Pacemaker Therapy

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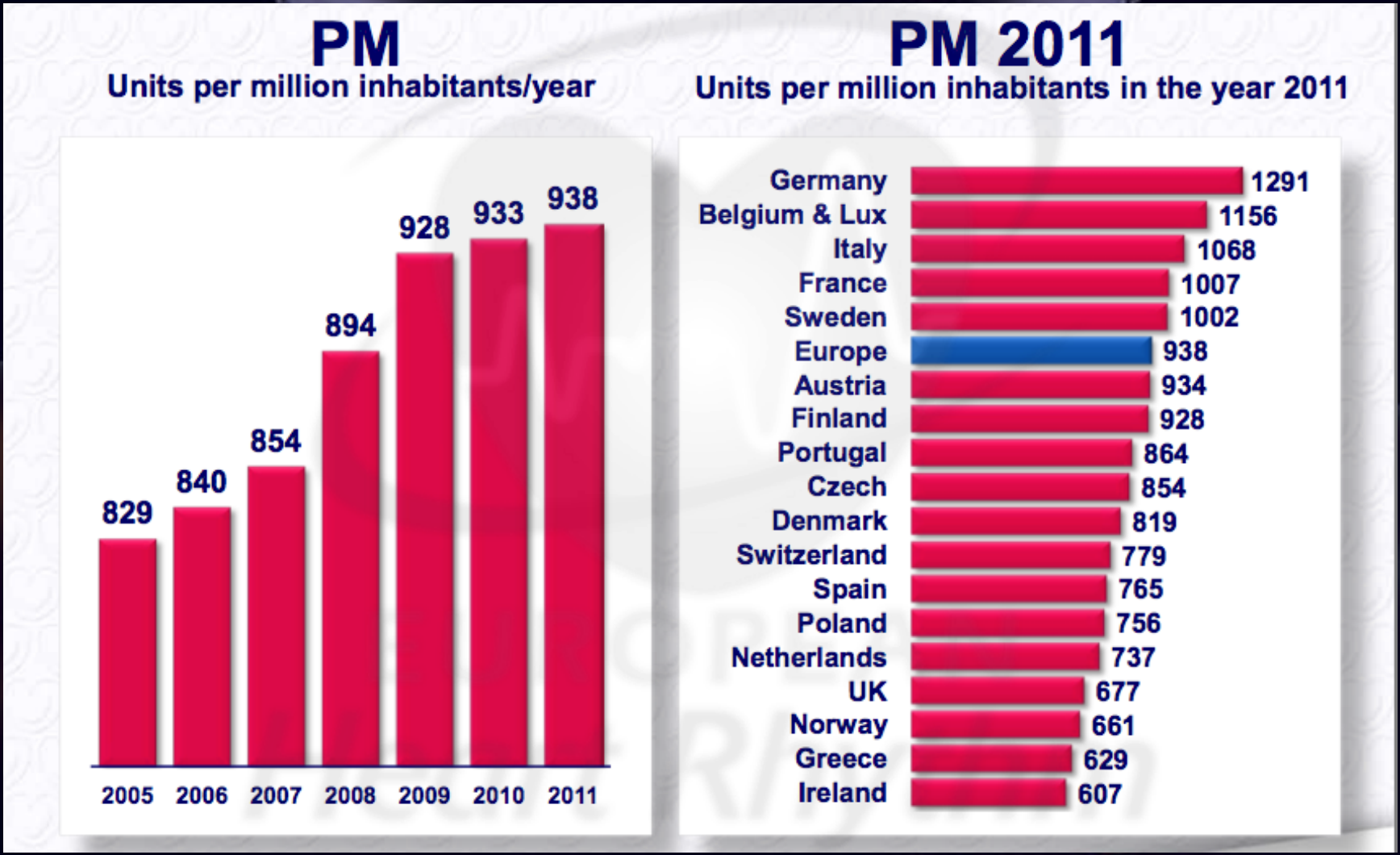
Pacemaker therapy
(CIED-Cardiac Implantable Electronic Devices)

- Temporary or permanent
- Temporary :
  - transcutaneous (rare, only during resustitation)
  - transvenous (v. jugularis, subclavian or femoralis vein)
  - transoesophegeal (nowadays very rare)
  - pull-back (during heart surgery)

History

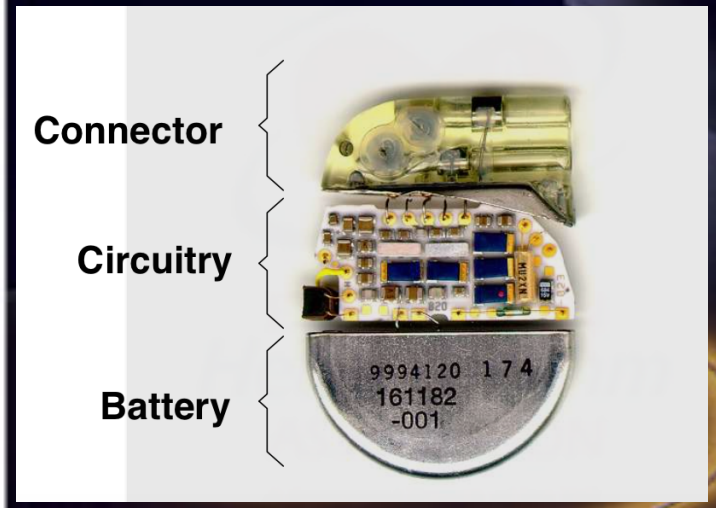
- The first artificial pacemaker was designed and built in 1950 by the Canadian electrical engineer, John Hopps.
- It was not implanted into the body and relied on external electrodes that had to be plugged into a wall outlet.
- Patient could go only as far as the extension cord and a power blackout was of constant concern.
- In 1958 the first pacemaker was implanted into the body which had a battery life of ~12 to 18 months.
- **First ICD implantation: 1980**

Implantation rate in Europe



Parts of Pacemaker

- Pulse generator.
  - Produces impulses and houses the electrical circuitry.
  - Constructed of titanium and contains a lithium battery with a life of ~8 to 15 years
  - The battery will provide a low warning months before it has fatigued.
  - Generator sends out electrical impulses through leads that are attached to the myocardium.



Parts of Pacemaker

- Lead(s)
  - Insulated wires that not only receive impulses, but carry signals back from the heart to the generator.
  - Lead(s) are steroid eluting to decrease the inflammation of the interface between the distal tip of the lead and myocardium.

Two types:

active (screw)- less disloc., easy extract

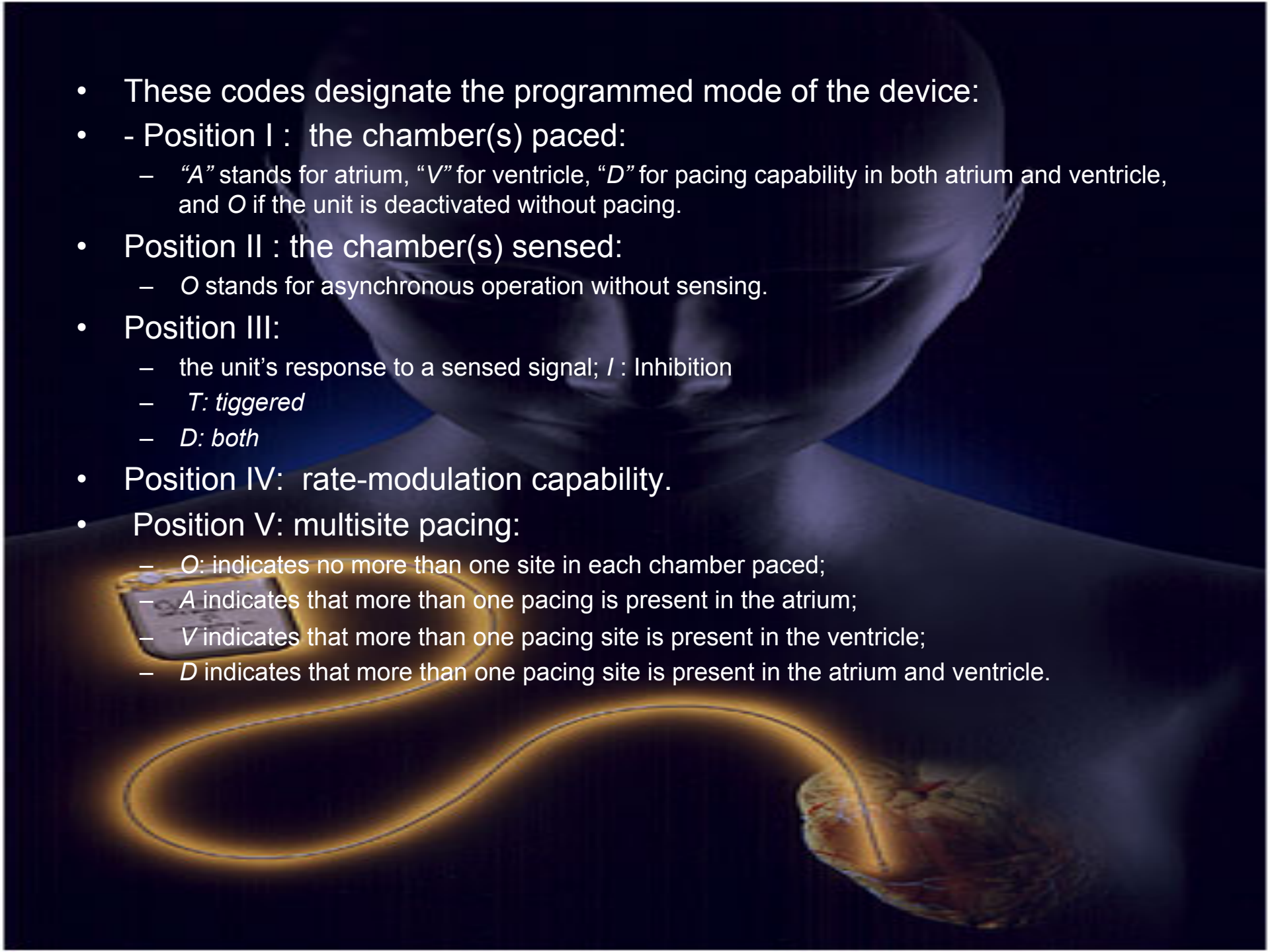
passive (anchor)- less perforation

NASPE- Code sytem

TABLE 8-6 Revised NASPE/BPEG Generic Code for Antibradycardia Pacing

Modified from Bernstein AD, Daubert JC, Fletcher RD, et al. The Revised NASPE/BPEG Generic Code for antibradycardia, adaptive-rate, and multisite pacing. *PACE* 25:260-264, 2000.

