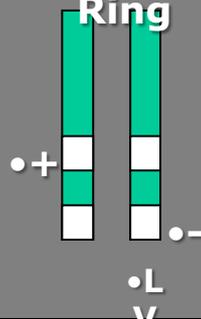
Copyright 2008 Boston Scientific Corporation or its affiliates. All rights reserved.



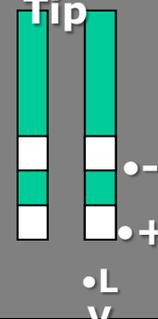
•LV-Tip → LV-Ring



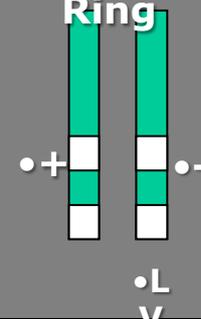
•LV-Tip → RV-Ring



•LV-Ring → LV-Tip



•LV-Ring → RV-Ring



•ICD SOFTWARE

QuickOpt™
TIMING CYCLE OPTIMIZATION

OPTIMAL CRT PROGRAMMING

Change in optimal AV and V-V delay over time in the total patient cohort

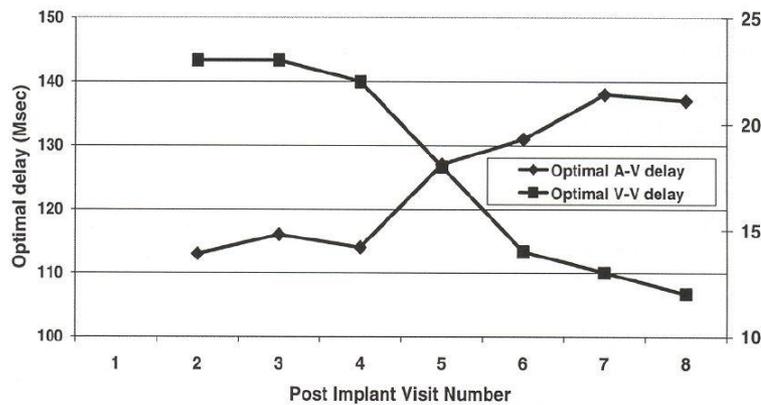


Figure 1. Temporal variation in optimal V-V and AV delays over the eight post-implant visits in the overall patient cohort. There is a significant reduction in LV predominance of the V-V delay and a significant increase in optimal AV delay.



•ICD SOFTWARE

QuickOpt™
TIMING CYCLE OPTIMIZATION

ST JUDE MEDICAL Atlas®+ HF Model: V-343 Serial: 159260 1 Jun 2006 16:38

Surface ECG (2.7 mV/cm)

Defib Status

A Intervals 1855 1859 1855 1859 1855 1859 1855 1859 1855 1859
 U Intervals 1859 1852 1855 1859 1852 1855 1859 1852 1855 1859
 A Events P P P P P P P P P P
 U Events JBU JBU JBU JBU JBU JBU JBU JBU JBU JBU

70 bpm
855 ms

QuickOpt™ Timing Cycle Optimization: Details

AV/PV Delay Optimization

Atrial Wave Duration Tests

A Sense: 61 ms (62, 62, 62, 62, 62, 55, 62 ms) AV Delay: 150 ms Optimize: 170 ms

A Pace: EGM stored PV Delay: 100 ms Optimize: 120 ms

Interventricular Pace Delay Optimization

RV-LV Interval Tests

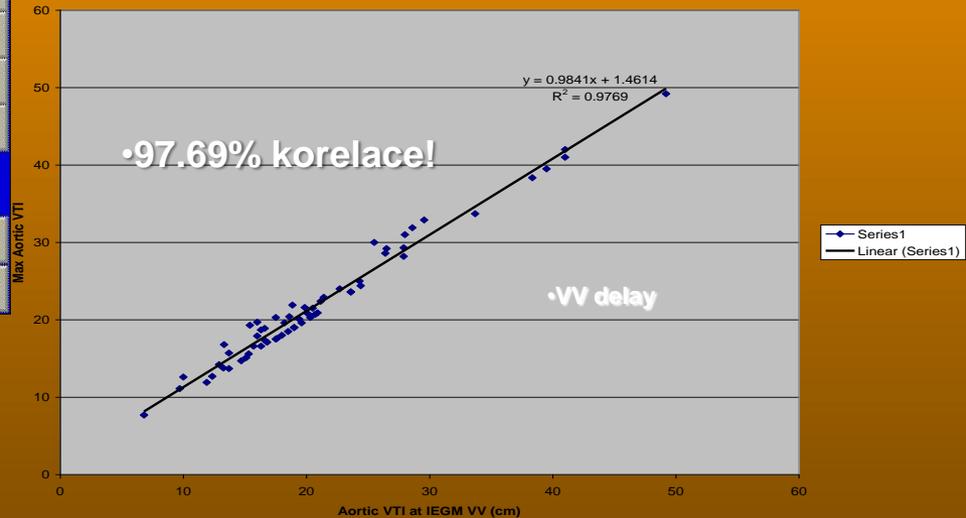
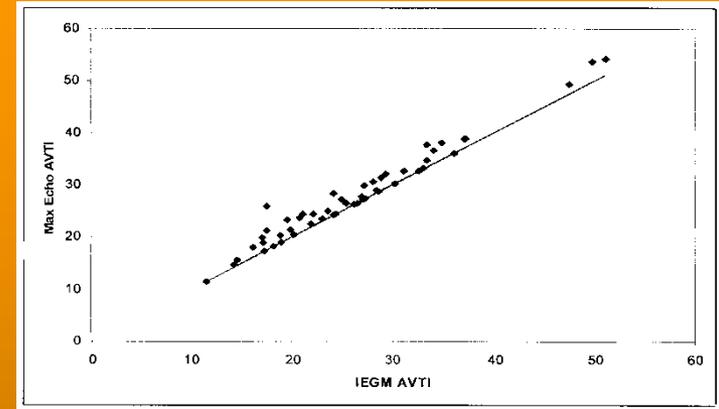
V Sense: 22 ms (LV First) (23, 23, 23, 31, 23, 23, 8, 23 ms) Interventricular Pace Delay: Simultaneous

RV Pace: 16 ms (16, 16, 16, 16, 16, 16, 16 ms) Optimize: 15 ms (LV First)

LV Pace: 4 ms (0, 16, 0, 0, 0, 23, 0, 0 ms)