Position I (Chambers Paced)	Position II (Chambers Sensed)	Position III (Response to Sensing)	Position IV (Rate Modulation)	Position V (Multisite Pacing)
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	T = triggered	R = rate modulation	A = atrium
V = ventricle	V = ventricle	I = inhibited		V = ventricle
D = dual (A + V)	D = dual (A + V)	D = dual (T + I)		D = dual (A + V)
S = single (A or V)*	S = single (A or V)*			

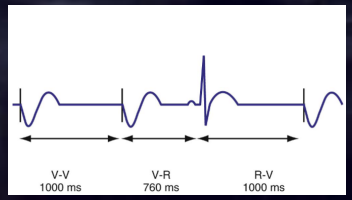


- These codes designate the programmed mode of the device:
- - Position I : the chamber(s) paced:
  - *"A"* stands for atrium, "*V"* for ventricle, "*D"* for pacing capability in both atrium and ventricle, and *O* if the unit is deactivated without pacing.
- Position II : the chamber(s) sensed:
  - *O* stands for asynchronous operation without sensing.
- Position III:
  - the unit's response to a sensed signal; *I* : Inhibition
  - *T: tiggered*
  - *D: both*
- Position IV: rate-modulation capability.
- Position V: multisite pacing:
  - *O*: indicates no more than one site in each chamber paced;
  - *A* indicates that more than one pacing is present in the atrium;
  - *V* indicates that more than one pacing site is present in the ventricle;
  - *D* indicates that more than one pacing site is present in the atrium and ventricle.

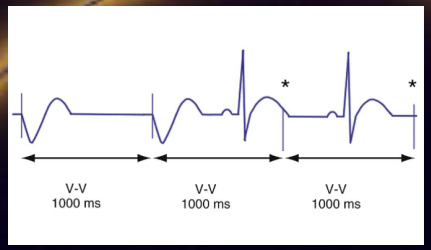
Pacing modes

Asynchronous	**Principle**	permanent pacing at the programmed rate
AOO, VOO, DOO	Advantages	ensures a fixed cardiac rhythm
	Drawbacks	no sensing of the spontaneous cardiac events
On Demand AAI, VVI, VDD DDI, DDD	**Principle**	- no spontaneous rhythm: permanent pacing at the programmed rate - when spontaneous rhythm < programmed rate: permanent pacing - when spontaneous rhythm > programmed rate: inhibition of the PM
	Advantages	no competition with the spontaneous rhythm
Synchronous	**Principle**	spikes triggered by spontaneous events
AAT, VVT	Advantages	no spontaneous events = cardiac pacing at the prog. rate interferences or artifacts = no pacing inhibition
	Drawbacks	early battery depletion

- SSI Mode:
  - The AAI and VVI modes act in a comparable manner, with pacing and sensing in the same chamber (atrium or ventricle) and the pacing output is inhibited by a sensed event in that chamber. For practical purposes, AAI and VVI modes may be considered to be a common SSI mode.
- VVI Mode:
  - In the VVI mode, the ventricular inhibited pacing mode, the pacemaker senses and paces in the ventricle. This mode is most appropriate for patients in chronic *atrial fibrillation* (AF) in whom atrial sensing or pacing is not needed

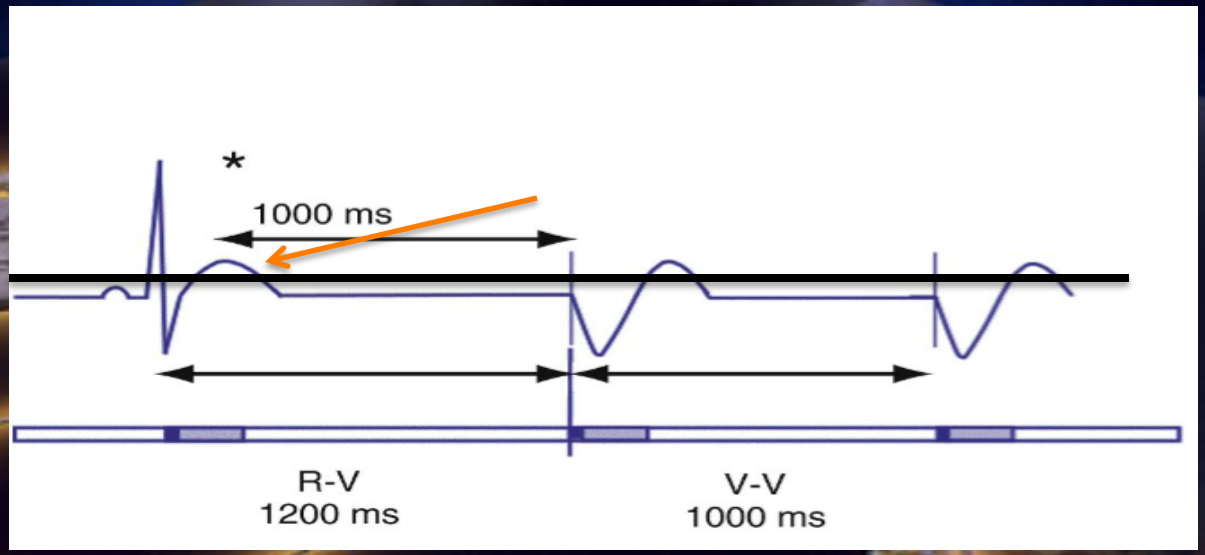


- V00 Mode:
  - In the VOO mode, ventricular pacing without ventricular sensing is present No intrinsic events are sensed, and therefore ventricular pacing occurs independent of the intrinsic rhythm. VOO is programmed *on* to prevent EMI from resulting in ventricular inhibition in the pacemaker-dependent patient.



Oversensing and Undersensing

- **Oversensing:** The PM senses an event that does not represent ventricular depolarization.
- Possible causes: parts of the QRS complex, the T wave, afterdepolarizations, atrial activity, noise, myopotencials, electromagnetic interference (EMI)

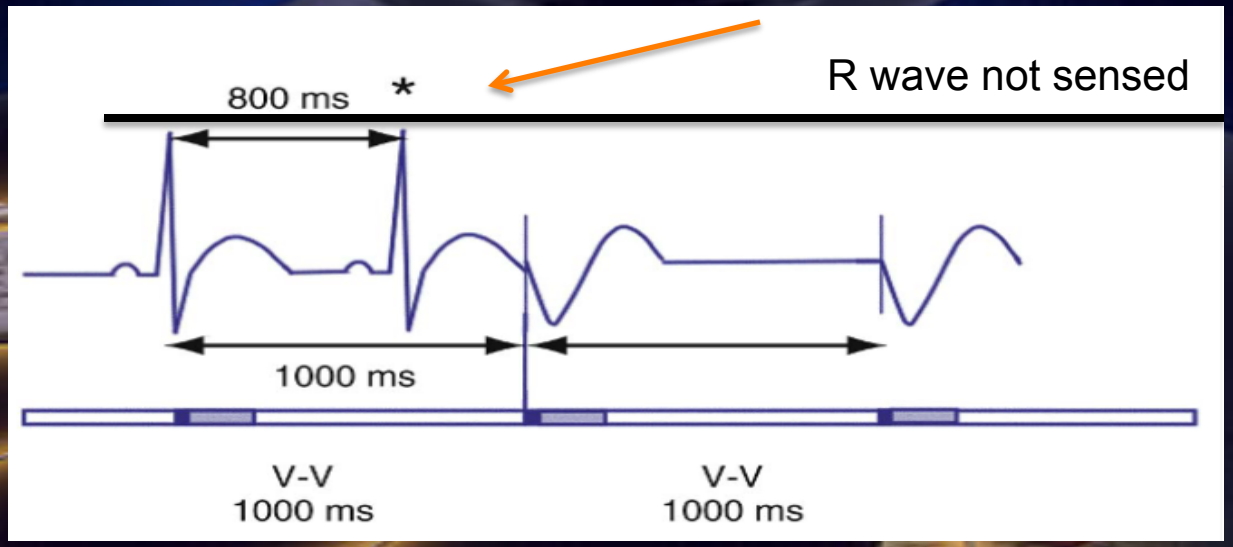


Undersensing:

T wave is sensed as „R" wave

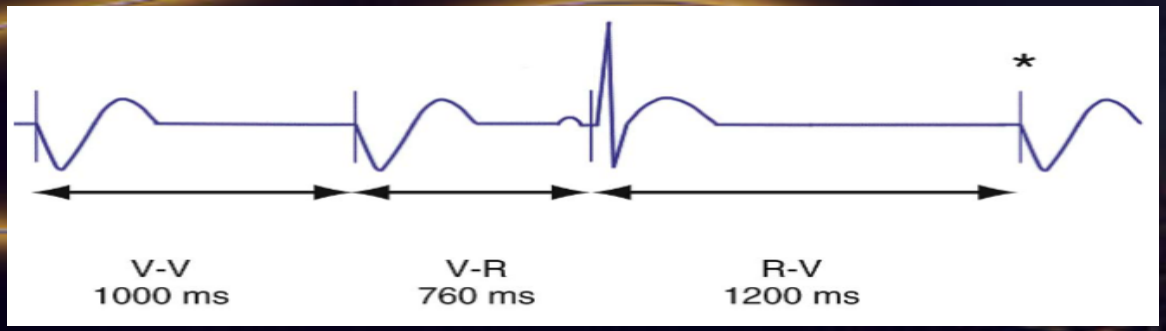
In *undersensing:* the pacemaker does not sense an intrinsic ventricular depolarization (R wave)

The pacing interval will be shorter than the pacing cycle length



Hysteresis Rate

- Hysteresis: longer ventricular escape interval from the last ventricular sensed event to the first ventricular paced event (R-V, *hysteresis interval*) but no change in the time from the last ventricular paced event
- allows the intrinsic heart rate to be lower before pacing occurs, but when pacing occurs, it will occur at a faster rate.
- E.g.: if the hysteresis pacing rate is 50 beats per minute and the base pacing rate is 60 bpm, pacing will not occur if the patient continues to maintain rates above 50 bpm. When the heart rate falls below 50 bpm, however, pacing will occur at 60 bpm.

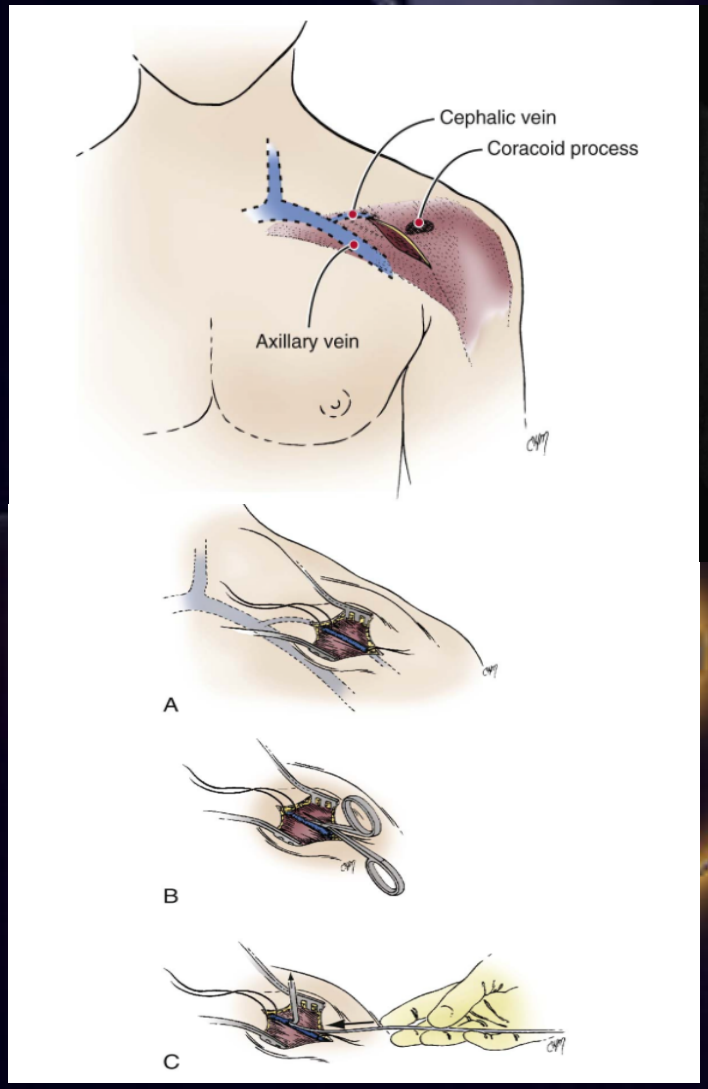


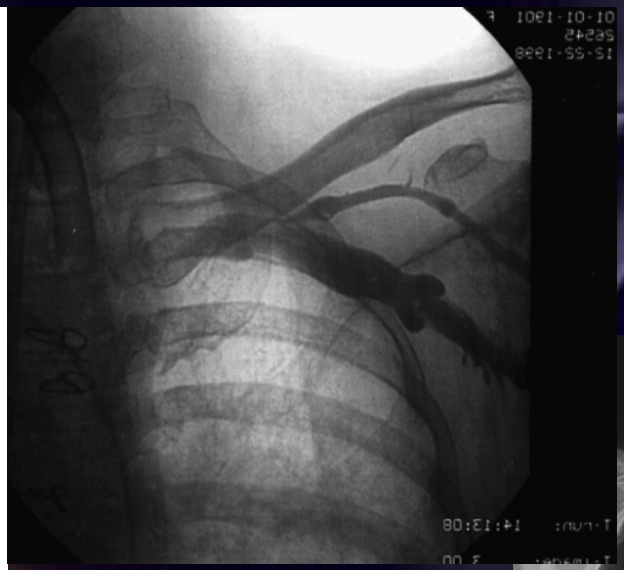
Implantation procedure

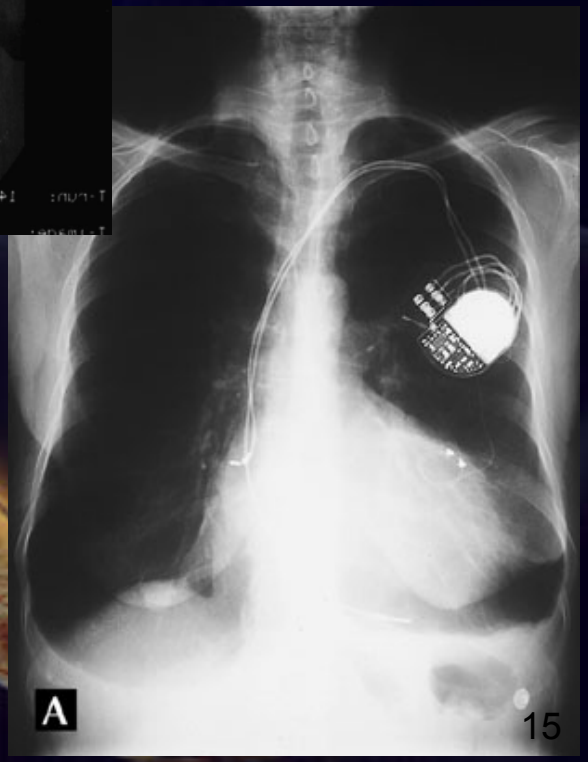
- A small 5-10 cm incision
- Small pocket will be made under skin for generator and lead(s).
- Under fluoroscopy, lead(s) travel through the subclavian vein, brachiocephalic vein, superior vena cava, and finally into the desired chamber.

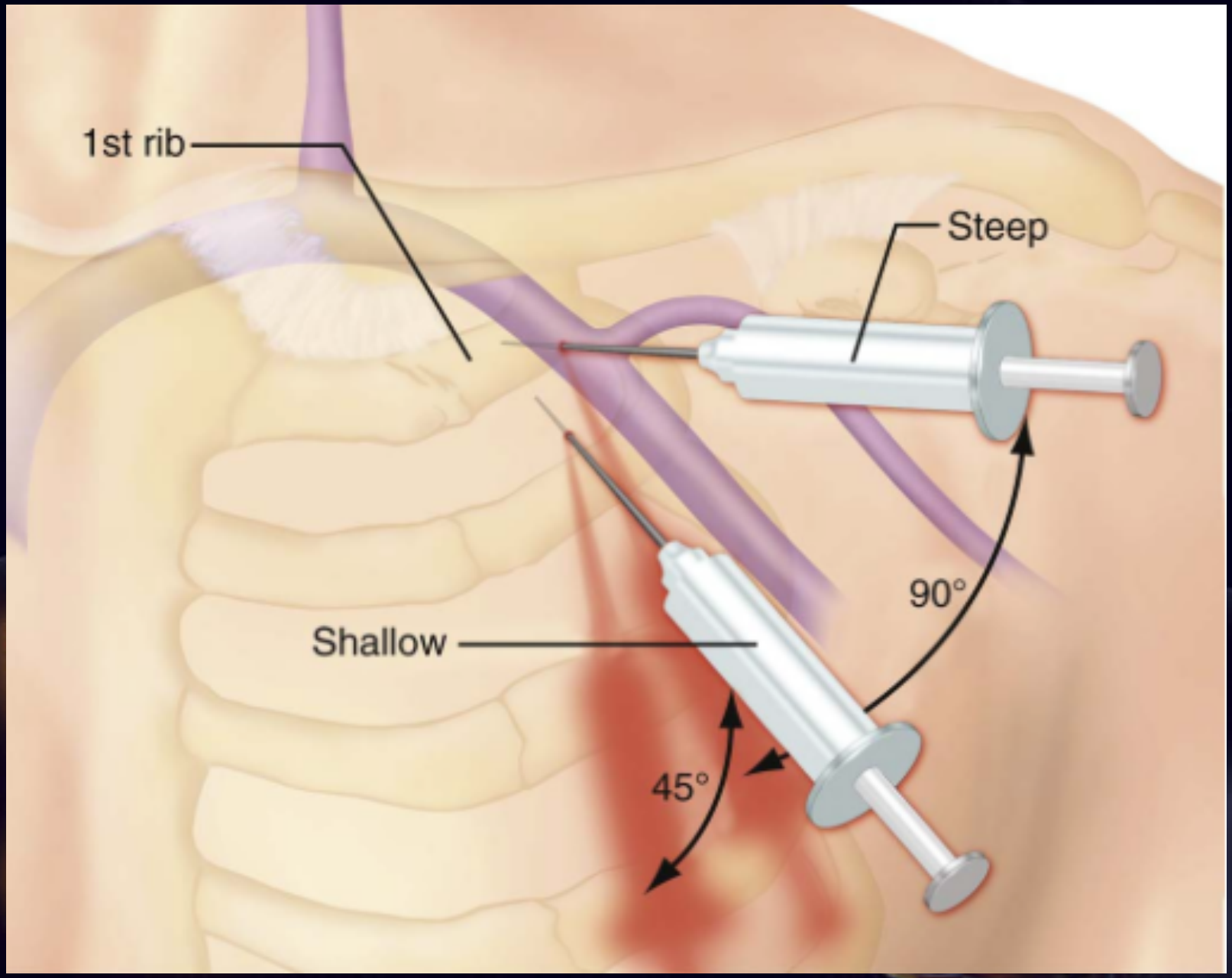
- Hospitalization usually last 1 to 3 days.