Print Report Close

Freeze
EGG/ECG Control
Summary
Parameters
Diagnostics
Episodes
Tests
Measured Data
Main Menu
End Session



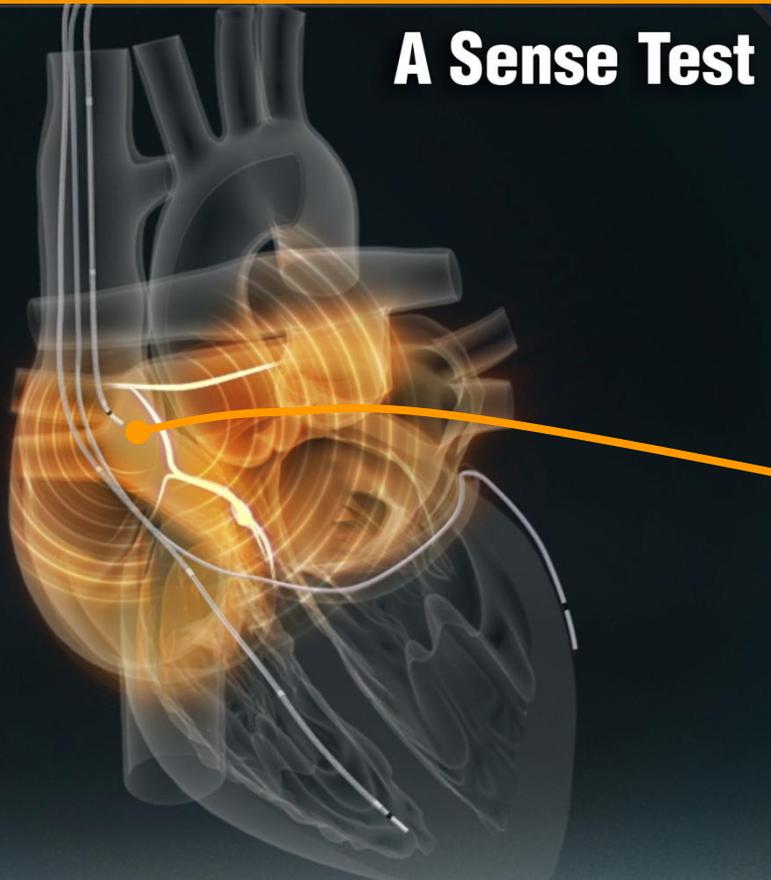
•Porterfield, et al. "Device based intracardiac delay optimization vs. echo in ICD patients (Acute IEGM AV/PV and VV Study)" Europace Vol 8 Supp 1 July 2006 [abstract #6178].

•Meine, et al. "An Intracardiac EGM Method for VV Optimization During Cardiac Resynchronization Therapy" Heart Rhythm Journal 3 (5) May 2006 [abstract AB30-5]

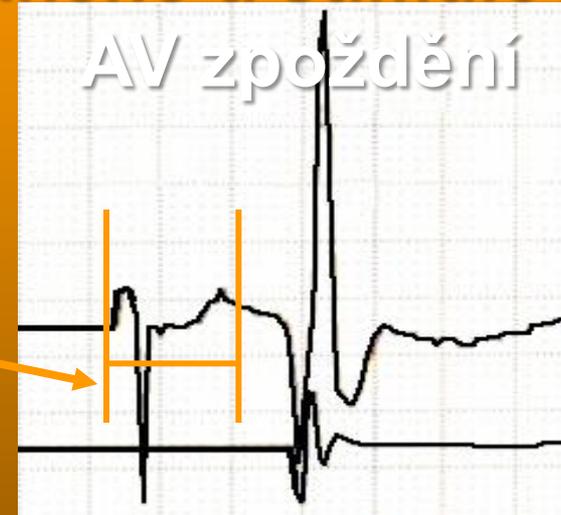
•ICD SOFTWARE

QuickOpt™
TIMING CYCLE OPTIMIZATION

A Sense Test



- Elektronická optimalizace snímaného a stimulovaného

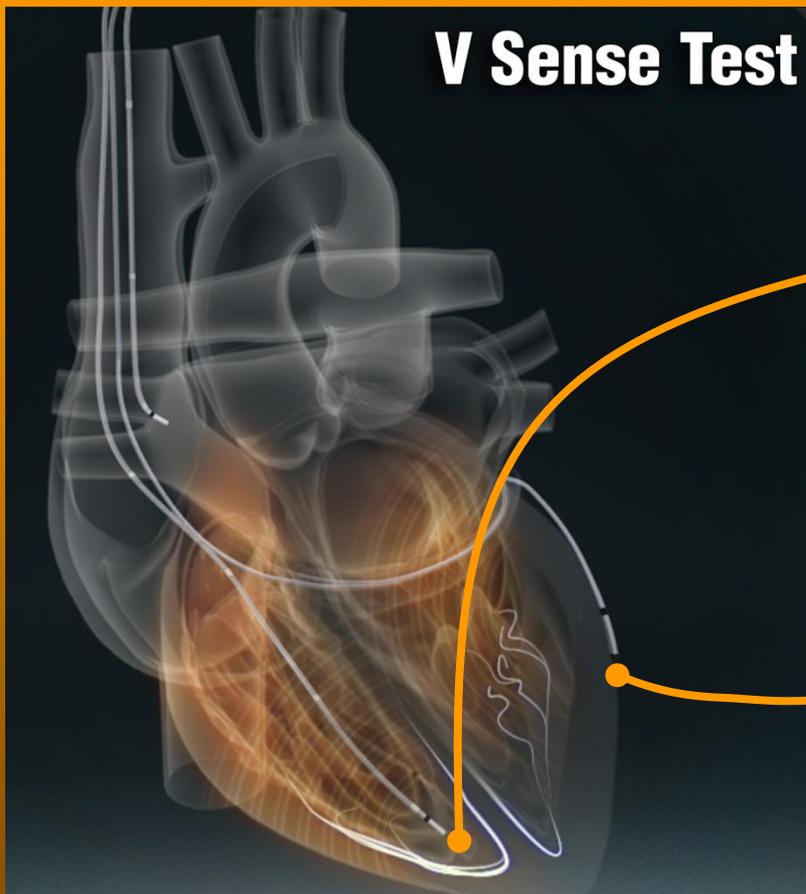


- Trvání P vlny je odrazem aktivace PS + LS. QuickOpt™ na základě tohoto intervalu počítá optimální S/P AV zpoždění k zajištění max. preloadu a umožnění správného načasování uzávěru Mi chlopně.