Műtéti technika

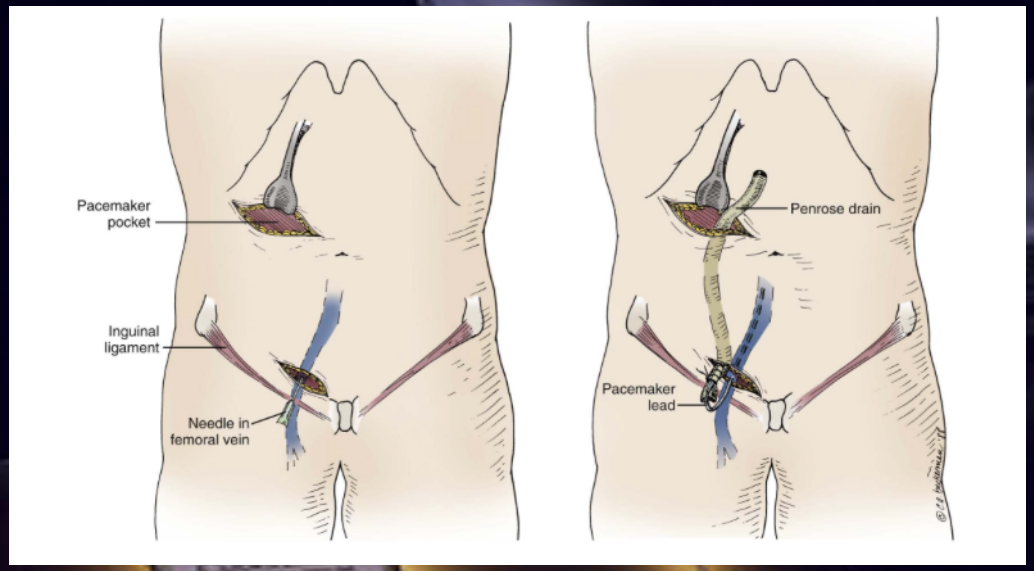


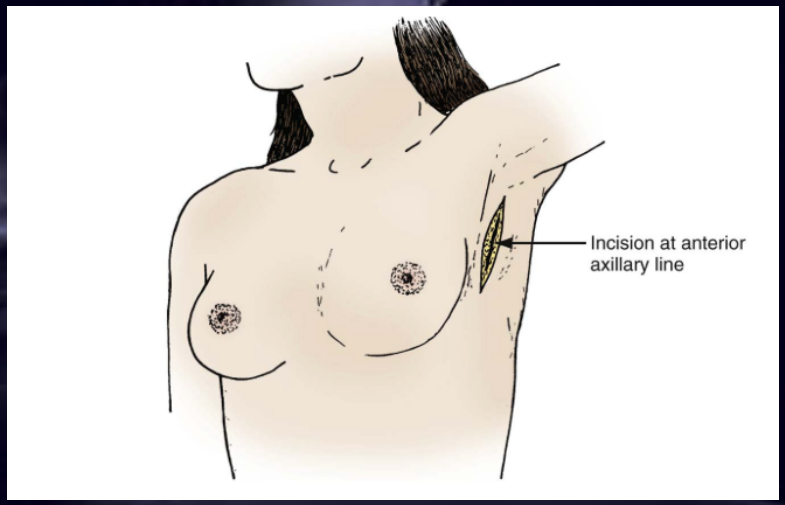


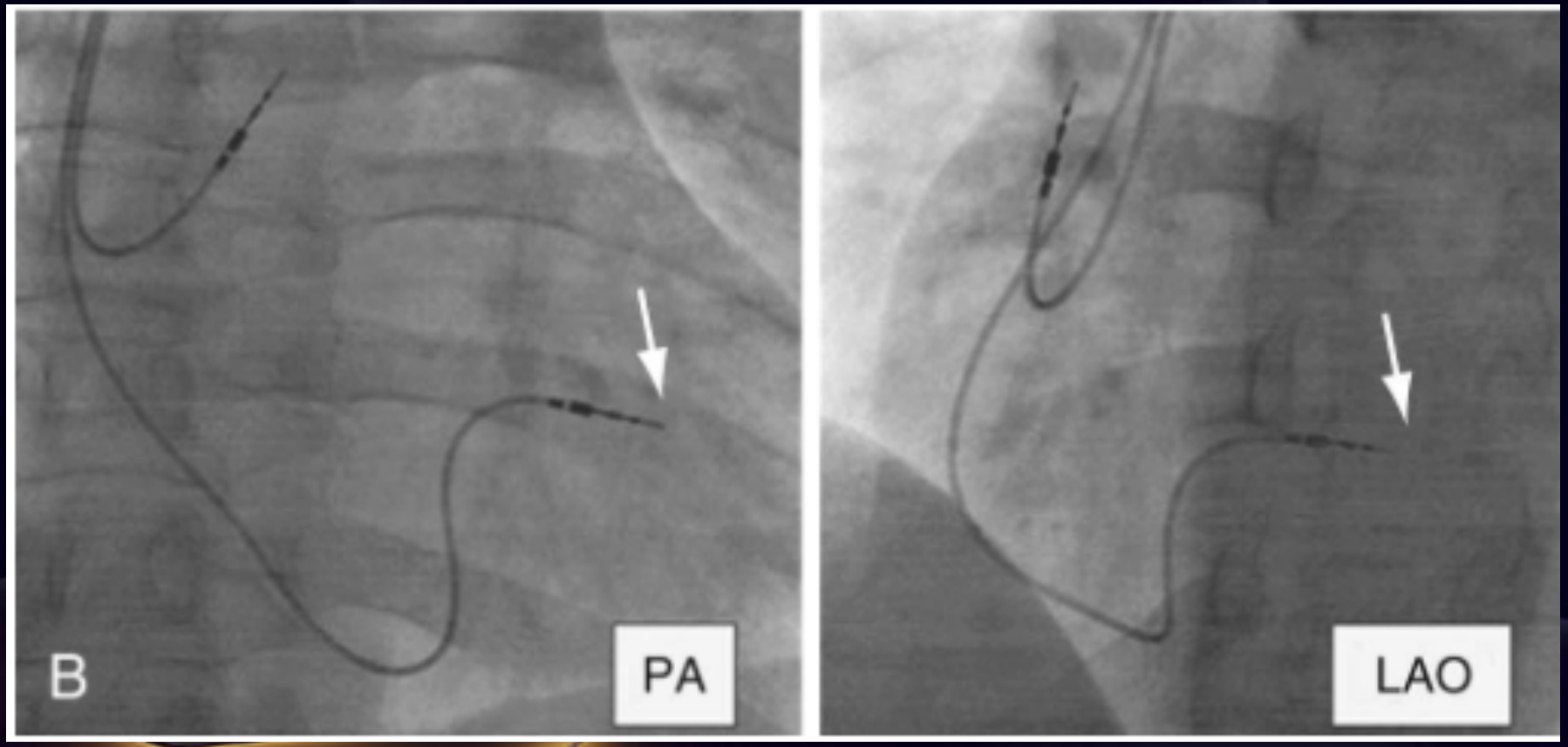



1st rib
Steep
90°
Shallow
45°

Alternatív műtéti technikák







Adverse events: pneumothorax, bleeding, hemopericardium, infection

Indication for pacing in patients with persistent bradycardia

Recommendations	Class	Level
1) Sinus node disease. Pacing is indicated when symptoms can clearly be attributed to bradycardia.	I	B
2) Sinus node disease. Pacing may be indicated when symptoms are likely to be due to bradycardia, even if the evidence is not conclusive.	IIb	C
3) Sinus node disease. Pacing is not indicated in patients with SB which is asymptomatic or due to reversible causes.	III	C
4) Acquired AV block. Pacing is indicated in patients with third- or second-degree type 2 AV block irrespective of symptoms.	I	C
5) Acquired AV block. Pacing should be considered in patients with second-degree type 1 AV block which causes symptoms or is found to be located at intra- or infra-His levels at EPS.	IIa	C
6) Acquired AV block. Pacing is not indicated in patients with AV block which is due to reversible causes.	III	C

Indication for pacing in intermittent documented bradycardia

Recommendations	Class	Level
1) Sinus node disease (including brady-tachy form). Pacing is indicated in patients affected by sinus node disease who have the documentation of symptomatic bradycardia due to sinus arrest or sinus-atrial block.	I	B
2) Intermittent/paroxysmal AV block (including AF with slow ventricular conduction). Pacing is indicated in patients with intermittent/paroxysmal intrinsic third- or second-degree AV block.	I	C
3) Reflex asystolic syncope. Pacing should be considered in patients ≥40 years with recurrent, unpredictable reflex syncopes and documented symptomatic pause/s due to sinus arrest or AV block or the combination of the two.	IIa	B
4) Asymptomatic pauses (sinus arrest or AV block). Pacing should be considered in patients with history of syncope and documentation of asymptomatic pauses >6 s due to sinus arrest, sinus-atrial block or AV block.	IIa	C
5) Pacing is not indicated in reversible causes of bradycardia.	III	C

Indication for cardiac pacing in patients with BBB

Recommendations	Class	Level
1) BBB, unexplained,syncope and abnormal EPS. Pacing is indicated in patients with syncope, BBB and positive EPS defined as HV interval of ≥70 ms, or second- or third-degree His-Purkinje block demonstrated during incremental atrial pacing or with pharmacological challenge.	I	B
2) Alternating BBB. Pacing is indicated in patients with alternating BBB with or without symptoms.	I	C
3) BBB, unexplained syncope with non-diagnostic investigations. Pacing may be considered in selected patients with unexplained syncope and BBB.	IIb	B
4) Asymptomatic BBB. Pacing is not indicated for BBB in asymptomatic patients	III	B

Indication for cardiac pacing in patients with undocumented reflex syncope

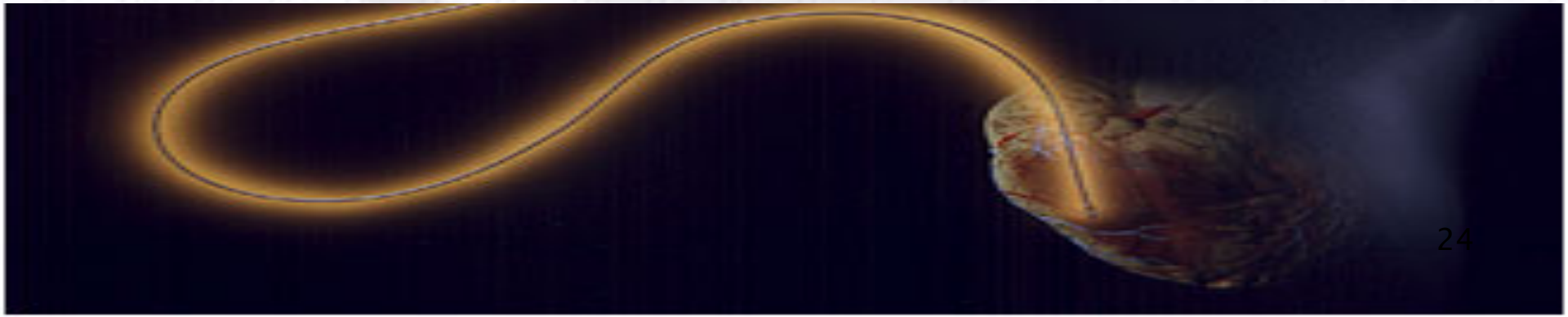
Recommendations	Class	Level
1) Carotid sinus syncope. Pacing is indicated in patients with dominant cardioinhibitory carotid sinus syndrome and recurrent unpredictable syncope.	I	B
2) Tilt-induced cardioinhibitory syncope. Pacing may be indicated in patients with tilt-induced cardioinhibitory response with recurrent frequent unpredictable syncope and age >40 years after alternative therapy has failed.	IIb	B
3) Tilt-induced non-cardioinhibitory syncope. Cardiac pacing is not indicated in the absence of a documented cardioinhibitory reflex.	III	B