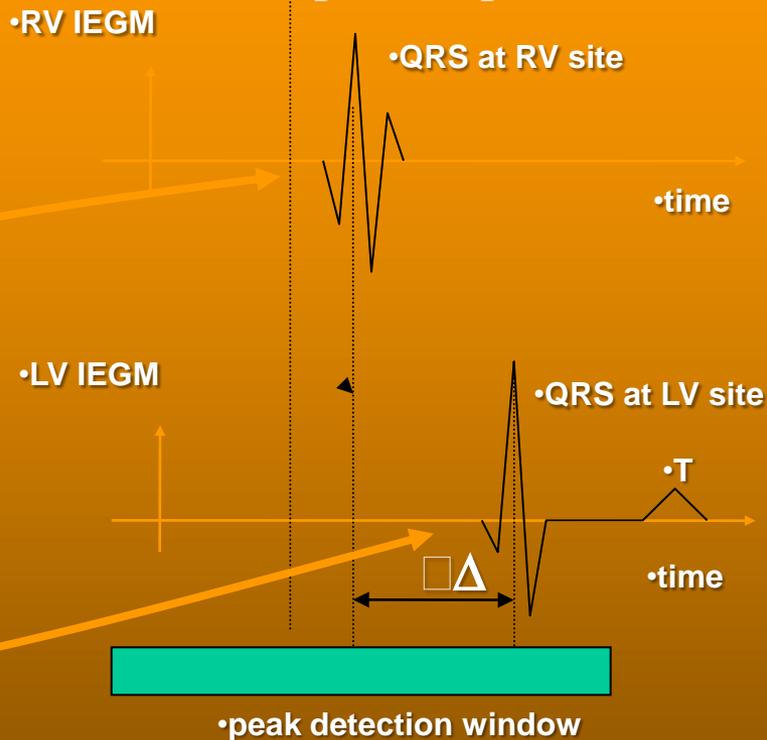
¹ Worley, et.al "Optimization of cardiac resynchronization: left atrial electrograms measured at implant eliminates the need for echo and identifies patients where AV optimization is not possible" *Journal of Cardiac Failure* Aug. 2004 Vol. 10, Issue 4, Pg S62.

•ICD SOFTWARE

QuickOpt™
TIMING CYCLE OPTIMIZATION



V-V optimalizace:
rozdíl časování spont. depolarizace (Δ)

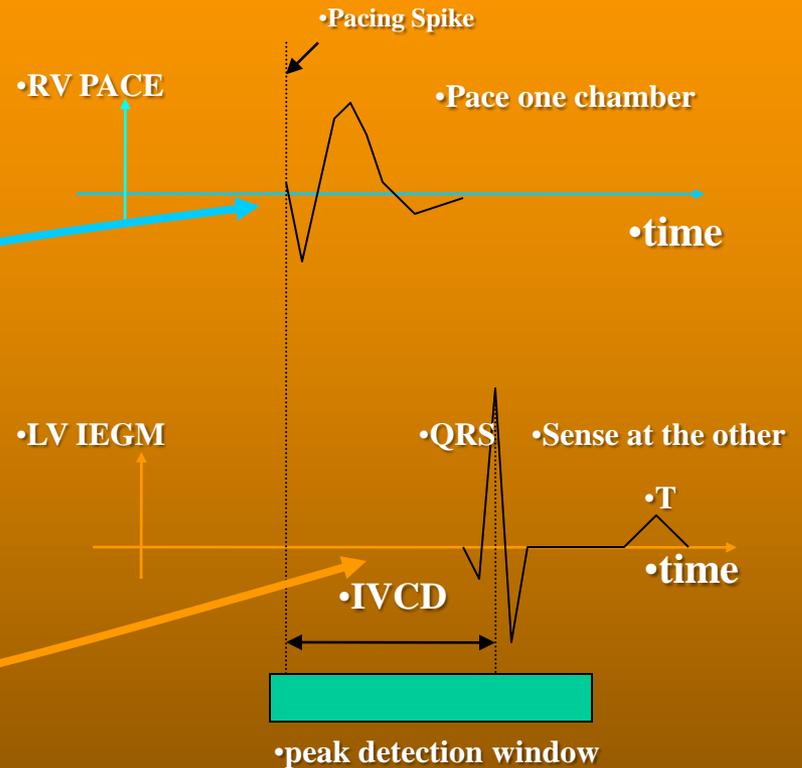
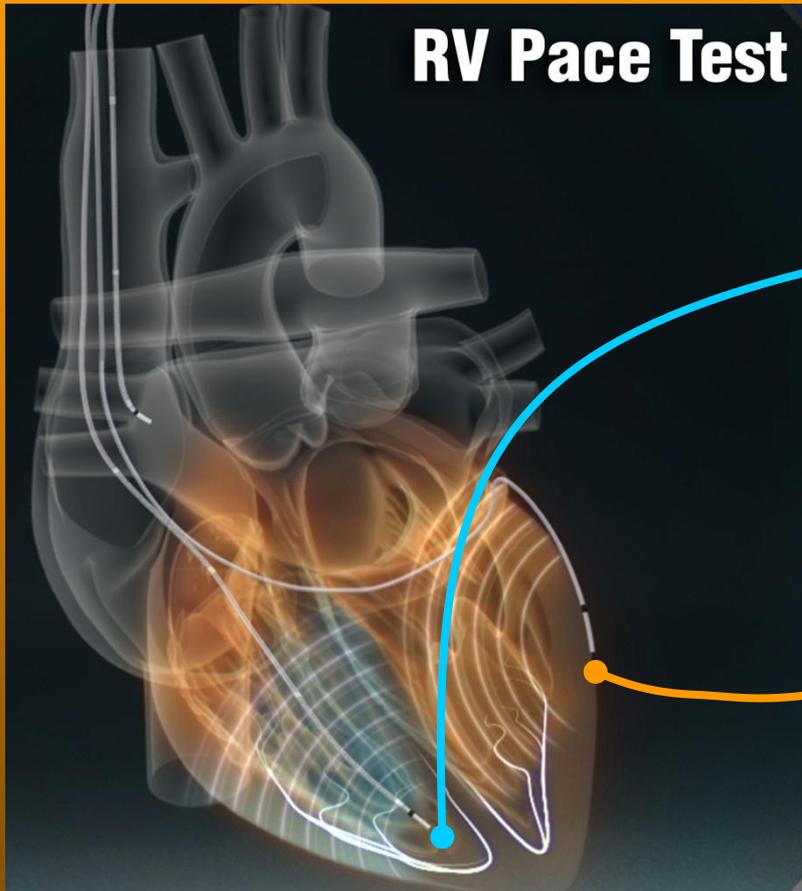


•Vedení v myokardu je definováno P/S testem. Cílem je aktivovat PK i LK tak, aby se stimulované elektrické aktivační vlny setkaly na IVS.

•ICD SOFTWARE

QuickOpt™
TIMING CYCLE OPTIMIZATION

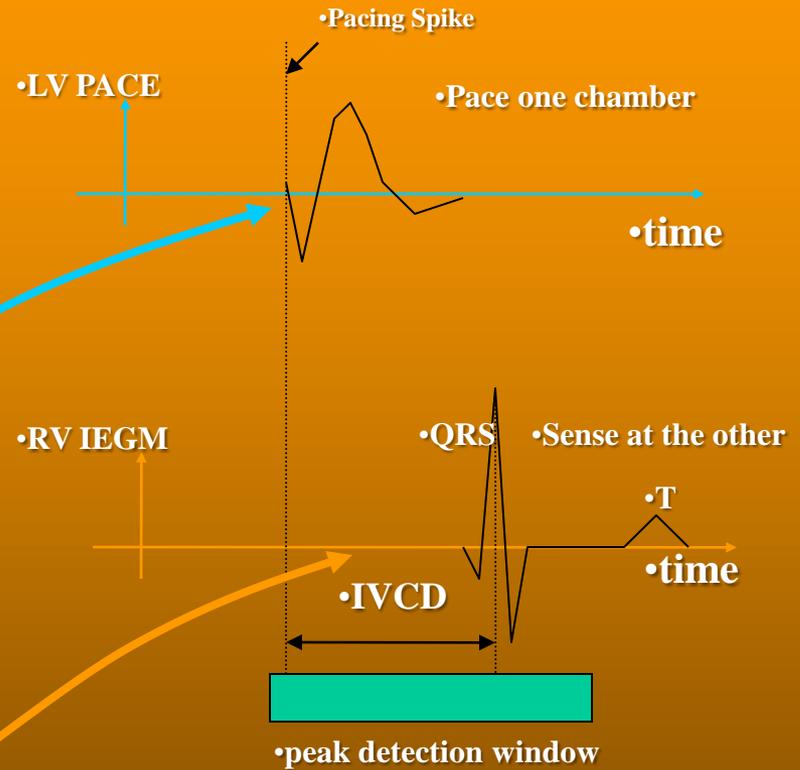
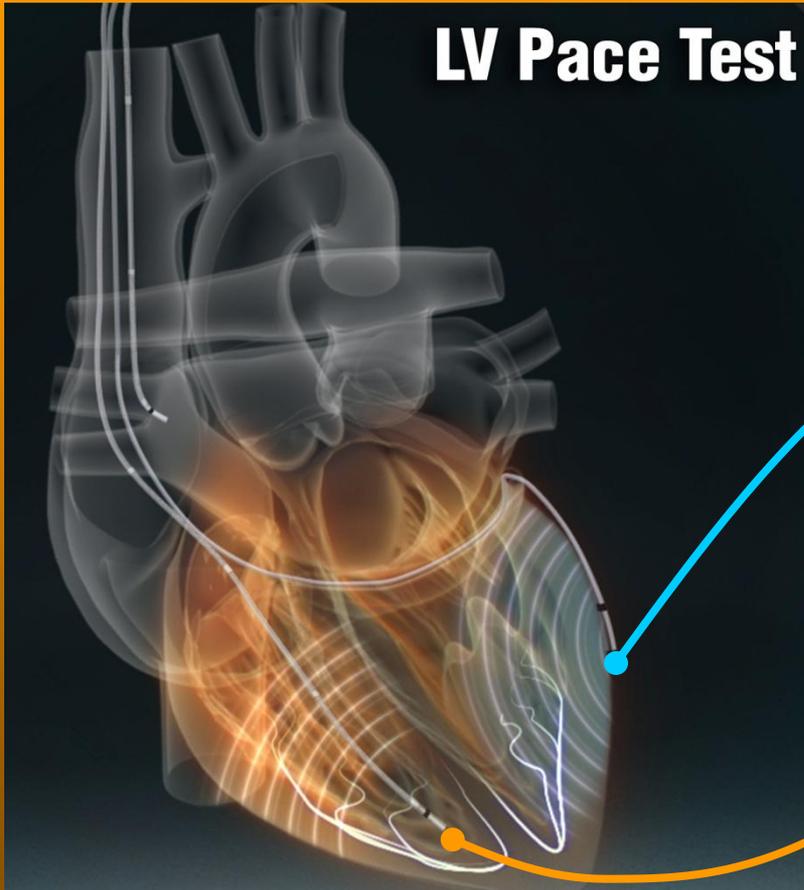
V-V optimalizace: rychlost aktivačních vln (ϵ)