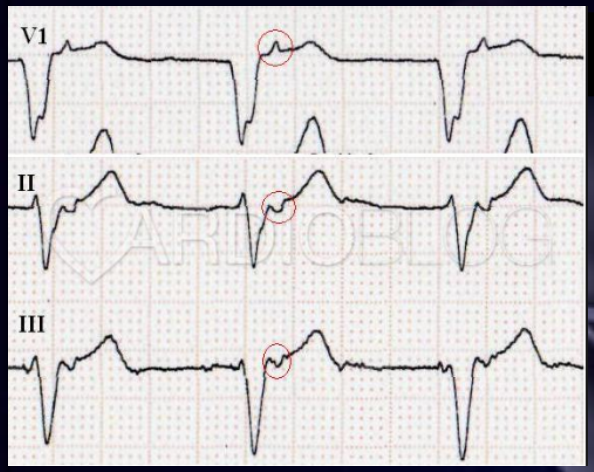
Indication for cardiac pacing in acute myocardial infarction

Recommendations	Class	Level
1) In the rare cases in which AV block becomes permanent, cardiac pacing is indicated with the same recommendations in section 2.1.	I	C
2) Cardiac pacing is not indicated after resolution of high degree or complete AV block complicating the acute phase of myocardial infarction.	III	B

Indication for cardiac pacing for first-degree atrioventricular block

Recommendations	Class	Level
Permanent pacemaker implantation should be considered for patients with persistent symptoms similar to those of pacemaker syndrome and attributable to first-degree atrioventricular block (PR >0.3 s).	IIa	C





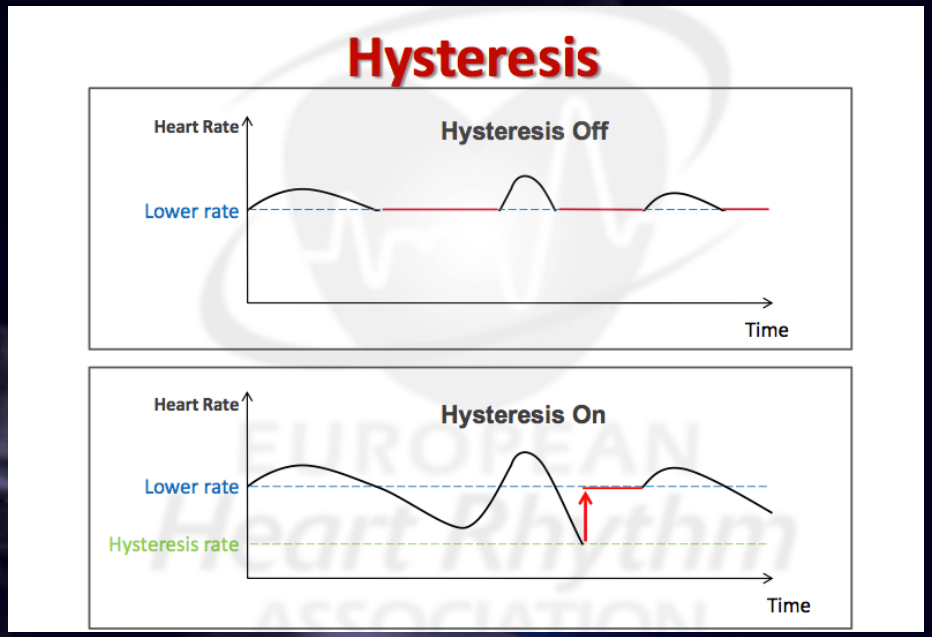
single chamber- dual chamber
MRI compatible
special features AF monitoring, sleep apnoe monitoring, minimal ventricular capture,
remote ambulatory monitoring

Dual-chamber versus ventricular pacing

Outcome	Dual-chamber benefit over ventricular pacing
All-cause deaths	No benefit
Stroke, embolism	Benefit (in meta-analysis only, not in single trial)
Atrial fibrillation	Benefit
HF, hospitalization for HF	No benefit
Exercise capacity	Benefit
Pacemaker syndrome	Benefit
Functional status	No benefit
Quality of life	Variable
Complications	More complications with dual-chamber

Programmable parameters:

- basic rate
-lower limit
-upper limit
-night rate
-rate respons
-auto capture
-hysteresis
-mode switching



ICD – history

Michel Mirowski, M.D.
1924-1990

- **1966:** a near friend started having VT episodes and eventually died while at dinner with his family.
- **Idea:** a defibrillator could be implanted in the body.
- **Challenges**:
  - Deliver sufficient energy
  - Leads carrying high energy
  - Detection of arrhythmias
  - Automated algorithms
  - Implantable size
  - …

ICD – history

1969: First experimental model

1969: First transvenous defibrillation

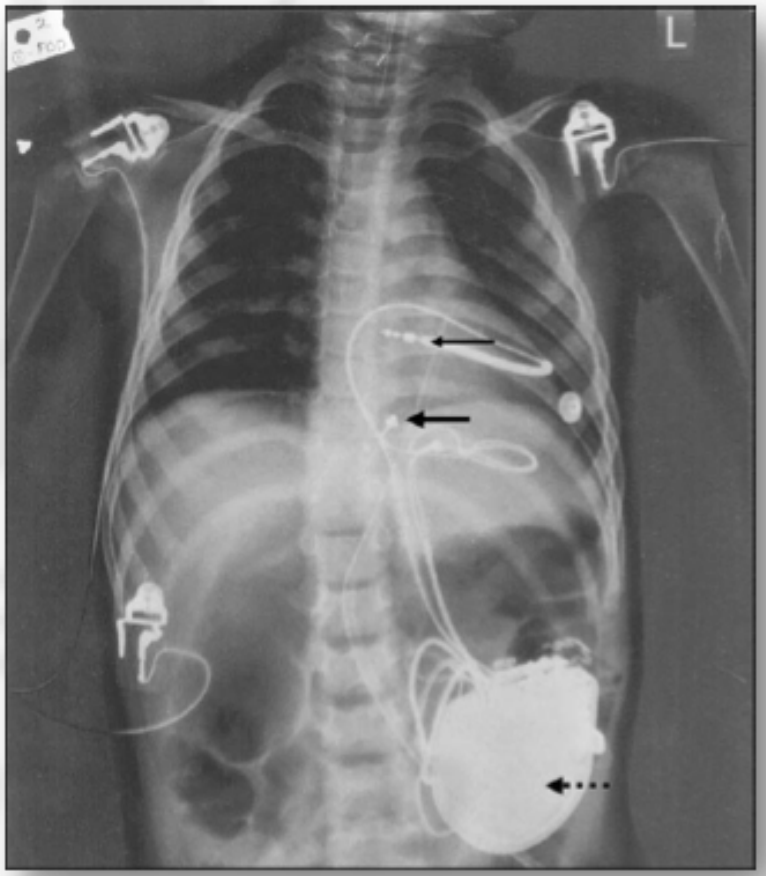
1975: First animal implant

1980: First human implant

1981: Addition of Cardioversion

1985: FDA approval

1988: First programmable ICD implanted in a human



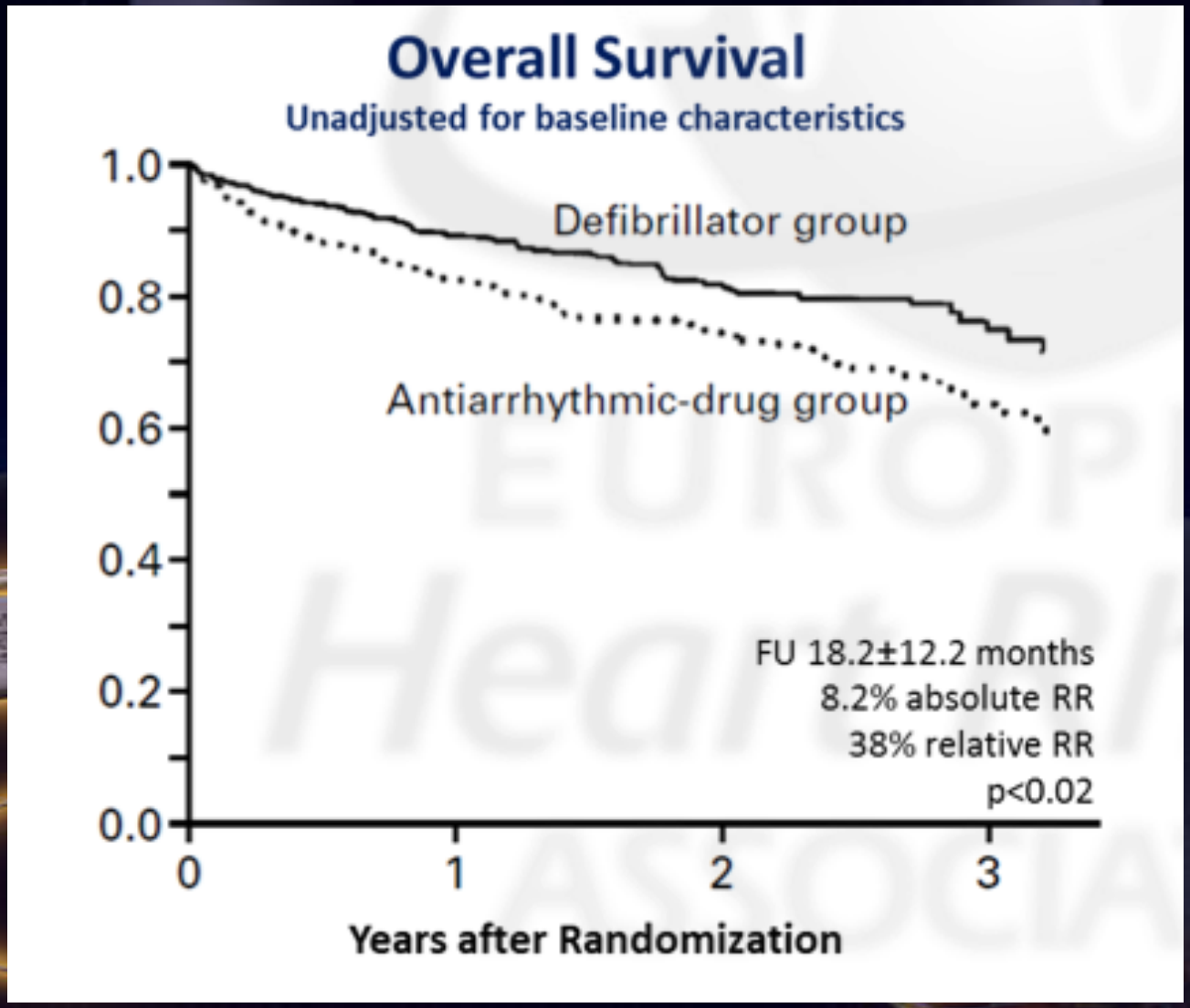
ICD – history

THEN

- Open-chest
- Large device
- Extended hospital stay
- Abdominal implant
- No or very limited programmability

NOW

- Minimally invasive pectoral implant
- Pacemaker size
- Outpatient procedure
- Pectoral implant
- Extensive programmability
- Extended service life
- Full pacing capability (CRT)
- ATP
- Smart algorithms
- Built-in sensors
- Remote monitoring


Overall Survival
Unadjusted for baseline characteristics
1.0
0.8
0.6
0.4
0.2
0.0
Defibrillator group
Antiarrhythmic-drug group
FU 18.2±12.2 months
8.2% absolute RR
38% relative RR
p<0.02
0
1
2
3
Years after Randomization

Primary prevention: MADIT

The Multicenter Automatic Defibrillator Implantation Trial

NYHA I/II/III
prior MI
LVEF ≤ 35%

↓

Holter: asymptomatic, non-sustained VT

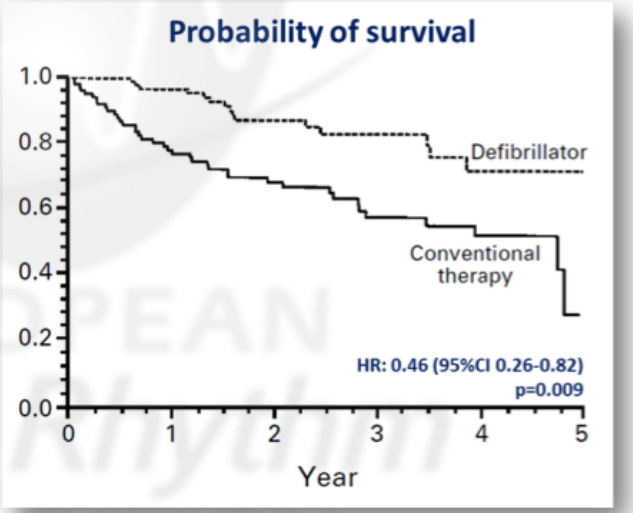
↓

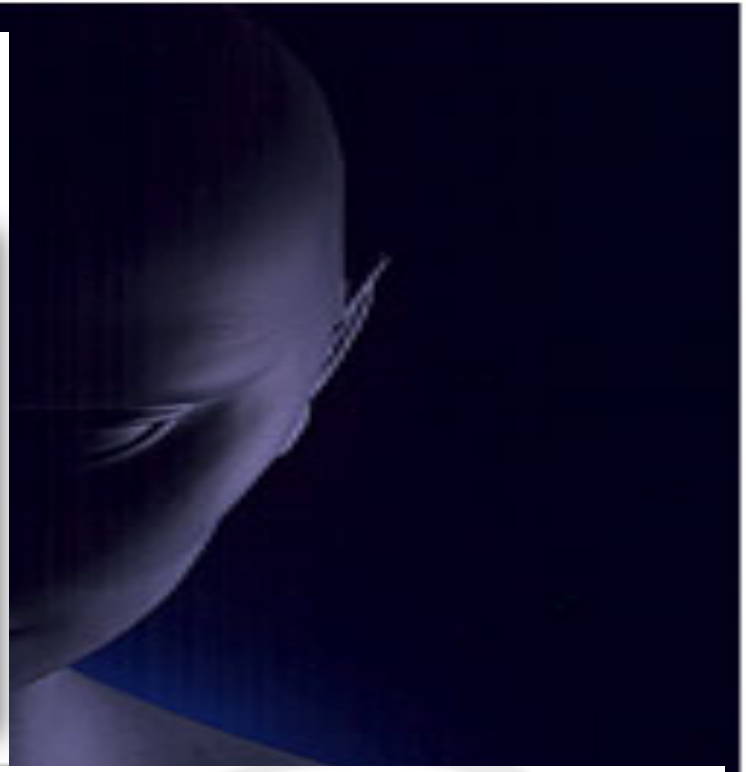
EPS: inducible VT, non-suppressible by drugs (n=196)

↓

Conventional medical therapy (74% amiodarone) (n=101)

ICD (n=95)



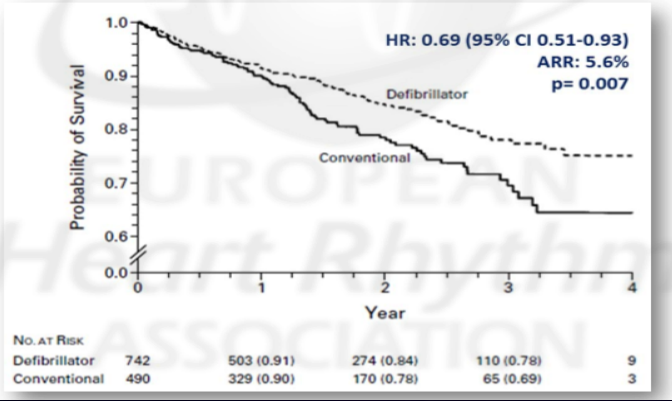


Primary prevention: MADIT II

The Multicenter Automatic Defibrillator Implantation Trial II

1) Prior MI (>1 month); 2) EF ≤ 30%

(no requirement of previous arrhythmia event or inducibility on EPS)



Primary prevention: MUSTT

The Multicenter Unsustained Tachycardia Trial

CAD
LVEF ≤ 40%
NSVT
(n=2202)

↓

EPS:
inducible sustained VT
(n=767)

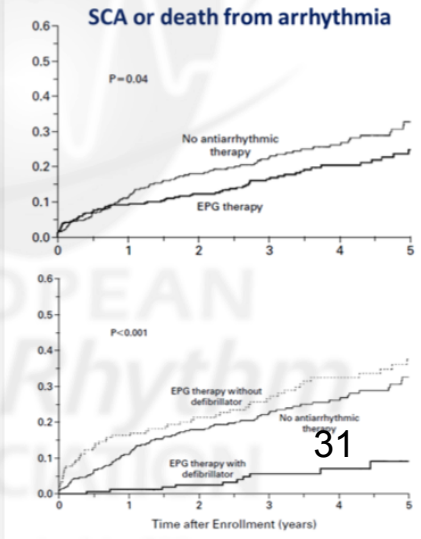
↓

EP-guided AA-therapy (n=351)

No AA-therapy (n=353)

↓

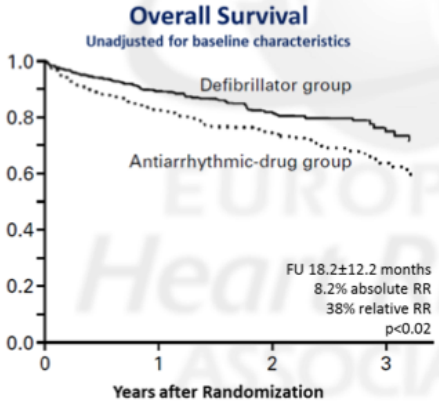
ICD
(if AA-therapy not effective; n=161)

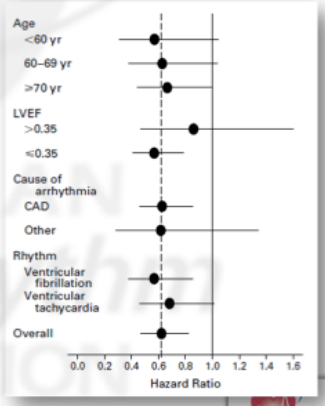


Secondary prevention: AVID

Antiarrhythmics vs Implantable Defibrillators Trial

n=1016: a) VF with SCA; b) sust VT with syncope;
c) sust VT with hemodynamic compromise and EF ≤ 40%

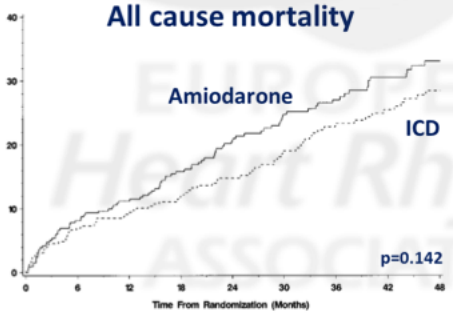




Secondary prevention: CIDS

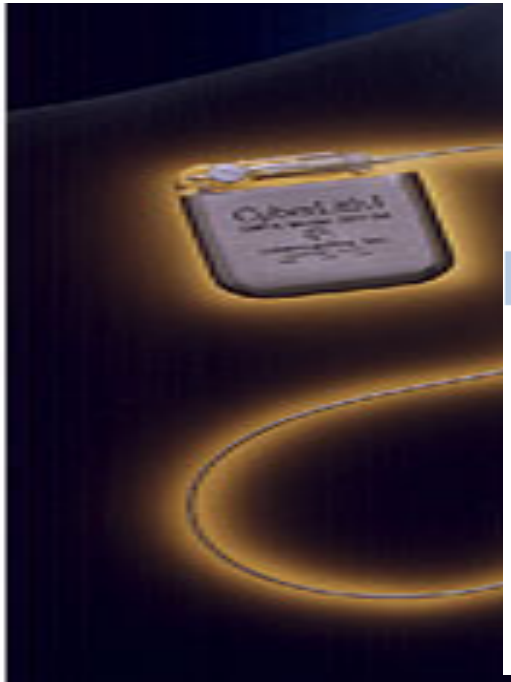
Canadian Implantable Defibrillator Study

n= 659; (1) VF; (2) SCA with cardioversion/defibrillation;
(3) sustained VT with syncope
(4) sustained VT ≥150/min with pre-syncope/angina *and* EF ≤ 35%;
(5) syncope with subsequent spontaneous/inducible VT