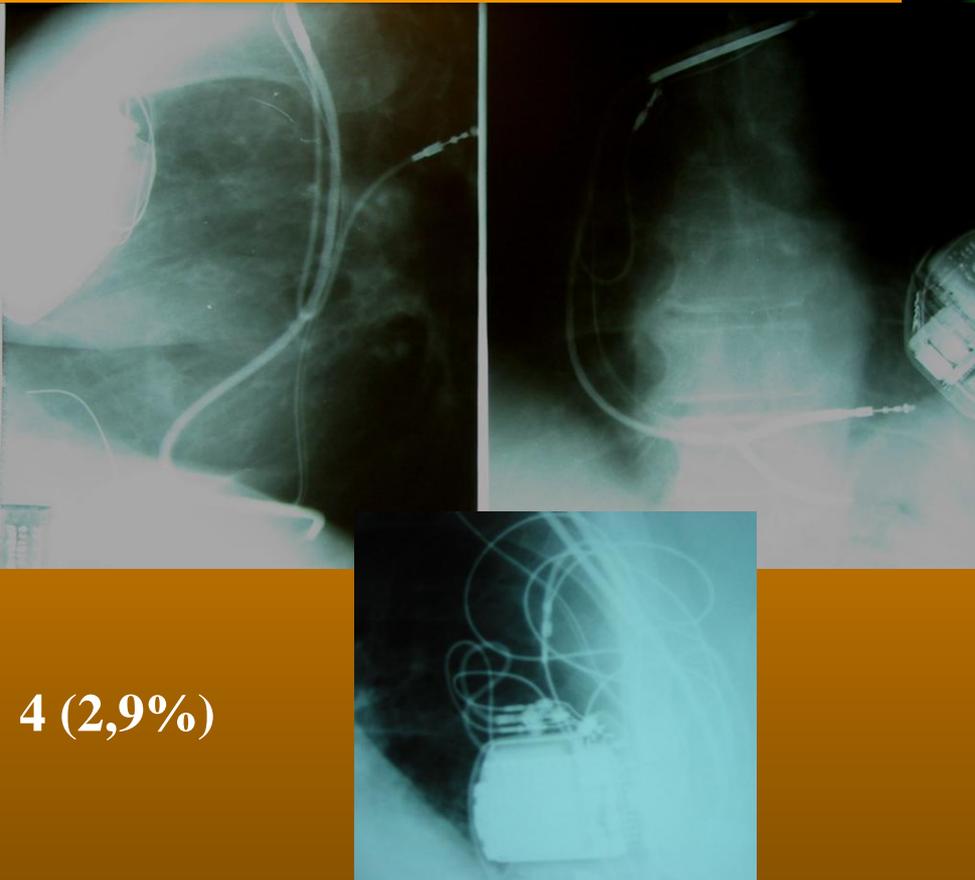
•ICD SOFTWARE

QuickOpt™
TIMING CYCLE OPTIMIZATION

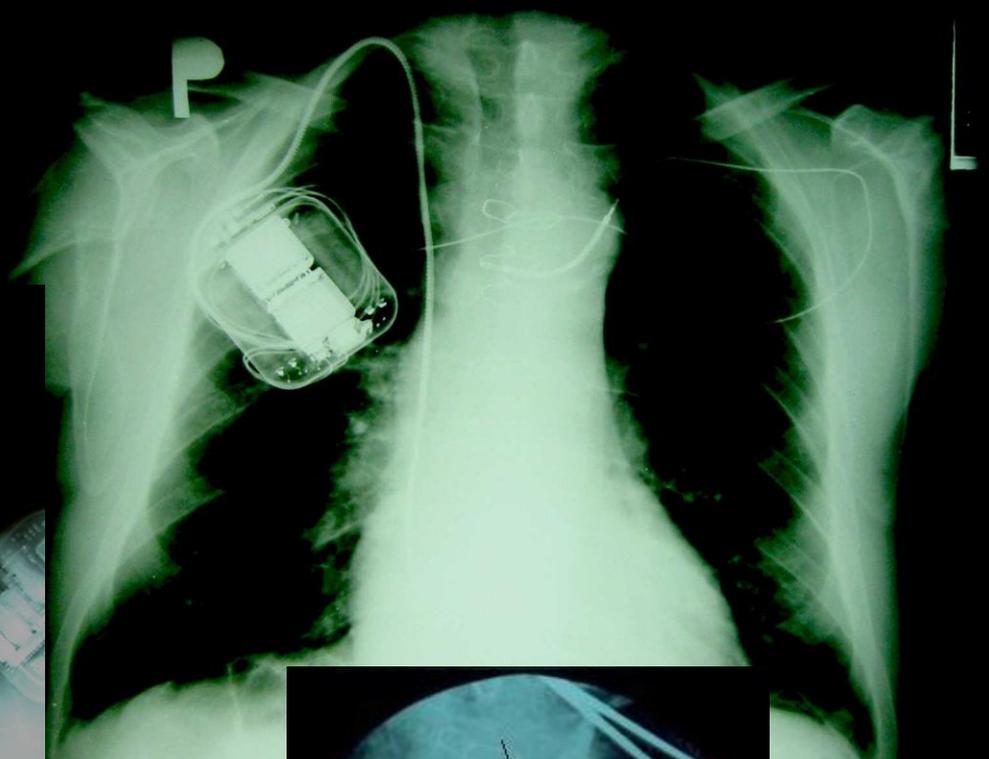
LV Pace Test



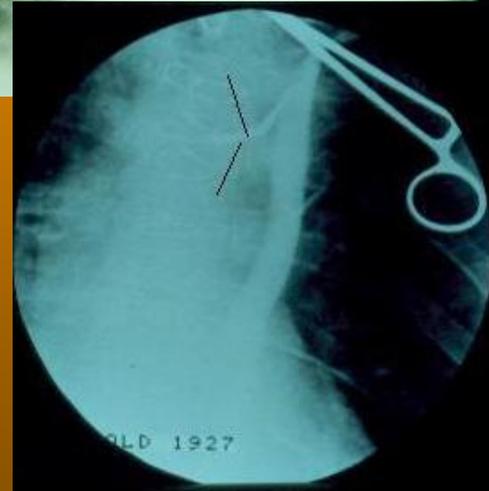
TROUBLESHOOTINGS



4 (2,9%)



5 (3,6%)



Kozák M, Sepši M, Křivan L et al. Cardiol 2002;11(4):259-263

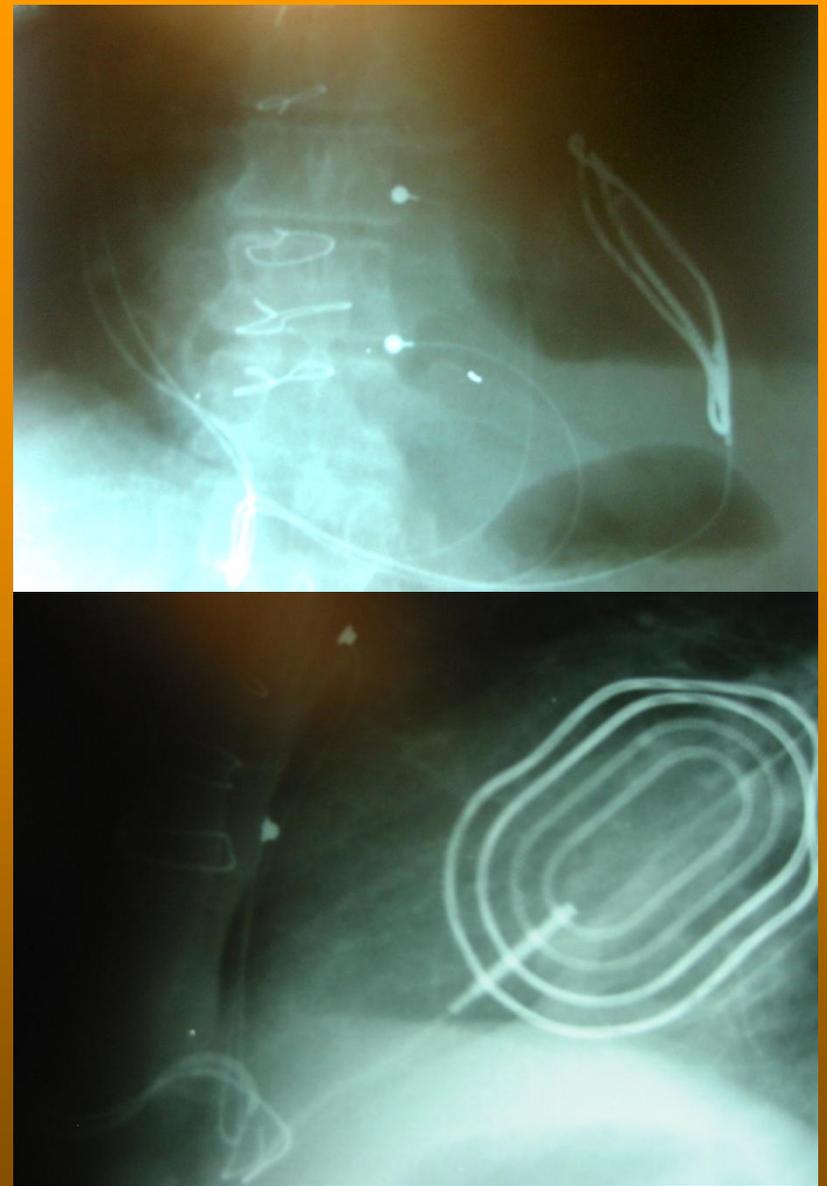
Kozák M, Křivan L, Semrád B et al. Cor Vasa 1999;41(5):252-254



TROUBLESHOOTINGS



4 pac, 2x ICD kapsa, 2x IE



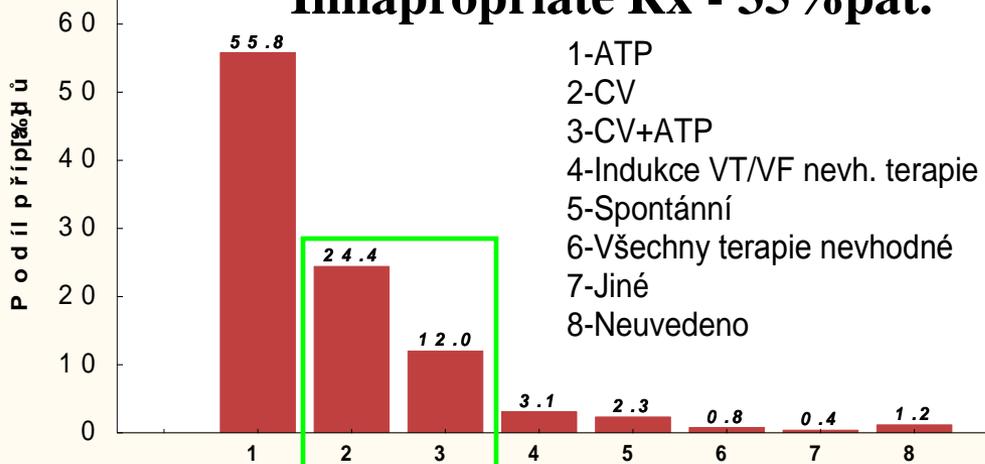
Křivan L, Kozák M, Sepši M et al. Cardiol 2001;10(5):238-242

Křivan L, Kozák M, Sepši M et al. Čas Lék čes 2004;143:521-525



TROUBLESHOOTINGS

Innapropriate Rx - 35%pat.

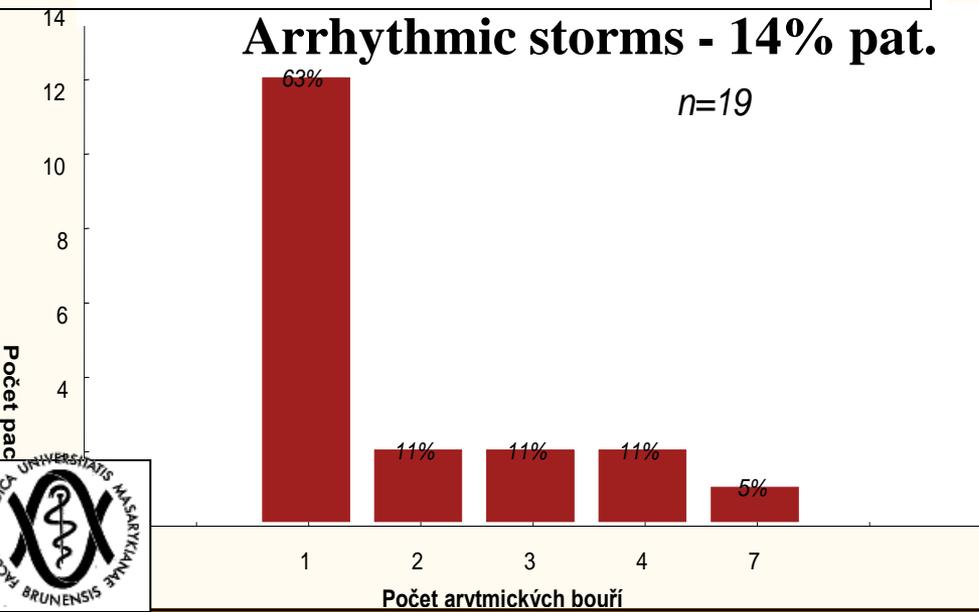


- 1-ATP
- 2-CV
- 3-CV+ATP
- 4-Indukce VT/VF nevh. terapie
- 5-Spontánní
- 6-Všechny terapie nevhodné
- 7-Jiné
- 8-Neuvedeno