ICD arm:
- 10% thoracotomy
- 5% no ICD
- 34% on betablocker

Amiodarone arm:
- 89% received amio (1y)
- 16% received ICD
- 21% on betablocker

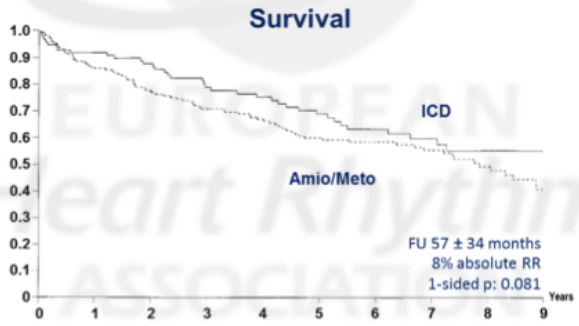


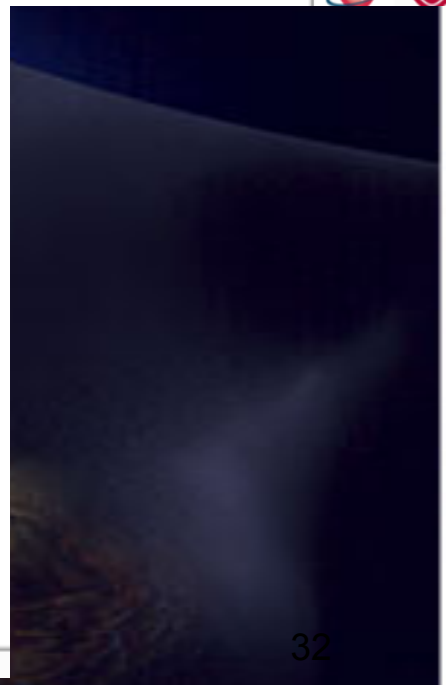
Secondary prevention: CASH

Cardiac Arrest Study Hamburg

SCA due to VT/VF (n=288)

ICD (n=99)	Amiodarone (n=92)	Metoprolol (n=97)	Propafenone (n=58) *Discontinued*

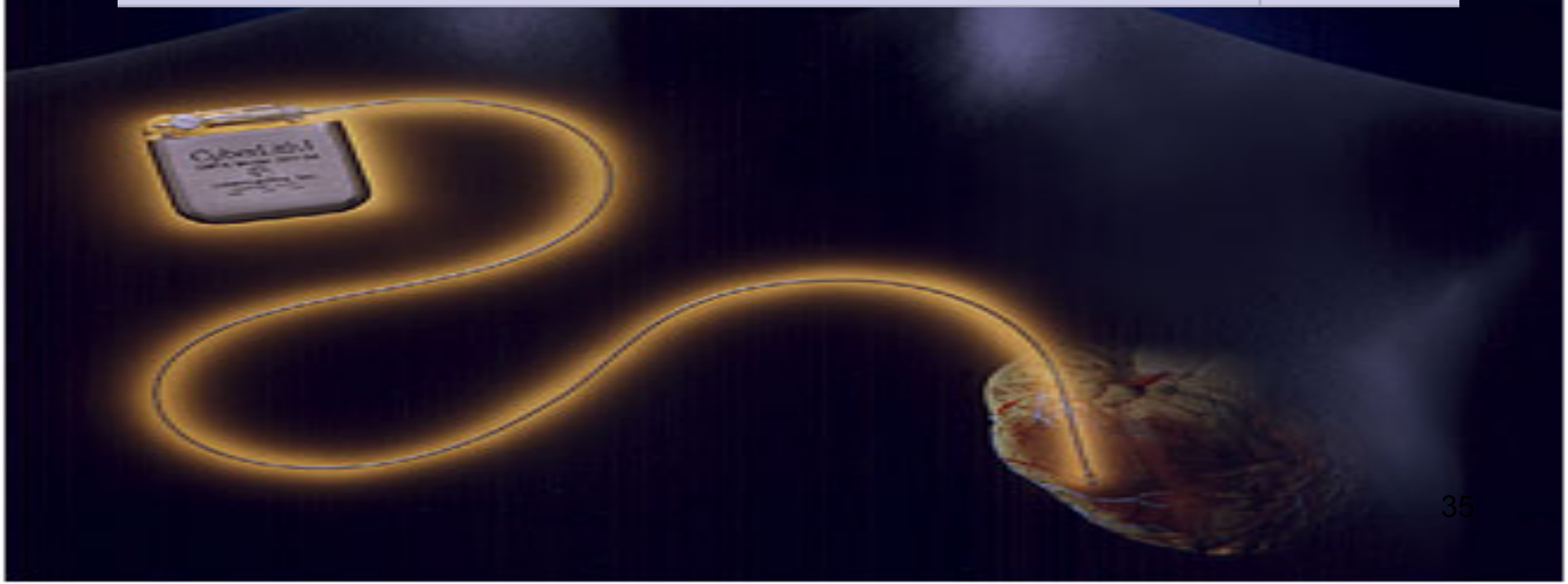




Recommendations	Level of Evidence
Class I (General Agreement of Benefit with ICD Therapy)	
1. Cardiac arrest due to VF or unstable sustained VT after evaluation in the absence of completely reversible causes.	A
2. Structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable.	B
3. Syncope of undetermined origin and clinically relevant, sustained VT or VF induced at electrophysiologic study.	B
4. LVEF ≤0.35 due to prior MI who are at least 40 days post-MI and NYHA II or III.	A
5. LVEF ≤0.35 due to a nonischemic cause and NYHA II or III.	B
6. LVEF ≤0.30 due to prior MI, at least 40 days post-MI and are NYHA I.	A
7. Nonsustained VT due to prior MI, LVEF ≤0.40, and inducible VF or sustained VT at electrophysiologic study.	B

Class IIa (Weight of Evidence in Favor of Usefulness of ICD Therapy)	
1. Unexplained syncope, significant LV dysfunction related to a nonischemic cause.	C
2. Sustained VT and normal or near-normal left ventricular systolic function.	C
3. Hypertrophic cardiomyopathy with one or more major risk factors for sudden death (see text).	C
4. Arrhythmogenic right ventricular cardiomyopathy (ARVC) with one or more risk factors for sudden death.	C
5. Long-QT syndrome with syncope and/or VT while receiving β-blockers.	B
6. Nonhospitalized patients awaiting transplantation.	C
7. Brugada syndrome with syncope.	C
8. Brugada syndrome with documented VT that has not resulted in cardiac arrest.	C
9. Catecholaminergic polymorphic VT with syncope and/or documented sustained VT while receiving β-blockers.	C
10. Cardiac sarcoidosis, giant cell myocarditis, or Chagas disease.	C

Class IIb (Efficacy of ICD Therapy Is Less Well Established)	
1. LVEF ≤ 0.35 due to nonischemic cause and NYHA I.	C
2. Long-QT syndrome and risk factors for sudden death.	B
3. Advanced structural heart disease and syncope of unknown cause despite invasive and noninvasive investigations.	C
4. Familial cardiomyopathy associated with sudden death.	C
5. Left ventricular noncompaction.	C

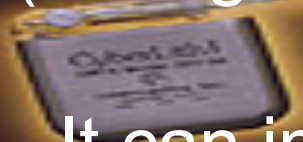


Cardiac Resynchronisation Therapy (CRT)

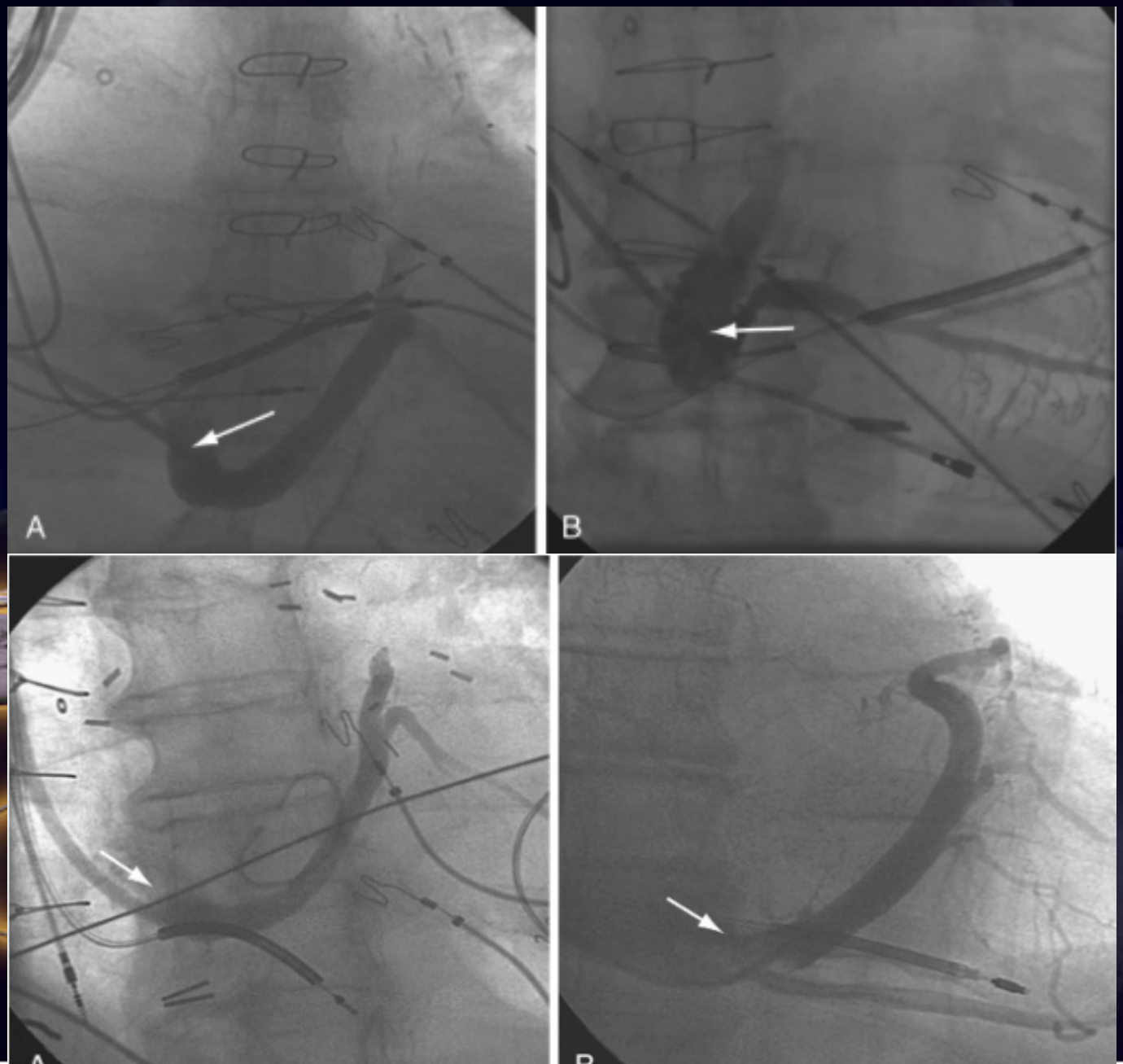
-Heart failure + conduction delay (LBBB)
-dyssynchrony: echo, MRI
-Impaired left ventricular function (EF« 35%)