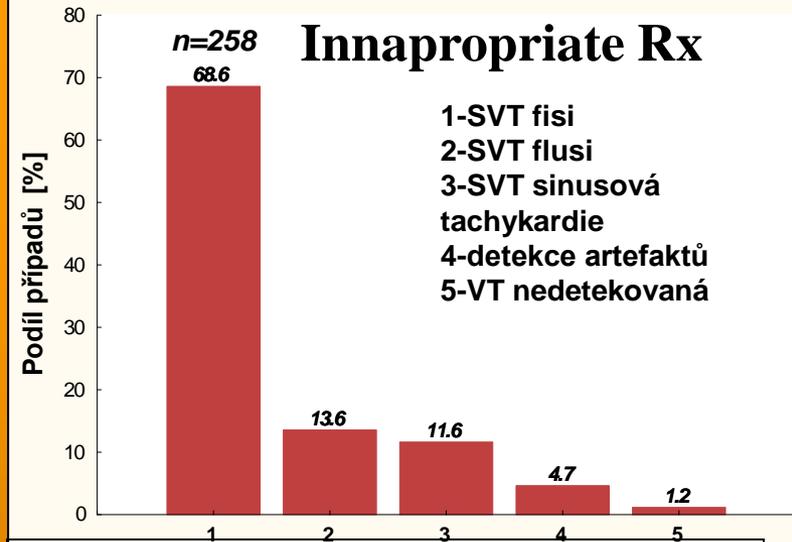
ATP - antitachykardická stimulace, CV - kardioverze

Arrhythmic storms - 14% pat.

n=19

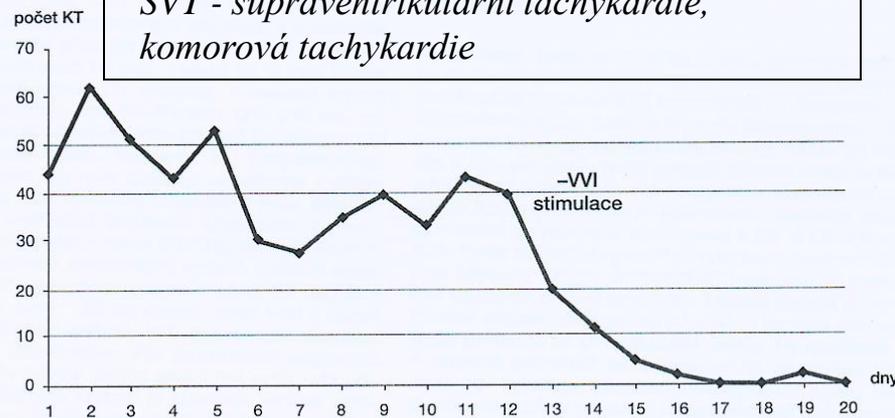


Innapropriate Rx



- 1-SVT fisi
- 2-SVT flusi
- 3-SVT sinusová tachykardie
- 4-detekce artefaktů
- 5-VT nedetekovaná

SVT - supraventrikulární tachykardie, komorová tachykardie



1 Četnost epizod KT během arytmiické bouře a po programaci VVI stimulace

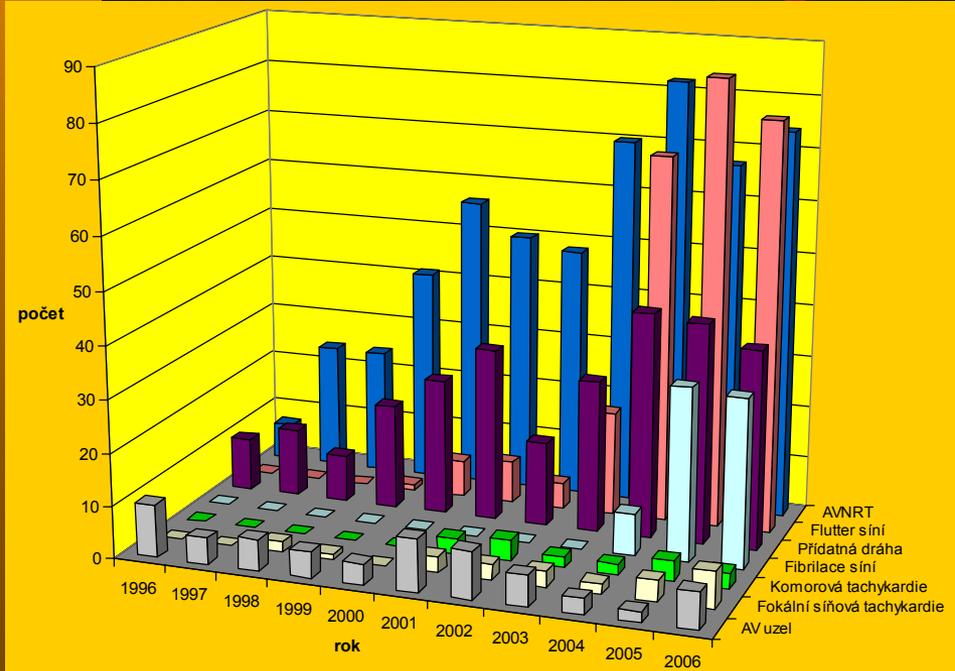
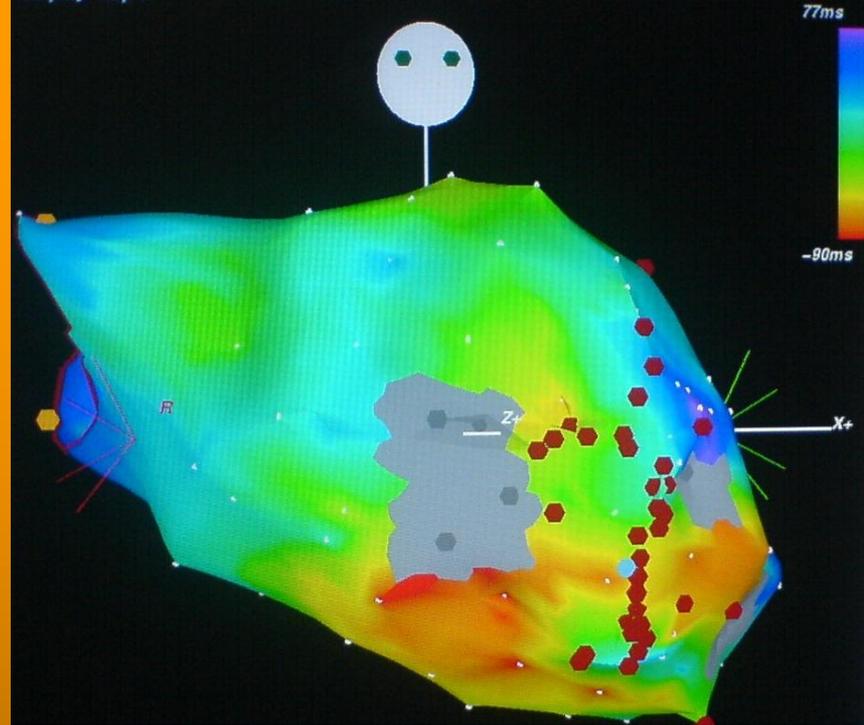
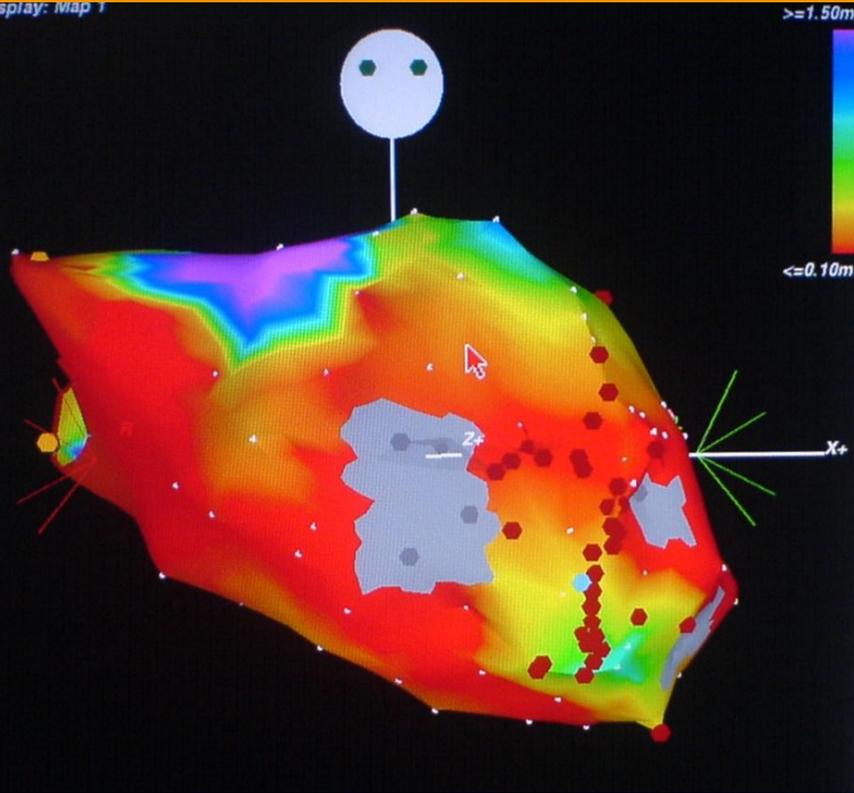
Křivan L, Kozák M, Vlašínová J et al., Cor Vasa 1999;41(2):112-115

Křivan L, Kozák M, Sepši M et al., Med Sci Monit 2005;11(9):CR426-429



RFA VT

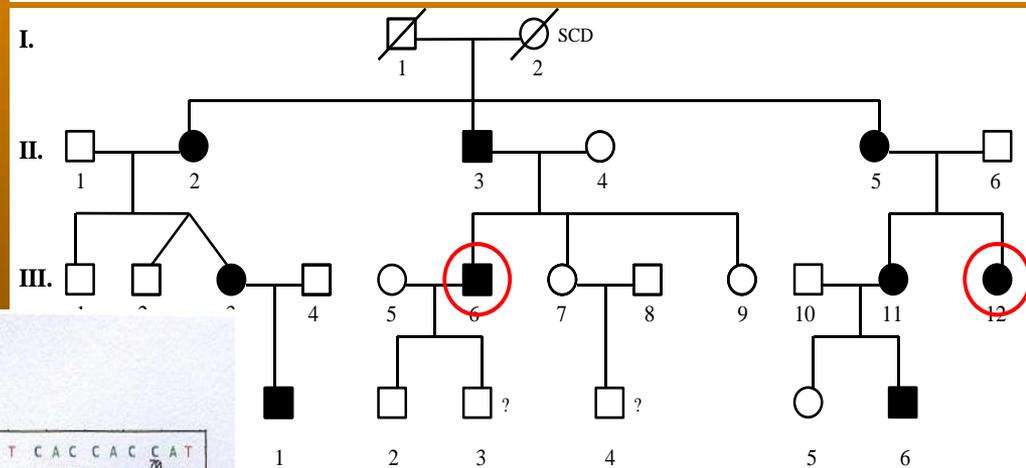
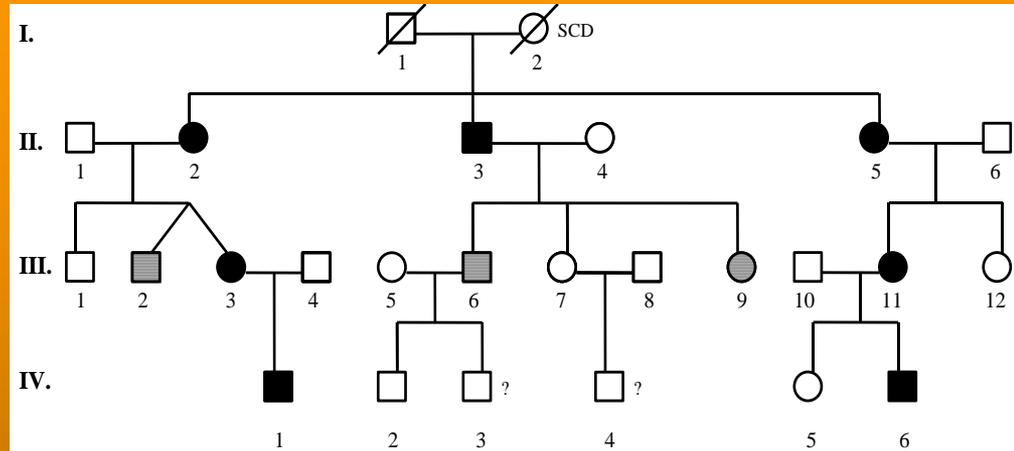
Display: Map 1



PREVENTIVE PROGRAMMS

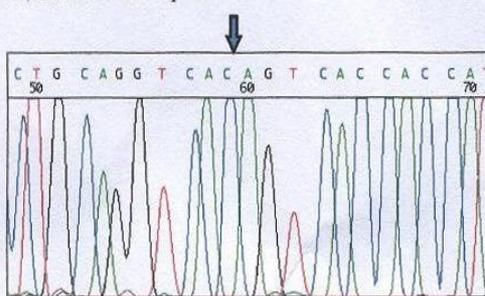
LQTsy

FENOTYPE



GENOTYPE

A) normal DNA sequence



B) mutant DNA sequence