CRT-P: No ICD function
CRT-D: with defibrillator function

3 leads: right atrium, right ventricle, left ventricle (through CS)



- It can improve EF, less mortality rate, improved clinical outcome, less hospitalisation because of HF, better clinical condition (improved NYHA class)


A
B

Magnitude of benefit from CRT

Highest (responders)

Wider QRS, left bundle branch block, females, non-ischaemic cardiomyopathy

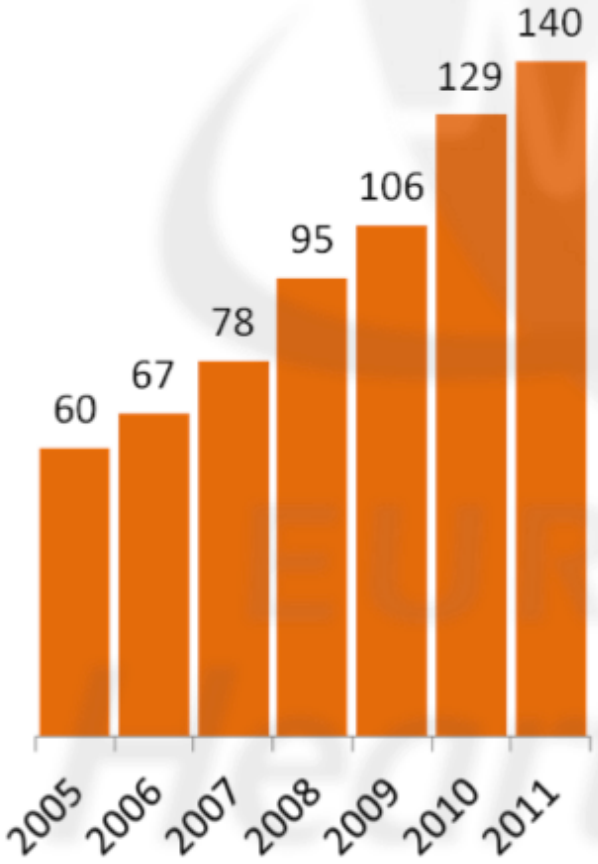
Males, ischaemic cardiomyopathy

Lowest (non-responders)

Narrower QRS, non-left bundle branch block

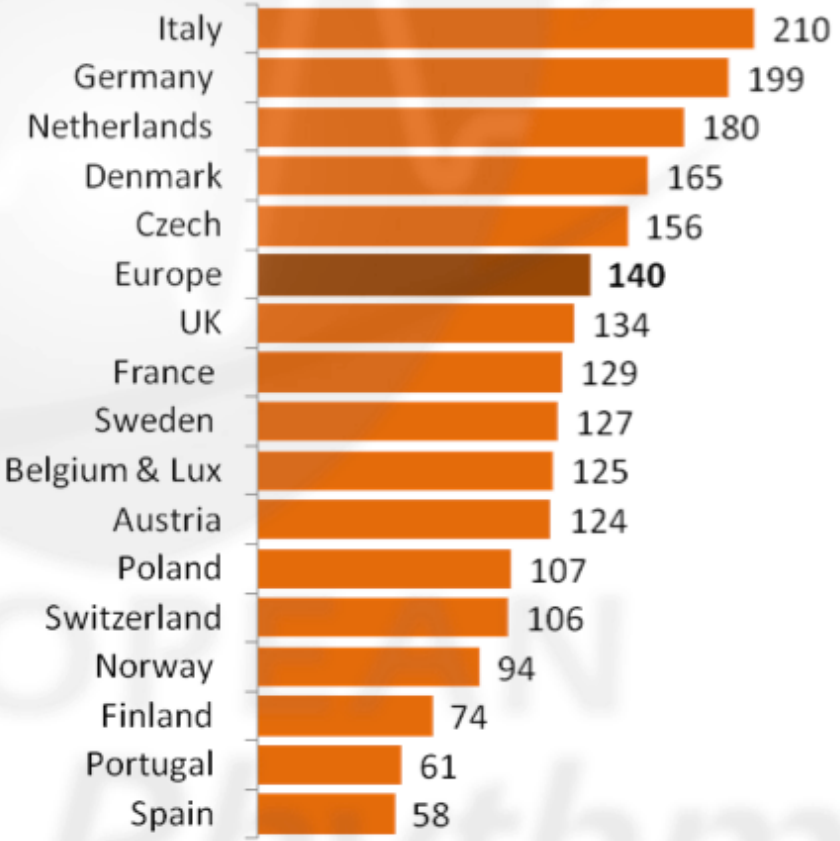
CRT

Units per million inhabitants



CRT 2011

Units per million inhabitants

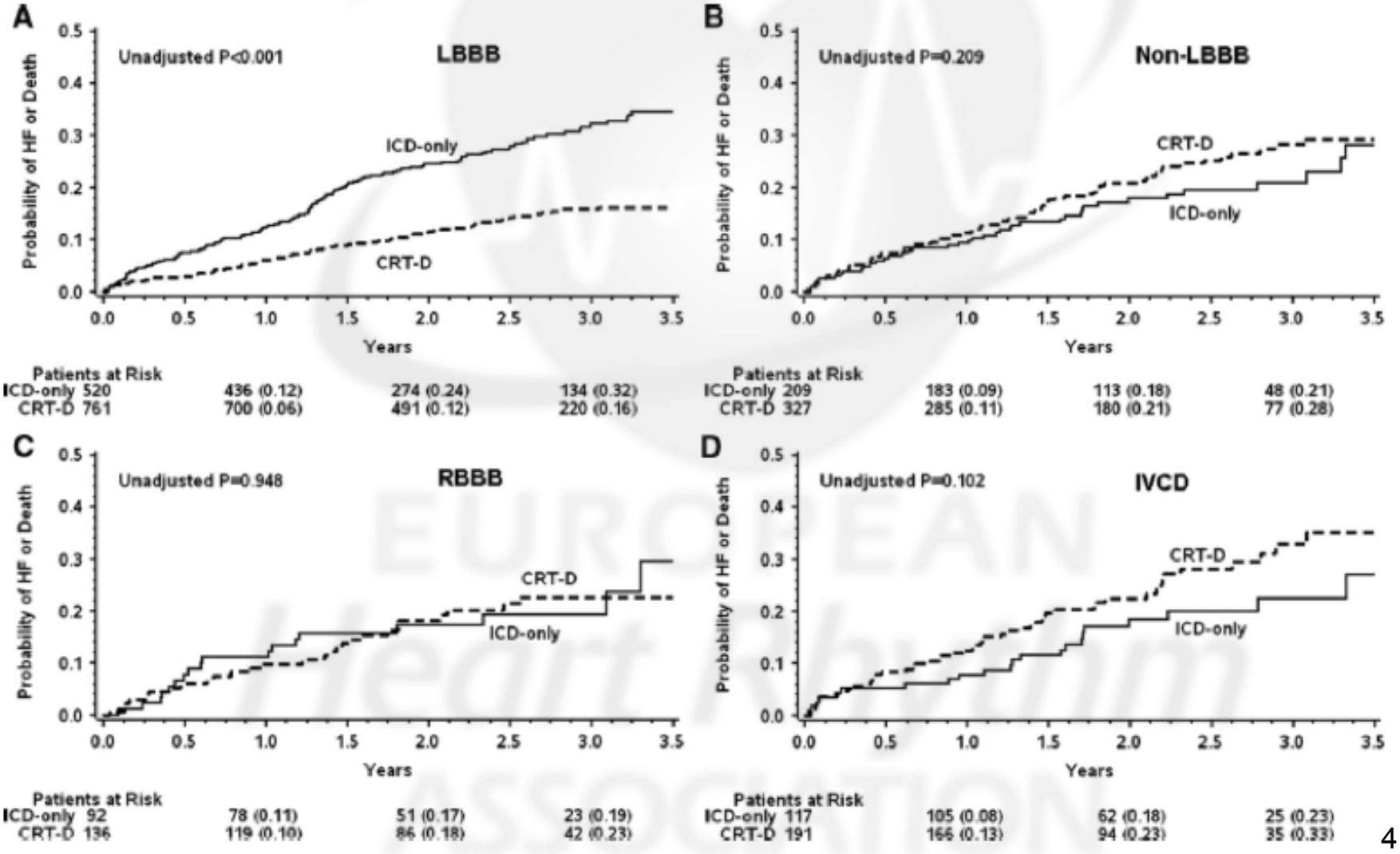


Recommendations	Class [a]	Level [b]	Ref. [c]
1) LBBB with QRS duration >150 ms. CRT is recommended in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment. [d]	I	A	48–64
2) LBBB with QRS duration 120–150 ms. CRT is recommended in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment. [d]	I	B	48–64

Recommendations	Class [a]	Level [b]	Ref. [c]
3) Non-LBBB with QRS duration >150 ms. CRT should be considered in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment. [d]	IIa	B	48–64
4) Non-LBBB with QRS duration 120–150 ms. CRT may be considered in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment. [d]	IIb	B	48–64

Recommendations	Class [a]	Level [b]	Ref. [c]
5) CRT in patients with chronic HF with QRS duration <120 ms is not recommended.	III	B	65, 66

Importance of conduction disorders



Future perspectives

- Subcutaneous ICD
- Leadless PM
- Cardiac Contractility Modulator-
- Biological PM